

Letter

Arterial air embolism

SIR.—Arterial air emboli produce multifocal neurological dysfunctions mainly in the brain, owing to transient occlusion of the major cerebral arteries.¹ We report an unusual cause of Brown-Sequard syndrome caused by air emboli.

A 55-year-old man with chronic bronchitis underwent thoracotomy for right anterior pleural lesion. No hypotensive episodes happened during surgery; surgical manoeuvres did not affect arteries concerned with spinal cord circulation. On awakening, the patient complained of right leg weakness and complete blindness. Seizures were never observed. The ocular fundi appeared normal. The neurological examination revealed a normal mental state, bilateral amaurosis with equal pupils showing normal symmetric responses to light. There was a paralysis of the right leg with hyperreflexia, spasticity and up-going toes. The position sense of the right leg was impaired. There was hypesthesia for thermo-algic modalities below a level at T₆ on the left. An EEG was normal. Lumbar puncture showed a protein content of 195 mg/100 ml, a glucose level of 100 mg/100 ml, seven white cells and no red cells per mm³. Eight hours later the patient's condition had not improved and hyperbaric therapy was begun consisting of compression for two hours with three atmospheres of pure oxygen. During compression, the patient presented several grand-mal seizures. One hour after therapy, he was able to distinguish shadows. Twenty hours later, his vision was completely restored. The spinal deficit gradually recovered in two weeks.

Involvement of the spinal cord, manifested by paraplegia occurs frequently in decompressive sickness. Nitrogen is absorbed in the scuba-diver's tissues to a concentration depending on the partial nitrogen pressure in the inspired air. When the diver ascends too quickly, a fall in ambient pressure leads to the formation of gas bubbles. These bubbles can directly damage the tissues and disturb the flow in the epidural vertebral venous system. As they enter the large veins, they may obstruct the radicular veins draining the spinal cord, producing venous spinal infarction.² Hyperbaric re-

compression allows the nitrogen bubbles to redissolve. The treatment's efficacy in this situation is well known.

Arterial air embolism is a different entity which can occur in various situations.^{3,4} In these clinical settings, spinal cord damage is unusual in contradistinction to decompressive sickness.⁵ However, if an anterior cord syndrome can be expected following a vascular accident, even by air emboli when the anterior spinal artery has been occluded, the Brown-Sequard syndrome exhibited by our patient is more surprising. We believe this is the first reported case of Brown-Sequard syndrome due to air emboli. We postulate a transient block of a thoracic subcommissural artery which could account for the ischemia of one-half of the spinal cord.

On the other hand, bilateral amaurosis is known to be the most common ocular finding in air embolism. The loss of vision results from involvement of both retineae or the visual cortex of both occipital lobes.⁶ Fundoscopic examination in our patient was negative and failed to show the typical bubbles with their convex meniscus at either end in the retinal arteries.⁷ Moreover, reaction to light was present. Therefore the blindness was attributed to transient occlusion of the posterior circulation leading to ischaemia in both visual cortices.

Often in arterial air embolism, large amounts of air suddenly rush into the right heart and the pulmonary vessels producing acute right heart failure and air passage into the left circulation with secondary cerebral hypoxia producing syncope and seizures. Here again, as in decompressive sickness, high pressure (six atmospheres) hyperbaric therapy is mandatory.⁸ It allows the size of the air bubbles to diminish to one-sixth of their original volume, thereby reducing the obstructive phenomenon. But when multifocal neurological deficits are the only signs of air embolisation without any features of acute heart failure, one can argue that there is no rationale to embark upon sophisticated treatment to reverse ischaemic lesions. It is questionable whether air bubbles responsible for these lesions are still obstructing the vascula lumen or whether they have dissipated after a few minutes,¹ long before the treatment is initiated. Nevertheless, cases of recovery, days after gas embolism, when treated with hyperbaric therapy of six atmospheres of air have

been reported. In experiments involving baboons, hyperbaric therapy at six atmospheres for 50 minutes has been the preferred treatment.⁹ Although we only applied three atmospheres of pure oxygen to our patient because of technical limitations, he exhibited a favourable evolution. Whether or not this was due to the treatment remains open to doubt.

References

- 1 Menkin M, Schwartzman RJ. Cerebral air embolism. Report of five cases and review of the literature. *Arch Neurol* 1977; **34**:168-70.
- 2 Hallenbeck JM, Bove AA, Elliott DH *et al*. Mechanisms of spinal cord injury in decompression sickness. *Neurology (Minneapolis)* 1975; **25**:308-16.
- 3 Winter P, Alvis HJ, Gage AA. Hyperbaric treatment of cerebral air embolism during cardiopulmonary bypass. *JAMA* 1971; **215**:1786-8.
- 4 Baskin SE, Wozniak RF. Air embolism occurring during hemodialysis. *N Engl J Med* 1975; **293**:184-5.
- 5 Garland H, Greenberg J, Harrington GF. Infarction of the spinal cord. *Brain* 1966; **89**:645-62.
- 6 Hermans G, Szyper C. Cécité optique par embolie gazeuse. Effet de l'oxygénothérapie hyperbare. *Bull Soc Belg Ophtal* 1971; **159**:604-2.
- 7 Walsh FB, Hoyt WF. *Clinical neuro-ophthalmology*. Baltimore: Williams and Williams Co, 1969; Vol 3, 2448-52.
- 8 Takita H, Olszewski W, Shimert G, Lonphier EH. Hyperbaric treatment of cerebral air embolism as result of open-heart surgery. Report of a case. *J Thorac Cardiovasc Surg* 1968; **55**:682-5.
- 9 Gosset A, Cambarelli-Dubois D, Riche D, Fructus X, Naquet P. Tentatives thérapeutiques par l'hyperbarie de l'embolie gazeuse carotidienne chez le babouin. *Rev Neurol* 1969; **121**:531-48.

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