Lidocaine in the treatment of decompression illness: a review of the literature

S J Mitchell

Key words
Lidocaine, lignocaine, decompression illness, arterial gas embolism, neuro-protection, recompression

Abstract

While recompression and hyperbaric oxygen administration remain the mainstays of treatment for decompression illness (DCI), drugs that might improve outcomes or prove beneficial in first aid management have been sought. There has been much interest in lidocaine, a sodium channel-blocking agent used clinically as an antiarrhythmic and local anesthetic. The relevant literature is reviewed. Lidocaine is neuro-protective in cerebral arterial gas embolism (CAGE) in vivo, and in a variety of in vivo and in vitro models of ischemic brain injury. There has been limited in vivo investigation of efficacy in DCI where bubbles have formed from dissolved nitrogen. Mechanisms of neuro-protection by lidocaine include deceleration of ischemic ion fluxes across the neuronal cell membrane and prevention of the consequent neurotoxic events. In addition, lidocaine lowers neuronal metabolism, exerts advantageous effects on cerebral hemo-dynamics, and is a potent anti-inflammatory. There is one randomized double blind study that demonstrates improved neuropsychological outcomes in cardiac surgery patients receiving lidocaine. Clinical evidence of efficacy in DCI is limited to anecdotal reports. Expeditious administration of lidocaine is justified in cases of unequivocal CAGE. Speculative use may be justified in severe neurologic DCI after patient counseling and consent.

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A summary of this paper was presented by Dr Mitchell at the SPUMS ASM, 2001, Madang, PNG.

Editor's comment: Dr Mitchell reviewed the literature on lignocaine in the treatment of decompression illness (DCI) for his SPUMS Diploma thesis seven years ago.[1] Since then, our understanding of the neuro-protective actions of lignocaine has progressed, not least as a result of Mitchell's own work; the randomised study mentioned in the abstract.[2] The present, comprehensive review, for which there is not space here to republish in full, brings up to date the body of knowledge underlying his final conclusions. The Slark Hyperbaric Unit, Auckland, commenced a randomised study on lignocaine in DCI some years ago, which has not been completed to date.[3] Likewise, James Francis has described how setting up an international multi-centre trial proved impossible.[4] The role of lignocaine in diving DCI, therefore, is not proven clinically, and nothing is known of its potential in altitude DCI, eg. in extra-vehicular space activities.

References

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Treatment of multiple sclerosis with hyperbaric oxygen therapy

M Bennett and R Heard

Key words
Multiple sclerosis, hyperbaric oxygen, treatment

Absract

Despite considerable research effort, there is little controlled evidence that a course of hyperbaric oxygen therapy (HBO_2T) results in any benefit for patients with multiple sclerosis (MS). The great majority of randomized trials involved investigating a course of 20 treatments at pressures between 1.75 and 2.5 atm abs daily for 60–120 min over 4 wk against a placebo regimen. None has tested the efficacy of HBO_2T against alternative current best practice. A systematic review of this randomized evidence suggests there is no significant benefit from the administration of HBO_2T (Improved EDSS after HBO_2T: OR = 2.02, 95% CI 0.63–6.43. Improved sphincter function: OR = 1.3, 95% CI 0.8–2.11). On average, 42 patients would need to be treated before we could expect one individual to benefit with an improved disability status score; however, we cannot be confident that the number we would need to treat is less than infinite (NNT = 42, 95% CI 15 to infinity). There is some case for further investigation of possible therapeutic effects in selected sub-groups of patients and for the response to prolonged courses of HBO_2T at more modest pressures; however, the case is not strong. At this time, we cannot recommend the routine treatment of MS with HBO_2T.

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Editor's comment: Michael Bennett has created a very useful database of randomised trials of hyperbaric oxygen, both in animal research and clinical practice. This is regularly updated. Each reference is followed by a critical review in an evidence base style .[1] This may be found on the UHMS web site <http://www.uhms.org> or is available at <http://www.hboevidence.com/>. Members are recommended to read Bennett's earlier SPUMS Journal article on evidence based journal reading.[1]

Reference

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Effects of water immersion on pulmonary function in asthmatics

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Key words
Diving, asthma, pulmonary function, exercise, closing volume, pulmonary barotrauma

Abstract

Immersion induces air trapping in the lungs, as does asthma. Consequently, when using diving apparatus, asthmatics may face greater risk than non-asthmatics of pulmonary barotrauma (PBT) during ascent. We studied the pulmonary airflows and closing capacities (CC = closing volume + residual volume) in subjects with exercise-induced asthma (A, n = 12) and in healthy controls (C, n = 11) under four conditions: dry and immersed, both before and after exercise (treadmill running, non-immersed). Immersed, both C and A had significant and equivalent reductions in vital capacity, FEV_1, FEV_1/FVC, and $FEF_{25\%-75\%}$. Post-exercise and immersed, pulmonary airflows deteriorated further in A but were better in C: FEV_1 (A, 3.6 ± 0.8 liter vs. 3.3 ± 0.8 liter, p = 0.001; C, 3.9 ± 0.5 liter vs. 4.1 ± 0.6 liter, p = 0.006), $FEF_{25\%-75\%}$ (A, 3.5 ± 1.0 liter · s^{-1} vs. 3.0 ± 0.8 liter · s^{-1}. p < 0.05; C, 4.0 ± 0.9 liter · s^{-1} vs. 4.3 ± 0.9 liter · s^{-1}, p <0.05). Therefore, in contrast to C, A subjects had reduced pulmonary airflows during immersion after exercise. Furthermore, A subjects more often had no closing volume phase IV when immersed after exercise than C (p = 0.005). Interpreting the absence of phase IV as indicative of more air trapping in the asthmatics during immersion after exercise would be consistent with the reductions in airflow.

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Commentary on Leddy et al by Paul Thomas

This interesting study tries to tease out what happens to exercise-induced asthmatic subjects during immersion, and compares the results with the pre-exercise values, and with normal control subjects. After immersion, they demonstrate a reduction in all lung volumes, including FEV_1 in both groups. After exercise, there is a fall in FEV_1 in the asthmatic group, which is part of the inclusion criteria for participation. A complex technique is used for estimating the point during expiration at which many airways close, but the data are only interpretable in half of the asthmatic subjects, thus this measurement did not show any significant changes. The authors had postulated that this measurement could be used to show that premature airway closure might occur during immersion, and that this would be more pronounced in asthma.

The study confirms that lung volumes are reduced by immersion as has been documented previously.[1] Immersion therefore reduces lung volume, which in turn allows airways to narrow and collapse in both subject groups. They are unable to demonstrate whether this leads to gas trapping, but speculate that this might be the case. What we need is a better method of measuring gas trapping in these asthmatic divers, if it occurs.

In subjects with mild obstruction secondary to smoking, when measuring gas volumes before and after hyperbaric oxygen therapy, we were unable to show a difference by body plethysmography.[2] The inaccessibility of the water medium not only means that diving is for the determined, but that physiologists find it troublesome to perform their measurements too. Given the 'head-out' immersion, the findings of this study are applicable to swimming at the surface. Should all asthmatics be advised not to swim? Not from these data!

References

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