Sirs: We describe a patient with bilateral cerebral air embolism caused by a fistula from the oesophagus to pulmonary veins next to the left atrium during gastroscopy and biopsy of a Barret's ulcer.

A 56-year-old woman was admitted to hospital after a right-hemispheric transient ischaemic attack causing a left-sided hemiparesis for two hours. Stroke risk factors were hypertension and hyperlipidaemia. One year previously the patient had undergone ovariectomy for a carcinoma. Cranial CT was unremarkable. The next day, haematemesis occurred. Endoscopy revealed oesophageal bleeding caused by a perforated Barret's ulcer. Bleeding could be stopped by adrenaline application. One hour later, the patient had a generalized seizure and developed a left-sided hemiparesis for four hours. She received anti-convulsants, and a central venous catheter was placed. Cranial CT performed 30 minutes later was normal. Two days later, endoscopic follow-up examination showed a large Barret's ulcer. Histological examination showed no neoplastic changes. Just after the second endoscopy, the patient suddenly became somnolent and vomited. To prevent aspiration, intubation was performed without complication. Cranial CT performed immediately afterwards showed bilateral air emboli (Fig. 1 A). Intensive care unit therapy including administration of 100% oxygen was performed. After two hours, the patient could be extubated. An electrocardiogram (ECG) demonstrated transient T-wave inversion in I, II, and V2–6, echocardiography showed hypokinesia of the anterior wall of left ventricular apex and a slightly reduced left ventricular ejection fraction. Five hours after detection of bilateral cerebral air embolism, follow-up cranial CT showed that the gas had disappeared. Five days later, transoesophageal echocardiography and ECG were entirely normal. Follow-up MRI seven days after detection of the bilateral cerebral air embolism revealed bilateral T2w-hyperintense infarction areas in those locations, where air embolism had taken place (Fig. 1 B). The apparent diffusion coefficient (ADC) was normalized. MR-angiography of the circle of Willis, ultrasonography of carotid and vertebral arteries, angiography of aortic arch, brain supplying arteries, and cardiac vessels, cerebrospinal fluid analysis, chest radiographs, sonography of the epigastrium, transoesophageal echocardiogram, ultrasonographic bubble-examination for a right-to-left shunt were entirely normal. High-resolution CT showed no pulmonary arteriovenous malformations. Laboratory investigation was unremarkable. CT of the mediastinum eight days after the detection of bilateral cerebral air embolism revealed next to the Barret's ulcer an air-isodense oesophageal second lumen leading to the pulmonary veins next to the left atrium (Fig. 1 C). Conservative treatment was favoured, because the patient had recovered well since the cerebral air embolism had taken place eight days before and no clinically silent infarctions had been detected on diffusion-weighted MRI. Follow-up gastroscopy showed a complete healing of the Barret's ulcer after medical treatment. The patient was discharged without focal neurological deficits.

Cerebral gas embolism is known to occur in several clinical settings such as trauma, surgery, sudden air pressure changes, central venous catheter placement [3], mechanical ventilation [7], lung biopsy, angiography [11], ingestion of concentrated hydrogen peroxide [5], endoscopy, or with an oesophageal-atral fistula [10]. It can easily be diagnosed with CT, if the scan is performed shortly after symptom onset [3, 4]. Intracerebral gas embolism is a serious disorder often causing dramatic manifestations with diffuse brain damage and has a high mortality rate [3, 9]. The most commonly observed symptoms are hemiparesis or hemianopia due to cerebral infarction and seizure activity [3, 9]. In some patients with minor cerebral involvement, when gas has cleared and capillary flow returns as in our patient, a sudden recovery may follow [7]. Air entering the venous system and crossing over to the arterial system through an open foramen ovale or the pulmonary bed, even without the presence of a macroscopic arteriovenous shunt has been described. In those cases, air may have passed across the pulmonary capillary bed or through microscopical pulmonary arteriovenous anastomoses [1].

In our patient, cerebral air was detected just after gastroscopy and not after central venous catheter placement. Functional crossing of air from the venous to the arterial vessel system could be excluded by bubble-examination. Pseudo-normalization of the apparent diffusion coefficient of infarction areas,
typically observed approximately on day nine after a stroke, proved the relationship between air embolism and infarction in our patient [2]. Cerebral arterial gas embolization typically involves the migration of gas to small arteries with an average diameter of 30 to 60 µm leading to occlusion or reduction in perfusion distal to the obstruction depending on the amount of air that entered the arterial system [9]. In our case, the air was symmetrically distributed on the border of the anterior and middle cerebral artery territories, so that the air embolism may have affected the functional end arteries on the border of the anterior and middle cerebral artery territories causing the border-zone infarctions visible on MRI. The bilateral pattern of cerebral air embolism presumably resulted from air entry somewhere between pulmonary veins, left atrium and aortic arch before branching of arteries supplying the brain. Mediastinal CT demonstrated a second oesophageal lumen, which partially could have been a mucosal fold. However, the fistula-like part perpendicular to the oesophagus was suspiciously like a connection between oesophagus and pulmonary venous system next to the left atrium, presumably resulting from the perforated Barret's ulcer or from biopsy of the ulcer. The formation of an oesophageal-left atrial fistula as a rare and often lethal complication of chronic oesophagitis like Barret's oesophagitis has already been described [6, 8]. In our case, an inflammatory fistula formation seems more likely, as it could explain why the patient's state deteriorated just after both endoscopies with air-insufflation. The episode with a generalized seizure might be associated with cerebral air embolism without detection by CT.

The findings in our patient also demonstrate that emboli from the pulmonary venous bed can reach the brain and can cause infarctions.

References