

Dennis M. Fisher, M.D., Editor

Anesthesiology

1999; 90:564-75

© 1999 American Society of Anesthesiologists, Inc.

Lippincott Williams & Wilkins, Inc.

Systemic Air Embolism after Lung Trauma

Anthony M.-H. Ho, M.D., M.Sc., F.R.C.P.,* Elizabeth Ling, M.D., M.Sc., F.R.C.P.C.†

LUNG trauma involving laceration of air passages, lung parenchyma, and blood vessels may result in direct communication of these structures. Systemic air or gas embolism (SAE) occurs when air or gas enters the pulmonary venous system as a result of a positive gradient caused by low pulmonary venous pressure (as in hypovolemia) or increased airway pressure (as in positive pressure ventilation [PPV], coughing, or tension pneumothorax). Pulmonary venous air embolizes to the heart and to coronary and cerebral arteries with catastrophic consequences (fig. 1).¹ Two milliliters of air injected into the cerebral circulation can be fatal.^{2,3} Likewise, 0.5–1 ml of air injected into a pulmonary vein can cause cardiac arrest from coronary air embolism and ischemia.⁴

Clinical systemic air or gas embolism has been recognized increasingly since 1973,⁵ albeit with controversy and skepticism.⁶ Trauma clinicians have witnessed the sudden collapse of these patients, often after tracheal intubation and initiation of assisted ventilation.^{5,7–17} The collapse is typically unresponsive to conventional resus-

citation.^{5,7–17} In the ensuing emergency thoracotomy, the nonfunctional heart shows little evidence of direct trauma, and air is seen in the coronary arteries, left cardiac chambers, or in the aortic root.^{5,7–16} Less frequently the development of central nervous system (CNS) dysfunction in previously conscious patients without obvious head injury may implicate cerebral air embolism.¹⁷

To date most reports of SAE have been published in the surgical literature.^{1,5–18} Not surprisingly the solutions offered have largely been surgical. These solutions include immediate thoracotomy to clamp the ipsilateral hilum (if the source of SAE is unilateral), arresting the continuous passage of air entering the left heart and systemic circulation.^{8,19} Hyperbaric oxygen therapy (HBOT) has also been used to manage cerebral dysfunction with good results.¹⁷ The *airway and breathing* portions of resuscitation, unfortunately, have received little attention.

In a letter to the editor, Moon and Piantadori²⁰ suggested selective unilateral lung ventilation to prevent further gas embolization from the involved lung. This idea was dismissed by Trunkey²¹ as impractical. In all published cases of SAE, no special airway or ventilation intervention was used until recently. In 1995, Saada *et al.*⁷ described the use of high-frequency ventilation (HFV) and small ventilatory volume in three patients with SAE. Using transesophageal echocardiography (TEE), they documented that air embolism ceased without thoracotomy simply by altering ventilation.

In the anesthesiology literature, SAE is discussed briefly in subspecialty texts dedicated to trauma.^{22,23} These discussions echo viewpoints previously published in the surgical literature and offer no new insights into airway and ventilatory management. Proper airway and ventilatory interventions remain unexplored issues within the domain of the specialty of anesthesiology and may be important in the prevention and management of SAE. Unless cardiac tamponade or mediastinal hemor-

* Associate Professor, Department of Anaesthesia and Intensive Care, Faculty of Medicine, The Chinese University of Hong Kong, and Associate Clinical Professor, Department of Anaesthesia, McMaster University Faculty of Health Sciences.

† Assistant Clinical Professor, Department of Anaesthesia, McMaster University Faculty of Health Sciences, and Staff Anaesthetist, Hamilton Health Sciences Corporation.

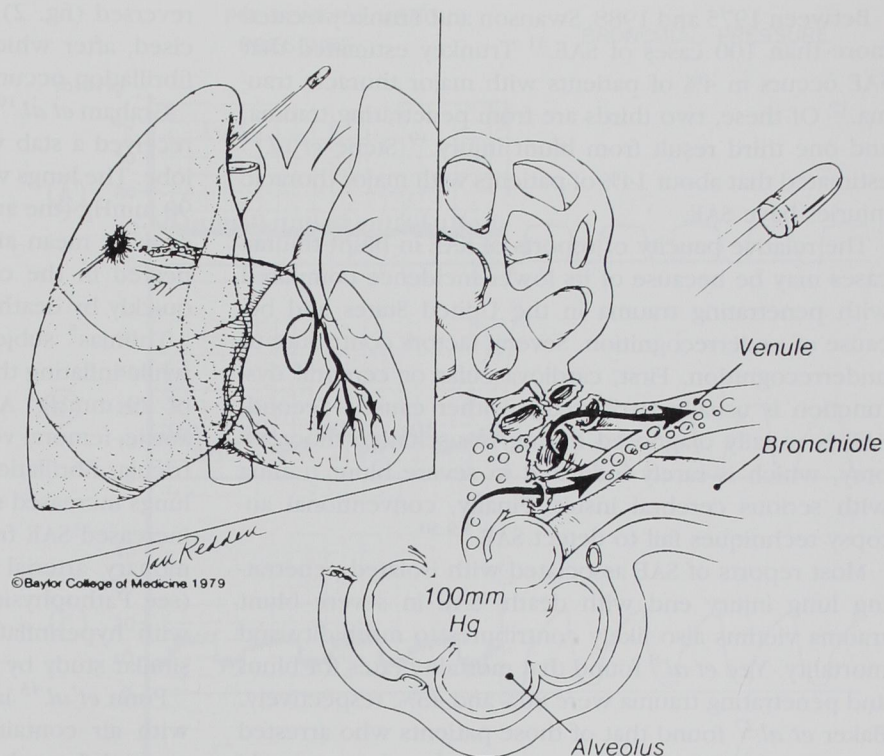
Received from the Department of Anaesthesia and Intensive Care, Faculty of Medicine, The Chinese University of Hong Kong, Shatin, NT, Hong Kong, the Department of Anaesthesia, McMaster University Faculty of Health Sciences, and Hamilton Health Sciences Corporation, Hamilton, Ontario, Canada. Submitted for publication October 1, 1997. Accepted for publication July 14, 1998. Support was provided solely from institutional and/or departmental sources.

Address reprint requests to Dr. Ho: Department of Anaesthesia and Intensive Care, The Prince of Wales Hospital, Shatin, NT, Hong Kong. Address electronic mail to: hoamh@hotmail.com

Key words: Airway pressure; bronchiole-pulmonary venous fistulas; cardiac and cerebral ischemia; lung isolation.

SYSTEMIC AIR EMBOLISM AFTER LUNG TRAUMA

Fig. 1. Air embolization through a direct bronchiole-pulmonary venule communication created in lung trauma. From Mattox.¹



rhage is present or suspected, thoracotomy may be avoided or at least delayed until the patient is stabilized and brought to the operating room (OR).

The objective of this article is to discuss the recognition and management of SAE resulting from fistulization between the pulmonary veins and the airway. A portion of this article is devoted to the hypothesis that early lung isolation in the management of SAE may decrease morbidity and mortality. Lung isolation using special tracheal tubes will not occur in a trauma setting unless the anesthesiologist, surgeon, and emergency physician consider the possibility and understand the pathophysiology of SAE.

Epidemiology of Traumatic Systemic Air Embolism

Almost half of all trauma patients sustain some degree of thoracic injury.²⁴ In the United States, 20% of all trauma deaths per year are attributable to chest trauma. This is approximately 16,000 deaths per year.²⁵

Before the early 1970s, SAE after trauma was largely unrecognized.⁵ The first seven cases of traumatic SAE were published by Thomas and Stephens of the San Francisco General Hospital in 1973⁵ and 1974.⁹ Since those two reports,^{5,9} more than 100 cases^{1,7-18,26-30} of

SAE, mostly from penetrating trauma, have been published in the English language literature.

