It is true that Tony was probably a little hypothermic at the end of the dive, and he could not even be bothered taking his gear off when we were hauled on board.

The diving was sometimes spectacular, usually beautiful, and always interesting. There were iridescent blue ice caves to negotiate, free floating ice packs to dive under, and large irregular shaped icebergs floating by us. Hundreds of penguins, demonstrating a speed and dexterity that they lack on the surface, joined us during the dives and welcomed their underwater visitors.

It is true that we encountered some minor problems with cold fingers and toes. To prevent this, we experimented with the CSIRO heat producing iron/magnesium sachets. Held in gloves and bootees, these produced enough warmth when contact was made with sea water to allow us to continue the dive. Although they look and feel almost the same as commercial tea bags, they produce only a lukewarm beverage of inferior quality, very metallic and devoid of the tannin aroma.

These little heat producing sachets can also be interspersed through the wet suit, ensuring that the whole body is kept warm throughout the dive. If the particle size of the iron and magnesium is too small, excessive heat is produced and burns can be experienced to the skin.

With Antarctic diving, if Scuba gear is used, it as essential to have a line connecting the surface to the diver. It is of value in finding the body afterwards. It may even help the diver retrace his pathway, in the event of accidents. Another similar approach is to use a compressor with a surface supply. It works the same as the safety line, and ensures an adequate supply of gas. With either Scuba or the surface supply, a bale-out or pony bottle is essential.

It was not relevant in the summer diving, but in winter a real problem is to ensure that the hole in the ice is kept open. The water freezes over rapidly even though it is only zero degrees, because the air is at -30° or -40° Centigrade. In the Australian base we had a large shelter which protected the surface crew from freezing, and also allowed a heater to be used which tends to keep the ice hole open. The hole must also be capable of taking at least two people so that a rescue procedure can be conducted in the event of incapacity of one of the divers.

Diving is now being performed throughout the year, and if needed they can use the underwater oxygen unit for emergency treatment of decompression sickness. Of course, the ideal is to ensure that one does not get this illness, by remaining well within depth and duration guidelines.

There are other ways to keep warm, and these are very effective. If you wear a wet suit under a dry suit, this is the most effective. Another technique is to have a hot water supply similar to that used by the abalone divers, ie. with that water being heated from the outflow of the compressor, and being pumped from the ocean around the outflow and then back down to the inside part of the wet suit of the diver. The degree of heat then is related to the gearing up of the compressor.

No-one goes to the Antarctic without falling in love with it. Everyone wants to go back, and many expeditions have been down there on numerous occasions. The attraction is in the beauty and majesty of the land. I was fortunate enough to extend the experience to include the underwater scene.

DROWNING

John Doncaster

Drowning can be defined as death through asphyxiation following immersion in a liquid medium.

Near drowning refers to the survivor of submersion, to which may be added resuscitation and survival, for 24 hours, whether or not fluid has been aspirated into the air passages.

INCIDENCE

The incidence world-wide is roughly three to ten per 100,000 depending on the figures given. In our Geelong area, in the past 24 years, we have had 258 deaths from a population in the summer which is around 200,000 and that gives a yearly average of about 5 per 100,000 per year. Less than 10 per cent of the deaths have occurred from our surrounding beaches. Since 1964, we have had 5,000 admissions to our Intensive Care Unit at the Geelong Hospital, and of these 43 were immersion victims. There were four moribund on admission, the remainder survived.

Not everyone who enters the water drowns, some come out of it in a delightful way. There is usually some problem which helps set the scene, such as alcohol, boats, poor swimming ability, unexpected currents, fatigue, waves, cramp or entanglement in wires, ropes, etc. While increasing the bravado, alcohol lessens the ability to cope, and it impairs intellectual and motor function and reaction times lengthen. Haight and Keating have shown that small volumes, just 30 ml of alcohol, without any carbohydrate ingestion, superimposed on exercise will lead to marked hypoglycaemia, with resultant weakness and confusion. Impaired temperature regulation is also associated with alcohol ingestion leading to a rapid cooling. At Easter, just before coming away, we had a chap who wandered off the edge of the pier falling into the bay. He was drunk, even though it was only 10.00 am. He was rescued virtually immediately by a bystander, who jumped in after him and dragged him out of the cold water. By the time he got to the Casualty Department of Geelong Hospital, which was half a kilometre away, his rectal temperature was 33° which shows how the cooling effect can occur quite rapidly.

Plueckhahn in the Geelong Studies of 238 drownings, found that 168 were accidental, with 117 deaths occurring in adults. Thirty-four were associated with boating accidents: overcrowding, overturning, inexperience, lack of lifejackets and inability to swim. Seventy two were swimming, surfing or in falls from wharves, jetties and rocks, or diving or in vehicles. In more than 50 per cent of the accidents, there was an association with alcohol. In 58 males in the over 30 age group, 53 per cent had blood alcohol greater than 0.08% and 45 per cent were greater than 0.15% at autopsy, and figures from the Surf Life-saving Association of Australia suggest that about one third of near-drowning adults had recently ingested alcohol.

Panic may well be the response to the problem, what ever it is, and this can lead to purposeless struggling, with consequent rapid shallow breathing and this can lead to loss of buoyancy, and submersion, exhaustion and collapse follow. Especially with water in the pharynx in the vicinity of the larynx, breath-holding will occur, water in the pharynx is swallowed, which could well lead to vomiting and gasping with subsequent aspiration into the lungs, leading to blood stained, frothy sputum. Drowning without aspiration occurs in about 10-20 per cent of victims due to laryngo-spasm

and asphyxia.

