Quadriplegia following venous air embolism during posterior fossa exploration

A case report

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Summary

A frequent complication of posterior fossa exploration with the patient in the sitting position is venous air embolism. Spinal cord deficits following such a mishap have rarely been reported. We report a case in which a patient who suffered venous air embolism developed quadriplegia from the C6 level. The mechanisms of passage of air into the arterial system are discussed.

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Surgical procedures in the region of the posterior fossa with the patient in the sitting position are associated with a number of complications. Venous air embolism is the most common complication and has been documented frequently.[1,2]

The reported incidence of venous air embolism in the sitting position varies from 2%[2] to 29%[3] depending on the sophistication of the methods used. The mortality associated with this complication ranges from nil[3] to 73%.[4] Neurological complications of venous air embolism have rarely been reported.[5] We report the development of quadriplegia following successful resuscitation of a patient who sustained this complication during posterior fossa surgery.

Case report

A fit 26-year-old woman was scheduled for posterior fossa exploration for a suspected cerebellar tumour. The operation was to be performed with the patient in the sitting position. The tumour was situated in the rostral vermis area of the cerebellum, and histological examination of a biopsy specimen established the diagnosis of haemangioblastoma.

Premedication of lorazepam 2 mg was given orally 2 hours before the operation. On arrival at the theatre labetalol 25 mg was given intravenously. The patient was pre-oxygenated for 4 minutes, and induction with thiopentone 300 mg was followed by alcuronium 15 mg. Prior to intubation she was ventilated with 100% oxygen.

The trachea was sprayed with xylocaine 4% and a number 8 cuffed endotracheal tube was inserted. Anaesthesia was maintained with a nitrous oxide-oxygen mixture, the fractional inspired oxygen concentration being 0,5. Halothane (0,4-1,0%) and fentanyl 0,5 mg in divided doses were added. The patient was ventilated with a Draeger Spiromat 650. Shortly after induction administration of 20% mannitol (1 g/kg) was commenced.

The following monitors were used: (*i*) a 20G radial artery catheter was inserted and the arterial pressure continuously monitored with a Simonsen and Weel AE 840 pressure transducer and oscilloscope; (*ii*) a central venous line was inserted via a basilic vein and a radiograph taken to confirm that the tip lay opposite the third right costal cartilage; (*iii*) an intra-oesophageal stethoscope; (*iv*) an intra-oesophageal temperature probe; (*v*) continuous electrocardiographic monitoring; (*vi*) expired carbon dioxide was continuously monitored with a Godart infant capnograph; (*vii*) measurement of the peripheral pulse rate; and (*viii*) measurement of urine volume.

The patient was gradually eased into the sitting position with minimal upset in the readings. The pulse rate was 75/min, the central venous pressure was 5 cm H_2O and the end-tidal carbon dioxide concentration was 4 vol. %. The arterial pressure was 105 mmHg.

Anaesthesia was maintained for $4^1/_2$ hours with only minor changes in the above readings. During approximation of the muscle layers at the end of the operation, however, the systolic blood pressure dropped to 60 mmHg in 1 minute, the end-tidal carbon dioxide concentration dropped to 2 vol. %, and the central venous pressure rose to 17 cm H_2O. The ECG remained normal. Venous air embolism was suspected. No cardiac murmurs were detected through the oesophageal stethoscope.

The operating table was levelled and 100% oxygen administered. Aspiration on the central venous catheter produced 130 ml air. Within 5 minutes the patient's condition improved — all the readings were back to the previous levels and the operation could be continued. Methylprednisolone 30 mg/kg was administered intravenously. The operation ended 35 minutes after the incident, and although the patient was fully conscious within 20 minutes it was decided that she should be ventilated overnight.

