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Venous air embolism: Clinical and experimental considerations

STEVEN L. OREBAUGH, MD

Objective: To examine the existing literature concerning venous air embolism. Causes, pathophysiology, and management are emphasized.

Data Sources: The literature that was reviewed was retrieved from the MEDLINE System under the headings "venous air embolism," "air embolism," "therapy of air embolism," "etiology of venous air embolism," and "pathophysiology of venous air embolism" for the years 1970 to 1991. A manual search, derived from the references of these papers, was performed to obtain relevant citations for the years preceding 1970.

Study Selection: Experimental (animal) data, case reports, case series, and clinical investigations are included.

Conclusions: Venous air embolism is an infrequent complication of invasive diagnostic and therapeutic maneuvers. The cardiovascular, pulmonary, and central nervous systems may all be affected, with severity ranging from no symptoms to immediate cardiovascular collapse. Therapeutic interventions include mechanical measures, such as positioning, withdrawal of air from the right atrium, and measures aimed at reducing bubble size. Hyperbaric oxygen therapy holds some promise in accomplishing the latter, but randomized, controlled trials demonstrating efficacy have yet to be performed. (*Crit Care Med* 1992; 20:1169-1177)

KEY WORDS: air embolism, venous; catheters, central venous; pressure, intrathoracic; invasive procedures; dyspnea; complications, iatrogenic;

catheter, pulmonary artery; air embolus, paradoxical; air embolism, cerebral; hyperbaric oxygen

Venous air embolism is an infrequent occurrence that may be an iatrogenic complication of invasive procedures or may result from accidental trauma. Signs and symptoms vary from nonexistent to complete cardiovascular collapse, depending on the amount and rate of air entry. This review focuses on the pathophysiologic consequences and clinical manifestations of venous air embolism, appropriate therapeutic interventions, and the importance of preventive measures.

Etiology and Occurrence. Admission of air into the venous system may occur if several conditions exist: a source of gas (usually the atmosphere), a communication between the venous system and this source, and a pressure gradient favoring ingress of the gas. In the most direct example (1), air can be injected through a catheter into a peripheral vein or infused inadvertently via an improperly vented iv administration set, although this occurrence is infrequently reported. Likewise, air can enter the thoracic veins directly during central catheter insertion or during use after placement, due to the potential for subatmospheric intrathoracic venous pressure (2-14). Venous air embolism has been reported in penetrating chest trauma in humans (15) and in animals (16), and during surgical procedures involving lung parenchyma (17), as well as with bronchopulmonary venous fistulas caused by blunt chest trauma (18).

Neurosurgical and head/neck surgical procedures in the sitting position, and insufflation of air or other gases into the peritoneum, are other settings in which venous air embolism frequently has been described (19-22). Durant et al. (23) reported the occurrence of air embolism in the setting of maxillary sinus lavage, pneumothorax, and pneumoperitoneum. In total hip arthroplasty, venous air embolism is more frequent than previously thought, and is a probable cause of hemodynamic disturbances that formerly were attributed to methylmethacrylate cement (24-26). Use of the neodymium yttrium-argon-garnet laser in uterine surgery

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has resulted in several deaths attributed to air embolism, as the instillation of air, CO₂, or nitrogen gas at high flow rates is required to cool the sapphire tip during the procedures (27). Positive-pressure ventilation in infants with hyaline membrane disease, and high peak airway pressure in mechanically ventilated adults both have been reported to cause venous air embolism (28–30). Vaginal insufflation during orogenital sex in pregnancy has also resulted in venous air embolism (31, 32).

The true occurrence of venous air embolism is uncertain, as this entity may be obscured by resultant cardiovascular, pulmonary, or neurologic manifestations that are felt to be primary in nature (33). If autopsy examination is delayed, air in the venous system or capillaries may be resorbed, contributing to the difficulty in diagnosis (21). In the operating room, where constant monitoring is done in high-risk patients, the occurrence rate of venous air embolism in neurosurgery or head/neck surgery when the patient is in the seated position is reported to be 21% to 40% (33).

