circumstances of a critically ill patient seeking advice concerning a life saving procedure and those in which a person is contemplating taking up a new recreational activity. A court is more likely to sympathize with a position of a medical adviser seeking to assist a critically ill patient, where time is of the essence, than with a doctor consulted by a prospective diver.

It is largely for these reasons that we have formed the view that a doctor performing an assessment of fitness to dive ought to inform the candidate about PFO, the implications the latent condition has for divers and the technique available for its diagnosis and the risks associated with it. In so advising the patient the hyperbaric doctor greatly increases the probability that the obligation imposed upon him by the law will be discharged.

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THE PATHOLOGY OF AIR EMBOLISM OF THE BRAIN IN DIVERS

Des Gorman and Stephen Helps

Introduction

The conventional pathophysiological model of air embolism of the brain circulation does not fit either animal or human data well. A revised model is proposed, based on bubbles precipitating deleterious effects in blood vessels and in blood constituents.

Aetiology

Bubbles can enter the brain arteries of divers either after pulmonary barotrauma or in decompression sickness (DCS).[1-6]

Pulmonary barotrauma is largely seen in novice and trainee divers[7] and occurs in 1:2,500 free/buoyant-ascents performed by submariners in training.[8,9] The latter occurs despite these candidates having a normal chest X-ray and a spirometric ratio of FEV_1 to FVC of greater than 75%.

The lung vessels act as a filter for venous bubbles in DCS,[10,11] but bubbles can overload this mechanism and can also by-pass it via shunts such as a patent foramen ovale.[4-6] Arterial gas embolism (AGE) may underlie much of the brain damage in DCS.[1]

The incidence of AGE of the brain in Australasian divers and trainees is unknown.

Bubble distribution

Bubbles distribute in large vessels in accordance with blood flow and their buoyancy relative to blood, and in small vessels with flow alone.[12-15] In divers this distribution and the invariable upright posture on ascent explains the preponderance of brain involvement.[8,9] Bubbles entering one carotid system tend to distribute ipsilaterally and the middle cerebral artery is primarily affected.[8,9,16]

These bubbles usually do not become trapped and pass through the arteries, arterioles and capillaries to the veins;[13,14,17] to be collected in jugular vein air traps introduced into experimental animals.[15,17-20] This passage of bubbles is promoted by the relatively large calibre of the venous end of capillaries, the hypertension and vasodilatation that follow embolism of the brain-stem vasomotor centres and the local vasodilatory response to bubbles.[16,17]

Indeed, bubbles will only become trapped when they are large enough to occupy several generations of branching arterioles such that net surface tension pressure exceeds cerebral perfusion pressure.[13,14,17] The vessels at the junction of the grey and white matter may be predisposed to such trapping.[21]

Effects of bubble trapping

Very large bubbles or bubbles in a hypotensive diver may be trapped to block flow in a region of the brain; the degree of ischaemia and the development of an infarct is dependent upon the adequacy of the collateral circulation.[2,18,19,21-24] Most of these larger bubbles will however only be trapped temporarily and will eventually be dis-

placed by blood that advances progressively with cardiac systole.[13,14,16,17] Such interruptions of flow are poorly tolerated and even after flow is restored brain function may remain suppressed.[25] Also, if redistribution of bubbles does not occur within 20 minutes of embolism, vessels may collapse so that subsequent reperfusion will require extremely high pressures.[17]

Effects of bubble transit

Fortunately most bubbles pass through the brain circulation and do not become trapped.[13,14,15,17,20] During bubble passage local brain function is lost, but returns as bubbles clear.[26-30] These mobile bubbles damage endothelial cells,[31-33] perhaps by stripping them of surfactants, cause a persistent vasodilatation,[16,25] which may itself be a result of the endothelial damage, but is not indicative of vasoparalysis,[13] and activate platelets and leucocytes both to aggregate and adhere to vessel walls.[26-29,34-37]

The accumulation of leucocytes is rheologically important[34] and making animals leucocytopenic, but with normal red blood cell and platelets, prevents the typical decline in cerebral blood flow seen after AGE of the brain.[38] This endothelial damage will also potentiate leucocyte adherence and probably underlies the immediate but temporary (several hours) increase in extravasation of fluid across the blood-brain-barrier into the brain interstitium.[23,32,39-42]

The flow of this increasingly viscous blood[43] through these damaged vessels consequently decreases even though bubbles may no longer be present,[16,25] and may eventually become inadequate for neuronal function.[26,29,35,44] This can explain the typical history of AGE in divers; sudden loss of brain function, early recovery and perhaps a deterioration or relapse within several hours.[30] A sudden relapse, particularly if it involves an originally affected area of brain, may also be due to re-embolism.[9]

AGE and DCS

The pathological consequences of AGE will also depend upon whether the diver has DCS or significantly increased tissue insert gas tensions.[45] DCS may even be precipitated by AGE (Type III DCS).[45] The outcome after AGE of the brain consequently worsens with increasing dive duration/depth.[46]

Summary

Arterial gas embolism of the brain is not a simple mechanical occlusive event. Much of the brain dysfunction/damage is due to the effects that bubbles have on blood vessels and on the blood itself.