At the San Francisco General Hospital, the charts of 447 trauma patients between 1970 and 1981 were reviewed.⁸ Complete data to diagnose SAE were found for 61 patients (14%); of them, 15 sustained blunt trauma, and 46 sustained penetrating trauma. Between 1972 and 1978, there were 168 cases of emergency room (ER) thoracotomy at that institution, 11 (7%) of which were to manage SAE.²⁷ Robison *et al.*²⁶ reported 5 deaths among 18 cases of emergency lung resection after penetrating chest injuries; two were from SAE. Wiencek *et al.*²⁸ examined 25 cases of central lung injuries involving hilar vascular structures; 8 (32%) deaths were the result of hemorrhage, and 6 (24%) were caused in part by SAE. The same authors²⁹ also reviewed 138 cases of emergency thoracotomies for penetrating trauma; they found 6 patients (4%) with gunshot wounds to the chest with hemoptysis who arrested immediately after intubation and institution of mechanical ventilation. Air was found in the coronary arteries on autopsy. King *et al.*³⁰ studied 12 deaths in which the only thoracic injury after penetrating chest trauma was to the pulmonary parenchyma. Dissecting the isolated heart under water revealed left cardiac air emboli in nine cases.

Between 1975 and 1988, Swanson and Trunkey treated more than 100 cases of SAE.³¹ Trunkey estimated that SAE occurs in 4% of patients with major thoracic trauma.¹⁹ Of these, two thirds are from penetrating trauma, and one third result from blunt injury.¹⁹ Stene *et al.*²² estimated that about 14% of patients with major thoracic injuries have SAE.

The relative paucity of reports of SAE in blunt trauma cases may be because of its lower incidence compared with penetrating trauma in the United States and because of underrecognition. Several factors contribute to underrecognition. First, cardiovascular or cerebral dysfunction is usually attributed to other causes. Second, SAE is usually diagnosed during resuscitative thoracotomy, which is rarely indicated in severe blunt trauma with serious cerebral insult. Finally, conventional autopsy techniques fail to detect SAE.^{5,9,30}

Most reports of SAE associated with isolated penetrating lung injury end with death. SAE in severe blunt trauma victims also likely contributes to morbidity and mortality. Yee *et al.*⁸ found that mortality rates for blunt and penetrating trauma were 80% and 48%, respectively. Baker *et al.*²⁷ found that of those patients who arrested because of SAE requiring emergency thoracotomy, only 9% survived. For those who required emergency thoracotomy for hypovolemic arrest or tamponade, survival rates were 14% and 38%, respectively.²⁷

The association of SAE with primary blast injury, a special form of blunt trauma, is well known.³²⁻⁴⁰ Blast injury is common in military conflict but also can be seen in civilian practice, given the rising incidence of terrorism and the occurrence of industrial and domestic explosions.

The incidence of SAE in primary pulmonary blast injury is unknown. The fact that it does not occur in all blast injuries is highlighted by cases of blast-induced ruptured viscus that were repaired under general anesthesia using PPV and N₂O.^{39,40} There was no apparent cardiovascular or neurologic sequelae despite the presence of extensive lung injury and hemoptysis.

Animal Studies

Chiu *et al.*⁴¹ measured bronchial, pulmonary venous, and left atrial pressures in four ventilated dogs. When bronchial pressures were low, pulmonary venous pressures were comparatively higher. As airway pressure increased, pulmonary venous pressure increased but to a lesser degree so that the pressure gradient eventually

reversed (fig. 2). SAE occurred when the lung was incised, after which coronary air emboli and ventricular fibrillation occurred in all dogs.

Graham *et al.*¹⁰ studied eight ventilated dogs. Each dog received a stab wound with a scalpel to the left upper lobe. The lungs were ventilated with pressures averaging 90 mmHg (the authors did not state whether these were peak or mean airway pressures). Within 5 min, air appeared in the coronary arteries in all cases, followed quickly by death.

Thomas⁵ subjected 14 dogs on PPV to a lung stab while inflating the lungs to a mean peak airway pressure of 29 mmHg. After injury, air was seen in implanted aortic-femoral vein shunts. Cardiac arrhythmia and ventricular fibrillation rapidly ensued. Hyperinflation of the lungs increased shunt air flow in some cases, suggesting increased SAE from barotrauma or *via* bronchiole-pulmonary arterial fistulae and transpulmonary passages (see Pathophysiology section). The occurrence of SAE with hyperinflation of the lungs was also found in a similar study by Meier *et al.*⁴²

Ponn *et al.*⁴³ lacerated the lungs of 18 dogs ventilated with air containing charcoal powder. The dogs progressed through stages of open chest, tension pneumothorax, and hypovolemia. Charcoal was found in blood, internal organ, and skeletal muscle specimens taken at the end of each stage in all dogs. An ultrasonic flowmeter positioned over the right carotid artery in six dogs detected air through all three stages. Circulatory collapse did not occur, but there were episodes of ventricular arrhythmias, all resolving spontaneously. The authors contend that lacerations produce less air fistulization and thus less SAE than stab wounds. This contention was supported by similar experiments performed by Waldo *et al.*⁶ and by Meier *et al.*⁴²

Chiu *et al.*⁴⁴ proposed that the lacerations produced by Ponn *et al.*⁴³ and by Waldo *et al.*⁶ might have decompressed themselves and that the induced tension pneumothorax could have decreased the possibility of large air emboli by compressing lacerated veins and perhaps bronchioles. By comparison experimental stab wounds produced a relatively closed channel between the bronchial tree and the pulmonary venous system. This type of injury would mitigate against escape of air into the pleural space or the atmosphere and thus approximate more closely a bronchopulmonary venous fistula that would not be easily subject to spontaneous decompression.

A study to link SAE with blast lung injury was conducted by Mason *et al.*,⁴⁵ who subjected a dog to a near-lethal air blast. An implanted ultrasonic cuff around