Venous Air Embolism in Central Venous Catheterization. In the ICU, venous air embolism is most likely to be encountered during or after insertion of a central venous catheter. The occurrence rate of this problem is variable, reported from one in 47 central venous catheter insertions (34) to none in 355 (35, 36). Feliciano et al. (14) noted two cases in their report of >1500 subclavian vein catheterizations; James and Myers (10) reported only one case, a fatality, in 3,000 attempts at supraclavicular subclavian catheter insertion. The mortality rate of venous air embolism is significant: by 1987, a total of 79 cases had appeared in the world literature, and 25 (32%) of these cases had had a fatal outcome (3).

Since normally protective extrathoracic venous valves are transgressed when a central venous catheter is inserted, free communication may develop between the intrathoracic vasculature and the atmosphere. Admission of air then occurs whenever iv pressure decreases below ambient air pressure (33). Negative intrathoracic venous pressure is most likely to occur with deep inspiration, dyspnea, or hypovolemia, and in the upright position (5). During strained respiration, thoracic vein pressure may decrease 25 mm Hg below atmospheric pressure (21).

Numerous case reports (2–14) of venous air embolism related to central venous catheter insertion or use exist in the medical and surgical literature. Air may enter the great veins directly after the needle is inserted, if the hub is not conscientiously occluded (2, 4, 5, 14). However, the majority of cases occur not with the insertion of the catheter, but during use or maintenance of the catheter (2). Catheter hub fracture

or disconnection has been associated with 63% to 93% of episodes of venous air embolism related to central venous catheters (5). Even a tract that forms during the use of a catheter-through-the-needle device, or a tract that remains after removal of a long-indwelling catheter can lead to venous air embolism (37, 38). Gas sequestered in the pleural space has been described as a source of embolism with subclavian vein catheterization (39).

Pulmonary artery catheter introducers have been implicated as causes of venous air embolism as well. Soon after the development of pulmonary artery catheters, anesthesiologists noted a potential for air embolism through introducers that were left in place for iv access after removal of the catheter (34, 40, 41). However, changes made by manufacturers followed, and self-sealing valves in introducers became standard (38). Some (40) suggested that valves in introducers might malfunction after being violated by pulmonary artery catheters, but *in vitro* studies (42) of four common brands failed to demonstrate airleaks in the unused state or after 48 hrs of cannulation by a catheter. Nevertheless, it is recommended that the introducer be removed along with the pulmonary artery catheter, or that an obturator be used to occlude the introducer if left in situ (40). Pulmonary artery catheters have been reported to cause venous air embolism related to pulmonary artery rupture after balloon inflation (43).

Pathophysiology. The alterations in cardiovascular and pulmonary physiology, resulting from admission of air into the venous system, have been well studied in animals. In 1947, Durant and colleagues (23) reported that the most important factors in determining mortality after experimental venous air embolism in dogs were: a) the amount of air entering the veins, b) the speed with which it enters, and c) the position of the body at the moment of embolization. Adornato et al. (44) distinguished between the cardiovascular effects of bolus venous air embolism and the effects of slow air infusion as can occur with a break in an iv line. With a bolus, the authors (44) noted an increase in central venous pressure, a decrease in pulmonary artery pressure, ST-segment depression on the electrocardiogram, and shock. These changes were thought to be due to an "air lock" in the right heart that was obstructing outflow. Slow, continuous infusion of air yielded an increase in central venous pressure and abrupt increase in pulmonary arterial pressure, decreases in systemic vascular resistance and mean arterial pressure (MAP), and a compensatory increase in cardiac output. Thus, the basic disturbance of cardiovascular function in venous air embolism is obstruction to right ventricular ejection. This disturbance may be at the pulmonary outflow tract, as in bolus air embolism, or at the level of

the pulmonary arterioles, as seen with slower infusions (21). Small bubbles lodge in pulmonary arterioles and create mechanical obstruction to flow, causing pulmonary arterial hypertension (7, 33, 45). Pulmonary vasoconstriction, demonstrated by Berglund and colleagues (46) in a canine model subjected to a slow air infusion, results in further compromise of right heart function. Durant et al. (23) reported rapid right ventricular dilation with evidence of ischemia after injection of an air bolus in dogs.

