nomic imperatives of running a competitive recreational diving enterprise. It is inevitable then that at times SPUMS will disagree with the practices of these training organisations. This is appropriate and should not consistently lead to sustained conflict.

Contrary to some current claims from both sides of the debate, Drew Richardson is not the first non-medico to become a full member of SPUMS. From its foundation, SPUMS has had non-medical full members. John Pennefather, the foundation Treasurer, Glen Egstrom and Peter Bennett are good examples. The requirement was and is a commitment to diving medical research.

Given the prevalent paranoia, cynicism and hostility (as manifested during recent code of practice discussions in Queensland), SPUMS' attempts at maintaining a balanced position and of sustaining communications is very much like lambada dancing on a tightrope. The Executive Committee believes that all our members and associates must have access to our Journal, regardless of the polarity of their opinions. However, on some issues, regardless of the debate, there will be no compromise by the Society on such matters as the obligate need for all scuba diving training candidates to have a medical fitness assessment performed by a trained physician. Finally, we will welcome active participation in our annual scientific meeting (1993 - Palau, 1994 - Rabaul) by all of our members and associates. The alternative is an inevitable regression to open warfare, and consequent little benefit to anyone involved.

Des Gorman
President of SPUMS

ORIGINAL PAPERS

CARBON MONOXIDE POISONING: A REVIEW

Paul Mark

This paper reviews recent advances in our knowledge of the pathophysiology of CO poisoning and describes its clinical presentation. It outlines the management of the CO poisoned patient in the emergency department and discusses the indications for referral to a hyperbaric facility.

Introduction

Carbon monoxide (CO) is a colourless, odourless, tasteless and non-irritant gas. It is the commonest agent used in suicide by poisoning in the United States[1], Britain[2,3] and Australia.[4] In addition eighty percent of immediate deaths in burning buildings are due to CO.[5,6]

Following non-fatal poisoning, 10-40% of victims develop neurological or psychiatric sequelae.[7] The risk of death or major disability is increased in the young, the elderly and those with cardiovascular, cerebrovascular, or pulmonary disease.[8]

Hyperbaric oxygen (HBO) was first used to treat CO poisoning by Smith in 1960.[7] Numerous published series have established its benefit when compared with historical controls.[8-15] CO poisoning is regarded as an "accepted" indication for Hyperbaric Oxygen Therapy by the Undersea and Hyperbaric Medical Society.[16]

A recent review of thirteen published series containing 3,441 CO poisoned patients has shown clearly that administration of hyperbaric oxygen at 2-3 atmospheres absolute (ATA) soon after admission to hospital and repeated daily, or as made necessary by the patient's condition, is the only effective treatment of CO poisoning yet demonstrated.[17]

Circumstances of poisoning

The clinical features of CO intoxication are non-specific and may outlast the detection of carboxy-haemoglobin (COHb) in the blood. A thorough history often suggests the diagnosis in less obvious cases.

ATTEMPTED SUICIDE

Patients attempting suicide usually park their vehicles in isolated places with a hose connecting the exhaust pipe to the interior of the vehicle. Occasionally they park in a closed garage with the vehicle windows open. Even if the motor stops, the exhaust fumes persist for hours. The Australian change to unleaded petrol for new vehicles should reduce the opportunity for suicide as catalytic convertors significantly reduce the output of CO.[18-20]

FIRE

Persons trapped in building fires usually collapse from CO poisoning before being burnt. The mortality from CO poisoning is four times higher when it is complicated by smoke induced chemical pneumonitis.[21] The delayed sequelae of smoke inhalation greatly increase the mortality from cutaneous burns.[22] This may be partly due to the concomitant production of cyanide which is difficult to detect specifically.[5,23] A number of other irritant chemicals such as

acrolein, hydrochloric acid, toluene diisocyanate and nitrogen dioxide are produced by fires. These cause bronchospasm and pulmonary oedema.[24,25]

ACCIDENTAL

It is likely that many cases of accidental exposure go unrecognised.[26] Mechanics occasionally poison themselves when the car engine is run with the garage doors closed, especially in cold weather. It has been shown that a lethal concentration of CO can be reached in a closed garage in 10 minutes.[27]

Faulty exhaust systems in cars or home heaters can result in whole families being affected. Following severe exposure the patients may present in coma or with altered mental states. Confusingly with less severe exposure they may present with symptoms similar to a viral illness.[28]

