

## Inadvertent Venous Air Embolism from Pressure Infuser Bag Confirmed by Transesophageal Echocardiography

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Most reports on the topic of venous air embolism (VAE) deal primarily with surgical procedures where the operative site is above the level of the heart. Accidental administration of air while using a pressure infuser bag is rare. We report a case of cardiovascular collapse while applying pressure over a hard plastic bottle for rapid fluid infusion. Massive air bubbles in the right side of the heart were confirmed by intraoperative transesophageal echocardiography (TEE). This case demonstrates that TEE plays an important role in prompt diagnosis and management of VAE in anesthetized patients. The patient gave written permission to the authors to publish this report.

### Case Reports

A 54-year-old woman, weighing 56 kg, has had a known history of severe mitral stenosis for 4 years. Although mitral valve replacement was recommended to her, she refused to undergo surgery. She was seen in the hospital with a one-year history of worsening exertional dyspnea (NYHA grade III), deteriorating exercise tolerance and episodic paroxysmal nocturnal dyspnea. Past medical history included diabetes mellitus and hypertension 7 years earlier.

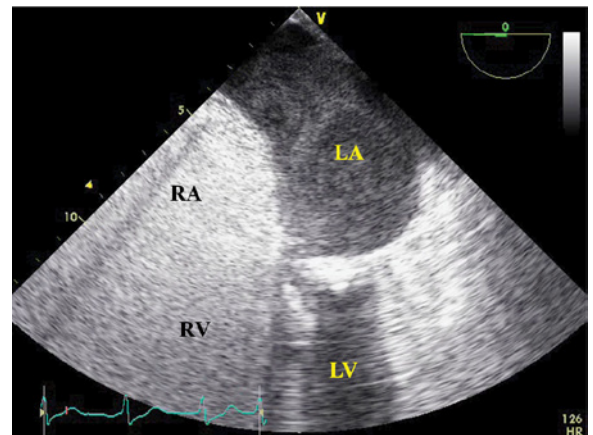
The preoperative echocardiogram reported a severe mitral valve stenosis with calcified valve leaflet. The left ventricular ejection fraction (LVEF) was 81%, with no regional wall motion abnormality. The estimated right ventricular systolic pressure was 94 mmHg. The baseline electrocardiogram (ECG) showed an atrial fibrillation rate of 70 beats/min. A coronary angiography revealed single vessel disease (80% stenosis of proximal left anterior descending artery). There was no significant baseline pulmonary dysfunction. Preoperative blood chemistry tests were normal. She was scheduled for mitral valve replacement with coronary artery bypass graft (CABG) surgery.

The patient received orally 7.5 mg midazolam 30 minute before transfer to the operating room. In addition to routine monitoring, an arterial line and a pulmonary artery (PA) catheter were inserted under local anesthesia. The preoperative blood pressure was 170/90 mmHg, there was an irregular pulse rate of 100 beats/min, and the PA pressure was 44/25 mmHg. The right atrial pressure was 12 mmHg. General anesthesia was induced with fentanyl 300 µg, propofol 100 mg titrated and pancuronium 6 mg. The patient was intubated and ventilated with oxygen in air and sevoflurane. A TEE probe was placed in the esophagus without difficulty.

The patient developed rapid atrial fibrillation and hypotension ten minutes after induced anesthesia. TEE revealed that the left ventricle was relatively small and hyperdynamic. A pressure infuser bag was applied on a hard plastic bottle for rapid infusion via a 14G peripheral line. Norepinephrine 16 µg was titrated. Five minutes after vigorous volume replacement, the hemodynamic parameters were worse: the systolic arterial pressure had fallen to 70 mmHg, the heart rate was 180 beats/min, the pulmonary artery systolic pressure rapidly had increased to 65 mmHg, and the central venous pressure had changed from 12 to 19 mmHg. Simultaneously, TEE showed opacity at the right atrial and

right ventricle, and the interatrial septum deviated to the left. It was strongly suspected there was evidence of a massive venous air emboli (Figure 1, 2). At that time, the operation had not been started and we suddenly found the pressured crystalloid bottle was empty and the IV set had air in it. ET<sub>CO</sub><sub>2</sub> decreased from 30 to 17 mmHg while O<sub>2</sub> saturation dropped to 74%.

The patient was placed in Tr endelenberg position. Hemodynamic was supported with dobutamine 6 µg/kg/min and norepinephrine 20 µg in divided doses. An emergency cardiopulmonary bypass (CPB) was performed immediately. CPB was instituted using bicaval and aortic cannulation, and the venous return lines demonstrated a significant amount of air bubbles. The CPB time was 167 minutes and the ischemic time was 107 minutes. A saphenous vein graft was anastomosed to LAD, and a mitral valve replacement was done uneventfully. A patent foramen ovale (PFO) was not found. Weaning from CPB was achieved with epinephrine 0.16 µg/kg/min, dobutamine 5 µg/kg/min, milrinone 0.6 µg/kg/min and 10 µg of nebulized prostacyclin. The postbypass TEE revealed good function of prosthetic valve. There was no significant amount of intracardiac air bubbles and a fair LV contraction.