SYSTEMIC AIR EMBOLISM AFTER LUNG TRAUMA

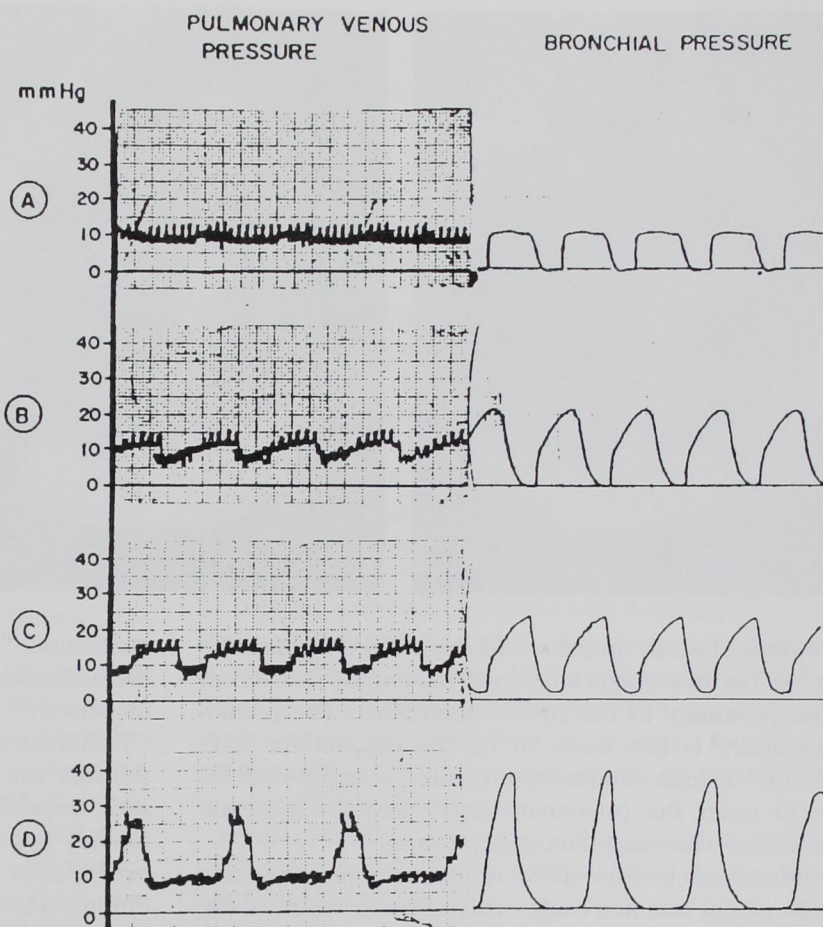


Fig. 2. The changes in pulmonary venous pressure in response to increments of bronchial pressure during positive-pressure ventilation. The gradient between the two pressures increases as bronchial pressure increases. From Chiu.⁴¹

a carotid artery detected large numbers of emboli, presumably air, for 30 min after injury.

In summary, animal studies strongly support the concept of bronchopulmonary venous fistulization in penetrating and blast lung trauma. In the presence of a favorable pressure gradient created by PPV, the likelihood of air embolization to the coronary and cerebral vessels appears greater than that currently recognized in humans.

Pathophysiology

Tissue disruption from chest trauma may result in direct communication between the pulmonary vasculature and the bronchial tree. One mechanism of SAE after penetrating trauma is illustrated in figure 1. In chest contusion, scattered loss of integrity of the pulmonary blood vessels, air passages, and alveoli also creates communicative pathways.

In a spontaneously breathing patient, pressure in the airway is lower than that of the pulmonary veins. This pressure gradient may reverse if pulmonary venous pressure is low or if airway pressure is high. Figure 2 demonstrates airway and pulmonary venous pressures in ventilated dogs.⁴¹ As airway pressure increases, pulmonary venous pressure also increases but to a lesser extent. Further increases in airway pressure eventually reverse the gradient so that air or gas enters the pulmonary circulation.

Figure 3 shows the TEE of a patient after blunt thoracic trauma.⁷ The left panel is taken while the patient is apneic, and the right panel is taken during a ventilator-generated inspiratory cycle. Gas is abundant in the left cardiac chambers during the positive pressure phase. Figure 4 was recorded during ventilator-generated inspiration.⁷ The amount of air in the left cardiac chambers was noticeably less when the inspiratory volume was decreased from 650 ml (right panel) to 350 ml (left panel).

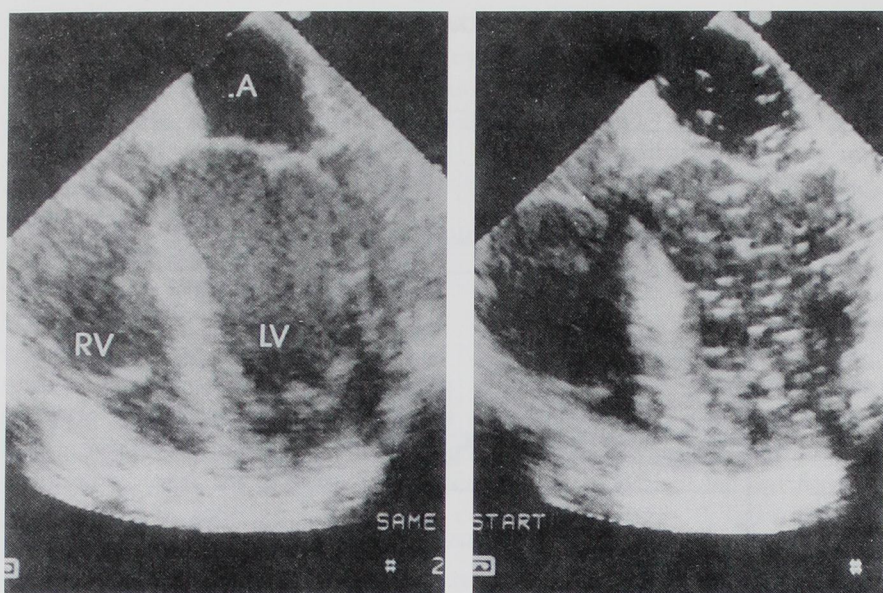


Fig. 3. Transesophageal echocardiography of a trauma patient. The left panel (four chamber view) was taken during apnea, and the right panel during a machine-driven inspiratory cycle. Air bubbles are seen in the left atrium and ventricle only during positive-pressure ventilation. RV = right ventricle; LV = left ventricle. From Saada.⁷

Injuries close to the hilar region yield the highest risk for SAE because that is where the pulmonary vein lies in close proximity to the airway branches.¹¹ The pulmonary artery is less likely to be the conduit for SAE, although at high ventilatory pressures, it is possible for gas to enter the pulmonary artery and the systemic circulation *via* transpulmonary passages.⁴⁶

Another mechanism of SAE requires the presence of a probe patent foramen ovale, which occurs in 30% of the normal population.⁴⁷ Right-to-left shunting may occur through the patent foramen in a hemodynamically significant venous air embolism (VAE) and with mechanical

ventilation,⁴⁸ positive end-expiratory pressure (PEEP),⁴⁹ discontinuation of PEEP,⁴⁹ coughing,^{50,51} and Valsalva's maneuver.⁵⁰

Paradoxical air embolism also occurs when air is pushed through the pulmonary circulation, as in those with adult respiratory distress syndrome (ARDS),⁵² severe pulmonary hypertension,⁵³ or even with mean pulmonary pressures of 34 mmHg⁵⁴ and 42 mmHg.⁴⁶ In these conditions, SAE occurs when air volume overwhelms the filtration capacity of the pulmonary vasculature.^{46,54-56} Butler and Hills⁵⁵ determined the pulmonary filtration threshold in healthy sheep to be 0.30–0.35 ml · kg⁻¹ ·

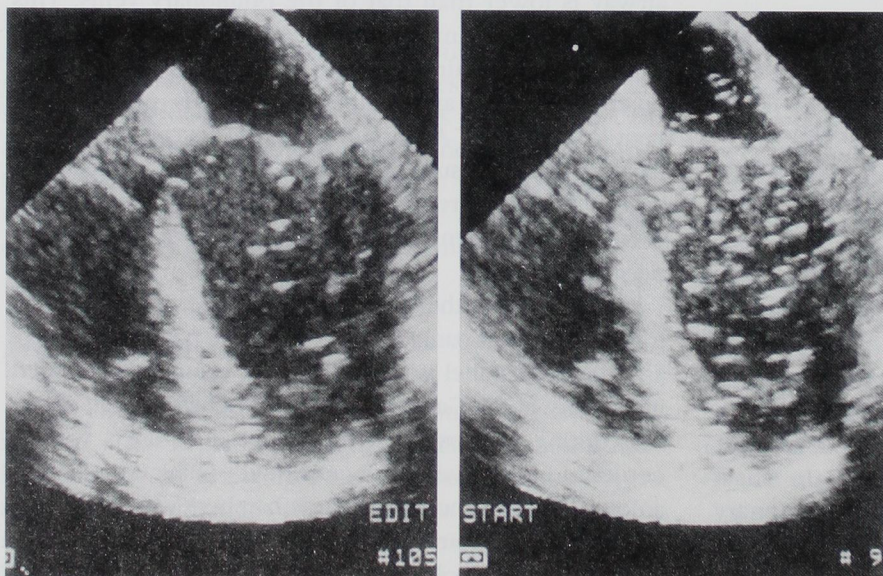


Fig. 4. The effect of 350 ml (left panel) and 650 ml (right panel) tidal volumes on the amount of gas bubbles in the left cardiac chambers of a ventilated trauma patient is shown in these transesophageal echocardiograms. A higher tidal volume, hence higher ventilatory pressures, led to more gas embolization. From Saada.⁷