Most paint strippers contain methylene chloride which may be inhaled or absorbed through the skin. It is metabolised to CO.[30] The COHb level continues to rise after the patient has been removed from the source of poisoning and then falls slowly.[30,31] However the clinical disturbance is not as severe as the COHb level would indicate.[30]

Divers using a compressor for surface supplied air (hookah) are often poisoned when the air inlet is down wind of the exhaust from the pump motor. If scuba divers' cylinders are filled with CO containing air several persons are likely to be affected. CO poisoning can be mistaken for cerebral arterial gas embolism.[32,33]

Pathophysiology

HYPOXIA

A small amount of CO is produced by the normal enzymatic breakdown of haem. Unless there is significant haemolysis the normal COHb level is less than two percent. However smokers may have up to 10% of their haemoglobin (Hb) bound to CO.[34]

Research by Haldane demonstrated that Hb binds CO 240 times more avidly than it binds oxygen.[35,36] This creates a functional anaemia which is exacerbated if the patient has a true anaemia.

CO moves the oxy-haemoglobin (oxy-Hb) dissociation curve to the left, further reducing oxygen delivery to the tissues.[37] This will be exacerbated by alkalosis, hypothermia and concomitant poisoning with cyanide.[38] Mild to moderate acidosis is said to improve tissue oxygen delivery.[37]

HISTOTOXIC EFFECTS

A further consequence of poisoning is the binding of CO to mitochondrial enzymes, including reduced cytochrome A3 oxidase, reduced cytochromes of the P450 type and tryptophan dioxygenase.[20,36,47,48] This prevents cells from utilising oxygen and is thought to be a major cause of the disordered physiology. In 1975 Goldbaum replaced two thirds of a laboratory dog's haemoglobin with COHb. There was no deleterious effect. However, when dogs were allowed to breathe CO until an equivalent COHb level was reached they died.[49] It is the CO dissolved in the tissues that is important in producing clinical effects.[50]

Short term exposure, especially in an exercising fireman, will lead to high COHb levels but little CO in the mitochondria. In such cases collapse may be due solely to hypoxia.[3] Longer exposure in an overdose victim will give lower COHb levels but more tissue bound CO.[21] This the setting most likely to give rise to neurological damage including delayed neuropsychiatric sequelae.[51] Concomitant intoxication with ethanol may afford some protection, probably because of an ethanol induced decrease in blood flow and CO intake.[52,53]

Cytochromes have been shown, in-vitro, to bind oxygen nine times more readily than they bind CO.[54] Tissue hypoxia, due to cardiopulmonary or cerebrovascular disease and to the effects of COHb, promotes CO binding with cytochromes.[46] With time, the mitochondria may be overwhelmed as the cytochrome-CO bond is probably not entirely competitive.[47]

The tissues with the highest metabolic rate suffer the most damage especially in areas of poorly developed collateral blood supply.[42,44] Children are more at risk than adults.[8]

THE FOETUS

The foetus is particularly susceptible to the effects of CO.[61-63] Foetal Hb binds CO more avidly than adult Hb resulting in a half life twice as long as that of maternal COHb and COHb levels 10-15% higher. The oxy-Hb dissociation curve is further to the left and the low arterial partial pressure of oxygen facilitates CO binding to cytochrome. The risk period extends into the first year of life, as at three months of age the child still has 25% of foetal Hb.

OTHER TISSUES

Injury to other tissues is often complex.[34] Trauma to muscle, chemical damage to the lung and sludging in the glomeruli as a result of rhabdomyolysis all complicate the direct cellular damage produced by CO.[44]

DELAYED SEQUELAE

The events at cellular level are not clear. Early deterioration may be due to death of tissue with previously impaired blood supply or to cerebral oedema. This does not explain why some patients improve for up to three weeks

before relapsing. One theory suggests that enzyme synthesis disruption occurs but the cells continue to function until the existing enzymes are used up. The ability of hyperbaric oxygen therapy to improve delayed function is possibly due to reactivation of these enzyme systems.[6] A variation of this model emphasises the strong binding of carbon monoxide to solid surfaces and its ability to compete for receptor sites slowing the rates of physiological reactions.[17,55]

