Air Embolus in the Cardiac Catheterization Laboratory – A Highly Preventable Procedural Complication

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I was recently asked to review a case of a patient who had a myocardial infarction after a coronary angiogram, caused by an air embolus. The patient recovered, but I realized I had incomplete knowledge of this complication. I thought I'd review the subject here for our cath lab colleagues.

Air embolisms (AEs) occur when air enters or traverses a vessel or chamber. Air can enter either into the venous or arterial circulation, moving along a pressure gradient or traveling with gravity. AEs are usually iatrogenic (as opposed to gas formed during some infections) and are frequently associated with various invasive procedures or interventions (Table 1). AEs can transiently obstruct the coronary blood flow, producing ischemia (Figure 1), typically detected during or immediately after a procedure by symptoms such as chest pain, dyspnea, confusion, or altered consciousness (eg, encephalopathy). AEs may also be silent and be detected incidentally on repeat imaging. The most common AEs occur during central venous catheterization. With

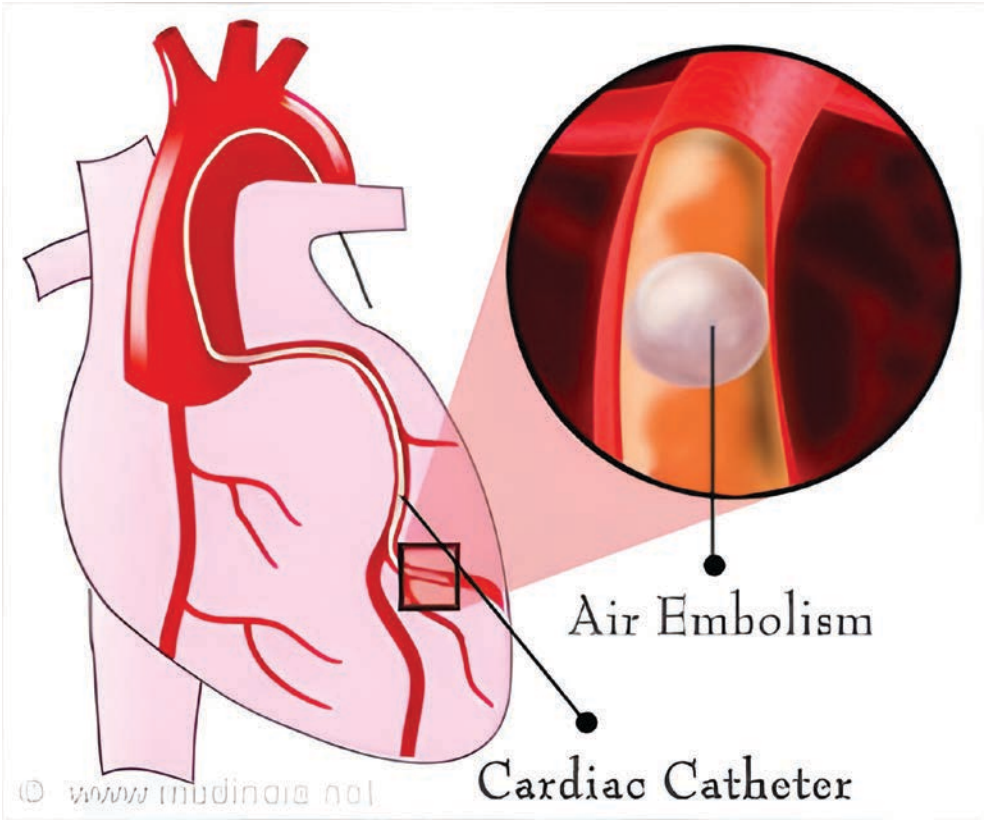

Figure 1. Diagram of air bubble in a coronary. A catheter has been inserted in an attempt to aspirate the air.

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TABLE 1. Procedures associated with air embolism.