To our horror it was discovered 4 hours later that she was quadriplegic, with a motor and sensory level below C6. On the 2nd postoperative day some movement of the left big toe was possible. Reflexes in the legs were slightly depressed, but the arms exhibited a flaccid paralysis. Shoulder movement was normal. Motor function in the legs improved progressively over 7 days up to 50% of normal power, but with an element of spasticity. The flaccidity of the arms remained unchanged. At first urinary retention was a problem, but bladder function had returned to normal within 3 weeks. There was loss of pain sensation below C6, but proprioceptive sensation was normal. When discharged 28 days later she could stand with support, but there was no improvement in the flaccid paralysis of the hands.

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Discussion

Venous air embolism is regarded as a particular hazard during posterior fossa exploration in the sitting position. A number of clinical signs of air in the circulation and methods of detecting it have been described,[6-14] the placing of a Swan-Ganz catheter in the pulmonary artery[15,16] being the most recent.

Our patient developed quadriplegia after air embolism, an extremely rare complication. We have failed to find another such report in the literature of the past 10 years. Paraplegia has been reported following venous air embolism resulting from a pressurized scalp vein infusion in a neonate.[17]

Emerson *et al.*[18] injected air into the rat pulmonary artery but failed to detect bubbles in the pulmonary vein or left auricle. They concluded that under physiological conditions it is difficult, if not impossible, for gaseous emboli to pass through the pulmonary circulation in the absence of cardiac defects. Although cardiac catheterization to exclude a cardiac defect was not performed in our case there was no clinical evidence of any abnormality. However, Butler and Hills[19] demonstrated in dogs that air could pass through the pulmonary circulation when a large bolus (30 ml) was injected into the right ventricle or pulmonary artery. The bubbles were detected by a Doppler probe positioned transcutaneously over a femoral artery. When their experimental animals were pretreated with aminophylline, a pulmonary vasodilator, quantities of air not exceeding 2 ml injected into the right ventricle, with a bubble size of 130 μm, passed through the pulmonary circulation in every case. This observation has important clinical implications.

There are reports[20,21] of observation of air in the arterial system in man after inadvertent venous leaks. Vourc'h[20] observed air in the cerebral arterioles following venous air embolism during posterior fossa exploration. At autopsy no cardiac defect could be demonstrated. Baskin and Wozniak[22] presumed that the air traversed physiological arteriovenous shunts.

It has been demonstrated in man that venous air embolism raises the pulmonary arterial pressure, the rise being directly related to the amount of air entrained.[17] In our case a large amount of air (130 ml) was aspirated, which would have raised the pulmonary arterial pressure markedly. In addition halothane, a pulmonary vasodilator which also suppresses the hypoxic pulmonary vascular reflex responsible for vasoconstriction, was used,[23] resulting in the conditions found by Butler and Hills[19] to be necessary for a venous air embolus (a large bolus of air with raised pulmonary artery pressure and vasodilation) to pass the pulmonary circulation.

Nitrous oxide was used as an anaesthetic agent at the time of the air embolism in our case, and its gradient and solubility between blood and the air bubbles would have resulted in a marked increase in the size of the latter.[24] After an embolic episode one is never sure when the circulation has been cleared of air, and nitrous oxide should therefore not be used again.

Air bubbles in cerebral or spinal arteries produce an immediate transient block. After minutes the air moves on, leaving arterial spasm, followed by dilatation and stasis.[25] The localization of the embolus and the extent of ischaemia determine the degree of neurological deficit, the brain being far more vulnerable than the spinal cord,[21] and there is also selective vulnerability of some areas of the brain and spinal cord.[26] Although a hypotensive anaesthetic technique was not used in this case there was a period of less than 5 minutes during which the systolic pressure dropped to 8 kPa (60 mmHg), when ischaemic damage to the spinal cord could have occurred. However, in view of the recovery of normal cerebral function it seems most unlikely that this was the case; furthermore, in the sitting position the arterial pressure at the C6 level would have been higher than at cranial level. We cannot implicate the posterior fossa surgery either because of the level of the spinal lesion.

In conclusion, passage of air into the arterial circulation, although a rare extension of venous air embolism, may cause catastrophic neurological damage and should be added reason for particular vigilance during procedures which put patients at risk of this complication.