References

1 Adkisson GH, Macleod MA, Hodgson M et al. Cerebral perfusion deficits in dysbaric illness. *Lancet* 1989;2 (8655): 119-121

2 Catron PW, Hallenbeck JM, Flynn ET, Bradley ME and Evans DE. Pathogenesis and treatment of cerebral air embolism and associated disorders. *NMRI Report* 84-20, Bethesda, Maryland: 1984

3 Lynch PR, Brigham M, Tuma R and Wiedeman MP. Origin and time course of gas bubbles following rapid decompression in the hamster. *Undersea Biomed Res* 1985; 12 (2): 105-114

4 Moon RE and Camporesi EM. Right-to-left shunting and decompression sickness. *Undersea Biomed Res* 1988; 15 (Suppl): 18

5 Moon RE, Camporesi EM and Kisslo JA. Patent foramen-ovale and decompression sickness in divers. *Lancet* 1989; 1 (8637): 513-514

6 Wilmshurst PT, Byrne JC and Webb-Peploe MM. Neurological decompression sickness. *Lancet* 1989; 1 (8640): 731

7 Gorman DF, Pearce A and Webb RK. Dysbaric illness treated at the Royal Adelaide Hospital 1987, a factorial analysis. *SPUMS J* 1988; 18 (3): 95-101

8 Brooks GJ, Green RD and Leitch DR. Pulmonary barotrauma in submarine escape trainees and the treatment of cerebral air embolism. *INM Report 13/85.* Alverstoke, U.K.: 1985

9 Gorman DF. Arterial gas embolism as a consequence of pulmonary barotrauma. In: Desola J ed. *Diving and hyperbaric medicine.* Barcelona, Spain: EUBS, 1984: 348-368

10 Butler BD and Hills BA. The lung as a filter for microbubbles. *J Appl Physiol* 1979; 47: 537-543

11 Butler BD and Hills BA. Transpulmonary passage of venous air emboli. J *Appl Physiol* 1985; 59: 543-547

12 Butler BD, Katz J, Leiman BC, Waters RD and Sutton T. Cerebral decompression sickness: bubble distribution in dogs in the Trendelenberg position. *Undersea Biomed Res* 1987; 14 (2) (Suppl): 15

13 Gorman DF and Browning DM. Cerebral vasoreactivity and arterial gas embolism. *Undersea Biomed Res* 1986; 13: 317-336

14 Gorman DF, Browning DM, Parsons DW and Traugott FM. Distribution of arterial gas emboli in the pial circulation. *SPUMS J* 1987; 17 (3): 101-115

15 Van Allen CM, Hrdina LA and Clark J. Air embolism from the pulmonary vein - a clinical and experimental study. *Arch Surg* 1929; 19: 567-599

16 Helps SC, Parsons DW, Reilly PL and Gorman DF. Gas emboli invoke changes in cerebral blood flow, pial arterial diameter, and neural function in rabbits. *Stroke* 1990; 21: 94-99

17 Gorman DF. *The redistribution of cerebral arterial gas emboli.* PhD Thesis in Medicine, Sydney University Library, 1987

18 de la Torre E, Mitchell OC and Netsky MG. The seat of respiratory and cardiovascular responses to cer-

ebral air emboli. *Neurol* 1962; 12: 140-147

19 Fries CC, Levowitz B, Adler S, Cook AW, Karlson KE and Dennis C. Experimental cerebral gas embolism. *Ann Surg* 1957; 145: 461-470

20 Pate JW and Birdsong S. Carotid air embolism. *Arch Surg (Chicago)* 1964; 89: 685

21 Dutka AJ, Kochanek PM, Hallenbeck JM and Storey JR. Air embolism may cause unrecognized ischaemia of the grey-white junction. *Undersea Biomed Res* 1988; 15 (2): 99-106