SYSTEMIC AIR EMBOLISM AFTER LUNG TRAUMA

min⁻¹ of air. Barotrauma and volutrauma can rupture respiratory ducts and alveoli that are distended beyond their tensile strength. The escaping air may be entrained into the pulmonary veins, resulting in SAE, as seen in laboratory animals,⁵⁷ neonates,^{58,59} and adults.⁵²

In those with blast injuries, the shock wave front passing through the interfaces between air, alveolar tissue, and blood vessels causes the blood vessels to burst and the alveolar septa to tear (spalling effect).³²⁻³⁸ The alveoli reexpand rapidly after the shock wave front passes (implosion).³²⁻³⁸ With acceleration, lung parenchyma may shear away from the vascular tree.⁶⁰ The torn alveoli, bronchiole branches, and pulmonary vessels communicate, resulting in hemoptysis and possibly SAE. The Spalling effect and implosion affect all gas-containing organs (*i.e.*, ears, lungs, gut, and cranial sinuses), especially the ears.³²

Discussion of Previously Published Cases

We conducted computerized MEDLINE searches from 1966 to April 1998 using the Subject Headings *air embolism* and *blast injury*. Pertinent articles were obtained, and additional relevant citations were identified and retrieved. One hundred seven cases of SAE after lung trauma^{5,7-18,30,61} and six definitive cases of SAE after primary pulmonary blast injuries^{35,37} were identified. Clinical details are lacking in individual cases from case series. Many cases of SAE presented immediately after tracheal intubation and ventilation.^{5,7-9,11-17,30,61} Ventilatory pressures were reported as high in some cases,^{7,10} normal (maximum airway pressure of <25-30 cmH₂O) in one,¹⁶ and not reported in most others. Lung isolation techniques were not mentioned and presumably not used in any of the cases. Saada *et al.*⁷ was the only group that used definitive ventilatory intervention. They demonstrated the effectiveness of HFV and the influence of a lower ventilatory volume on the amount of gas embolism produced.

The majority of the reported cases involved penetrating lung trauma,^{5,8-17,30} in which SAE was diagnosed during thoracotomy or autopsy. SAE was diagnosed in a few cases of blunt trauma with the use of TEE.⁷

Evidence of CNS embolism was presented in a minority of cases based on computed tomography (CT),⁶¹ neurologic status,³⁶ ophthalmoscopy,³⁶ autopsy finding of cerebral arterial air,³⁶ or favorable response to HBOT.¹⁷ In others with neurologic impairment, the presence of hypotension, possible drug effects, and conceivable head trauma makes implicating SAE difficult.

Overall these cases illustrate the often fatal nature of SAE and the lack of awareness of the potential effectiveness of airway and ventilatory maneuvers to minimize morbidity and mortality.

Clinical Presentation and Diagnosis of Systemic Air Embolism

In those with lung trauma, the combination of hemoptysis, circulatory and CNS dysfunction immediately after initiation of PPV, and air in retinal vessels is sufficient to make the provisional diagnosis of SAE, without confirmation by ancillary diagnostic tools or the direct visualization of coronary or cardiac air during thoracotomy. Hemoptysis suggests communication between pulmonary blood vessels and the airway. It may be present in 22% of SAE cases.³¹ In Yee *et al.*'s series,⁸ 10 (16%) of the 61 patients with SAE presented with hemoptysis, and 12 patients (20%) developed circulatory arrest immediately after tracheal intubation and administration of PPV. Shock immediately after initiation of PPV also can occur in the presence of hypovolemia, cardiac tamponade, tension pneumothorax, cardiac injury, or exacerbation of underlying heart disease.

Focal neurologic changes, seizures, and other CNS dysfunction in the absence of head injury or other obvious causes also suggest SAE, especially if onset is immediately after tracheal intubation and the initiation of PPV.

Fundoscopy examination of the retinal vessels may reveal air presenting as streaming bubbles or pale silvery sections, confirming SAE.^{19,36} During thoracotomy, air may be seen or aspirated from the coronary arteries, left atrium and ventricle, and the aorta. Aspiration of air from arteries (*e.g.*, radial, femoral) may likewise provide confirmation.¹⁹ Similar to VAE, an abrupt decrease in the end-tidal CO₂ may occur with SAE as a result of decreased cardiac output. Other clinical signs may include marbling of the skin⁵² or mottling of the tongue (Liebermeister's sign).⁶²

Chest radiograph and CT may show pneumohemothorax, pneumatocele, interstitial emphysema, infiltrates, or contusion. CT of the thorax showing intravascular and intracardiac air has never been reported in a live patient. Postmortem and preautopsy CT documentation of air in all cardiac chambers in a patient who suffered barotrauma and SAE on rapid ascent from submersion, presumably against a closed glottis, has been reported.⁶³ CT of the head may reveal intracerebral air as negative density streaks reproducing a cast of the cerebral arter-

ies;⁶¹ as well-defined areas of low density, often multiple and bilateral;⁶⁴ or as cerebral edema or infarction.⁶⁵ Magnetic resonance imaging and single photon emission tomography techniques may be useful to document cerebral gas collection.⁶⁶

Transesophageal echocardiography can detect gas bubbles as small as 2 μ m in diameter.⁶⁷ In one study, 0.02 ml of air infused over 1 min into the right atrium was detectable.⁶⁸ TEE may be used to diagnose SAE, VAE, cardiac abnormality, and great vessel injury and to assess volume status and contractility.^{69,70} Its use in trauma is limited by considerations for traumatic upper airway and esophageal injuries.⁷⁰ With increased availability of TEE for trauma management, the diagnosis of SAE resulting from pulmonary contusion may increase. Transthoracic echocardiography is noninvasive but has poorer image resolution than TEE.⁷¹

Precordial Doppler can detect VAE produced by the infusion of 0.12 ml of air over 40 s into the right atrium.⁶⁸ It and the esophageal Doppler may be useful in detecting air in the left ventricle. Transcranial Doppler monitoring, preferably of the right middle cerebral artery, may detect cerebral gas embolization.⁷²

In those with blast injuries, it is important to note that the tympanic membrane has a lower threshold for injury than the lungs and gut.^{32,38} If both tympanic membranes are intact, it is unlikely that the lungs or gut are injured.³³ In those with air blast injuries, the lungs are more susceptible than the gut to injury, whereas in those with underwater blast injuries, the gut is more susceptible.^{33,37} Primary blast internal injuries are not necessarily accompanied by external signs of trauma.

Management

Spontaneous ventilation is preferred in any patient at risk for SAE. When PPV is necessary, the injured lung should be isolated. Lung isolation devices should therefore be readily available in the ER. In those with bilateral lung injuries, emboli may originate from either lung, so lung isolation is not a first option. Ventilatory pressures and volumes should then be kept as low as possible to maintain adequate oxygenation and a tolerable level of hypercapnia (with considerations for its effect on intracranial pressure). Transportation of the patient to a facility where HFV and echocardiographic monitors are available may be required.

