

agonal ear-lobe crease is positively related to coronary heart disease<sup>2,3</sup> and not related solely to advancing age, as has been suggested.<sup>4</sup> Also, the data suggest that the crease can be used as an indicator of operative risk, since intraoperative and postoperative cardiovascular complications occurred more frequently in patients with the crease than in age-matched controls without the crease. The latter finding also was observed when patients with and without the crease were matched for type of surgical procedure and anesthetic technique.

Although the time of origin and the mechanism of formation of the ear-lobe crease have not been investigated, it has been assumed that the crease is not present at birth but develops later in life, either as a result of late penetration of a genetically-determined characteristic or as a result of localized vascular disease with atrophy of the skin.<sup>2</sup> That only three of 120 patients less than 40 years old had the crease supports the assumption that the crease develops in later life. However, since the ages

of these three patients were 3, 14, and 17 years, it would appear that the crease occasionally may be present at birth or may develop shortly thereafter. The significance of the crease at such an early age remains to be determined.

In summary, it appears that, for the patient not already identified to be at risk, the ear-lobe crease may be a useful indicator of the likelihood of development of intraoperative and postoperative cardiovascular problems.

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## Pulmonary Interstitial Edema after Multiple Venous Air Emboli

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Venous air embolism is a well-recognized complication in patients undergoing neurosurgical procedure in the sitting position. Although cardiopulmonary changes associated with air embolism during the operative course have been described, little attention has been focused on pulmonary changes in the postoperative period. The following report describes the occurrence of postoperative pulmonary interstitial edema in a healthy patient in whom eight separate episodes of venous air embolism and systemic arterial hypotension were documented during intracranial operation in the sitting position.

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#### REPORT OF A CASE

A 25-year-old, 100-kg man had occasional frontal headaches and decreasing hearing in the left ear associated with low-pitched tinnitus. Past medical history was not contributory, and laboratory studies, physical examination, roentgenogram of the chest, and ECG disclosed no abnormality. Audiograms and tomograms were consistent with a diagnosis of 1.5-cm acoustic neuroma. The patient was classified ASA physical status I.

The patient received 50 mg hydroxyzine, 100 mg meperidine, and 0.4 mg atropine, im. An hour later anesthesia was induced with thiopental. Tracheal intubation was facilitated with 100 mg succinylcholine, iv. Anesthesia was maintained with 50 per cent nitrous oxide and 1 per cent halothane in oxygen and pancuronium. Ventilation was controlled with a volume-limited ventilator attached to a circle absorber system. The ECG recorded cardiac rate and rhythm, and a stethoscope and a thermistor probe were inserted into the esophagus. A catheter was placed in the right radial artery, and the tip of a Swan-Ganz catheter<sup>1</sup> was positioned in the pulmonary artery, as confirmed by a transduced pressure-wave pattern. End-tidal CO<sub>2</sub> concentration

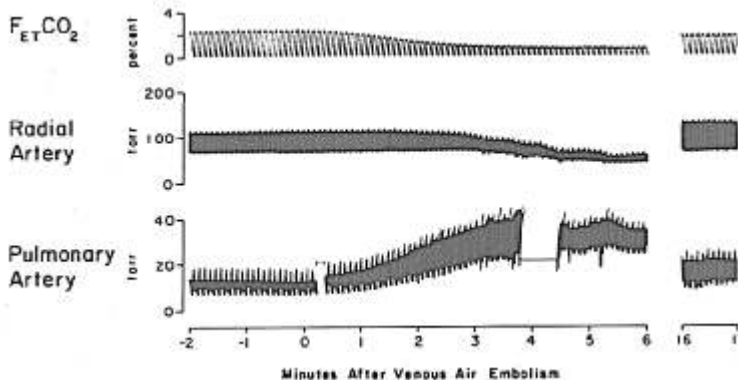


FIG. 1. Cardiopulmonary responses during the third episode of venous air embolism. Note irregular tracing of radial arterial and pulmonary arterial pressures, reflecting premature ventricular contractions. Interruptions in the pressure tracings represent catheter irrigation and attempted air aspiration, respectively.

