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.

Reprinted with kind permission from *Undersea Hyperb Med* 2001; 28: 165-174

S Mitchell was based at the Wesley Centre for Hyperbaric Medicine, Brisbane, Australia at the time of writing.
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

1 Mitchell SJ. The role of lignocaine in the treatment of decompression illness: a review of the literature. *SPUMS J* 1995; 25: 182-194
2 Mitchell SJ. *Prevention of brain injury in cardiac surgery*. Thesis for PhD in Medicine. Auckland: University of Auckland, 2000
3 Gorman D, Drewry A, Mitchell S. A progress report on diving medicine studies in the RNZN. *SPUMS J* 1994; 24: 161-163
4 Francis J. A randomised prospective trial of lignocaine in the management of acute neurological decompression illness - an update. *SPUMS J* 2002; 32: 97-105

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.

Reprinted with kind permission from *Undersea Hyperb Med* 2001; 28:117-122