When SAE is evolving, isolating the injured lung should theoretically stop the flow of gas into the circulation,

thus increasing the likelihood that resuscitation would be successful. Although emergency thoracotomy may be unavoidable, the patient may be stabilized first by isolating the lung before transporting to the OR. In addition, lung isolation may be required to prevent spillage of blood into the uninjured lung. Fiberoptic bronchoscopy is recommended to assess airway trauma and to confirm placement after lung isolation.⁷³

Selective lung ventilation may be achieved using double-lumen tubes (DLTs), Univent tubes,⁷⁴ and bronchial blockers. Placement of these may be difficult in certain trauma situations. For the patient already intubated with a standard endotracheal tube, the lung may be isolated by passing a Fogarty balloon-tipped catheter beside the tracheal tube with the aid of a fiberoptic bronchoscope.⁷⁵

The choice of lung isolation technique should be individualized. A DLT is unsuitable for nasotracheal intubation and offers significant airflow resistance, and its relatively small endobronchial tube makes bronchial toilet and bronchoscopy less efficient. However, it allows the lungs to be ventilated separately and differentially and is familiar to most anesthesiologists. The Univent tube is suitable for nasotracheal intubation in large patients,⁷⁶ permits HFV through the bronchial blocker lumen,⁷⁷ allows efficient pulmonary toilet and bronchoscopy,⁷⁸ may be less traumatic to the airway mucosa, and using a J wire as a guide, can selectively block certain pulmonary lobes.⁷⁹

For practitioners not comfortable with the above equipment or when none is available, intubating with an uncut tracheal tube will allow right-sided endobronchial intubation 99% of the time by simply sliding the tube distally.⁸⁰ Occlusion of the right upper lobe bronchus is a concern. Intubation of the left main stem bronchus is 92% successful if the patient's head is turned to the right and if the endotracheal tube is rotated 180° and passed distally.⁸⁰ This maneuver is contraindicated when cervical spine injury is suspected. Alternatively, passing a fiberoptic bronchoscope through the tracheal tube into the appropriate main bronchus will aid endobronchial intubation with a standard uncut tracheal tube. If the *in situ* endotracheal tube is already cut, it may be lengthened by connecting it to another tracheal tube of 1.5–2 mm ID smaller or larger.

With successful lung isolation, the lung containing the bronchopulmonary venous fistula can rest. If oxygenation becomes a problem, the lowest possible airway pressure, in the form of continuous positive airway pressure, intermittent PPV, oxygen insufflation, or HFV, can

SYSTEMIC AIR EMBOLISM AFTER LUNG TRAUMA

be used on the injured lung. Rankin *et al.*⁸¹ maintained satisfactory gas exchange for 5 days in a patient with massive unilateral traumatic air leak through a bronchopleural fistula by using high-frequency intermittent PPV to the injured lung and PPV to the uninjured lung *via* a DLT.

Immediate thoracotomy to allow hilar clamping or isolation of the injured lung segment may be needed if selective lung isolation and resuscitation are unsuccessful. Whether selective lung isolation and differential ventilation obviate the need for thoracotomy to remove the bronchopulmonary venous fistula is unknown. Factors favoring conservative management over surgical repair include an improving clinical picture, resolution of hemoptysis, stable or improving radiographic picture, cessation of SAE (clinically and echocardiographically), and lack of proximal lesion on bronchoscopy.

Nonemergent surgery should be delayed. Regional and local anesthesia are preferred for those undergoing immediate surgery.³³ General anesthesia should be delayed to allow time for the bronchopulmonary venous lesions to close. The time it takes for the lesions to close is unknown and probably depends on many clinical factors. Normalization of hemostatic parameters should promote such closure.

Nitrous oxide should be avoided because it increases the size of bubbles *in vivo*. Further, N₂O may induce regional myocardial ischemic changes through epicardial arterial constriction.⁸² It has been observed that swine with artificially induced coronary air embolism died when ventilatory gases contained N₂O but survived when ventilated with oxygen alone.⁸² Because the solubility of oxygen in blood is almost twice that of nitrogen, oxygen emboli dissolve faster than air emboli. Ventilating with high oxygen concentrations is therefore recommended.

An alternative to lung isolation is HFV. Its effectiveness in management of SAE and bronchopleural fistula is documented.^{7,81} Saada *et al.*⁷ also demonstrated that decreasing the ventilatory volume (and pressure) may reduce or eliminate the amount of air embolized. This is a simple first step whenever SAE is suspected and when preparations are underway for selective endobronchial intubation.

Hyperbaric oxygen therapy has been successful in treating those with CNS air embolism.^{17,83-85} A popular protocol is outlined in the US Navy Compression Table 6A.⁸³ At a pressure of 2.8 atmospheres (as used in the US Navy protocol⁸³), bubble volume is reduced by two thirds.⁸⁶ Benefits of HBOT include improved tissue oxy-

genation, decreased intracranial pressure,^{86,87} decreased reperfusion injury,⁸⁶ and reduced mortality among severely brain-injured patients.⁸⁷ Patients with SAE should be treated in the HBOT chamber as soon as initial resuscitation, diagnostics, and surgery are completed. Generally prognosis is good if HBOT is started within 6 h of the insult.⁶² Delays caused by other priorities should not discourage the use of HBOT. Dexter *et al.*⁸⁸ calculated that large (>4-mm radius) cerebral oxygen and nitrogen emboli may take more than 20 and 63 h, respectively, to absorb. Sadan *et al.*⁸³ successfully treated air embolism in a parturient possibly up to 40 h after initial onset of neurologic deficit and 16 h after further acute neurologic deterioration. Cases of improvement after 14-60 h have also been reported.^{17,83,89} Clinicians involved in trauma care should acquaint themselves with the nearest hyperbaric facilities. If transportation is by air, the lowest and safest altitude should be used because bubble size increases with altitude. For example, the atmospheric pressures at 1,000 feet and 1,500 feet above sea level are 674 and 634 torr, respectively. Based on Boyle's Law, bubble size increases in volume by 11% and 20%, respectively. Weiler-Ravell *et al.*³⁶ reported five blast injury victims with SAE being transported in a helicopter to a hospital. Two of the patients died and one's condition deteriorated rapidly *en route* and eventually also died despite HBOT. Although the authors did not state whether expansion of air emboli could have contributed to the poor outcome, we speculate that flying at altitude increased the volume of bubbles.

Cardiopulmonary bypass has been used to successfully treat a case of catastrophic VAE and paradoxical air embolism during laparoscopy and hysteroscopy.⁹⁰

Other adjunctive maneuvers in the management of SAE include Trendelenburg positioning and having the injured lung dependent to the left atrium to increase pulmonary venous pressure. Low intravascular volume and hypotension should be avoided. Fluid balance may be a challenge when the presence of ARDS contradicts aggressive fluid administration. Moreover, a recent US study done in urban settings suggests that immediate (*vs.* delayed) aggressive fluid resuscitation may decrease survival associated with penetrating torso trauma.⁹¹ Fluid therapy should thus be individualized. Hyperglycemia should be avoided because of its potential harmful effects on the ischemic brain.⁹² Hypocapnia, often used to reduce intracranial pressure, may exacerbate cerebral and coronary ischemia. The contribution of hypothermia to bubble size, based on Charles' Law, is negligible.

Currently pneumatic antishock garment (PASG) has a

controversial and decreasing role in trauma. In the presence of closed head injury, pregnancy, and pulmonary edema, PASG may actually be harmful.⁹³ Additionally PASG may adversely affect the outcome for patients with cardiac and thoracic vascular injuries.⁹⁴⁻⁹⁷ As indicated previously, immediate aggressive fluid augmentation of the central circulation in patients with penetrating torso trauma in urban settings may decrease survival.⁹¹ Although use of PASG may increase pulmonary vascular pressures⁹⁸ and may discourage the formation of SAE, its use in chest trauma cannot be recommended as a standard practice at this time.

