

submarine medicine, volume 3. Department of the Navy Publication, Washington DC, 1966

- 4 Edmonds C. *The Diver. Project 2/72. Royal Australian Navy School of Underwater Medicine Report.* 1972
- 5 Bachrach AJ and Egstrom GH. *Stress and Performance in Diving.* San Pedro, California: Best Publishing, 1987
- 6 Edmonds C. *The Abalone Diver.* Morwell, Victoria: National Safety Council of Australia, 1987
- 7 Morgan WP. Anxiety and panic in recreational scuba divers. *Sports med* 1995; 20 (6): 398-421
- 8 Morgan, WP and Raglin JS. Psychological considerations in the use of breathing apparatus. In *Proceedings of Undersea and Hyperbaric Medical Society Workshop, Physiological and Human Engineering Aspects of Underwater Breathing Apparatus.* Claes Lundgren. Ed. UHMS. 1989
- 9 Raglin JS, O'Conner J, Carlson N and Morgan WP. Response to underwater exercise in scuba divers differing in trait anxiety. *Undersea Hyper Med* 1996; 23 (2), 77-82
- 10 Williamson A, Edmonds C and Clarke B. The neurobehavioural effects of professional abalone diving. *Brit J Ind Med* 1987; 44, 459-466
- 11 Morris PE, Leach J, King J and Rawlings JSP. *Psychological and Neurological Impairment in Professional Divers. P2050 Final Report.* London: Dept of Energy, 1991
- 12 Edmonds C and Hayward L. Intellectual impairment with diving. A review. In *9th International Symposium on Underwater and Hyperbaric Physiology.* Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987
- 13 Edmonds C and Coulton T. Multiple aptitude assessments on abalone divers. In *The Abalone Diver.* Edmonds C. Ed Morwell, Victoria: National Safety Council of Australia, 1986; 149-156
- 14 Williamson A, Clarke B and Edmonds C. The influence of diving variables on perceptual and cognitive functions in professional shallow-water divers. *Environ Research* 1989; 50: 93-102
- 15 Andrews G, Holt P, Edmonds C et al. Does non-clinical decompression stress lead to brain damage in abalone divers? *Med J Aust* 1986; 144: 399-401

LATE SEQUELAE OF CARBON MONOXIDE POISONING 2 CASE REPORTS

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Key Words

Carbon monoxide, hyperbaric oxygen, sequelae, treatment.

Introduction

Neuropsychiatric manifestations of acute carbon monoxide (CO) poisoning may include non-focal changes in mental state, seizures, amnesia, apraxia, agnosia, Parkinsonism, cortical blindness, incontinence and peripheral neuropathy. A lucid period of up to twenty one days may occur followed by the delayed sequelae of CO poisoning which may include aphasia, apathy, disorientation, psychosis, gait disturbances, faecal and urinary incontinence and bradykinesia. Cognitive and neurological deficits may also be present, as can personality changes with impulsiveness, violence, verbal aggressiveness and mood changes.¹

This syndrome has a reported incidence of 3% to 40%,² with a set of risk factors having been identified within the group of affected patients.³ The neuropsychological deficits associated with CO poisoning are highly variable despite exposure to similar levels of CO poisoning.⁴ The white matter of the frontal lobe is involved but the pathological mechanism leading to demyelisation, petechiae, oedema and necrosis is poorly defined. Depressed cardiovascular function induced by CO, and a limited cerebral blood flow, may be major factors leading to neurologic cellular damage from CO poisoning.¹

Case histories

The Hyperbaric Unit at Fremantle Hospital actively treats CO poisoning with about 30 cases per annum being referred from Perth and more remote regions. The unit recently treated two cases with apparent late sequelae with resulting clinical improvement.

