Early Detection of Venous Air Embolism Using a Swan-Ganz Catheter

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Venous air embolism is a dreaded complication that may occur when patients undergo intracranial surgery in the sitting position. The following report describes such a complication which occurred early in the course of operation and required postponement of the procedure. Subsequently the operation was completed successfully utilizing anesthesia and monitoring techniques not previously reported for neurosurgical procedures.

REPORT OF A CASE

A 27-year-old woman was scheduled for surgery to remove a cerebellar pontine-angle tumor. The patient, otherwise healthy, was classified as an ASA physical status I. Following establishment of anesthesia with nitrous oxide, 67 per cent in oxygen, morphine, and *d*-tubocurarine, she was placed in a sitting position. Catheters were placed in the radial artery and the right atrium using pressure-recording confirmation. Lead II of the electrocardiogram (ECG) was displayed on an oscilloscope. Esophageal temperature was recorded with a telethermometer, and heart sounds were monitored with an esophageal stethoscope.

After the suboccipital incision was made, arterial blood pressure and heart rate remained stable for 90 minutes at 110/80 torr and 70/min, respectively. For the next 30 minutes, at which time the skin and muscles were being opened, the systolic blood pressure became unstable, fluctuating between 140 and 110 torr. The heart rate showed a gradual increase to 100/min. No change in heart sounds was detected with the esophageal stethoscope; however, a single analysis of arterial blood showed Pa_{O_2} 85 torr ($F_{I_{O_2}}$ = .32) and Pa_{CO_2} 34 torr. Two hours after the operation began, just as the muscles were being stripped from the bone, an open emissary mastoid vein was noted. Simultaneously, the blood pressure decreased suddenly from 120/80 torr to a systolic level of 20 torr, and a harsh, systolic murmur was detected. The patient was placed supine, nitrous oxide was discontinued, and the lungs were ventilated with pure oxygen.

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At this time 40 ml of gas were aspirated from the right-atrial catheter. Ten minutes later the blood pressure had returned to 80/40 torr, and 15 minutes after the onset of hypotension, the arterial blood pressure was 100/70 torr. Arterial blood–gas analysis showed Pa_{O_2} 400 torr and Pa_{CO_2} 46 torr.

The operative wound was closed and the patient was taken to the intensive care unit, where ventilation was assisted with a mechanical ventilator. Within an hour she became responsive and was able to move all extremities. A chest roentgenogram was normal. Three hours after the onset of the venous air embolism, a measurement of wasted ventilation showed a V_D/V_T ratio of .46. The endotracheal tube was removed four hours after the onset of embolism, at which time arterial blood gas values were normal.

Reoperation was undertaken a week later. Informed consent was obtained after the nature of the proposed procedures had been fully explained. Anesthesia was maintained with halothane at an inflow concentration of less than .7 per cent in combination with nitrous oxide, 50 per cent in oxygen. The patient was placed in the sitting position. Ventilation was controlled with a volume-limited ventilator attached to the circle absorber system. The ECG, temperature, and heart sounds were monitored as before. Prior to operation, a catheter was placed in the radial artery and a #7 F triple-lumen Swan-Ganz catheter[1] was advanced into the pulmonary artery, as confirmed by pressure tracings. End-tidal carbon dioxide concentration ($F_{ET_{CO_2}}$) was analyzed with a Godart Capnograph and recorded on a polygraph along with radial and pulmonary arterial blood pressures.

When the operation commenced, nitrous oxide was discontinued and the inflow halothane concentration was increased to 1 per cent. Arterial and pulmonary arterial blood samples were obtained intermittently for analysis of Pa_{O_2}, Pa_{CO_2}, pH_a and calculation of the total venous admixture (Q_A/Q_T). These values and the radial and pulmonary arterial pressures are shown in table 1. The first three hours of anesthesia were uneventful and there was no indication of intravascular aspiration of air. As the muscles were being stripped from the bone, nitrous oxide was added to the circle inflow gases (N_2O 8 l/min–O_2 2 l/min). Almost immediately, $F_{ET_{CO_2}}$ and radial arterial pressure decreased and pulmonary arterial pressure increased. The surgeons searched but were unable to find a source of air entry. After 5 minutes, nitrous oxide was discontinued and all three values rapidly returned toward control levels. At 4.5 hours, nitrous oxide again was administered as a challenge for silent venous air embolism. No change in $F_{ET_{CO_2}}$ or pulmonary arterial pressure was found. At six hours

the acoustic neuroma had been removed, with facial-nerve preservation. As the surgeons were closing the dura after having obtained hemostasis, there was a gradual decrease in FET_{CO_2} and an increase in pulmonary arterial pressure (fig. 1). Within 7 minutes, pulmonary arterial pressure had reached 22/10 torr, representing an approximately twofold increase. At the same time, FET_{CO_2} had decreased to about half its pre-embolism level. Nitrous oxide, 80 per cent, again was added to the circle inflow gases. After 1.5 minutes and exactly 12 ventilatory cycles, pulmonary arterial pressure further increased to 36/16 torr. The patient then began to "fight" the ventilator. This response persisted for 3 minutes. There was no change in radial arterial pressure or development of cardiac arrhythmias or murmurs. However, an accentuation of the second heart sound (S2) was heard through the esophageal and precordial stethoscopes. Nitrous oxide was discontinued, and in 20 minutes pulmonary arterial pressure and FET_{CO_2} returned to pre-embolism levels.