More recent research has concentrated on the similarity to re-perfusion injury following cardiac arrest.[56,57] Thom has shown that exposure of laboratory rats to carbon monoxide is associated with an increase in brain lipid peroxidation, which commences only after the animals are returned to CO free air.[58,59] Hyperbaric oxygen at 3 ATA, but not oxygen at 1 ATA (sealevel), was shown to antagonize lipid peroxidation possibly by increasing the scavenging of oxygen free radicals by superoxide dismutase.[59,60]

POST MORTEM

Post mortem findings show widespread petechial haemorrhages consistent with hypoxia, also infarction, particularly in the globus pallidus, substantia nigra and myocardium, and cerebral oedema.[26,34,43,64] In those who die after a delay, the findings are those of cell degeneration and demyelination.[65]

Clinical features

MILD EXPOSURE

Low level exposure causes protean symptoms which may easily be mistaken for influenza or gastroenteritis.[6,66] Headache is the most frequent symptom and is often accompanied by nausea and light headedness.[6,34,67] Fatigue, muscle pains, diarrhoea, vomiting and difficulty in concentrating occur as exposure increases.[68] Dilation of superficial veins occurs early and flame-shaped retinal haemorrhages may occur if exposure has lasted twelve hours or more.[69] A sudden increase in angina or the occurrence of palpitations may result from occult exposure.[21,34]

MODERATE EXPOSURE

More severe exposure leads to subtle neurological dysfunction reflecting diffuse damage to the higher centres. Many of these patients appear vague and psychometric testing will uncover deficits. Neurologic examination often shows generalized muscle weakness, impaired balance and diplopia.[70]

Studies have shown that psychometric scores correlate better with the eventual outcome than does the COHb level measured on arrival at hospital.[71,72] Psychometric scoring measures the actual neurological deficit. When there has been a delay between the CO exposure and the taking of the blood sample some CO will have been released from the haemoglobin, but not necessarily from the tissues. The correct first aid treatment of 100% oxygen by mask can reduce the COHb level very rapidly.

A brief psychometric format appropriate to an emergency department includes orientation, short term memory and recall, serial 7's, spelling a word backwards, drawing a three dimensional house, writing a sentence, naming items and following verbal and written instructions. It is useful to have a standard formula available, such as the Mini-Mental State Examination (MMSE).[73]

SEVERE EXPOSURE

Severe exposure results in gross neurological abnormalities, lethargy, coma, agitation and convulsions.[26] Often these patients are abusive and combative when disturbed. They may appear to be hyperventilating, hysterical or frankly psychotic.[6,42] Muscle spasm, including trismus, has been described.[34] Peripheral neurological involvement is infrequent.[74]

DELAYED SEQUELAE

Delayed neuropsychiatric sequelae develop between two and twenty one days following exposure, often after apparent recovery from the acute insult.[10,75] Higher functions are impaired, especially short term memory. Psychometric abnormalities may reappear. Korsakoff's syndrome may occur. Personality changes range from apathy to unconcerned incontinence. Ataxia, incontinence and Parkinsonism may develop.[34,44] Almost any neurological syndrome may be produced.

In a series of 206 patients treated by Smith and Brandon in the 1960's, only the more severely affected were given 100% oxygen at atmospheric pressure. There was a 33% mortality rate. Only 2.2% of the 138 survivors had neuropsychiatric sequelae at discharge.[76] When reviewed three years later the situation was considerably different as 10.8% had gross neuropsychiatric deficits, 28.4% had an obvious personality deterioration and 36.5% had some loss of memory.[7]

The clinical state at the scene of poisoning correlates better with the risk of sequelae than does the clinical state on arrival at hospital.[10,14,77] Variable recovery occurs in 75% of survivors at one year.[78] Characteristically the most severe delayed sequelae are seen in the elderly and in children.[8] Delayed sequelae are more common following severe poisoning.