( $F_{ETCO_2}$ ) was monitored with a Godart capnograph and recorded on a Grass polygraph with the pulmonary and radial arterial pressures. A transthoracic ultrasound Doppler probe was positioned on the chest in the fourth intercostal space at the left sternal border.

At 0930 the patient was placed in the conventional supine sitting position. Head positioning and preparation of the surgical field required 90 minutes, during which time the first measurement of arterial

blood-gas tensions was made (table 1). At 1100 the surgical incision was made. Fifteen minutes later, during neck muscle dissection, a "murmur" was detected with the Doppler monitor, pulmonary arterial pressure (PAP) increased from 16/5 torr to 40/15 torr,  $F_{ETCO_2}$  decreased from 2.6 to 1.0 percent, arterial blood pressure fell from 110/85 to 75/60 torr, and premature ventricular contractions were seen. A tentative diagnosis of venous air embolism was made, nitrous oxide was discontinued, and the

TABLE 1. Arterial Blood-Gas Tensions and Calculated Intrapulmonary Shunt Values during and after Halothane Anesthesia

Time	$F_{I_{O_2}}$	PEEP (torr)	$P_{a_{O_2}}$ (torr)	$P_{a_{CO_2}}$ (torr)	pH <sub>a</sub>	$Q_{\dot{V}_A}/Q_{\dot{V}_T}$
Operative day						
1025	.50	0	106	27	7.53	—
1230	1.0	0	220	32	7.47	.34†
1415	1.0	0	327	41	7.37	.32†
1640*	1.0	0	96	42	7.35	—
1925	1.0	10	161	40	7.36	—
2200	1.0	14	182	37	7.31	.28†
First postoperative day						
0045	.65	14	223	44	7.37	.12§
0430	.36	12	149	39	7.41	.07§
1400	.30	4	121	41	7.47	—
Second postoperative day						
0250	.21	0	73	40	7.50	—

\* Ten minutes after transport to the SICU.

† Calculated from blood-gas values in blood simultaneously drawn from the radial artery and the pulmonary artery; ‡ or right atrium.§

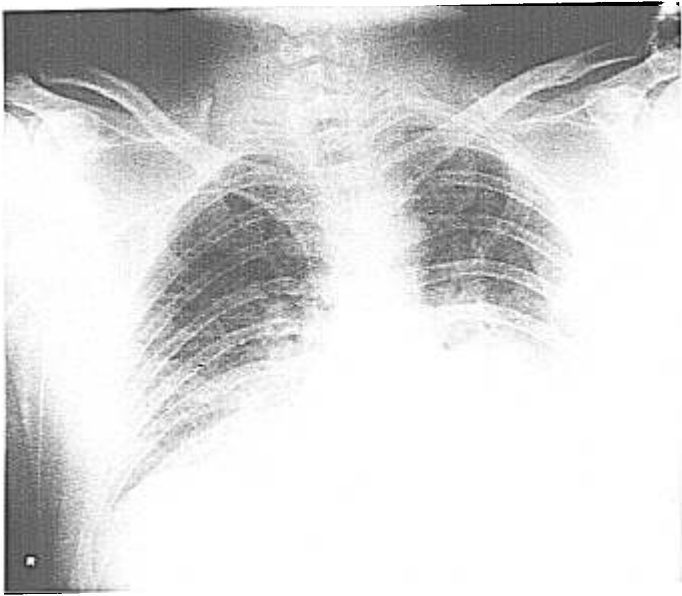


FIG. 2. Roentgenogram of the chest obtained one hour postoperatively, showing basilar alveolar and interstitial infiltrates.

patient was ventilated with 100 per cent oxygen. Fifty milligrams of lidocaine, given iv, abolished the cardiac arrhythmias, and systemic blood pressure returned to pre-embolism levels. At 1145, a similar episode occurred. In addition to administering lidocaine, the patient's position was lowered to 35 degrees from vertical. Ephedrine, 50 mg, was administered iv.