After closure of the muscle layer, halothane was discontinued and anesthesia maintained with nitrous oxide, 75 per cent, in O_2. After 15 minutes the patient again developed the same cardiorespiratory changes, suggesting intrapulmonary air embolism. Syringe aspiration through pulmonary-artery and central-venous catheter lumens recovered no gas. Five minutes after ventilation with oxygen, pulmonary arterial pressure and FET_{CO_2} returned to pre-embolism levels.

On completion of the operation the patient awakened quickly and showed no residual effect of air embolism. A roentgenogram of the chest revealed the tip of the Swan-Ganz catheter to be in the right mid-chest area. The following morning the endotracheal and vascular catheters were removed. The patient recovered satisfactorily, and on the seventh postoperative day a determination of wasted ventilation showed a V_D/V_T ratio of .29.

Comment

Various methods of monitoring have been advocated for the early detection of venous air embolism. These include measuring FET_{CO_2} during constant-volume ventilation[2,3] and using a stethoscope[4] or transthoracic ultrasonic Doppler device[5,6] to detect intracardiac gas. A central-venous or right-atrial catheter also facilitates the aspiration of gas once venous air embolism is apparent.[5] With these diagnostic and therapeutic measures, the incidence of venous air embolism during neurosurgical procedures may be as great as 40 per cent.[3]

We believe that during the initial operative procedure our patient probably experienced showers of silent air emboli for at least 30 minutes prior to the time when an open emissary vein and acute hypotension were noted. This is supported by the presence of arterial blood pressure fluctuations, development of tachycardia, and a Pa_{O_2} value of 85 torr during exposure to an FI_{O_2} of .32. That the occurrence of venous air embolism went undetected by vigilant and continuous monitoring of heart sounds through an esophageal stethoscope suggests that this

TABLE 1. Cardiorespiratory Changes in a Neurosurgical Patient during Halothane Anesthesia

	Radial Arterial Pressure (torr)	Pulmonary Arterial Pressure (torr)	$F_{ET}CO_2$ (Per Cent)	Pa_{O_2}* (torr)	Pa_{CO_2} (torr)	pH	Q_s/Q_T
3 hours							
Control	120/80	12/4	3.6	452	34	7.40	
After 5 min N_2O†	95/65	22/12	2.1				
After 8 min O_2	110/70	12/3	3.1	411	32	7.51	0.23
4.5 hours							
Control	95/70	14/4	3.9	289	38	7.37	0.20
After 5 min N_2O†	85/55	13/5	4.0				
After 8 min O_2	105/75	14/4	4.0	382	38	7.37	0.20
6 hours							
Control	100/70	12/3	3.8				
Air embolus	100/70	22/10	2.1				
After 1.5 min N_2O†	95/65	36/16	1.8				
After 5 min O_2	100/70	18/16	3.6	280	41	7.30	0.32

* $FI_{O_2} = 0.99$.
† $FI_{O_2} = 0.20$.

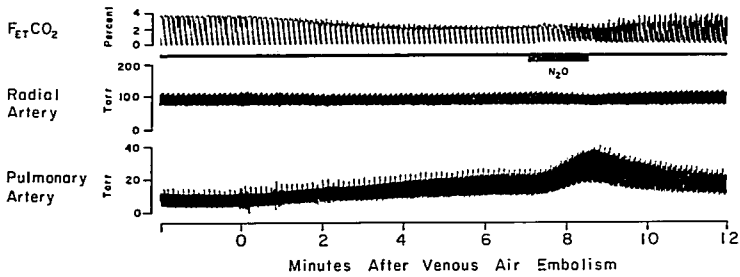

FIG. 1. Changes in blood pressures and $F_{ET_{CO_2}}$ during venous air embolism for a neurosurgical procedure on a patient placed in the sitting position after 6 hours of halothane anesthesia (corresponding cardiorespiratory values are shown in table 1). After 7 minutes of a gradual decrease in wasted ventilation and simultaneous increase in pulmonary arterial pressure, 80 per cent N_2O was added to the inspired gases. Within 12 ventilatory cycles (1.5 min), pulmonary arterial pressure increased markedly. Twenty minutes after N_2O was discontinued, cardiorespiratory changes had returned to normal.

