Is Emergency Thoracotomy Always the Most Appropriate Immediate Intervention for Systemic Air Embolism After Lung Trauma?*

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Abbreviations: PPV = positive pressure ventilation; SAE = systemic air (or gas) embolism

Lung trauma involving laceration of air passages, lung parenchyma, and blood vessels may result in direct communication among these injured entities. Systemic air (or gas) embolism (SAE) occurs when gas enters pulmonary veins—the result of low pulmonary venous pressure (as in hypovolemia) or increased airway pressure (as in positive pressure ventilation [PPV], coughing, or tension pneumothorax), or both. Pulmonary venous gas embolizes to the heart and to the coronary and cerebral arteries with catastrophic consequences. Although iatrogenic SAE from therapeutic pneumothorax, thoracentesis, lung resection, and biopsy1–7 has been shown to occur for many decades, it was not until 1973 that a description of this clinical condition as a sequela of lung trauma was first published.8

The incidence of SAE after severe lung trauma has been estimated at 4 to 14%.9,10 Of these, two thirds resulted from penetrating lung injury and one third from chest contusion.10 SAE is often diagnosed when sudden circulatory collapse of a traumatized patient occurs immediately after tracheal intubation and the initiation of PPV. The collapse is typically unresponsive to conventional resuscitation. 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 the aortic root.1,9,11–20 Less frequently, the development of neurologic deficit or seizures in previously conscious patients without obvious head injury may implicate cerebral air embolism.11,16

The current recommended treatment of SAE associated with unilateral lung injury is immediate thoracotomy, in the emergency department if necessary, to clamp the hilum of the injured lung to arrest the continuous passage of air into the coronary, cerebral, and other systemic arteries.9,10 Other measures include cross-clamping the aorta; cardiac massage; aspirating air from the left ventricle, aortic root, and pulmonary veins; volume infusion; cardiotonic drug administration; and optimization of metabolic and respiratory status.1,9,10 Hyperbaric oxygen therapy has been used in some cases to treat cerebral air embolism with good results.19 Despite such heroic measures, the morbidity and mortality associated with SAE are high. Most reported cases of SAE associated with isolated penetrating lung injury have a fatal outcome.1,8,9,11–20 In one series of 61 patients with documented SAE, 15 patients had blunt trauma and 46 patients had penetrating trauma.9 The blunt group had a mortality rate of 80%, and the penetrating group rate was 48%.9 In another series of cases involving 168 patients who required emergency department thoracotomy for circulatory collapse, SAE was diagnosed in 11 patients, hypovolemia in 115 patients, and tamponade in 42 patients. The number of deaths were 10 (91%), 109 (86%), and 26 (62%), respectively.21

The injection of 2 or 3 mL of air into the cerebral circulation can be fatal.22 Likewise, injecting 0.5 to 1.0 mL of air into a pulmonary vein can cause cardiac arrest from coronary air embolus.23 Air embolism exerts its adverse effects by obstructing blood flow and by causing an acute intravascular inflammatory response.24

In a patient who has sustained pulmonary trauma, blood in the tracheal tube (hemoptysis) should raise the suspicion of communication between pulmonary blood vessels and airway.9,10,16 Normally, in a spontaneously breathing person, pulmonary venous pressures exceed airway pressures and blood tends to flow from the pulmonary vasculature into the bronchial tree. Increased airway pressure may reverse
that gradient. Thus, circulatory collapse, neurologic
deterioration immediately after initiation of PPV,
(or both), coughing (caused by irritation of airway by
blood or equipment), and tension pneumothorax are
the classical presentations of SAE.9,10 Yee et al9
found that 36% of patients with SAE present with
either hemoptysis or cardiac arrest after intubation
and initiation of PPV. Fundoscopic examination may
reveal air in the retinal vessels, and arterial aspirates
may contain bubbles.10 Echographic equipment de-
tects intracardiac air with high sensitivity.11 Ideally,
transesophageal echocardiography should be used in
all suspected and confirmed cases of SAE. CT scans
may reveal cerebral25 and thoracic vascular air. Bron-
choscopy may be useful in diagnosing injuries to the
bronchial tree and in identifying the source of
hemoptysis, and is indispensable during the place-
ment of bronchial tubes and blockers (see below).
During thoracotomy, air in coronary arteries, left
atrium and ventricle, and the aorta may be seen or
aspirated.8,9,11–20