Other Causes of Air Embolism in Nontraumatic and Traumatic Clinical Settings

Several cases of SAE have been reported with needle biopsy of a lung lesion during general anesthesia with PPV.^{99,100} Clinically, frothy blood is seen in the breathing circuit with sudden severe cardiac dysfunction under PPV. The finding of air in the left ventricle and coronary arteries and on CT of the head suggests that SAE occurred by the same mechanism as traumatic lung injury. Biopsy through air-space disease may be the most dangerous scenario for development of SAE. The incidence of this complication for 16- to 20-gauge needles is 0.5-0.8/1,000.^{99,100} Percutaneous procedures with 20-gauge or smaller needles are considered safe, although cerebral air embolism after transthoracic aspiration with a 23-gauge needle has been reported.^{99,100}

Other situations associated with SAE include rapid decompression after deep-sea diving,⁶² cardiac surgery,⁷² cardiopulmonary bypass,⁷² arterial¹⁰¹ and venous² instrumentation, laparoscopy,¹⁰² and chest operations.¹⁰³

Chest trauma may also lead to VAE.^{104,105} In one case, a subclavian vein was lacerated by a stab; deep inspirations by the fleeing victim resulted in fatal VAE.¹⁰⁴ In another case, a subclavian vein injury resulted in fatal VAE on removal of a fence post impaled in the victim's chest.¹⁰⁵ VAE has also resulted from an open dural sinus after a traumatic skull fracture¹⁰⁶ and from surgeries above the level of the heart. Generally small VAE are well tolerated unless a shunt is present. In addition, external chest compression can cause lung trauma and set the stage for SAE.^{83,107}

Summary

Systemic air or gas embolism has been increasingly recognized as a complication of serious chest trauma and often presents with catastrophic circulatory and cerebral events. The classic findings are hemoptysis, sudden cardiac or cerebral dysfunction after initiation of PPV, air in retinal vessels, and air in arterial aspirations. The clinician must be wary of more subtle presentations. Several diagnostic tools (TEE, Doppler, CT) can detect intracardiac and cerebral air, but they may not be necessary to confirm the diagnosis of SAE.

Cessation of SAE is essential for successful resuscitation. In those with unilateral lung injury, this can theoretically be achieved by isolating and ventilating the noninjured lung. Sole reliance on immediate thoracotomy for hilar clamping to stem the flow of gas emboli is a concept that needs to be challenged. Whether airway and ventilation interventions will eliminate, delay, or decrease the need for thoracotomy and improve the prognosis of SAE remains to be seen. There is little reported in the literature regarding such interventions. Airway management of a patient at risk for SAE should include a technique that can selectively ventilate each lung. Patients with bilateral sources of SAE may benefit from the avoidance of high airway pressures.

Regional anesthesia should be considered when appropriate. HBOT is useful in managing cerebral air embolism and should be incorporated as soon as possible.

Clinicians involved in trauma care must be familiar with SAE. By adopting a problem-based solution through innovative airway and ventilation management, anesthesiologists may significantly alter and improve the morbidity and mortality rate of SAE resulting from chest trauma.

The authors thank Dr. Les Greene, Dr. Irene Cybulsky, and Ms. Ginette Lalonde-Kontio of McMaster University for their constructive criticism of the manuscript, and the various authors and publishers who kindly granted permission to have figures 1-4 reproduced in this article. The authors also thank Ms. LeeAnne H. Contardi for her expert secretarial assistance.

References

1. Mattox KL: Thoracic trauma, Glenn's Thoracic & Cardiovascular Surgery, 5th Edition. Edited by Baue AE, Geha AS, Naunheim KS. Norwalk, CT, Appleton & Lange, 1991, p 94
2. Michel L, Poskanzer DC, McKusick KA, Nardi GL, Malt RA: Fatal paradoxical air embolism to the brain. Complication of central venous catheterization. JPEN 1982; 6:68-70

