GLEANINGS FROM MEDICAL JOURNALS

The following articles have come to the notice of the editorial staff and these notes are printed to bring them to the attention of members of SPUMS. They are listed under various headings of interest to divers. Any reader who comes across an interesting article is requested to forward the reference to the Journal for inclusion in this column.

DECOMPRESSION PROBLEMS

Cerebral perfusion deficits in dysbaric illness
G H Adkisson¹, M A Macleod², M Hodgson¹, J J W Sykes¹, F Smith¹, C Strack¹, Z Torok⁴, R R Pearson¹. Cerebral perfusion deficits in dysbaric illness. Lancet 1989; ii: 119-121.

From Institute of Naval Medicine, Alverstoke, Hants PO12 2DL ¹, Royal Naval Hospital, Haslar, Gosport ², Aberdeen Royal Infirmary, Aberdeen ³, and Admiralty Research Establishment, Alverstoke ⁴.

Summary
Decompression sickness (DCS) is usually categorised as type I (mild; peripheral pain, non-neurological) or type II (serious; neurological). Type II is regarded as predominantly a spinal cord disease with infrequent cerebral involvement. Cerebral perfusion was studied by injection of ⁹⁹ Tc-hexamethylpropyleneamine oxime and single photon emission tomography in 28 divers with confirmed incidents of DCS and cerebral arterial gas embolism (CAGE). Cerebral perfusion deficits were present in all 23 cases of type II DCS and in all 4 cases of CAGE. No deficits were present in the single case of type I DCS. Type II DCS should be recognised as a diffuse, multifocal, central nervous system disease.


(Letter) Wilmshurst P and Nunan T.O. From the Department of Cardiology, St Thomas’ Hospital, London SE1 7EH, England.

(Reply) Adkisson G.H. From the Institute of Naval Medicine, Alverstoke, Hants PO12 2DL.

CARBON MONOXIDE POISONING

Hyperbaric and Normobaric Oxygen in Acute Carbon Monoxide Poisoning

(Letter) James P.B. From Department of Community Medicine, Wolfson Institute of Occupational Health, Ninewells, Dundee DD1 92Y, Scotland.

OXYGEN TOXICITY

Biochemistry of reoxygenation injury.

This paper summarizes current knowledge on the biochemistry of oxygen toxicity in general and ischemia-reoxygenation tissue injury in particular. The superoxide radical, hydrogen peroxide, and the hydroxyl radical in cells can be formed enzymically or non-enzymically. Primary effects of oxygen radicals result in lipid peroxidation, which is believed to be initiated by a perferryl radical. Secondary effects are believed to be due to a disturbance in cellular calcium homeostasis. Reactions and treatment potentials are highly complex and their effects on cells, tissues, and organism are difficult to predict. Treatment potentials include superoxide dismutase, catalase, calcium entry blockers, iron chelators, xanthine oxidase inhibitors, and agents to prevent leukocyte adhesion. Reoxygenation injury mechanisms during resuscitation from clinical death can be studied in animals by evaluating the effects of antireoxygenation injury therapies and by monitoring free radical reactions.

Keywords reoxygenation/oxygen toxicity/hydrogen peroxide.

IMMERSION

The resuscitation of immersion victims.

This paper reviews the outcome of 150 victims of drowning and near-drowning brought to a district general hospital close to the sea. The pathophysiology of the drowning process is reviewed. Patients were treated immediately on arrival by a resuscitation team. Respiratory difficulties were relieved as quickly as possible. Common problems were hypoxia, hypothermia, acidosis and low blood pressure. The apparently dead were assessed very carefully. The results show an excellent prognosis for those patients who had not suffered cardiac arrest before arrival. There were, however, two survivors from the cardiac arrest group. Survival rates in this group will only improve if the hypoxia is relieved before cerebral damage occurs.

Keywords near-drowning/hypoxia/cardiac arrest/death.

SELECTED ABSTRACTS

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