Iatrogenic arterial gas embolism in Australia – a demographic perspective
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Arterial gas embolism (AGE) is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation. Air may enter the arteries directly (arterial air embolism (AAE)), or may enter via the venous system (venous air embolism (VAE)), passing into the arterial circulation through right-to-left shunts, or by overwhelming the filtering capacity of the pulmonary circulation. Bubbles passing through the cerebral circulation cause endothelial damage, with resulting cerebral oedema due to capillary leak, and multiple areas of local ischaemia due to arterial occlusion.1 There have recently been a number of articles on the topic of gas embolism in the medical literature and to those of us working in the field of Hyperbaric Medicine, it is surprising that there is still little understanding of the condition in the general medical community, in particular the role of hyperbaric oxygen therapy (HBOT).2,3 This presentation will concentrate on AGE resulting from iatrogenic causes, and attempt to define the demography of iatrogenic AGE in Australia, based on data collected at the major hyperbaric medicine units over the past 10 years.

A total of 39 cases of iatrogenic air embolism were seen and treated in Australian hyperbaric medicine units in the last 10 years. In approximately half of these the arterial gas embolism occurred during cardiac surgery. Most were diagnosed post-operatively after a relatively uneventful bypass procedure, when the patients manifested neurological signs, most commonly hemiparesis or focal seizure activity on awakening. Some were treated on the basis of a witnessed event in which a large amount of air was seen to pass into the circulation during the operation. The remaining cases had a varied aetiology, most commonly radiological procedures (lung biopsy, coronary or cerebral angiography), laparoscopic surgery, hysteroscopy, and air entrainment into venous access lines. There was one case of massive helium embolism following direct inhalation of helium from a gas cylinder at a party, and one blast injury.

The most common treatment regime for AGE in Australia is the Royal Navy Table 62, with extensions as required, followed by daily oxygen treatments at 2.8 or 2.4 ATA as needed. Lignocaine was used as an adjunct to HBOT in 66% of cases. The time delay between injury and treatment with hyperbaric oxygen ranged from one hour to several days. Sixty-two per cent were treated within six hours, with two-thirds of these patients gaining a complete recovery. Overall, 60% of patients had a complete recovery to normal neurological function, and a further 15% were significantly improved, regardless of delay to treatment.

Benefits of HBOT for treatment of gas embolism include elimination of gas by establishment of a diffusion gradient, reduction in bubble size as a direct result of increased ambient pressure, prevention and treatment of cerebral oedema, and reduced leukocyte adherence to damaged endothelium.4 Immediate recompression produces the best response, but there is growing evidence that delayed HBOT is still beneficial, especially in AAE.5

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References

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