Similar effects are evident in humans. Bedford and co-workers (47) evaluated 100 seated patients during neurosurgical procedures with invasive hemodynamic monitoring and precordial Doppler ultrasound, and they detected 80 episodes of air embolism. Of these patients, 36 demonstrated increased pulmonary artery pressure, of whom a minority became hypotensive. Only those patients with an increase in pulmonary arterial pressure developed a decrease in MAP. A correlation was noted between the severity of hypotension and the degree of increase of pulmonary arterial pressure (47). Others (48) reported increased pulmonary arterial pressure in venous air embolism in humans as well. Venous air embolus occurring during surgery on the right side of the heart, resulting in postoperative pulmonary hypertension, has also been described (45).

Adornato et al. (44) found that air infusion rates of >1.8 mL/kg/min were fatal in dogs. They reported that a bolus of air >200 mL rapidly injected caused ventricular tachycardia and death in these animals. They (44) concluded that amount of air and speed of infusion are important factors affecting the mortality rate of venous air embolism (23). The lethal volume decreases as the rate of air entry increases. In humans, the fatal dose is uncertain, but estimates vary between 300 and 500 mL of air at 100 mL/sec (7). Such flow rates are attainable through a 14-gauge needle, if a pressure gradient of 5 cm H₂O exists (8). Lower volumes may prove lethal to the critically ill patient with compromised hemodynamics.

Microbubbles introduced into the pulmonary circulation result in significant ultrastructural changes and abnormalities of lung extravascular water content (49–51). The bubbles themselves obstruct flow and increase pulmonary arterial pressures; on the bubbles' surfaces, a network of fibrin, platelets, RBCs, and fat globules is constructed, which serves to further restrict blood flow (33). In sheep, Albertine et al. (51) demonstrated that neutrophils clumped around small bubbles in pulmonary arterioles are attached to the endothelium of these vessels. Ultrastructural changes included gaps between endothelial cells and disruption of the underlying basal lamina. This disruption produced an

increase in lung lymph flow and protein flux, which was also demonstrated after infusion of 300- μ m-diameter bubbles in sheep by Ohkuda et al (52).

The role of the neutrophil in lung damage produced by venous air embolism was further emphasized by Flick et al. (53), who demonstrated a greater increase in lung lymphatic flow after air infusion in sheep with normal numbers of circulating leukocytes, as compared with those sheep that had been rendered leukopenic. Jérôme and colleagues (54) demonstrated protection from an increase in lung lymph flow and granulocytes in lung lymph when methylprednisolone was given before or during venous air embolism. Flick and co-investigators (55) pursued the nature of lung microvascular injury by pretreating a sheep model of venous air embolism with heparin and superoxide dismutase. The characteristic increase in lung lymph flow and lymph protein content observed with venous air embolism was effectively prevented. The investigators (55) concluded that the superoxide anion, probably produced by leukocytes, plays an important role in the pulmonary microvascular damage of air embolism. In a related study (56), Flick et al. pretreated sheep subjected to air embolism with 50 mg/kg of iv catalase, which also prevented the expected effects of venous air embolism on lung lymph flow and protein content, supporting a role for the hydrogen peroxide molecule in the mechanism of pulmonary injury in this setting.

These histopathologic and cellular changes led to prominent abnormalities of pulmonary function. Lung compliance is reduced as a result of increased permeability and increased interstitial edema, and airway resistance is thereby increased (33). Hlastala et al. (57), using the inert gas elimination technique, demonstrated abnormalities of pulmonary ventilation-perfusion matching with resultant increases in physiologic dead-space and physiologic shunting. These aberrations underlie the hypoxemia universally found in victims of clinically important venous air embolism, both human and animal.

