1DF, 0.83; P>0.75. 95% Confidence Limits of difference 28% fewer incomplete resolutions in <12 hour group to as much as 9.2% fewer in the >12 hour group). It should be stressed that the second analysis is on somewhat rocky statistical ground in view of the small numbers involved in one of the groups and there is a reasonable case for use of a more conservative approach using conditional probabilities, such as Fisher's exact test. These calculations were the basis of my statement concerning the lack of statistical support for improved outcome with early retrieval and treatment.

I wholeheartedly agree that the presence of many of the more acutely unwell patients in the shorter delay to treatment groups is a substantial bias against finding a positive correlation between shorter times to treatment and improved outcome. It was my intention to make this point in the final paragraph of the discussion in my paper and I thank Dr. Davies for making this clear if I did not do so.

Finally, I wonder if we know sufficiently well the natural history of relatively minor grades of DCI to establish just how good a 75% resolution rate is in these patients?

Mike Bennett

Key Words

Decompression illness, letter, transport, treatment sequelae.

LIGNOCAINE AND DCI

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27/4/96

Dear Editor

I read with great interest Dr Simon Mitchell's informative review of lignocaine use in decompression illness (DCI). As well as the potential protective mechanisms described, I wonder whether he has considered the action lignocaine might have on the neuropathic process itself. Damaged nerves tend to produce new sodium channels and lignocaine appears to suppress sodium channel formation. It has been suggested that lignocaine may work in neuropathic pain by preventing this "wind-up" phenomenon.

Could the action of lignocaine in DCI, therefore, be due at least in part to symptomatic suppression of this phenomenon, not only in pain pathways, but also other abnormal sensory changes? To take this a step further, has anyone reported the use of lignocaine in the suppression of chronic pain syndromes that undoubtedly occur in some patients following neurological DCI?

Mike Davis

Key Words

Decompression illness, drugs, treatment.

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16/5/96

Dear Editor

I would like to thank Mike Davis for his constructive questions. In my literature review I restricted discussion of lignocaine's possible protective mechanisms to those which provided rational explanations for the protective phenomena that have been observed, viz: preservation of the somatosensory evoked response (SER) and reduction in infarct size, both following in vivo cerebral arterial gas embolism (CAGE) or cerebral ischaemia. It seems unlikely that the rapidly apparent preservation of the SER, for example, could be accounted for by down-regulation of sodium channel formation in compromised neurones.

Dr Davis begins his second paragraph by suggesting that "in part", the "action of lignocaine in decompression illness (DCI)" may be attributable to down-regulation of sodium channel formation. It is important to emphasise that apart from anecdote, no action by lignocaine in DCI per se has been demonstrated. I am reluctant to speculate on the mechanism of effects that have not yet been reported. However, I agree that a putative analgesic action by lignocaine in DCI patients can be based on reports that this drug is analgesic in both neuropathic pain[1] and postoperative pain.[2] Down-regulation of sodium channels may well contribute to this action. A purely analgesic effect by lignocaine in DCI is probably undesirable as it will merely mask the underlying pathology.

In answer to Dr Davis' final question, I am not aware of any references to the use of lignocaine in chronic pain syndromes following neurological DCI.

Simon Mitchell

References

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Key Words

Decompression illness, drugs, treatment.