PROCEDURE
Abdominal aortic aneurysm repair
Abdominoperineal resection
Atrial septal defect closure
Bronchoscopy
Coronary artery bypass grafting
Cardiac ablation
Cardiac catheterization
Inadvertent air bolus via IV line
Central vascular access
Endoscopic Retrograde Cholangio-Pancreatography (ERCP)
Hysteroscopy
Laparoscopic liver resection
Necrotic bowel resection
Endovascular neurointervention
Neurosurgery (open procedures)
Pacemaker placement
Spinal surgery
Transarterial chemoembolization (TACE)
Varicose vein injection

From McCarthy CJ, Behravesh S, Naidu SG, Oklu R. Air embolism: diagnosis, clinical management and outcomes. *Diagnostics (Basel).* 2017 Jan 17;7(1):5. doi:10.3390/diagnostics7010005. Licensed under a Creative Commons Attribution-Non Commercial 4.0 International License. https://creativecommons.org/licenses/by-nc/4.0/deed.en

Figure 2. Cineframe during right coronary angiography. During percutaneous coronary intervention for an acute ST-elevation myocardial infarction (STEMI) of the right coronary artery, angiography demonstrates circular lucent filling defect. Air versus thrombus? Top small arrow shows a round lucency (maybe a thrombus tail) and the lower arrow shows an ulcerated plaque with thrombus related to the STEMI.

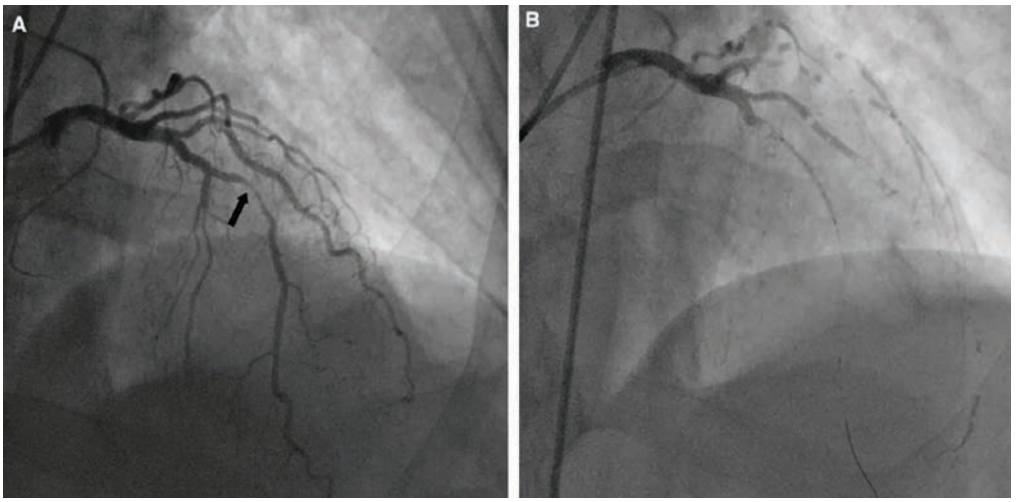

Figure 3. Diagnostic coronary angiography of the left anterior descending artery in the right anterior oblique cranial projection, showing (A) a severe mid-left anterior descending artery lesion (black arrow) and (B) several coronary intraluminal filling defects attributable to massive air embolism.

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central venous pressure catheter placement, an air embolus may occur in approximately 0.2-1% of patients.[1] Treatment may prove futile if the air bolus is >50 ml.[1]

In the cath lab, the most common occurrence of an air embolus results from inadvertent injection of air into a coronary artery through the manifold, tubing, and coronary catheter. An air embolus can also occur during any large-bore procedure, or even during flushing of intravascular imaging or aspiration catheters. Air bubbles may be injected if there is insufficient aspiration of blood during the clearing of the catheter or with poor flushing techniques. Rarely, an AE can occur during device exchanges or loose port connections where removal of a large catheter creates a transient vacuum and air enters the guide catheter. Iatrogenic introduction of air during catheter manipulation or exchange is rare and usually harmless (1 in 3000 cases).[2]

Massive air embolism in cardiac bypass procedures is rare, occurring in between 0.003% and 0.007% of cases, with 50% having adverse outcomes.[1] AE is most associated with otolaryngology and neurosurgical procedures due to the location of the surgical incision. The sitting position in posterior craniotomies is deemed especially risky and procedure-related complications of venous air have been estimated to be between 10% and 80%.[3] This fact should be kept in mind for procedures in the cath lab when the patient's head is in an elevated position.

While air in the coronary arteries may impede coronary flow producing ischemia, air in the left ventricle impedes diastolic filling. During systole, air is pumped into the coronary arteries, disrupting coronary perfusion, and leading to acute hypoxemia and hypercapnia. Air may produce acute changes in right ventricular (RV) pressure, resulting in RV strain, which can lead to right heart failure, decreased cardiac output, RV ischemia, and arrhythmia, followed by systemic circulatory collapse and even death.