At 1230, radial and pulmonary arterial blood-gas analysis revealed a calculated right-to-left pulmonary shunt ( $Q_{wp}/Q_t$ ) of 34 per cent.<sup>2</sup> At 1320, inhalation of nitrous oxide was reinstated in 50 per cent concentration for 5 minutes. An increase in pulmonary arterial pressure (13/7 to 17/8 torr) and a decrease in  $F_{I\text{O}_2}$  (2.0 to 1.8 per cent) indicated the presence of residual air in the pulmonary circulation.<sup>2</sup> Nitrous oxide was discontinued and anesthesia maintained for the duration of the procedure with halothane in oxygen. Between 1335 and 1515, six more episodes of venous air embolism occurred, showing the same changes in blood pressure and  $F_{I\text{O}_2}$  as described above. The changes recorded during the third episode are shown in figure 1. On several occasions we aspirated small amounts (0.5 to 1.0 ml) of gas through the distal port of the Swan-Ganz catheter.

After the fourth episode we were unable to aspirate through the pulmonary catheter, so the distal port was withdrawn to the right atrium. At 1545, the final venous air embolism occurred during muscle closure. At this time we were able to aspirate a few bubbles of gas from the right atrium. Intraoperative blood replacement (450 ml) was equal to loss, and replacement with lactated Ringer's solution exceeded urinary output by 600 ml.

The patient was taken to the surgical intensive care unit at 1630 with the endotracheal tube in place, breathing spontaneously at 12/min. Breath sounds were equal bilaterally, but rhonchi and basilar inspiratory rales were present. A roentgenogram of the chest revealed bilateral diffuse alveolar and interstitial infiltrates of both lungs (fig. 2). Positive end-expiratory pressure (PEEP), incrementally increased to 14 torr,<sup>4</sup> was added to the spontaneous ventilation pattern, and 40 mg furosemide were administered iv in divided doses. In the next four hours urinary output was 1800 ml. A second roentgenogram of the chest, obtained six hours postoperatively, showed marked clearing of the alveolar and interstitial infiltrates.  $F_{I\text{O}_2}$  and PEEP were progressively lowered, and oxygenation continued to im-

prove. A roentgenogram of the chest obtained 16 hours postoperatively showed continued clearing. The endotracheal tube was removed 24 hours postoperatively, and a roentgenogram of the chest on the third postoperative day disclosed no abnormality.

### DISCUSSION

There have been few clinical reports of postoperative pulmonary complications following intraoperative venous air embolism. Still, Lederman and Renn<sup>5</sup> reported a case of pulmonary edema which they believed was the result of air embolism. However, pulmonary hypertension was not documented, and they assumed that local hypoxia in conjunction with pulmonary hypertension predisposed to the formation of pulmonary edema. Our patient had eight episodes of venous air embolism with pulmonary arterial systolic pressures greater than 40 torr. In addition, these episodes were associated with systemic arterial hypotension with pressures as low as 50 torr systolic, which led to multifocal premature ventricular contractions. Our patient was young, previously healthy, and manifested no sign of postoperative heart failure. Cardiomegaly was absent and postoperative pulmonary-artery wedge pressures were within normal limits. Overtransfusion was unlikely since blood replacement was equal to loss, and crystalloid infusion exceeded urinary output by 600 ml.

The mechanism of pulmonary edema induced by air embolism is not clear, and conflicting reports exist in the literature. Venous air embolism is believed to produce pulmonary hypertension from mechanical obstruction of the precapillary arterioles,<sup>6</sup> as well as reflex vasoconstriction of pre- and post-capillary vessels.<sup>7,8</sup> Pulmonary edema has been reported to occur after venous air embolism in the dog.<sup>9,10</sup> Dahlgren and Josephson<sup>10</sup> showed that pulmonary edema developed in only 12 of 28 dogs and no correlation with increased pulmonary arterial pressure or hypoxemia was observed. In fact, pulmonary edema was found in two dogs in the absence of increased pulmonary arterial pressure. Moss<sup>11</sup> and Daicoff *et al.*<sup>12</sup> have shown that increased resistance distal to pulmonary capillaries from either air or blood clots results in a "tourniquet effect," which also produces pulmonary edema. A centrinergic relationship between the

increased resistance of the post-capillary vessels and inadequate cerebral perfusion also exists. In rats subjected to rapid decompression, intravascular air bubbles cause damage and herniation of capillary endothelial cells of the mesentery through fenestrations in the more rigid vessel wall structures.<sup>13</sup> Mechanical obstruction of pre-capillary arterioles also occurs from embolism of proteinaceous material of platelets and fibrinogen; lipids adhere to the bubble at the gas-blood interface.<sup>13</sup>