monitoring system may have limited value in such circumstances. During the second operative procedure, when changes in $F_{ET_{CO_2}}$ and pulmonary arterial pressure indicated the presence of intrapulmonic air, the only change in heart sounds detected was an increased S2. These observations suggest that showers of silent emboli easily pass through the heart to rest in the pulmonary outflow tract without producing a cardiac murmur. We have yet to determine whether transient intracardiac gas would have gone undetected by precordial Doppler monitoring. The increases in wasted ventilation that we measured after venous air embolism are similar to observations by others.[2,3,7] The effect of nitrous oxide on pulmonary arterial pressure and the apparent reflex tachypneic response at the point of maximal pressure increase are similar to the results obtained in studies in dogs.[7] We believe that sufficient arterial oxygenation was maintained in our patient during the 1.5 minutes of nitrous oxide inhalation ($F_{I_{O_2}}$ = .20) that hypoxemia probably was not a contributing factor to the observed changes in pulmonary arterial pressure.

Bubbles of embolized gas in pulmonary arterioles can readily affect changes in $F_{ET_{CO_2}}$ and pulmonary arterial pressure but remain inaccessible to an intravenous catheter whose tip lies in the superior vena cava or right atrium. Only following massive venous air embolism, or when the cumulative volume of gas exceeds pulmonary arterial capacity (approximately 5 ml/kg body weight), does intracardiac gas appear.[8] While pulmonary outflow tract gas can be aspirated directly through a Swan-Ganz catheter, removal of right ventricular or right atrial gas easily can be achieved either by utilizing the third lumen (CVP port, 20 cm from the catheter tip) or by withdrawing the catheter while applying syringe aspiration until gas is obtained.

The use of nitrous oxide in animals has been shown to accentuate the cardiorespiratory efforts of sublethal venous air embolism, or render lethal otherwise innocuous injections of embolized air.[9] However, it might be argued that the use of nitrous oxide could be beneficial because, by converting small and otherwise asymptomatic air emboli into larger bubbles, the signs would be more easily recognized and diagnosed by the clinician. Under these circumstances the elimination of nitrous oxide from alveolar gas and pulmonary blood would allow more rapid diffusion of the nitrous oxide from the gas emboli of blood to alveolar gas. The demonstration that the onset and recovery from nitrous oxide effects occur within a few breaths agrees with animal studies and supports the concept that nitrous oxide diffusion into precapillary pulmonary arterioles occurs at the gas exchange level of the lung. However,

the removal of nitrous oxide from intracardiac gas would first require loss of nitrous oxide from venous blood. This process takes longer since it is dependent on the washout of nitrous oxide from the vessel-rich tissues. Removal of intracardiac gas would occur only while adequate cardiac function is maintained, suggesting that the use of nitrous oxide requires proper patient monitoring to ensure early diagnosis of intrapulmonic gas.

Until a more complete evaluation of the incidence and severity of venous air embolism during nitrous oxide anesthesia can be made, we believe it is best avoided in patients in whom the risk of air embolism is great. However, the intermittent use of nitrous oxide as a diagnostic test for the presence of early and asymptomatic venous air embolism, in combination with continuous monitoring of $F_{ET}CO_2$ and pulmonary arterial pressure, is a safe and useful procedure.

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More Failsafe Failsafes

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Failsafe devices for anesthesia machines are, as most of us know, not failsafe.[1] Despite these devices, an anesthesia machine can still deliver pure nitrous oxide. Solutions to this problem have not been generally applied, perhaps because they are too expensive or restrict the versatility of the standard types of machines.[2,3]

The two devices presented here greatly decrease the chances for delivering hypoxic mixtures without altering the way flows are selected. They are simple, relatively inexpensive, and can be applied to most existing machines. Both have performed without failure in extensive laboratory trials.

The fluidic device is simply an OR/NOR logic gate† which controls a pressure-electric switch.‡ The nitrous oxide is connected to the power supply position of the OR/NOR gate (fig. 1); if insufficient diverting flow of oxygen is present, the nitrous oxide will exit via outlet 2, close the pressure-electric switch and sound an alarm. If an adequate flow of oxygen is present, then the nitrous oxide flow will be diverted to exit via outlet 1 and the alarm switch remains open. The components used here have a maximum operating pressure of 10 psi, so 3-psi pop-off valves§ were used to allow extra flow to

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† Corning Glass Works, Fluidic Products Department, Corning, N.Y. 14803, Catalog number 191453.

‡ Corning Glass Works, Catalog number 191491.

§ Nupro Company, 15635 Saranac Road, Cleveland, Ohio 44110, Model number 4C.