22 Brierly JB, Brown AW, Meldrum BS and Tiche D. The time course of ischaemic neuronal changes in the primate brain following profound arterial hypertension, air embolism and hypoglycaemia. *Phys Soc* 1980: 59-60

23 Garcia JH, Klatzo I, Archer T and Lossinsky AS. Arterial air embolism: structural effects on the Gerbil brain. *Stroke* 1981; 12: 414-421

24 Kogure K, Busto R, Alonso OF and Samson R. Effects of recompression treatment on cerebral energy metabolism in arterial air embolism of the rat brain. In: Hallenbeck JM, Greenbaum LJ Jr Eds. *Air embolism and acute stroke*. Bethesda, Maryland: UMS, 1977: 105-122

25 Helps SC, Meyer-Witting M, Reilly PL and Gorman DF. Increasing doses of intra-carotid air and CBF in rabbits. *Stroke* 1990; 21: 1340-1345

26 Hallenbeck JM, Leitch DR, Dutka AJ and Greenbaum LJ Jr. The amount of circumscribed brain edema and the degree of post-ischemic neural recovery do not correlate well. *Stroke* 1982; 13: 797-804

27 Hallenbeck JM, Leitch DR, Dutka AJ, Greenbaum LJ Jr and McKee AE. Prostaglandin I2, indomethacin, and heparin promote post-ischemic neuronal recovery in dogs. A*nn Neurol* 1982; 12: 145-156

28 Kochanek PM, Dutka AJ, Kumaroo KK and Hallenbeck JM. Effects of prostacyclin, indomethacin, and heparin on cerebral blood flow and platelet adhesion after multifocal ischaemia of canine brain. *Stroke* 1988; 19: 693-699.

29 Obrenovitch T, Kumaroo K and Hallenbeck JM. Autoradiographic detections of III Indium-labeled platelets in brain tissue sections. *Stroke* 1984; 15: 1049-1056

30 Stonier JC. A study in prechamber treatment of cerebral air embolism patients by a first provider at Santa Catalina Island. *Undersea Biomed Res* 1985; 12 (Suppl): 58

31 Haller C, Sercombe R, Verrecchia C, Fritsch H, Seylaz J and Kuschinsky W. Effect of the muscarinic agonist carbachol on pial arteries in vivo after endothelial damage by air embolism. *J Cereb Blood Flow Metab* 1987; 7: 606-611

32 Persson LI, Johansson BB and Hansson HA. Ultrastructural studies on blood-brain barrier dysfunction after cerebral air embolism in the rat. *Acta Neuropathol (Berl)* 1978; 44: 53-56

33 Warren BA, Phillip RB and Inwood MJ. The ultrastructural morphology of air embolism: platelet adhesion to the interface and endothelial damage. *Br J Exp Pathol* 1973; 54: 163-172

34 Dutka AJ, Kochanek PM and Hallenbeck JM. Influence of granulocytopenia on canine cerebral ischaemia induced by air embolism. *Stroke* 1989; 20: 390-395

35 Hallenbeck JM,Dutka AJ,Tanishima T. Polymorphonuclear leucocyte accumulation in brain regions with low blood flow during the early postischaemic period. *Stroke* 1986; 17 (2): 246-253

36 Hallenbeck JM, Furlow TW Jr, Ruel TA and Greenbaum LJ Jr. Extra-corporeal glass-wool filtration of whole blood enhances post-ischemic recovery of the cortical sensory evoked response. *Stroke* 1979; 10: 158-164

37 La Celle PL. Alterations by leucocytes of erythrocyte flow in microchannels. *Blood Cells* 1986; 12: 179-189

38 Helps SC and Gorman DF. The effect of air emboli on brain blood flow and function in leucocytopenic rabbits. *Undersea Biomed Res* 1990; 17 (Suppl): 71-72

39 Ah-See AK. Permeability of the blood-brain barrier to FITC labeled dextran in massive cerebral air embolism. In: Hallenbeck JM, Greenbaum LJ Jr Eds. *Air embolism and acute stroke*. Bethesda, Maryland: UMS, 1977: 43-48

40 Hekmatpanah J. Cerebral micro-vascular alterations in arterial air embolism. *Adv Neurol* 1978; 20: 245-253

41 Lee JC. The blood-brain barrier and cerebral air embolism. In: Arfel G, Naquet R, eds. *Colloque en L'embolic gazeuse du systeme carotidien*. Dion, Paris, 1974: 158-164

42 Nishimoto K, Wolman M, Spatz M and Klatzo I. Pathophysiologic correlations in the blood-brain barrier damage due to air embolism. *Adv Neurol* 1978; 20: 237-244