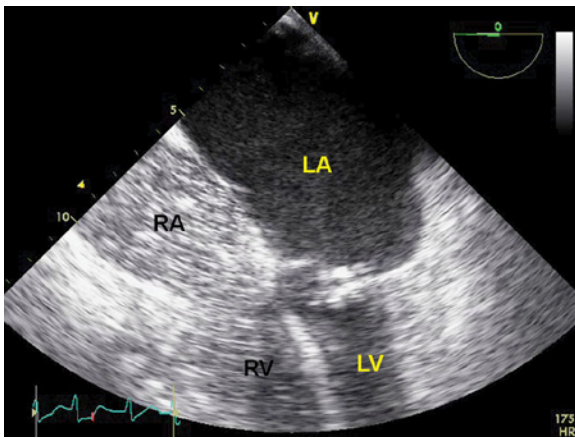
**Figure 1:** Mid esophageal four-chamber view: TEE demonstrated that the right atrial and right ventricle were opacified with a massive amount of air.

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**Figure 2:** Mid esophageal four-chamber view: 10 minutes after Figure 1. Snow-storm appearance in the right atrial and ventricle.

The patient was transferred to postoperative ICU. She regained consciousness with no neurologic deficits. Her hemodynamic parameters were stable, and inotropic drugs could be weaned off 14 hours postoperatively. She was extubated after 18 hours and discharged home 21 days after the operation.

## Discussion

There are two important keys in this. Firstly, massive air embolism can occur while infusing fluid under pressure with a pressure infuser bag. Secondly, recognition of the echocardiographic appearance of VAE will aid in prompt diagnosis and management.

VAE has been defined as the entrainment of air and/or medical gas introduced to the central venous intravascular space and embolized to the right heart or pulmonary arterial system. Arterial embolism results from the entry of gas into the left heart chambers, as with paradoxical venous embolism across an intracardiac shunt or during cardiac surgery, or directly into the arteries of the systemic circulation, such as during decompression barotraumas or a penetrating trauma involving an artery. According to a recent review of VAE [1], numerous surgical procedures and nonoperative procedures have been associated with this potentially fatal complication.

Air can also be introduced directly via peripheral venous cannulae, particularly when fluid is rapidly infused under pressure infusion devices. Gray et al reported two cases of VAE while infusing Haemaccel® from a pressurized plastic bottle with a standard administration set. They demonstrated that up to 45 ml of air expelled from IV set could be infused into a patient [2]. Linden et al reported five cases of fatal VAE after the readministration of recovered blood. All cases involved reinfusion of blood under pressure [3]. This interesting publication has been mentioned twice in the letter to the editor by Benumof JL [4]. ASA closed claims project database revealed ten claims for air emboli caused by IV infusion. Claims related to air embolism had the highest median payment and a rate of 100% payment-per-claim [5].

The incidence of VAE from pressurized fluid may be underreported because the diagnosis is difficult and usually made by exclusion. Currently, a novel method that can detect small air emboli (0.05mL) in a swine model is transvenous intracardiac echocardiography (ICE). Schäfer et al demonstrated that ICE has much greater sensitivity

compared to TEE and precordial Doppler monitoring (PCD). However, more clinical studies are needed to confirm the sensitivity of ICE [6].

The presented case demonstrates how massive VAE from accidental infused air through an IV line can be confirmed by TEE. TEE is a very sensitive technique for VAE detection in clinical practice. It offers direct observation of air in the heart, allowing anesthesiologists to make a definite diagnosis. Cardiac anesthesiologists frequently perform intraoperative TEE in cardiac surgery to diagnose intracardiac shunts, disturbances in valvular function, gas embolism, aortic dissection, myocardial ischemia, and to detect residual air when the patient is being weaned from CPB [7]. Diagnosis of VAE by TEE must be interpreted cautiously and differentiated from an echocardiographic contrast caused by air bubbles from a rapid IV infusion which do not lead to adverse sequelae. One limitation of TEE is the need for sufficient numbers of expert anesthesiologists. Ideally, one anesthesiologist should conduct anesthesia while another dedicated person performs TEE. However, the situation is not always possible in Thailand, especially in regional hospitals. We are currently seeking more manpower to combat this problem. Other limitations of TEE are that it is expensive and invasive.

The morbidity and mortality rates from VAE are determined by the volume of air entrained, the type of gas injected, the rate of entrainment, the patient's position, and the cardiac status [1,8]. One report could quantify the lethal volume of air at 200 mL [9]. Small acute volumes are often well tolerated whereas larger volumes (more than 2 mL/kg) have substantial effects predominating in the cardiovascular, pulmonary and cerebral organ systems [1]. VAE might have caused increasing pulmonary vascular resistance and right ventricular dysfunction in our case because multiple inotropic medications were needed for weaning from CPB.

Treatment of air embolism includes hemodynamic support, instituting high flow oxygen, reducing embolic obstruction, cardiopulmonary resuscitation and prevention of further air entry into the circulation [1]. Hyperbaric oxygen may be considered if there are neurological changes evident on physical examination [10]. The recommendations for this iatrogenic complication are avoiding the use of a pressure infuser bag applied over the hard plastic bottle, routine de-airing of all IV fluid bags, and the use of devices that incorporate automatic detection of air coupled with a shut-off mechanism [11,12].

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