Natural History of Cardiovascular and Pulmonary Alterations in Venous Air Embolism. The natural history of venous air embolism has been described in animal models (23, 46, 50, 57). Adornato et al. (44) noted the changes in vital signs and venous pressures that occur with both graded venous air infusion and bolus introduction of air. In air infusion, the central venous pressure, the right pulmonary arterial pressure, and the heart rate all increased in a dose-related fashion, with air infusion rates of ≥ 0.4 mL/kg/min. At rates of >1.5 mL/kg/min, bradycardia supervened and cardiovascular decompensation began. No significant change occurred in BP until a rate of 1.7 mL/kg/min had been reached; thereafter, BP decreased rapidly. When

canines were subjected to iv boluses of air, the central venous pressure and heart rate again increased linearly with the dose of air, while BP predictably decreased in a dose-related manner. No change occurred in pulmonary arterial pressures until a dose of >100 mL of air was given. A 200-mL dose resulted in a coarse "mill-wheel" heart murmur and the death of the animal. In an investigation (58) of the hemodynamic effects of venous air embolism in sheep, a 60-mL air bolus rapidly caused a significant decrease in cardiac output in the majority of subjects within 1 min, while recovery to normal values was evident by 10 mins. The alterations in ventilation-perfusion matching demonstrated by Hlastala et al. (57), resulting from gas bubble infusion into canines, returned to normal 30 mins after the pulmonary insult. Presson et al. (49) described the effect of venous air emboli in dogs by directly observing the pulmonary circulation with an *in vitro* microscopy technique while air was being infused into the animals. Microbubbles were observed to lodge in the pulmonary arterioles and underwent spontaneous resorption after 60 to 180 secs. Larger doses of air seem to take longer to resolve. In a canine study (58), air bubbles persisted in the pulmonary circulation for 17.8 mins after an air bolus of 2 mL/kg when infused at 2 mL/sec. Butler et al. (59) demonstrated that pulmonary vascular bubbles remained for a mean of 26.9 mins.

Systemic Embolism Resulting From Venous Air Embolism. Venous air emboli may also result in air reaching the systemic circulation (60, 61). This phenomenon is referred to as "paradoxical" air embolism and usually occurs in the setting of a patent foramen ovale (61). While disturbances of cardiopulmonary physiology may be profound with air embolus confined to the right heart or pulmonary circulation, the consequences of systemic embolization are even more severe. Even small amounts of air can produce end-organ ischemia and infarction: as little as 0.05 mL of air injected into a coronary artery may cause focal infarction (62). Air emboli to the cerebral circulation frequently result in focal neurologic deficits (60).

Clinical Manifestations. Venous air embolism is variable in its clinical expression, both in its severity and in the organ systems affected. In canines (44) and in humans (63), a "gasp" has been reported to follow the initial infusion of air into the pulmonary circulation, possibly a reflex response. Intrathoracic pressure is then decreased, facilitating greater air entry. Patients often complain of breathlessness, light-headedness, chest pain, or a feeling of impending death (4, 7, 33). In one series of 14 cases, 100% of patients had sudden onset of dyspnea (64).

Signs that reflect the severity of air embolism are seldom specific for this entity. Significant emboli result

in tachypnea, tachycardia, and frequently, hypotension. The only sign specific for air embolism, the rare "mill-wheel" murmur, is dependent on the presence of a large bolus of air in the right ventricle (21, 38, 63). A harsh systolic murmur (65) or normal heart sounds are more commonly found on examination. In a large proportion of patients, neurologic signs predominate. Kashuk and Penn (66) described a predominance of central nervous system manifestations in 42% of 24 instances of venous air embolism reviewed. These manifestations included altered mental status, frank coma, and focal deficits. Uncertainty exists as to whether hypoxia, hypotension, paradoxical embolism, or some combination of these factors are responsible for this neurologic compromise (4).

Minutes to hours after air embolism occurs, rales or wheezing can be detected on auscultation of the lung fields, evidence of pulmonary edema (66). In animal models (53-56, 67-69), the coexistence of increased pulmonary arterial pressure and pulmonary edema protein content with normal pulmonary artery occlusion pressures suggests that increased vascular permeability underlies the development of abnormal extravascular lung water.

Laboratory abnormalities also vary with the severity of the embolism, and lack specificity for this process. Electrocardiographic changes evident in canines include peaking of the P-wave and ST segment depression (23, 44). Reports in humans emphasize sinus tachycardia, nonspecific ST-segment and T-wave changes (13), and evidence of acute right heart strain (38, 70). Chest radiographs are usually normal initially, but later radiographs may show evidence of noncardiogenic pulmonary edema (67-69). An air fluid level in the central pulmonary artery or ventricle is rarely seen (6). Arterial blood gases reflect abnormalities of matching of ventilation and perfusion, usually as hypoxia, which may be profound (71). Hypercarbia is occasionally seen.