43 Whittaker SRF and Winton FR. The apparent viscosity of blood flowing in the isolated hind limb of the dog, and its variation with corpuscular concentration. *J Physiol (Lond)* 1933; 78: 338

44 Dutka AJ, Hallenbeck JM and Kochanek P. A brief episode of severe arterial hypertension induces delayed deterioration of brain function and worsens blood flow after transient multifocal cerebral ischemia. *Stroke* 1987; 18: 386-395

45 Neuman TS and Bove AA. Severe refractory decompression sickness resulting from combined no-decompression dives and pulmonary barotrauma: type III decompression sickness. In: Bove AA, Bachrach AS and Greenbaum LJ Jr, eds. *Underwater and Hyperbaric Physiology IX*, Bethesda, Maryland: UHMS, 1987: 985-992

46 Brew S, Kenny CTC, Webb RK and Gorman DF. Outcome of 125 divers with dysbaric illness treated by recompression at HMNZS PHILOMEL. *SPUMS*

J 1990; 20: 226-30

Key Words

Air embolism, gas embolism, arterial gas embolism, cerebral arterial gas embolism

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THE BS-AC '88 DECOMPRESSION TABLES

Greg Adkisson

The British Sub-Aqua Club 1988 (BS-AC '88) Decompression Tables were introduced, after numerous delays, into general usage in the latter months of 1988. I was first introduced to them by a patient I was treating for an episode of neurological decompression sickness (DCS). Before I create the wrong impression, I should state that the patient was not using the new tables, but had completed his dive in accordance with the older RNPL/BS-AC tables of 1972.

The dive in question was to 26 m for 31 minutes with appropriate decompression conducted for 5 minutes at both 10 m and 5 m depths. As I monitored this patient's extended RN table 62 treatment, I had a chance to read through the new tables. I was surprised to find that using the new tables, while the definitions were slightly different, a more lenient dive of 27 m for a bottom time of 32 minutes could have been performed and would require only a 1 minute decompression stop at a depth of 6 m. I examined them further and noted that, for the same 10 minutes of decompression, the allowable bottom time would have been 43 minutes. I was fascinated.

Call me old fashioned, call me conservative, but this notable reduction in decompression requirement or, conversely, increase in available diving time, seemed just too good to be true so I went looking for the magic formula upon which these changes were based. I found myself, within a very short time, embroiled in controversy with the BS-AC. I had the pleasure of several lengthy discussions with the author of the tables, Dr Tom Hennessey, but I find myself still in search of the magic formula and remain as firmly against their general use now as I have been since the first day I read them. I have been asked here to comment on the format, design, algorithms and testing of these tables.

The BS-AC gives four basic reasons for introducing new tables in their BS-AC '88 Decompression Tables Instructor's notes. Since the introduction of the RNPL/BS-AC tables, "the pattern of diving has changed, the possibilities available to sports divers have developed, much experience has been gained in the use of the tables and understanding of decompression has improved". To quote *DIVER*, the magazine of the British Sub-Aqua Club, August 1988, "it has become increasingly clear that the RNPL/BS-AC table is insufficiently flexible for the patterns of diving required by divers today".

It is my personal opinion that, despite other objectives, the single most important factor in the introduction of a new set of tables is that they must not increase the risk of DCS and other accidents in the general diving population. The introduction of a new set of diving tables is no easy task and for acceptance in the commercial diving world one of two conditions must apply. The tables must be more conservative than previous versions or must have undergone extensive testing and evaluation prior to their use. While it is laudable that the BS-AC would desire to give its members greater flexibility in their diving, it must also accept the responsibility that any new tables it introduces should be safe and well tested.

Testing and evaluation of the BS-AC '88 tables

To my knowledge, the BS-AC '88 tables have never been tested in any type of controlled situation. Dr. Hennessey maintains that the BS-AC '88 tables are more conservative and provide a "greater margin of safety than the classic military-based tables". This is despite significant reductions of in-water decompression requirements and with an emphasis placed on decompression stop and repetitive diving. When asked about the lack of testing, Dr. Hennessey has argued that actual in-water testing would be impossible to do across the range of the tables. He says that limited testing might be conducted, but would not be statistically valid, and relies on unproven theoretical considerations to claim that the tables are more conservative than their predecessors and do not, therefore, require testing.

Comparisons of BS-AC '88 and established tables

Admittedly, it is difficult to do straight across comparisons of diving tables. The wide variation of designs makes exact comparisons impossible but I believe that