CASE ONE

A 61 year old female patient, who attempted suicide by connecting the exhaust pipe of her car to the cabin, was found by her neighbour at about 0950 with the car engine still running. The Glasgow Coma Scale (GCS) at the site was reported to be 9/15. In the Emergency Department of a peripheral hospital the patient was noted to have deteriorated with hypotension (55/28 mm Hg). She had an oxygen

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saturation of 77% on air and an acidosis with a bicarbonate of 12.1 mmol/l (normal 22-32 mmol/l). Her ECG showed sinus tachycardia and carboxyhaemoglobin estimation revealed a level of 41.9% four hours after being removed from the vehicle. Transfer to Fremantle Hospital was accomplished with the patient intubated and ventilated.

In the Intensive Care Unit at Fremantle Hospital the patient was unrousable despite withholding all sedation. Initial treatment was performed using an 18 m for sixty minutes with a thirty five minute decompression (18:60:35) hyperbaric oxygen treatment (HBO₂T) table while the patient remained sedated and ventilated. Sedation was withdrawn overnight and extubation performed the following morning with improved and stable observations. The patient received two further daily treatments using the same treatment table. She was noted to have an improved mental state during and between visits to the chamber. Without sedation the patient responded to commands by eye opening only and was restless and disorientated during the second day. A fourth treatment using the RN 61 table was administered on day four after which it was noted that she was able to conduct a rational conversation and read printed material. The patient was handed over to the care of the medical team who discharged her seven days after admission. Appropriate psychiatric follow up had been arranged.

Eighteen days after her discharge the patient was readmitted with a marked deterioration in psychological, cognitive and neurological function after having been found at the side of the road in the driver's seat of her car and clothed only in a bath towel. Signs and symptoms included urinary and faecal incontinence, inability to perform routine activities of daily living, loss of short term memory, disorientation in time, place and person, ataxic gait, and inability to perform a Mini Mental State Examination (MMSE). Neuropsychological testing was difficult and attempted on two occasions with the latter revealing exceptional weakness in her Verbal Test Score and inability to negotiate Performance tests. Short and long term recall for verbal and non-verbal material was zero and the patient was unable to distinguish between her left and right hand.

The patient remained on a medical ward for four weeks at the end of which she was assessed to be suitable for nursing home care due to an inability to care for her self. A subsequent referral of the patient for hyperbaric treatment resulted in a series of 14 daily hyperbaric treatments using table 14:90:24 being administered with steady recovery almost on a daily basis. The patient regained urinary and faecal continence and independence in activities of daily living (ADLs). Post-treatment neuropsychological testing revealed an ability to cooperate with full scale IQ in the average range. Memory function assessment for verbal material was normal whilst non-verbal was moderately impaired.

The patient was discharged to live independently, although an assessment before hyperbaric treatment indicated permanent placement in hostel accommodation. At follow up interview the patient was functioning at a normal level living independently and requiring only the support of her local general practitioner.

CASE TWO

A 45 year old male had apparently been exposed to 30 minutes of car exhaust gas four days before assessment. Upon failing to follow through on his suicide attempt the patient had consumed beer and Prothiaden (dothiepin hydrochloride) without acute sequelae.

He presented to his General Practitioner with various symptoms and general malaise the day after the incident but was reassured there was nothing seriously wrong. No carboxyhaemoglobin estimation was performed at presentation. His condition deteriorated over several days following the poisoning with poor balance, headache, poor short-term memory and slow mentation noted by his wife. Initial assessment by the hyperbaric team four days later revealed a Sharpened Romberg of less than 5 seconds on three attempts, a Mini-Mental State Examination (MMSE) score of 17/30 and a GCS of 15/15.

Following a treatment using the 18:60:35 protocol four days after the poisoning the patient's MMSE improved to 23/30 and the Sharpened Romberg time was 40 seconds. The patient was given two further treatments with subsequent improvement arresting the decline in his condition. Treatment had to be curtailed for social reasons and the patient has not returned for review.