When utilized, invasive hemodynamic monitoring provides further evidence of clinically important air embolus. Increases in central venous pressure and pulmonary artery pressure are dose-dependent in animals subjected to slow air infusion (44). However, pulmonary arterial pressure may remain normal with an increased central venous pressure, presumably due to a large air bubble trapped in the right ventricle or pulmonary outflow tract. This phenomenon occurs in animals with massive air embolism (>5 mL/kg) (48). Of 40 cases of venous air embolism occurring during neurosurgery in the seated position in patients monitored with central venous catheters, 25% had an increase of central venous pressure (65). In a similar population of 100 patients monitored with precordial Doppler,

pulmonary artery catheters, and end-tidal Pco_2 equipment, Bedford et al. (47) described the occurrence of 80 cases of venous air embolism detected by Doppler, of which nearly half developed an increased pulmonary arterial pressure.

The utility of end-tidal Pco_2 monitoring to detect clinically important venous air embolism is well described (20, 21, 72). In the above-noted study of Bedford et al. (47), 30 of the 80 patients with venous air embolism reportedly had a decrease in end-tidal Pco_2 , reflecting disordered ventilation-perfusion matching and increased physiologic deadspace. Other useful indicators of venous air embolism include aspiration of air or foam from an indwelling central venous catheter or pulmonary artery catheter (48), and ultrasonography. Both Doppler ultrasound and precordial echocardiography are sensitive in the detection of intraoperative air embolism (71). However, only transesophageal echocardiography has proven useful in documenting paradoxical air embolism (73). Finally, Matjasko et al. (74) investigated end-tidal nitrogen monitoring for venous air embolism in canines and found that end-tidal nitrogen pathologically increases before the decrease in end-tidal Pco_2 in bolus embolism.

Differential Diagnosis. Venous air embolism may occur with a wide variety of signs and symptoms, and runs a continuum of severity from clinically insignificant to life-threatening, allowing an extensive differential diagnosis. When respiratory complaints and signs are primarily manifest, venous air embolism must be differentiated from other causes of sudden pulmonary compromise, such as pulmonary thromboembolism, pneumothorax, acute bronchospasm, and acute pulmonary edema. When no obvious predisposing factors or specific signs for venous air embolism are present to guide the clinician toward this diagnosis, consideration should be given to performing pulmonary angiography (or ventilation-perfusion scanning) to distinguish air embolism from thromboembolism, particularly in patients at high risk for the latter. If signs of central nervous system embarrassment predominate, the clinician must consider focal or global brain ischemia, hemorrhage into the parenchyma or subarachnoid spaces, hypoxia, trauma, and metabolic causes of rapid central nervous system deterioration such as hypoglycemia.

In many cases, cardiovascular compromise dominates the clinical picture. Usually, this situation takes the form of hypotension, in which case, venous air embolism must be considered, along with other causes of obstructive shock, hypovolemia, primary cardiac dysfunction, and acute vasodilatory states such as septic shock. Venous air embolism has also been reported (43, 75) to manifest as electromechanical

dissociation, a potential result of right ventricular outflow obstruction, reducing the cardiac output without initially disturbing cardiac electrical activity. Therefore, one should consider this entity in the differential diagnosis of electromechanical dissociation when the patient is at risk for venous air embolism.

Management of Venous Air Embolism. Identification of the source of air entry and immediate measures to counter this entry are the first priority in the treatment of venous air embolism. Subsequent management strategies are based on evidence derived from animal investigations and case series in humans. Five areas of therapeutic intervention are discussed in the following sections: patient positioning, closed-chest cardiac massage, removal of air from the venous circulation, attenuation of bubble size, and miscellaneous measures not yet thoroughly investigated but with some promise in alleviating the effects of air embolism.

