

References

- 1 *BSAC diving incident report archive 1980 to 1999*. Accessed 15 October 2013. Available from <http://www.bsac.com/page.asp?section=2619§ionTitle=Diving+Incident+Report+Archive>
- 2 *BSAC annual diving incident report 2000 to 2012*. Accessed 15 October 2013. Available from: <http://www.bsac.com/page.asp?section=1038>
- 3 Wilson CM. British Sub-Aqua Club (BSAC) diving incident report 2007. *Diving Hyperb Med*. 2008;38:165-6.
- 4 Wilson CM. British Sub-Aqua Club (BSAC) diving incident report 2011. *Diving Hyperb Med*. 2012;42:234-5.
- 5 Lippmann J, Walker D, Lawrence CL, Wodack T, Fock A, Jamieson S, et al. Provisional report on diving-related fatalities in Australian waters 2008. *Diving Hyperb Med*. 2012;43:16-34.

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Recreational diving, accidents, diving deaths, abstracts, case reports

Letters to the Editor

Despite animal studies, HBOT is the treatment of choice for cerebral gas embolism

Following our case report on retrograde cerebral venous gas embolism (CVGE),¹ we wish to draw the attention of readers of this journal to a paper in *Critical Care Medicine* by Weenink et al reporting that severe cerebral arterial gas embolism (CAGE) is a lethal injury in a swine model, not salvageable with hyperbaric oxygen.² This we already know from managing some very unfortunate patients.

Weenink and co-workers have done important research into CAGE, hyperbaric oxygen treatment (HBOT) and animal models.^{2,3} Unfortunately, the less well informed reader may find their title: "*Hyperbaric oxygen does not improve cerebral function when started 2 or 4 hours after cerebral arterial gas embolism in swine*" enough to abandon the idea of HBOT.² The more astute reader, we hope, will concur with us that this study has many shortcomings: the injuries inflicted were excessive, only one HBOT session was administered, no clinical follow up was possible, no imaging was done, the inflammatory process was not quantified, and no histology was undertaken.²

Administering smaller quantities of air and then correlating the effect with imaging to compare such moderately

severe cases of CAGE with retrograde cerebral venous gas embolism (CVGE) of similar quantity will be extremely useful.^{4,5} Comparing the duration of gas present in the cerebral arterial versus cerebral venous system will shed some important light on this newly recognised phenomenon of CVGE. Comparing imaging, outcomes, response to HBOT and histology will also be important.

Their assertion that clinical outcome variables are of vital importance is correct, but we disagree that studying animal models is the only way forward. With the human brain being such a complex structure with complex functions, we do not think outcomes can be accurately assessed in an animal model. With the recognition of CVGE as a separate entity we now have to look at this 'new' category, and need the co-operation of all clinicians dealing with air embolism to report their cases to our journals and also to the air embolism registry that has been established recently in the United Kingdom (<http://www.gasembolism.org.uk>).

We will be collecting extensive, anonymous data from all cases with CVGE and CAGE notified to the registry. Hopefully we may be able to see if patients with CVGE may 'get away' without HBOT in the recommended 6–7 hour time frame, which is often a difficult target if a hyperbaric unit is not available on site. Assessing the possibility of fewer HBOT sessions or any benefit at all for cases with CVGE will be important.^{1,6}

References

- 1 Bothma PA, Brodbeck AE, Smith BA. Cerebral venous air embolism treated with hyperbaric oxygen: a case report. *Diving Hyperb Med*. 2012;42:101-3.
- 2 Weenink RP, Hollmann MW, Vrijdag XC, Van Lienden KP, De Boo DW, Stevens MF, et al. Hyperbaric oxygen does not improve cerebral function when started 2 or 4 hours after cerebral arterial gas embolism in swine. *Crit Care Med*. 2013;41:1719-27.
- 3 Weenink RP, Hollmann MW, van Hulst RA. Animal models of cerebral arterial gas embolism. *J Neurosci Methods*. 2012;205:233-45.
- 4 Schlimp CJ, Loimer T, Rieger M, Lederer W, Schmidts MB. The potential of venous air embolism ascending retrograde to the brain. *J Forensic Sci*. 2005;50:906-9.
- 5 Schlimp CJ, Lederer W. Factors facilitating retrograde cerebral venous air embolism - correspondence. *J Child Neurology*. 2008;23:973.
- 6 Lai D, Jovin TG, Jadhav AP. Cortical vein air emboli with gyriform infarcts. *JAMA Neurol*. 2013;70:939-40.

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