Depression occurs frequently, even in patients who do not attempt suicide. Following an attempted suicide, it is often difficult to diagnose an antecedent psychiatric disorder due to the presence of the neurological and psychiatric sequelae of CO intoxication.[79]

CARIORESPIRATORY EFFECTS

The cardiovascular effects include ischaemia, arrhythmias and hypotension. Most early deaths from CO poisoning are due to ventricular arrhythmias and are more common in patients with coronary heart disease or cardiomegaly.[34,44]

Non-cardiogenic pulmonary oedema, due to other toxic agents in smoke, is particularly likely if the victim has been trapped by fire in an enclosed space.[46] Aspiration of fluids is a hazard in anyone who is unconscious and is often implicated in the death of patients who die a few days after admission.[80]

OTHER EFFECTS

In severe cases the effects of CO on other body systems become apparent. Skin blistering, which usually occurs in pressure areas, is uncommon unless the COHb has exceeded 40%.[81] The classically described cherry red discolouration of the skin is rare but venous blood samples may appear arterial in colour.[21]

Rare sequelae include rhabdomyolysis, acute tubular necrosis, pancreatitis, hepatitis, haemolytic anaemia and thrombotic thrombocytopenic purpura.[34] CO exposure during pregnancy may result in premature labour, stillbirth and severe neurological deficits in the infant.[61-63]

Laboratory investigations

Arterial blood gas analysis shows a normal partial pressure of oxygen if there is no lung pathology. Since the saturation figure given by most blood gas analysis machines is calculated from the measured oxygen partial pressure it will be falsely elevated. The true saturation is reduced by the percentage of Hb bound to CO. Most pulse oximeters are unable to distinguish between oxy-Hb and COHb and also over estimate the true oxygen saturation.[82]

The importance of metabolic acidosis as a measure of severity has recently been questioned. Several authors have demonstrated that both alkalosis and acidosis can accompany clinically severe poisoning.[83-86] The lactate level is probably a more reliable guide.[87]

The ECG commonly shows sinus tachycardia, often with ST depression and flat or inverted T waves.[17] The chest X-ray is often normal on admission but may display the features of pulmonary oedema or of aspiration. Haematological and biochemical analysis usually reveal a neutrophilia and may show hypokalaemia and hyperglycaemia.[21,88]

The COHb percentage can be measured readily by most laboratories and often helps to confirm the diagnosis. Whereas it was once the sole criterion upon which treatment was based it is now only one of a number of factors to be considered. Attempts to calculate the past likely maximum COHb are unreliable due to variations in its half life.[46]

Only when alternative diagnoses require exclusion should CT scanning from the emergency department be performed. The presence of bilateral low density regions in the globus pallidus 24 hours after poisoning has been found to predict a higher incidence of death or major disability but not all such patients show these lesions.[15,89]

Management

IMMEDIATE TREATMENT

Initial priorities are control of the airway and support of respiration and circulation. 100% oxygen should be given as soon as the diagnosis is suspected. In a co-operative patient this can be achieved with a tightly fitting face mask using a non-rebreathing valve and a reservoir bag. The Hudson mask is inadequate and should not be used as it will deliver no more than 50-60% oxygen even at 14 litres per minute oxygen flow. In a comatose or unco-operative patient aspiration must be avoided so endotracheal intubation, and possibly positive pressure ventilation, may be required. Before hyperbaric therapy the cuff of the endotracheal tube should be filled with water or saline. Cardiac monitoring is required and IV fluids should be started. Hypotension usually responds to IV fluids. A urinary catheter is required as in any other severely ill patient. Patients with chronic obstructive airways disease and hypoxia-dependent respiratory drive present problems and require careful assessment.

COMPLICATING FACTORS

Fire victims are at risk of airway problems from chemical or thermal injury and should be observed closely. Bronchospasm, pulmonary oedema and traumatic injuries may need to be treated. In a rapidly deteriorating patient who does not respond to oxygen, treatment for cyanide intoxication should be considered.[25] Sodium thiosulphate and cobalt EDTA are the preferred treatments.[23,25] Methaemoglobinaemia is dangerous in the presence of CO.[24,90]

Suicide victims should be assessed for the effects of ingested drugs, particularly tricyclic anti-depressants. Hypothermia should be corrected.

Intravenous solutions containing dextrose are best avoided as hyperglycaemia may adversely affect neurological outcome.[91] Increased interstitial and intracellular lactate levels produce acidosis which results in neuronal damage. Several retrospective studies both in CO poisoning and in other ischaemic neurological conditions support this theory.[92]

Metabolic acidosis should be treated if it is associated with resistant ventricular arrhythmias or decreased cardiac output not responding to intravenous fluids. The preferred method of correction, if the patient is ventilated, is by increasing the minute volume. Any sudden correction of acidosis will decrease the serum potassium and may precipitate arrhythmias.