Patient Positioning. Durant et al. (23), in their early work with air embolism in animals, demonstrated increased tolerance by dogs to air infusion when the animal was lying on the left side, and improvement in hemodynamics of a compromised animal when turned onto the left side. On the basis of observations in open-chest animals, they (23) concluded that the left lateral decubitus position placed the right ventricular outflow tract in a position inferior to the right ventricular cavity, so that an air bolus would migrate superiorly, removing the obstruction to blood flow. Placing the patient with suspected venous air embolism in this position remains a cornerstone of management today (64, 76). The head-down or Trendelenburg position is recommended as well, apparently with similar effects on the position of the offending "air lock" in the right ventricle (7).

Closed-Chest Cardiac Massage. Closed-chest cardiac massage has been recommended as a means to force air out of the pulmonary outflow tract and into small pulmonary vessels, as it was successfully used by Ericsson et al. (77) in the resuscitation of four of five patients sustaining venous air embolism during neurosurgical procedures. External cardiac massage has been evaluated in canines as therapy for venous air embolism (76) and was found to improve survival rate as effectively as use of the left lateral decubitus position or aspiration of air from the venous system.

Removal of Air From the Venous Circulation. A more direct approach to the treatment of air embolism is withdrawal of air from the right heart. In 1956, Stallworth et al. (78) reported a case of cardiac arrest from venous air embolism occurring in the operating room. The patient was treated with percutaneous needle aspiration of 15 mL of air from the right ventricle, resulting in prompt hemodynamic improvement. More

recent reports have focused on removal of intracardiac air via central venous catheters. In canines, Adornato et al. (44) demonstrated a "dramatic reversal of shock with aspiration of air from the atrium." A study (76) comparing intracardiac aspiration from the right ventricle to external cardiac massage and to the left lateral decubitus position in treatment of venous air embolism in dogs resulted in similar survival in all three groups but significantly more rapid resuscitation with air aspiration than mechanical measures. Colley and Artru (79) found that a greater proportion of injected air could be removed from the right atrium of dogs with venous air embolism using a multiple-orifice central venous catheter than by either a 7-Fr pulmonary artery catheter or single port central venous catheter, resulting in significantly improved survival rate.

Data in humans are less promising. Michenfelder et al. (65) evaluated right atrial catheters in 23 episodes of venous air embolism in upright patients undergoing neurosurgical procedures. Significant air retrieval with a favorable influence on resuscitation occurred in only eight cases. In the series by Bedford et al. (47) of 100 patients undergoing seated neurosurgical procedures, 80 episodes of venous air embolism were detected by Doppler, but recovery of air from the pulmonary artery and the right atrium was minimal, only 2 to 20 mL. The authors concluded: a) recovery of air was not a major factor in improving hemodynamics, and b) packing the site of air entry with sponges and discontinuing nitrous oxide were more efficacious. Thus, withdrawal of air from the right atrium or ventricle is useful in venous air embolism if a catheter is present, but it does not justify the time and effort necessary to insert a central venous catheter in the acute situation if not already in place.

The optimal position of a right atrial catheter for recovery of air in venous air embolism was evaluated by Bunegin and co-workers (80), who utilized a Silastic model of the right atrium with pumps, infusing normal saline to evaluate fluid and gas interactions. Maximum recovery of air was achieved when the multiorifice catheter tip lay 2.0 cm distal to the junction of the mock superior vena cava and atrial chamber at an inclination of 80° from the horizontal.

Attenuation of Bubble Size. To reduce the size of embolized bubbles, all patients with suspected venous air embolism should be placed on 100% oxygen (33), as this approach favors nitrogen diffusion out of the bubbles and into the alveoli (nitrogen "washout"). For patients under anesthesia, it is imperative that nitrous oxide be discontinued, as it diffuses rapidly into all air-containing cavities in the body, including bubbles, significantly increasing their size and the obstruction they present to the circulation (81).

