right-to-left shunting was observed as most likely a significant amount of air passed from the right over to the left ventricle, presumably because left atrial pressure was low because of postoperative dehydration, systemic hypotension, and an upright position.

This case presents the need for awareness of this rare but potentially life-threatening complication. Furthermore, sudden neurological or cardiac events after manipulation of a central venous line should call attention to a possible paradoxical air embolus. No specific treatment is available, but administration of oxygen 100% and i.v. fluids seems prudent. To prevent such events, any manipulation of central venous lines should be undertaken in the supine position while the spontaneous breathing patient is exhaling. However, even with severe neurological and cardiac effects, outcome may not be fatal.

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Editor—Intracoronary air embolism is a rare complication of open heart surgery which is characterized by the appearance of hypotension, ST elevation, and ventricular fibrillation after aortic clamping removal or in the last minutes of cardiopulmonary bypass (CPB). Several authors have demonstrated the effectiveness of monitoring transoesophageal echocardiography (TOE) during cardiac surgery for diagnosis of intracoronary air embolism as it detects the presence of air in the coronary arteries and intraoperative myocardial ischaemia due to alterations in segmentary ventricular contractility. We report a case of intracoronary air embolism detected by TOE in a patient undergoing aortic valve replacement for severe stenosis. Intraoperative TOE showed a marked depression in ventricular myocardial activity with severe hypokinesia and areas with hyper-refringency due to intracoronary air (Fig. 1).

A 43-yr-old, 91 kg man, with a history of hypertension and hypercholesterolemia who was treated with captopril, propranolol, and statins underwent general anaesthesia for valve replacement surgery for severe aortic stenosis secondary to bicuspid valve calcification. He had dyspnoea at rest for 8 weeks and episodes of upper retrosternal chest pain pressing in character. Transthoracic echocardiography

Fig 1 TOE: intracoronary air embolism with hyper-refringency areas in left ventricular anterior wall.
study showed a transvalvular gradient of 76 mm Hg and left ventricular hypertrophy with preserved systolic function. The patient was premedicated with oral diazepam 10 mg 1 h before surgery. In the operating theatre, arterial pressure was monitored at the left radial artery, with five-lead electrocardiography, pulmonary artery pressure by Swan Ganz catheter, regional cerebral oxygen saturation, bispectral index (BIS), pulse oximetry, partial pressure of end-tidal carbon dioxide, oesophageal temperature, and neuromuscular function monitoring. Intraoperative haemodynamic status was assessed by TOE, using 5 MHz frequency Philips Sonos multiplanar transducer. Induction was achieved with etomidate 0.3 mg kg⁻¹, cisatracurium 0.2 mg kg⁻¹, and fentanyl 4 μg kg⁻¹. Anaesthesia was maintained with sevoflurane 1–2%, remifentanil 0.3 μg kg⁻¹ min⁻¹ in the 40–60 BIS range. During surgery, haemodynamic stability was maintained with CBP uneventfully at 80 min with 60 min of aortic clamping, and sinus rhythm with adequate segmentary contractility in TOE. Fifteen minutes later, hypotension with a systolic pressure of 60 developed, despite apparent good function of the prosthetic valve with a transvalvular gradient of 23 mm Hg. There was a severely hypokinetic area on the anterior wall with high-refractivity areas attributable to intracoronary air, with calculated left ventricular ejection fraction of 20% and ST elevation in leads V1 and V2. This progressed to ventricular fibrillation in spite of internal defibrillation, and it was necessary to restore the CBP. With the diagnosis of intracoronary embolism, it was decided to increase arterial pressure by the infusion of levosimendan 0.1–0.2 μg kg⁻¹ min⁻¹ and also the flow speed of CBP, improving coronary perfusion, anterior wall segmentary contractility, and haemodynamic function, with subsequent disappearance of hyper-refractivity area. CBP and the inotrope were later withdrawn and normal arterial pressure maintained. The postoperative period in the ICU was uneventful with extubation 5 h after admission and return to the ward in 48 h.

Intracoronary air embolism during extracorporeal surgery is characterized by the presence of hyper-refractivity areas in the ventricular myocardium and left ventricular hypokinesia resulting from ischaemia. The right coronary artery is most frequently affected due to the anterior position of the right Valsalva sinus. In our patient, the hyper-refractivity area and myocardial hypokinesia was in the left ventricular anterior wall due to embolism of the anterior descending branch of the left coronary artery. TOE excluded early prosthetic dysfunction and the possibility of inadequate blood. The hyper-refractivity area and ventricular hypokinesia and immediate recovery after treatment with inotropic drugs facilitated the diagnosis of intracoronary air embolism as the cause of myocardial ischaemia, and excluded thrombosis and spasm. In intracoronary air embolism, symptomatic treatment with increase in coronary perfusion pressure and the use of inotropic agents is recommended. In our case, reconnection to CBP and levosimendan to increase myocardial contractility and coronary perfusion pressure returned left ventricular contractility to normal.

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4 Lam G, Auer J, Punzengruber C, Ng CK, Eber B. Intracoronary air embolism in open heart surgery—an uncommon source of myocardial ischaemia. Int J Cardiol 2006; 112: e85–6
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Non-cardiac surgery 2 weeks after percutaneous cardiac intervention

Editor—A 52-yr-old male was admitted with a fractured tibial plateau. He had undergone percutaneous cardiac intervention (PCI) 3 days previously, and had stent insertion three times previously in the last 12 years for coronary artery disease (CAD). The patient’s current medication included atenolol, atorvastatin, and oral hypoglycaemic drugs for type 2 diabetes. He was on oral aspirin and clopidogrel for anticoagulation. His ECG showed only left axis deviation and no evidence of ischaemia. The cardiologist advised continuation of the antiplatelet drugs because of the high risk of thrombosis of the stent due to epithelization up to 6 weeks. An echocardiogram showed an ejection fraction of 60% with no regional wall motion abnormalities. Surgery was planned for 11 days later with continued cover of aspirin and clopidogrel, under general anaesthesia.

On the morning of surgery, oral hypoglycaemic drugs were omitted whereas all other drugs were continued. A nitroglycerine patch was applied 1 h before surgery and plain insulin and dextrose infusion started for control of blood sugar during the perioperative period. Anaesthesia was induced with morphine 7.5 mg, thiopental, vecuronium, O₂, N₂O, and isoflurane. Airway management was accomplished with a Proseal LMA. Care was taken to maintain adequate depth of anesthesia and avoid sympathetic stimulation. A tourniquet was applied during surgery and no excessive bleeding was noted during surgery or after deflation of tourniquet. Reversal and postoperative period were uneventful.