Hyperbaric oxygen

RATIONALE OF THERAPY

The purpose of hyperbaric oxygen therapy is two fold.

Firstly, it accelerates the removal of CO from Hb, myoglobin and cytochromes. The half life of COHb in air is 320-480 minutes. This is reduced to 60-80 minutes by 100% normobaric oxygen and to 8-23 minutes by HBO at 2.8 ATA.[17,21,47,93] At this pressure the arterial partial pressure of oxygen is around 1,800 mm Hg which should displace any remaining CO from the cytochromes. Intense vasoconstriction due to hyperoxia rapidly reduces cerebral oedema while increasing cerebral oxygen flux. Numerous published case reports attest to the ability of HBO to awaken unconscious patients whose COHb level has been zero for hours and to the additional benefits of repeated treatments in patients with persistent deficits.[6,64,94,95]

Secondly, it prevents delayed neuropsychiatric sequelae. Myers studied two groups of patients.[10] Those with a normal psychometric score, normal ECG and a COHb level of less than 30% were given normobaric 100% oxygen. Delayed sequelae developed in 12%. The more severely affected patients, with gross neurological signs, psychometric abnormalities or a COHb level greater than 30%, received HBO at 2.8 ATA. There were no delayed sequelae in this group. Those patients who developed delayed sequelae following normobaric oxygen were later treated with HBO and all recovered.

While it was not a prospective controlled trial, this study demonstrates two important points. Patients who appear well when they reach the emergency department can develop sequelae, and HBO is effective both for the prevention and treatment of neurological sequelae.

Only one prospective controlled clinical trial of HBO in CO poisoning has been reported,[77] but this trial used oxygen doses which have been shown previously to be ineffective.[10,12,14,17,41,75,96]

In a recent review, Gorman and Runciman collated the results of 13 clinical series in which the long term mortality (at one month) could be determined.[17] A total of 1,847 patients had been treated with varying combinations of normobaric oxygen, a single HBO treatment or HBO therapy confined to the first 24 hours after admission. These therapies accelerated the natural early recovery from CO poisoning but did not prevent the delayed deteriorations.[55] Another group of 1,594 patients had been treated with HBO on admission and then either daily, or as made necessary by the patient's condition. Mortality in this group varied between 0 and 9.6% and was essentially seen only in those who had a cardiac arrest prior to arrival at hospital. In these patients long term morbidity varied between 0 and 4.4% and none suffered a late deterioration in cerebral function.

Indications for HBO

HBO should be given as early as possible to the unconscious patient and to the conscious patient with gross neurological or psychometric abnormalities, clinical or electrocardiographic evidence of ischaemia, arrhythmias, significant hypotension, pulmonary oedema or severe metabolic acidosis, even if this requires transfer. Normally transfer should not be attempted until the airway is secure and the cardiac rhythm and blood pressure are stable.

The COHb level upon arrival in the emergency department has repeatedly been shown to an unreliable basis for determining treatment.[8,10,11,14] Rather arbitrarily, an isolated level greater than 40% has been recommended as indication for urgent HBO.[21,34] Some authors recommend a level of 25% in the emergency department on the basis that the maximum level at the scene would have been higher.[97]

Patients who were unconscious or who had an abnormal mental state at the scene but who are clinically normal on arrival in the emergency department are at risk of delayed sequelae.[10,14,17] Adults with an isolated carboxyhaemoglobin level greater than 25% and children with a level greater than 15% are also at risk.[98] Transfer to a hyperbaric facility should occur within 24-28 hours of presentation.

In the absence of indications for early HBO therapy 100% oxygen should be administered for six hours.

The question of timing is important since most patients will have to be transferred for hyperbaric therapy. Goulon demonstrated improved results in comatose patients when HBO was given within six hours.[96] Norkool reviewed 115 cases over six years and concluded that it was possible to achieve good results as late as 20 hours after exposure.[11]

REMOTE LOCATIONS

Patients in remote locations with minor psychometric abnormality should be treated with 100% normobaric oxygen but if they do not improve within six hours, transfer should be arranged. If they improve they must be reviewed closely over the ensuing weeks for the development of delayed neuropsychiatric sequelae. If these occur they should be treated by HBO as soon as possible.[10]

SAFETY IN PREGNANCY

There has been concern about the safety of HBO in pregnant patients. A careful review of the literature by van Hoesen has revealed that fears regarding teratogenicity, foetal blindness and alterations in foetal circulation are unfounded.[61] It has been found that the maternal condition at the site of exposure predicts the risk of foetal sequelae.[62] Normobaric oxygen may decrease the maternal, but no the foetal, CO load.[99] HBO should be given for any maternal intoxication, foetal distress or a maternal COHb level exceeding 15%.[61] In all cases it should be given as early as possible.