For those patients not responding to these measures, hyperbaric oxygen therapy should be considered. While well-controlled, randomized trials comparing this therapy with standard measures have not been conducted, many physicians advocate hyperbaric oxygen use on the strength of numerous case reports (82–88), reflecting clinical improvement in cerebral air embolism of venous or arterial origin after hyperbaric oxygen therapy. This modality has long been used in the therapy of arterial gas emboli resulting from decompression sickness in divers (89), but only more recently has hyperbaric oxygen therapy been applied to cerebral injury related to air embolism from therapeutic misadventures (84). Murphy et al. (60) reported a case series of 16 victims of cerebral air embolism from a variety of causes with signs of central nervous system dysfunction in which hyperbaric oxygen therapy was used. Fifty percent of the patients had complete resolution of signs of cerebral air embolism, while 31% had partial improvement. A delay in therapy of >5 hrs reduced the likelihood of improvement. However, complete resolution of neurologic signs and symptoms after air embolism has been reported with hyperbaric oxygen therapy for patients brought to therapy ≤29 hrs after the initial insult (32). While most studies in humans focused on improvements in neurologic manifestations of venous air embolism with hyperbaric oxygen therapy, Baskin and Wozniak (84) reported reversal of all cardiopulmonary abnormalities in seven of nine patients who sustained venous air embolism from hemodialysis, with more gradual resolution in the other two patients.

Hyperbaric oxygen may benefit patients with air embolism in several ways: a) descent to six atmospheres of pressure significantly reduces bubble size and relieves obstruction to blood flow; b) the extremely high P_{aO_2} and arterial oxygen saturation of hemoglobin that are achieved improve oxygen delivery to tissues that are partially ischemic; and c) high alveolar P_{O_2} favors nitrogen diffusion from bubbles in the pulmonary circulation into the alveoli, again reducing bubble size (83, 86). Thus, hyperbaric oxygen may improve hemodynamic compromise from right ventricular or pulmonary arteriolar obstruction, as well as reduce end-organ ischemia.

Hyperbaric oxygen therapy must be carried out under the direction of a physician experienced and knowledgeable in this area. Despite the anecdotal evidence cited, recent doubt has been expressed (90) concerning the efficacy of hyperbaric oxygen in cerebral air embolism; scientifically valid data are needed in this area.

Miscellaneous Measures. Several measures reported to be useful in the therapy of venous air embolism have

not yet been adopted in the treatment of humans. Tuman et al. (91) evaluated the protective effects of a perfluorocarbon emulsion as pretreatment in a canine model of venous air embolism. Dogs so treated sustained less of a decrease in cardiac index, left ventricular stroke work index, and MAP than the untreated controls, as well as significantly less hypoxia and hypercarbia. Studies in different animal models (92, 93) also demonstrated protective effects of fluorocarbon emulsions in air embolism. Cardiopulmonary bypass has also been advocated in the therapy of venous air embolism to ameliorate right heart strain (14). However, this therapeutic modality has not been systematically evaluated and its utility remains dubious.

Prophylaxis. Most episodes of venous air embolism are related to central venous catheters, making preventive measures particularly important when these catheters are inserted, used, or removed. The patient to undergo catheterization of the central veins should be placed in Trendelenburg position, which increases the central venous pressure and reduces the likelihood of air entry through the needle or catheter (4, 33). After entering the vein, and before insertion of the guidewire during use of the Seldinger technique, one should occlude the needle hub, releasing the obstruction to insert the wire only after asking the patient to perform the Valsalva maneuver, or time the insertion of the wire to coincide with exhalation (5, 82). Ellermeyer and Keifer (94) developed a through-the-plunger guidewire system that may reduce the risk of air embolus.

Ensuring adequate hydration before catheter insertion also mitigates against the low venous pressures that will permit air entry (78). All connections to the central venous line should be tightly sealed using Luer-Lok connections (4). Placing an occlusive dressing over the catheter when in place, and over the tract for 24 hrs after removal, will also help prevent air emboli (5). As with insertion, removal of the catheter in a head-down position is recommended.

In the operating room, monitoring for air embolism during high-risk procedures with precordial Doppler and end-tidal P_{CO_2} monitoring has become standard (33). Gas insufflation for diagnostic purposes should be accomplished with CO_2 , not air, as the former has a low surface tension and causes less obstruction if embolism occurs.

Finally, if a patient experiencing air embolism can be ventilated with positive pressure, this procedure is preferable to spontaneous ventilation, as it reduces unfavorable pressure gradients permitting air entry. However, high peak inspiratory pressure causing barotrauma may, in itself, be a cause of venous air embolism and must be avoided (29).

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