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REFERENCES

1. Hunter AR. Air embolism in the sitting position. *Anaesthesia* 1962; **17**: 467-472.
2. Michenfelder JD, Martin JT, Altenburg BM, Rehder K. Air embolism during neurosurgery. *JAMA* 1969; **208**: 1353-1358.
3. Michenfelder JD, Miller RH, Gronert GA. Evaluation of an ultrasonic device (Doppler) for the diagnosis of venous air embolism. *Anesthesiology* 1972; **36**: 164-167.
4. Ericsson JA, Gottlieb JD, Sweet RB. Closed-chest cardiac massage in the treatment of venous air embolism. *New Engl J Med* 1964; **270**: 1353-1354.
5. Artu AA, Cucchiara RF, Messick JM. Cardiorespiratory and cranial nerve sequelae of surgical procedures involving the posterior fossa. *Anesthesiology* 1980; **52**: 83-86.
6. Mc Comish PB, Bodley PO. *Anaesthesia for Neurological Surgery*. London: Lloyd-Luke, 1971: 237-239.
7. Durant TM, Long J, Oppenheimer MJ. Pulmonary (venous) air embolism. *Am Heart J* 1947; **33**: 269-281.
8. Whitby JD. Electrocardiography during posterior fossa operations. *Br J Anaesth* 1963; **35**: 624-630.
9. Lewis JM, Rees GAD. Electrocardiography during posterior fossa operations. *Br J Anaesth* 1964; **36**: 63-64.
10. Mc Comish PB, Thompson DEA. Respiratory disturbances in air embolism. *Anaesthesia* 1968; **23**: 259-263.
11. Smith C. An endo-oesophageal stethoscope. *Anesthesiology* 1954; **15**: 566.
12. Marshall BM. Air embolus in neurosurgical anaesthesia, its diagnosis and treatment. *Can Anaesth Soc J* 1965; **12**: 255-261.
13. Maroon JC, Goodman JM, Horner TG, Campbell RL. Detection of minute air emboli with ultrasound. *Surg Gynecol Obstet* 1968; **127**: 1236-1238.
14. Edmonds-Seal J, Maroon JC. Air embolism diagnosed with ultrasound. *Anaesthesia* 1969; **24**: 438-440.
15. Munson ES, Paul WL, Perry JC, De Padua CB, Rhoton AL. Early detection of venous air embolism using a Swan-Ganz catheter. *Anesthesiology* 1975; **42**: 223-226.
16. Marshall WK, Bedford RF. Use of a pulmonary-artery catheter for detection and treatment of venous air embolism. *Anesthesiology* 1980; **53**: 131-134.
17. Willis J, Duncan C. Gottschalk S. Paraplegia due to peripheral venous air embolus in a neonate: a case report. *Pediatrics* 1981; **67**: 472-473.
18. Emerson LV, Hempleman HV, Lentle RG. The passage of gaseous emboli through the pulmonary circulation. *Respir Physiol* 1967; **3**: 213-219.
19. Butler BD, Hills BA. The lung as a filter for microbubbles. *J Appl Physiol* 1979; **47**: 537-543.
20. Vourc'h G. Paradoxical air embolism. *New Engl J Med* 1971; **285**: 56.
21. Menkin M, Schwartzman RJ. Cerebral air embolism. *Arch Neurol* 1977; **34**: 168-170.
22. Baskin SE, Wozniak RF. Hyperbaric oxygenation in the treatment of hemodialysis-associated air embolism. *New Engl J Med* 1975; **293**: 184-185.
23. Churchill-Davison HC. *A Practice of Anaesthesia*. 4th ed. London: Lloyd-Luke, 1978: 128-130.
24. Bethune DW. Air embolism during anaesthesia. *Anaesthesia* 1977; **32**: 669-670.
25. Hallenbeck JM, Bove AA, Elliot DH. Mechanisms underlying spinal cord damage in decompression sickness. *Neurology* 1975; **25**: 308-316.
26. Dommisse GF. The vasuclar system of the human spinal cord (M.D. thesis). Pretoria: University of Pretoria, 1973: 17-19.