It is difficult to attribute the pulmonary edema in our patient to any one of these factors alone. A cumulative effect of several factors may have occurred. During some periods, the effective cerebral perfusion pressure may have been less than the 50 torr recorded from the radial artery at heart level. It is possible that the systemic hypotension was sufficient to have evoked a centrinergic reflex and post-capillary vasoconstriction. The inhalation of 100 per cent oxygen for a relatively long period also may have contributed to local and central neurogenic edema formation.<sup>11</sup> The repeated episodes of pulmonary hypertension may have damaged pulmonary vascular endothelium. Protein aggregations formed at the gas-liquid interface also could have augmented pulmonary capillary blockage, which would persist into the postoperative period.

These clinical observations and experimental studies in animals suggest that pulmonary edema in the neurosurgical patient can occur following venous air embolism. If venous air embolism occurs, the clinician should closely observe the patient for the possible development of pulmonary edema.

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## Chronic Hematoma—A Complication of Percutaneous Catheterization of the Internal Jugular Vein

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Percutaneous catheterization of the internal jugular vein has become a widely accepted technique for placement of central venous catheters. Complications have been rare, and include hematoma formation, hemothorax, pneumothorax, hydrothorax, chylothorax, excessive or persistent bleeding, and cardiac arrhythmias.<sup>1-6</sup> Although many of these complications are potentially serious, hematoma formation has been described as rather common, benign, and without sequela. The following is a report of attempted percutaneous catheterization of the internal jugular vein resulting in the formation of a chronic hematoma, necessitating surgical removal two months later.

### REPORT OF A CASE

A 56-year-old white woman with a four-year history of progressive angina pectoris and two previous myocardial infarctions was admitted for an aorto-coronary-artery-saphenous-vein bypass procedure. Past history included adult-onset diabetes mellitus, hyperlipidemia, and an abdominal hysterectomy without anesthetic difficulty. Physical examination

was unremarkable with the exception of minimal obesity. Medications included digoxin, isosorbide dinitrate, nitroglycerin, and tolbutamide. Preoperative laboratory data, including results of clotting studies, were normal. An electrocardiogram revealed only nonspecific ST and T wave changes and a roentgenogram of the chest disclosed no abnormality. Cardiac catheterization revealed diffuse three-vessel disease with total occlusion of the right coronary artery and 80-90 per cent occlusion of the left anterior descending and circumflex arteries. In view of the severity of the disease, insertion of an intra-aortic balloon pump prior to induction of anesthesia was planned to prevent cardiac decompensation. After intramuscular injection of morphine sulfate, 10 mg, hydroxyzine, 100 mg, and atropine, 0.5 mg, the patient was brought to the operating room. Peripheral venous and left radial-artery catheters were inserted percutaneously. Blood pressure, electrocardiogram, and precordial auscultation were continuously monitored. The intra-aortic balloon pump device was inserted into the left femoral artery after infiltration of 1 per cent lidocaine locally and heparin, 50 mg, iv. General anesthesia was then induced with thiopental 325 mg, and succinyl choline, 100 mg, iv, and nasotracheal intubation performed. Maintenance anesthesia consisted of halothane, nitrous oxide, and oxygen. Due to the absence of suitable peripheral veins, percutaneous placement of a CVP catheter in the right internal jugular vein was attempted, using the method described by Vaughan with a Deseret Intracath.<sup>21</sup> Blood was easily aspirated through the 14-gauge needle on the first attempt; however, the 12-inch 16-gauge catheter could not be easily advanced. Upon needle removal, the catheter still could not be advanced, even though aspiration and injection were easily performed. The catheter was then withdrawn and a pressure dressing applied. Non-

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