On the venous side, great caution should be exercised in patients with a suspected patent foramen ovale (PFO). Paradoxical venous embolism in patients with PFO/shunts can produce a devastating stroke. The risk of venous air embolus is increased by low central venous pressure or inspiration, reducing intrathoracic pressure below atmospheric pressure.

Mechanisms of Symptoms

A coronary air embolism can be seen on angiography as a lucent spherical or circular lucency within the contrast-filled vessel or structure (Figures 2-3). The sequelae and pathophysiology of AEs arise from obstruction to blood flow, ischemia, and infarction from air entering the coronary arteries, brain, or other end organs resulting in reduced tissue perfusion. The lethal volumes of air in an acute bolus is approximately 0.5–0.75 ml/kg in rabbits and 7.5–15.0 ml/kg in dogs.[4] The lethal dose for humans is theorized to be 3-5 ml/kg, estimated at 300-500 ml of gas introduced at a rate of 100 ml/sec.[4] Air infusion rates of more than 1.5ml/kg/min are associated with bradycardia and cardiovascular decompensation.[3] The rate of accumulation and patient position contribute to the lethality.[4]

Diagnosis of Air Embolus

Prompt recognition and team coordination are critical. Early diagnosis depends on an index of suspicion for new clinical signs which

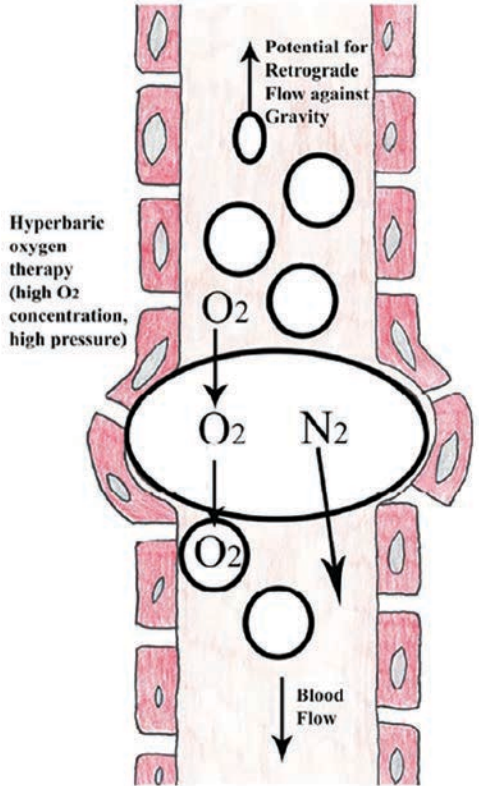

Figure 4. Diagram of an air bubble obstructing a vessel. As the concentration of O_2 becomes greater, nitrogen moves out of the bubble, reducing its size.

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Figure 5. Positioning and hyperbaric oxygen therapy chamber (bottom panel). Top left, Trendelenburg position (head down). Top right, patient is in left lateral decubitus position (also known as Durant's maneuver).[7] Left lateral decubitus positioning allows air to move toward the right ventricular apex, thereby relieving the obstruction of the pulmonary outflow tract.

may include sudden hypotension, bradycardia or tachycardia, chest pain or ST changes or seizures, and focal neurologic deficits. Cardiac arrest has been reported in rare and severe cases. Diagnostic modalities beyond angiographic or computed tomography angiography include Doppler ultrasonography, a sensitive and practical means of detecting intracardiac air, often used during neurosurgical procedures with the patient in a sitting position. Transesophageal echocardiography is more sensitive than Doppler and is frequently utilized by anesthesiologists to monitor patients during high-risk procedures.