A contributor to the poor prognosis associated
with SAE could be the low index of suspicion among
many physicians who deal with trauma. Successful
resuscitation is made difficult when the main source
of insult—continuous passage of gas from the venti-
lator into the heart and the brain—is not recog-
nized, or recognized late, after the chest is open.

Indeed, of the 107 or so published cases of
SAE,8,9,11–20 no effort to limit airway pressures or to
selectively ventilate the uninjured lung (in unilateral
lung trauma) was mentioned except in those de-
scribed by Saada et al.11 Using transesophageal
echocardiography, these authors demonstrated (1)
the cessation of intracardiac air emboli in between
positive pressure cycles, and (2) the decrease of
emboli when ventilatory pressures and volumes were
decreased.11

Based on intuition and the experience of Saada et
al,11 I believe it is appropriate to re-examine the
heretofore-neglected airway and breathing com-
ponents of the resuscitation of patients who are at risk
or suspected of having SAE. In such patients, PPV
should be avoided if possible. If PPV is required,
high pressures should be avoided. When the injury is
clearly unilateral, emergency thoracotomy for hilar
clamping may not always be the most appropriate
immediate intervention.26 It has been proposed that
selective ventilation of the uninjured lung may be
life-saving and should be implemented.26,27 This can
be achieved by using double-lumen tracheal tubes or
bronchial blockers (stand-alone or built into the
tracheal tube).26 Obviously, placement of these ap-
paratuses may be difficult in certain trauma situa-
tions because of possible cervical instability, risk of

Figure 1. Severe circulatory or neurologic dysfunction after lung trauma: suggested protocol for
resuscitation (adapted in part from Boyd et al28).
aspiration, head injury, hemodynamic instability, airway and mediastinal injury, and lack of patient cooperation. Emergency physicians and trauma team leaders should receive training on the use of such equipment. The early involvement of an anesthesiologist should be considered. Immediate thoracotomy for hilar clamping remains an important option, especially if tamponade and hemorrhage cannot be ruled out. However, such a heroic measure is no small undertaking even in centers with highly specialized and experienced professionals, and so far the results have been dismal. Therefore, every effort should be made to stop the air embolization at the source by proper airway and ventilatory management as early as possible.

If a patient’s cardiovascular status stabilizes on institution of one-lung or low-pressure ventilation, emergency thoracotomy may be unnecessary, or at least be less urgent. Further improvement as suggested by the cessation of hemoptysis, stable cardio-pulmonary parameters, echocardiography (preferably transesophageal), radiography, and ECG might obviate the need for thoracotomy and eventually allow the resumption of normal ventilation (Fig 1). Because proximal injury is more likely to be associated with SAE (the pulmonary vein lies close to the airway branches only in the hilar region), the lack of proximal lesions on bronchoscopy may favor conservative management. Normal hemostasis is important in promoting early closure of bronchopulmonary venous fistulas. Laboratory and clinical research will be needed, and are planned at this author’s institution, to support the thesis that conservative management is a viable alternative to emergency thoracotomy.

The injured patient should receive 100% oxygen, which should allow faster absorption of gas emboli because oxygen is more soluble than nitrogen. Nitrous oxide increases bubble size and should be avoided. Hypovolemia should be avoided to discourage the entrance of air into the pulmonary veins. However, decreased survival associated with initial zealous volume resuscitation was found in a United States study of patients with torso trauma in an urban setting, and excessive fluid may be detrimental in the presence of ARDS. Once the circulation is stabilized and immediate life-saving surgery is complete, the patient can then be transported (preferably by land) to the nearest hyperbaric facility for the treatment of any suspected cerebral air embolism.

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