Discussion

The use of HBO₂T for carbon monoxide poisoning remains contentious with polarised views about its efficacy. A recent review of the evidence available suggests that a substantial study with adequate controls and patient numbers still needs to be completed.⁵ These two cases had variable signs of delayed sequelae of carbon monoxide poisoning, however both responded to hyperbaric oxygen therapy. Clinical deterioration was reversed in both cases preventing nursing home placement in the female patient, who maintains her independence ten months after her treatment. Risk factors for neuropsychological sequelae include older age (50+ yrs), loss of consciousness, COHb > 25% and metabolic acidosis.³

Conclusion

In cases demonstrating neuropsychiatric sequelae due to CO poisoning it is worth considering hyperbaric oxygen

therapy to assist in the recovery from a debilitating syndrome.

References

- 1 Kindwall EP. *Hyperbaric Medicine Practice*. Flagstaff, Arizona: Best Publishing Company, 1995
- 2 Choi IS. Delayed neurological sequelae in carbon monoxide intoxication. *J Toxicol Clin Toxicol* 1982;19: 297
- 3 LK Weaver, RO Hopkins, S Churchill, KJ Chan, AH Morris, TP Clemmer, CG Elliott, JF Orme, FO Thomas and D. Risk factors associated with neuropsychological sequelae following carbon monoxide poisoning. *Undersea Hyper Med* 2001; 28 (Suppl): 16
- 4 Dunham MD and Johnstone B. Variability of neuropsychological deficits associated with carbon

monoxide poisoning: four case reports. *Brain Inj* 1999; 13 (11 Nov): 917–925

- 5 Bennett MH. HBO₂ for carbon monoxide poisoning: Well, what do we do now? Paper presented at the *Hyperbaric Technicians and Nurses Association 9th Annual Scientific Meeting, September 2001*

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THE WORLD AS IT IS

ABPM EXAM WHAT IT'S ALL ABOUT

By Dr Caroline Fife with modifications by Dr James Vanderploeg

Undersea and Hyperbaric Medicine Board Certification

The American Board of Medical Specialities

Due to the comments we have received regarding the subspecialty examination, some further clarification about the exam is necessary. In the United States, a Chicago-based organisation called the American Board of Medical Specialities (ABMS) determines not only WHICH specialties will be recognised in the US, but what the rules will be for specialty and sub-specialty certification across the nation. For example, only physicians completing residencies accredited by the Accreditation Council for Graduate Medical Education (ACGME) are eligible to sit for specialty board examinations. (This excludes most osteopathic physicians unless they complete a traditional allopathic residency). All specialties recognised by the ABMS must abide by the rules set by the ABMS. The requirements of the American Board of Medical Specialities apply to all of its Member Boards who offer certification in the United States.

What is important about ABMS recognition?

While a US physician might train in a field not recognised by the ABMS, certifications which are not recognised in some way by the ABMS are usually of limited

practical value in the US. The ABMS requirements for the recognition of a new medical specialty board are very stringent. The guidelines for the establishment of a new specialty or sub-specialty are such that no new specialties have been approved in the past 10 years. Furthermore, in order to maintain the designation of a "specialty" there have to be at least three residency programs in the US that are recognised by the ACGME. For these reasons (and because it is necessary to have a solid general medical background before concentrating on Undersea and Hyperbaric Medicine), it did not seem practical to seek approval of specialty certification in Undersea and Hyperbaric Medicine from the ABMS. However, a window of opportunity opened for Undersea and Hyperbaric Medicine to become a sub-specialty that would be recognised by the ABMS. Obviously, only physicians who already had an ABMS recognised specialty certification could get a sub-specialty certification. The question for the UHMS then became: Is it better to at least have an ABMS recognised **Sub-specialty** if the alternative is to continue trying to practice medicine outside of the mainstream? It seemed clear that the chance to be sub-specialty certified, despite all its limitations, was better than nothing.

It is important to understand that the UHMS has no authority to designate the rules by which either individual specialties or sub-specialties are recognised in the US. We could have chosen to do nothing, in which case no US physicians would have had the opportunity to become recognised as sub-specialists in Undersea and Hyperbaric Medicine. We could have waited several years hoping for a change in the philosophy of the ABMS and tried to gain specialty recognition. This might have allowed more US