Management of Air Embolus

Coronary air embolus is only one of the many potential complications related to coronary angiography. Table 2 lists some of the coronary and peripheral vascular complications and their management strategies. Treatment starts by stopping air entry into the system, followed by several other interventions such as Trendelenburg position and left lateral decubitus position (also known as Durant's maneuver).[5] Figure 4 illustrates how positioning and gravity can influence the movement of air against the blood flow direction and reduce the impact of embolic occlusion. Air in the heart can be stabilized within the left ventricular (LV)/RV apex. Left lateral decubitus positioning allows air to move toward the RV apex, thereby relieving the obstruction of the pulmonary outflow tract. Another method is to aspirate air via a guiding catheter.[6] A microcatheter may be able to reach more distally than a guide catheter. Some operators have suggested a forceful injection of saline or contrast to disperse air.[2]

Patients with an air embolus should receive 100% oxygen administration, which works by reducing the nitrogen content in the bubbles shrinking the bubble size. Air is mostly composed of nitrogen (78%), oxygen (21%), argon (1%), and carbon dioxide (0.04%).[7] Replacing nitrogen with O_2 at the higher concentration of O_2 helps reduce bubble obstruction.

Hyperbaric oxygen therapy (HBOT) should be considered early in severe cases of air embolus (Figure 5). HBOT has been used for years to treat divers who have rapidly ascended after prolonged exposure to deep ocean pressure. Under deep diving pressure exposure, air in the diver's inspired gases enters the blood and is compressed. On rapid decompression, the bubbles come out of solution and can block blood flow to various vital organs, but particularly the brain with central nervous system complications (decompression sickness, or the bends). For these patients, hyperbaric oxygen administration is the primary therapy.

Additionally, ventilation with 100% O_2 corrects hypoxemia and increases the diffusion gradient for nitrogen out of the bubbles, causing them to shrink.[8] HBO administration reduces bubble size due to the absorption of nitrogen from the bubble. Elevated ambient pressure reduces the bubble size in accordance with Boyle's law. At 282 kPa (kilopascal), a conventional HBO treatment pressure, gas bubble diameter will be reduced to 82%, a 45% decrease in volume, promoting passage through the microcirculation and resolution of embolic phenomena.[8]

In a review of a case series with 27 patients, Edsell et al[9] noted a substantial improvement in outcomes was shown in patients treated with

HBO. Of these patients, 346/441 (78%) who received HBO fully recovered and 20 (4.5%) died. Of the 288 with no recompression therapy, 74 (26%) fully recovered and 151 (52%) died.

Treating Coronary Slow/No Flow

Although thought to be mostly ineffective for air bubbles, therapies for coronary slow/no flow can be given during the institution of measures to stabilize blood pressure and any resuscitative attempts that may be required. First-line management includes intracoronary administration of adenosine (10-20 mcg bolus), verapamil (100-200 mcg bolus), and nitroprusside (50-100 mcg bolus). Alternative agents also thought to be effective include diltiazem (0.5-2.5 mg), papaverine (10-20 mcg), nicardipine (200 mcg), nicorandil (2 mcg), and epinephrine (50-100 mcg). Ineffective or contraindicated treatments for no reflow related to air embolus include intracoronary nitroglycerin, coronary artery bypass graft surgery, stent placement, and thrombolytics.

Innovations and Prevention

Staff education and air embolism checklists are an important starting point for lab improvement and protection. All personnel dealing with tubing, connectors, Luer-lock syringes, and air-eliminating devices should ensure tight connections and correct functioning. Do not attempt to bypass or silence bubble detectors. Inline microbubble detectors in bypass circuits and in the ACIST CVi Contrast Delivery System are designed as critical life-protecting implementations. For a quick, accurate assessment, operators can use real-time intracardiac echocardiography imaging to detect intravascular air.[10,11]

The Bottom Line

Air embolism is a preventable but serious complication. Early recognition and intervention reduce morbidity and mortality. Continued innovation in system protocols, equipment security for connections and leaks, with frequent checks on technique, and early-detection diagnostic tools will reduce complications and adverse outcomes. ■

TABLE 2. Coronary and peripheral vascular complications and their management strategies. Air embolus involves obstruction and occasionally no reflow.

	COMPLICATION	MANAGEMENT
	• Dissection	Stent
	• Thrombus	Aspiration, thrombolytics
Air	**• Embolus**	**Distal protection, thrombolytics**
Air	**• No reflow**	**Verapamil, adenosine, nipride**
Air	**• Occlusion**	**Rx cause, gravity positioning**
	• Perforation	Balloon tamponade, covered stent
	• Spasm	NTG, verapamil
	• Access bleed	Vascular closure devices

NTG = nitroglycerine

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Disclosures: Dr. Morton Kern reports he is a consultant for Abiomed, Abbott Vascular, Philips, ACIST Medical, and Opsens Inc.

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