SYSTEMIC AIR EMBOLISM AFTER LUNG TRAUMA

3. Sinner WN: Complications of percutaneous transthoracic needle aspiration biopsy. *Acta Radiol Diagn* 1976; 17:813-28
4. Goldfarb B, Bahnson HT: Early and late effects on the heart of small amounts of air in the coronary circulation. *J Thorac Cardiovasc Surg* 1980; 80:708-17
5. Thomas AN: Air embolism following penetrating lung injuries. *J Thorac Cardiovasc Surg* 1973; 66:533-40
6. Waldo WJ, Harlaftis NN, Symbas PN: Systemic air embolism: Does it occur after experimental penetrating lung injury? *J Thorac Cardiovasc Surg* 1976; 71:96-101
7. Saada M, Goarin J-P, Riou B, Rouly JJ, Jacques Y, Guesde R, Viars P: Systemic gas embolism complicating pulmonary contusion—Diagnosis and management using transesophageal echocardiography. *Am J Respir Crit Care Med* 1995; 152:812-5
8. Yee ES, Verrier ED, Thomas AN: Management of air embolism in blunt and penetrating thoracic trauma. *J Thorac Cardiovasc Surg* 1983; 85:661-8
9. Thomas AN, Stephens BG: Air embolism: A cause of morbidity and death after penetrating chest trauma. *J Trauma* 1974; 14:633-8
10. Graham JM, Beall Jr AC, Mattox KL, Vaughan GD: Systemic air embolism following penetrating trauma to the lung. *Chest* 1977; 72:449-53
11. Estrera AS, Pass LJ, Melvin RP: Systemic arterial air embolism in penetrating lung injury. *Ann Thorac Surg* 1990; 50:257-61
12. Donato AT, Arciniegas E, Lam CR: Fatal air embolism during thoracotomy for gunshot injury to the lung. *J Thorac Cardiovasc Surg* 1975; 69:827-9
13. Smith JM, Richardson JD, Grover FL, Arom KV, Webb TE, Trinkle JK: Fatal air embolism following gunshot wound of the lung. *J Thorac Cardiovasc Surg* 1976; 72:296-8
14. Campbell PR, Kerridge R: Fatal traumatic air embolism following a stab wound to the chest. *Aust N Z J Surg* 1993; 63:307-9
15. Hewitt PM, Knottenbelt JD, Mortimore S: Combined systemic and pulmonary air embolism after penetrating chest injury. *Injury* 1994; 25:553-4
16. Tracey JA, Vartian V: Systemic air embolism following manual lung inflation. *Can Anaesth Soc J* 1982; 29:272-4
17. Halpern P, Greenstein A, Melamed Y, Taitelman U, Sznajder I, Zveibil F: Arterial air embolism after penetrating lung injury. *Crit Care Med* 1983; 11:392-3
18. Enneking TJMQ, van der Warden C, de Wall Malifijt J: Bone necrosis of the femoral head after thoracic trauma. *Injury* 1994; 25:122-3
19. Trunkey D: Initial treatment of patients with extensive trauma. *N Engl J Med* 1991; 324:1259-63
20. Moon RE, Piantadori CA: Initial treatment of patients with extensive trauma (letter). *N Engl J Med* 1991; 325:1444-5
21. Trunkey D: Initial treatment of patients with extensive trauma (letter). *N Engl J Med* 1991; 325:1445-6
22. Stene JK, Grande CM, Bernhard WN, Barton CR: Perioperative anesthetic management of the trauma patient: Thoracoabdominal and orthopaedic injuries. *Trauma Anesthesia*. Edited by Stene JK, Grande CM. Baltimore, Williams & Wilkins, 1991, p 219
23. Jackson J: Management of thoracoabdominal injuries. *Trauma Anesthesia and Intensive Care*. Edited by Capan LM, Miller SM, Turndorf H. Philadelphia, JB Lippincott, 1991, p 486
24. Hill AB, Fleischer DM, Brown RA: Chest trauma in a Canadian urban setting—Implications for trauma research in Canada. *J Trauma* 1991; 31:971-3
25. LoCicero J III, Mattox K: Epidemiology of chest trauma. *Surg Clin North Am* 1989; 69:15-9
26. Robison PD, Harman PK, Trinkle JK, Grover FL: Management of penetrating lung injuries in civilian practice. *J Thorac Cardiovasc Surg* 1988; 95:184-90
27. Baker CC, Thomas AN, Trunkey DD: The role of emergency room thoracotomy in trauma. *J Trauma* 1990; 20:848-55
28. Wiencek RG Jr, Wilson RF: Central lung injuries: A need for early vascular control. *J Trauma* 1988; 28:1418-24
29. Wilson RF, Soullier GW, Wiencek RG: Hemoptysis in trauma. *J Trauma* 1987; 27:1123-6
30. King MW, Aitchison JM, Nel J: Fatal air embolism following penetrating lung trauma: An autopsy study. *J Trauma* 1984; 24:753-5
31. Swanson J, Trunkey DD: Trauma to chest wall, pleura, and thoracic viscera. *General Thoracic Surgery*, 3rd Edition. Edited by Shields TW. Philadelphia, Lea & Febiger, 1989, p 463
32. Mellor SG: The pathogenesis of blast injury and its management. *Br J Hosp Med* 1988; 39:536-9
33. Hamit IU: Primary blast injuries. *Indust Med* 1973; 42:14-21
34. Freund U, Kopolovic J, Durst AL: Compressed air emboli of the aorta and renal artery in blast injury. *Injury* 1980; 12:37-8
35. Hirst M, Bazini J: Blast injury of the chest. *Clin Radiol* 1969; 20:362-70
36. Weiler-Ravell D, Adatto R, Borman JB: Blast injury of the chest: A review of the problem and its treatment. *Israel J Med Sci* 1975; 11:268-74
37. Huller T, Bazini Y: Blast injuries of the chest and abdomen. *Arch Surg* 1970; 100:24-30
38. Candole CA: Blast injury. *Can Med Assoc J* 1967; 96:207-14
39. Caseby NG, Porter MF: Blast injuries to the lungs: Clinical presentation, management and course. *Injury* 1976; 8:1-12
40. McCaughey W, Coppel DL, Dundee JW: Blast injuries to the lungs: A report of two cases. *Anaesthesia* 1973; 28:2-9
41. Chiu C-J, Golding MR, Linder JB, Fries CC: Pulmonary venous air embolism: A hemodynamic reappraisal. *Surgery* 1967; 61:816-9
42. Meier GH, Wood WJ, Symbas PN: Systemic air embolism from penetrating lung injury. *Ann Thorac Surg* 1979; 27:161-8
43. Ponn RB, Zatarain G, Gerzberg L, Hottinger CF, Haase W, Nelson TS: Systemic air embolism in experimental penetrating lung injuries. *J Thorac Cardiovasc Surg* 1976; 74:296-8
44. Chiu C-J, Blaisdell FW, Symbas PN, Stivers RR, Lam CR: Letter to the editor. *J Thorac Cardiovasc Surg* 1976; 72:815, 948
45. Mason Wvan H, Damon EG, Dickinson AR, Nevison TO: Arterial gas emboli after blast injury. *Proc Soc Exp Biol Med* 1971; 136:1253-5
46. Thackray NM, Murphy PM, McLean RF, Delacy JL: Venous air embolism accompanied by echocardiographic evidence of transpulmonary air passage. *Crit Care Med* 1996; 24:359-61
47. Hagen PT, Scholz DG, Edwards WD: Incidence and size of patent foramen ovale during the first 10 decades of life: An autopsy study of 965 normal hearts. *Mayo Clin Proc* 1984; 59:17-20
48. Jaffe RA, Pinto FJ, Schnittger I, Brock-Utne JG: Intraoperative ventilator-induced right-to-left intracardiac shunt. *ANESTHESIOLOGY* 1991; 75:153-5
49. Black S, Muzzi DA, Nishimura RA, Cucchiara RF: Preoperative and intraoperative echocardiography to detect right-to-left shunt in patients undergoing neurosurgical procedures in the sitting position. *ANESTHESIOLOGY* 1990; 72:436-8
50. Dubourg O, Bourdarias J-P, Farcot J-C, Gueret P, Terdjman M, Ferrier A, Rigaud M, Bardet J-C: Contrast echocardiographic visualiza-

tion of cough-induced right-to-left shunt through a patent foramen ovale. *J Am Coll Cardiol* 1984; 4:587-94

51. Ulyatt DB, Judson J, Trubuhovich RV, Galler LH: Cerebral arterial air embolism associated with coughing on a continuous positive airway pressure circuit. *Crit Care Med* 1991; 19:985-7

52. Marini JJ, Culver BH: Systemic gas embolism complicating mechanical ventilation in the adult respiratory distress syndrome. *Ann Intern Med* 1989; 110:699-703

53. Black M, Calvin J, Chan KL, Walley VM: Paradoxical air embolism in the absence of an intracardiac defect. *Chest* 1991; 99:754-5

54. Spencer NT, Oyama Y: Pulmonary capacity for dissipation of venous gas emboli. *Aerospace Med* 1971; 42:822-7

55. Butler BD, Hills BA: The lung as a filter for microbubbles. *J Appl Physiol* 1979; 47:537-43

56. Bedell EA, Berge KH, Losasso TJ: Paradoxical air embolism during venous air embolism: Transesophageal echocardiographic evidence of transpulmonary air passage. *ANESTHESIOLOGY* 1994; 80:947-50

57. Kao DK, Tierney DF: Air embolism with positive-pressure ventilation of rats. *Am J Physiol* 1977; 42:368-71

58. Gregory GA, Tooley WH: Gas embolization in hyaline membrane disease. *N Engl J Med* 1970; 282:1141-2

59. Brown ZA, Clark JM, Jung AL: Systemic gas embolus. *Am J Dis Child* 1977; 131:984-5

60. Gans L, Kennedy T: Management of unique clinical entities in disaster medicine. *Emerg Med Clin North Am* 1996; 14:301-24

61. Muras I, Bonsignore R, Cesaro L, Frascadore L, Bernini FP: Post-traumatic cerebral air embolism—Case report. *J Neuroradiol* 1994; 21:267-9

62. Kindwall EP: Uses of hyperbaric oxygen therapy in the 1990s. *Cleve Clin J Med* 1992; 59:517-28

63. Williamson JA, King GK, Callanan VI, Lanskey RM, Rich KW: Fatal arterial gas embolism: Detection by chest radiography and imaging before autopsy. *Med J Aust* 1990; 153:97-100

64. Hirabuki BN, Miura T, Mitomo M, Kozuka T, Kitatani T, Terashima T, Okagawa K: Changes of cerebral air embolism shown by computerized tomography. *Br J Radiol* 1988; 61:252-5

65. Jensen ME, Lippe MH: CT in iatrogenic cerebral air embolism. *Am J Neuroradiol* 1986; 7:823-7

66. Moon RE, Vann RD, Bennett PD: The physiology of decompression illness. *Sci Am* 1995; 273:70-7

67. Practice guidelines for perioperative transesophageal echocardiography: A report by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. *ANESTHESIOLOGY* 1996; 84:986-1006

68. Glenski JA, Cucchira RF, Michelfelder JD: Transesophageal echocardiography and transcutaneous O₂ and CO₂ monitoring for detection of venous air embolism. *ANESTHESIOLOGY* 1986; 64:541-5

