LETTER TO EDITOR

Reply to comments on coronary air embolism

Jarosław Wośko

1st Department of Anaesthesiology and Intensive Therapy, Medical University of Lublin

Gas bubbles present in an animal were first described by Boyle in 1670. Boyle claimed that air bubbles were formed due to a rapid pressure decrease in the decompression chamber, in which the animal was placed. Moreover, he described blood flow abnormalities and tissue hypoxia caused by air emboli located in the tiny vessels. In 1934, Chase described the two-phase nature of the vascular reaction induced by air bubbles. Some years later, Durant explained in detail the mechanism of mechanical vessel closure combined with vascular spasm in the pathogenesis of tissue hypoxia due to air embolism [1].

Three hundred and forty years after the pioneer works of Boyle, rapid development of medicine multiplied the risks of air embolism. Undoubtedly, any invasive medical procedure, endangering the vascular continuity, is associated with the risk of gas entry to the bloodstream. Some treatment methods are particularly risky in terms of such complications, e.g. neurosurgical procedures in the posterior cranial cavity, which 15-20 years ago were performed with patients placed in reclining positions. According to some authors, the risk of air embolism in a patient operated on in such a condition is even 20-40% [2]. The more recent publications suggest lower percentages, i.e. about 9%.

Air embolism related to abdominal surgical procedures under pneumoperitoneum occurs in even 70-100% of patients [4, 5]. This is obviously a different issue due to the use of CO2; nevertheless, the link is the risk of massive gas penetration to the vascular bed. In 37.5% of cases, this results in haemodynamic disturbances [4]. Moreover, orthopaedic surgical procedures are associated with the risk of embolic complications; in some of them, air is the embolic material [6]. The largest group of patients at risk of air embolism includes those undergoing cardiac surgeries with extracorporeal circulation. Air bubbles are detected on transoesophageal echocardiography even in 79% of patients after heart valve surgeries and in 11% of aortocoronary bypass patients [7]. Some part of embolic gas material passes to the systemic circulation when the aorta camping is removed and haemodynamically efficient heart action is restored.

There are no doubts that intravascular gas emboli are associated with the risk of organ injuries, especially neurologic complications. Furthermore, nobody questions the fact that hyperbaric oxygen therapy is the most effective method of treatment of massive air embolism. According to Dexter, hyperbaric oxygenation should be always considered when the air bubbles are detectable on the brain CT scan [8]. Moreover, it is known that the therapy should be started as early as possible, although some believe that delayed institution is also associated with improvement of neurological condition. The incidence of air embolism cases seems definitely understated. The majority of cases are likely to present no explicit clinical manifestations, which is associated with moderate severity of the process and spontaneous absorption of air when its amount is low.

Indeed, in the case described there was the risk of severe neurologic sequels. However, the patient's general condition improved quickly and 2 h after the embolic episode verbal, logical communication with the patient was established. The in-depth neurological examination did not reveal any symptoms of focal brain damage.

Jarosław Wośko
I Klinika Anestezjologii i Intensywnej Terapii SPSK nr 4
ul. Jacezkiewicza 8, 20–950 Lublin
tel.: + 48 (81) 724 43 32

References