Treatment schedule

In 1992 HBO is a very safe treatment. Multi-place chambers allow medical staff to remain with any seriously ill patient. All patients are accompanied by a specially trained registered nurse. Intensive care, mechanical ventilation, therapeutic infusions and a range of other procedures can be performed in the chamber. Continuous cardiac monitoring and invasive blood pressure monitoring can be employed. Regular "air breaks" are provided which almost eliminate the risk of oxygen toxicity. The pressure changes are gradual and the difficulty in clearing the ears is little worse than descending in an aeroplane. Claustrophobia is overcome by distracting the patient, constant reassurance and the judicious use of benzodiazepines.

At Fremantle Hospital the initial treatment is conducted at 2.8 ATA. Following compression (often referred to as descent) there are two 25 minute oxygen sessions during which the patient breathes 100% oxygen, each followed by five minutes breathing air (air break). The patient also breathes oxygen during the 35 minute decompression (ascent) including a five minute stop at 1.9 ATA.

Follow up

Psychiatric review is important after attempted suicide. Precautions to prevent recurrence are essential after accidental cases. Reviewing patients seven days after exposure provides an opportunity to detect delayed sequelae. Relatives should be told to bring the patient back to hospital earlier if there is any change in their behaviour or personality.

Summary

Patients with carbon monoxide poisoning present many challenges to emergency physicians and hyperbaric unit staff. The role of hyperbaric oxygen has been well researched and indications for its use are now clear. It is essential treatment to prevent post-poisoning neuropsychological sequelae. In view of this, emergency physicians should know how to arrange treatment at the nearest hyperbaric facility.

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HAWAIIAN SCUBA DEATHS

Carl Edmonds and Roy Damron

Background

During the 1980's a number of surveys were carried out on the causes of recreational scuba diving deaths. Also, the death rate in recreational divers was revised upwards.[1] Previously and also during that time the National Underwater Accident Data Center (NUADC), under the control of John McAniff carried, out annual surveys on the causes of diving deaths.[2,3] NUADC have recorded almost 3,000 fatalities, but the documentation relies heavily on second and third hand information. Nevertheless the great numbers ensure that the information is of value. More recently the Divers Alert Network (DAN) has also become involved in the compilation and analysis of diving deaths in Northern America.

In the Australia and New Zealand survey (ANZ series), by Edmonds and Walker[4-6], the deaths were less numerous, but more data was available and it was far more comprehensively catalogued. It included, as a routine, comprehensive police reports, autopsy details, equipment analyses and re-enactment trials. The information so obtained was used as a basis for a series of reports showing the factors contributing to death, and not merely the "final" cause.

This paper covers a number of scuba deaths in Hawaii over the same period. It is reminiscent of the NUADC reports, relying more on newspaper and unofficial reports than did the ANZ series.

It is hard to quantify the relative amount of material available in the three series. The ANZ series had far more detail than either of the American series. The Hawaiian series may well be a little more comprehensive than the NUADC reports because of the simpler logistics of obtaining information from within a single State of the USA, as opposed to trying to obtain information from all States and overseas.

Methods

These case files were compiled by one of the diving experts in Hawaii (RD), initially obtained from newspaper reports but supplemented with follow up investigation, both on an official and personal level, to ascertain more details. The analysis was then made by an independent expert in this field (CE).

Diving deaths data

For the 80 deaths, the data was often not complete and therefore the percentages recorded are those of the number on which that specific data was available, usually not of the total 80 cases. The data available from this survey does give some appreciation of the population being studied.

Approximately half the deaths were in divers aged below 30. Another third were aged between 31 and 40. None were over 60 (Table 1). The great majority of the deaths were in males. Only eight out of the eighty (10%) were females.

Diving qualifications were not always recorded. This information was available for 53 (66%) of the 80 deaths (Table 2). Three died on their first dive; one alone and two with companions whose diving expertise was unstated. Although 15%died while under instruction during their basic "open water" diving certificate training, this figure