69. Vignon P, Lagrange P, Boncoeur M-P, Francois B, Gastinne H, Lang RM: Routine transesophageal echocardiography for the diagnosis of aortic disruption in trauma patients without enlarged mediastinum. *J Trauma* 1996; 40:422-7

70. Oxorn D, Edelist G, Smith MS: An introduction to transesophageal echocardiography: II. Clinical applications. *Can J Anaesth* 1996; 43:278-94

71. Johnson SB, Kearney PA, Smith NW: Echocardiography in the evaluation of thoracic trauma. *Surg Clin North Am* 1995; 75:193-205

72. Van der Linden J, Casimir-Ahn H: When do cerebral emboli appear during open heart operations? A transcranial Doppler study. *Ann Thorac Surg* 1991; 51:237-41

73. Klein U, Karzai W, Bloos F, Wohlfarth M, Gottschall R, Fritz H, Gugel M, Seifert A: Role of fiberoptic bronchoscopy in conjunction with the use of double-lumen tubes for thoracic anesthesia: A prospective study. *ANESTHESIOLOGY* 1998; 88:346-50

74. Inoue H, Shohtsu A, Ogawa J, Koide S, Kawada S: Endotracheal tube with movable blocker to prevent aspiration of intratracheal bleeding. *Ann Thorac Surg* 1984; 37:497-9

75. Harvey SC, Alpert CC, Fishman RL: Independent placement of a bronchial blocker for single-lung ventilation: An alternative method for the difficult airway. *Anesth Analg* 1996; 83:1330-1

76. Gozal Y, Lee W: Nasointubation and one-lung ventilation (letter). *ANESTHESIOLOGY* 1996; 84:477

77. Ransom E, Detterbeck F, Klein JJ, Norfleet EA: Univent tube provides a new technique for jet ventilation. *ANESTHESIOLOGY* 1996; 84:724-6

78. Gayes JM: The Univent tube is the best technique for providing one-lung ventilation. Pro: One-lung ventilation is best accomplished with the Univent endotracheal tube. *J Cardiothorac Vasc Anesth* 1993; 7:103-7

79. Cohen DJ: A unique use of the Univent tube (letter). *ANESTHESIOLOGY* 1995; 83:229

80. Kubota H, Kubota Y, Toyoda Y, Ishida H, Asada A, Matsuura H: Selective blind endobronchial intubation in children and adults. *ANESTHESIOLOGY* 1987; 67:587-9

81. Rankin N, Day AC, Crone PD: Traumatic massive air leak treated with prolonged double lumen intubation and high frequency ventilation: Case report. *J Trauma* 1994; 36:428-9

82. Tuman KJ, McCarthy RJ, Spiess BD, Overfield DM, Ivankovich AD: Effects of nitrous oxide on coronary perfusion after coronary air embolism. *ANESTHESIOLOGY* 1987; 67:952-9

83. Sadan O, Adler M, Ezra S, Zakut H: Air embolism due to pulmonary barotrauma in a patient undergoing cesarean section. *Acta Obstet Gynecol Scand* 1991; 70:551-3

84. Catron PW, Dutka AJ, Biondi DM, Flynn ET, Hallenbeck JM: Cerebral air embolism treated by pressure and hyperbaric oxygen. *Neurology* 1991; 41:314-5

85. Sukoff MH, Hollin SA, Jacobsen JH II: The protective effect of hyperbaric oxygenation in experimentally produced cerebral edema and compression. *Surgery* 1967; 62:40-8

86. Tibbles PM, Edelsberg JS: Hyperbaric oxygen therapy. *N Engl J Med* 1996; 334:1642-8

87. Rockswold GL, Ford SE, Anderson DC, Bergman TA, Sherman RE: Results of a prospective randomized trial of severely brain-injured patients with hyperbaric oxygen. *J Neurosurg* 1992; 76:929-34

88. Dexter F, Hindman BJ: Recommendations for hyperbaric oxygen therapy of cerebral air embolism based on a mathematical model of bubble absorption. *Anesth Analg* 1997; 84:1203-7

89. Bitterman H, Melamed Y: Delayed hyperbaric treatment of cerebral air embolism. *Israel J Med Sci* 1993; 29:22-6

90. Diakun TA: Carbon dioxide embolism: Successful resuscitation with cardiopulmonary bypass. *ANESTHESIOLOGY* 1991; 74:1151-3

91. Bickell WH, Wall MJ Jr, Pepe PE, Martin RR, Ginger VF, Alien MK, Mattox KL: Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso trauma. *N Engl J Med* 1994; 331:1106-53

92. Schleren CL, Berbowitz ID, Traystman R, Rogers MC: Controversial issues in cardiopulmonary resuscitation. *ANESTHESIOLOGY* 1989; 71:133-49

SYSTEMIC AIR EMBOLISM AFTER LUNG TRAUMA

93. Advanced Trauma Life Support Instructor Manual. Chicago, American College of Surgeons Committee on Trauma, 1993, pp 89-90
94. Chang AK, Dunford J, Hoyt DB, Rosen P: MAST 96. *J Emerg Med* 1996; 14:419-24
95. Honigman B, Lowenstein SR, Moore EE, Roweder K, Pons P: The role of the pneumatic antishock garment in penetrating cardiac wounds. *JAMA* 1991; 266:2398-401
96. Mattox KL, Bickell W, Pepe PE, Burch J, Feliciano D: Prospective MAST study in 911 patients. *J Trauma* 1989; 29:1104-12
97. Bickell WH, Pepe PE, Wyatt CH, Dedo WR, Applebaum DJ, Black CT, Mattox KL: Effect of antishock trousers on the trauma score: A prospective analysis in the urban setting. *Ann Emerg Med* 1985; 14: 218-22
98. Davies JW, McKone TK, Cram AE: Hemodynamic effects of MAST in experimental cardiac tamponade. *Ann Emerg Med* 1981; 10:185-6
99. Wong RS, Ketel L, Temes T, Follis FM, Ashby R: Air embolus complicating transthoracic percutaneous needle biopsy. *Ann Thorac Surg* 1995; 59:1010-1
100. Worth ER, Burton RJ, Landreneau RJ, Eggers GWN Jr, Curtis JJ: Left atrial air embolism during intraoperative needle biopsy of a deep pulmonary lesion. *ANESTHESIOLOGY* 1990; 73:342-5
101. Rand MV, Richard ARF: Cerebral air embolism occurring at angiography and diagnosed by computerized tomography. *J Neurosurg* 1984; 60:177-8
102. Schindler E, Muller M, Kelm C: Cerebral carbon dioxide embolism during laparoscopic cholecystectomy. *Anesth Analg* 1995; 81: 643-5
103. Khalil SW, Madan U, Rigor BM, Fields WS, Unger KM: Systemic air embolism following induction of artificial pneumothorax under anaesthesia with successful management. *Br J Anaesth* 1979; 51:561-5
104. Ellis GR, Brown JR: Massive air embolism due to gunshot wound. *JAMA* 1964; 189:177-9
105. Kron IL, Unger S, Crosby IK: Fence post impalement injury to the chest with air embolus: Case report. *Va Med Q* 1983; 110: 666-8
106. Iwama T, Andoh H, Murase S, Miwa Y, Ohkama A: Diffuse cerebral air embolism following trauma: Striking mostmortem CT findings. *Neuroradiology* 1994; 36:33-4
107. Shiina G, Shimosegawa Y, Kameyama M, Onuma T: Massive cerebral air embolism following cardiopulmonary resuscitation: Report of two cases. *Acta Neurochir (Wien)* 1993; 125:181-3