Diving is of interest to most of us here and the deliberate excursion beneath the surface brings added problems, such as entrapment by inanimate objects or in blind caves. Just before I left home, a diver in a local reservoir got too close to the outlet and was sucked into it and he subsequently popped out dead, and so this can be added to the list of inanimate objects which can trap divers. The equipment problems which may lead to aspiration can be from a faulty purge valve or damaged diaphragms or in the exhaustion of air supply, while faulty techniques, for example buddy breathing or free ascents or loss of a demand valve may all lead to disaster. The loss of consciousness, whilst an uncommon immediate cause of death on land, is lethal under water, and this can be due to air embolism or faulty gas supply or faulty usage. Hypothermia may result in impaired consciousness with subsequent drowning.

Coincidental medical problems such as myocardial infarction, cerebrovascular accident, diabetes, epilepsy and even head injury may occur. Drugs such as the central nervous system depressants, tranquillisers, anti-depressants and antihistamines may all alter the perception of, and the response to, potentially dangerous situations. Vomiting and aspiration of swallowed water, sea-sickness, hangover, or caloric stimulation from ear disorders may all lead to drowning, as may nitrogen narcosis. To put things in perspective however, in the Geelong region, of those past 238 deaths in the water, only eight were associated with diving. They were all males between 17 and 50 years.

PATHOPHYSIOLOGY OF DROWNING

The physiology of drowning is worth considering. The first thing is ventilation-perfusion imbalance occurring due to alveolar collapse, with the shunting of blood past unventilated areas of the lung. It is a common factor following aspiration of both fresh and salt water, due to alteration to, or destruction of surfactant. Foreign bodies such as sand, seaweed or vomit may block the airways. Falling lung compliance is due to the above plus reflex airway closure (through even a small volume of fluid aspirate) which may respond to vagal block and bronchodilators, as shown experimentally by Colebatch and Halmagyi. Pulmonary oedema is a very common, though sometimes a delayed, sequel to the presence of fresh or salt water in the lungs, due to osmolarity changes and the presence of particles of sand, weed, marine organisms or chemicals such as chlorine and gastric contents. An aspiration pneumonitis often results, with outpouring of a plasma-rich alveolar exudate. Alexander demonstrated detachment of the vascular endothelium from the basement membrane of the alveoli, along with alveolar cell oedema, following aspiration of both fresh and salt water. The biochemical changes include metabolic acidosis associated with impaired circulation, hypoxaemia due to pulmonary oedema and shunting, and increased oxygen use due to tachycardia caused by CSF acidosis and/or local pulmonary irritation. Unlike the animal experimental findings of a raised serum sodium, chloride and calcium in salt water aspiration, with the opposite findings for fresh water drowning (except for potassium (which raised due to the haemolysis)), minimal changes are often found in human victims, possibly because of the small volumes actually inhaled.

Hypokalemia may be observed during recovery, probably due to rapid correction of acidosis with bicarbonate, with the exchange of potassium and hydrogen ions across cell membranes. Rewarming, with rapid glucose metabolism, may also increase the movement of potassium into the cells.

Cerebral hypoxia will depress neural function and cause oedema, hyperaemia (through vase-motor paralysis) and acidosis within the cranial cavity.

Hypothermia may cause confusion below 35°C and unconsciousness below 32°C, and interfere with circulation by causing bradycardia, arrhythmias, ventricular fibrillation at about 25°C and asystole below about 22°C. When hypothermia develops before asphyxia, the brain may be protected for long periods of circulatory arrest, as oxygen requirements fall linearly with temperature.

Cerebral irritation may be manifested by straining or by grunting respiration, purposeless movements or even convulsions, all of which further raise cerebro-spinal fluid pressure.

Decerebrate rigidity, if present, raises the CSF pressure as does lowering the head or endotracheal suctioning with resultant straining.

Delayed necrotising pneumonitis may especially follow aspiration of contaminated water or of gastric content.

SURVIVAL

Survival from a near drowning accident is influenced by many factors. The physiological status has a big part to play, the young having a greater chance of neurological recovery after a period of immersion, possibly in part due to initiation of the diving reflex with selective sympathetic vasoconstriction producing a profound redistribution of blood flow to the heart and brain.

The clinical importance is that profound bradycardia and peripheral vaso-constriction may make the palpation of pulses difficult, which along with cyanosis or pallor, plus unreactive dilated pupils may lead to the unwarranted diagnosis of death.

The immersion medium may be significant, as survival from potentially fatal salt water immersion is about 80 per cent, whereas only about 50 per cent survive fresh water immersion. This may be related to the chance of being observed and rescued from the sea, rather than the nature of the fluid per se. These figures relate to children.

TABLE 1

CHILDHOOD ACCIDENTAL DROWNINGS

	Fatal	"Near"
Ocean or bay	17	10
Lakes or rivers	10	—
Dams, creeks	11	4 *
Home pools or ponds	10	3
Public patrolled pools	3	6

* 2 died subsequently.

There was quite a significant difference in recovery if the child was rescued from a pool by trained attendants, rather than if it was dragged out of a backyard swimming pool, dam or river etc. or even from the sea.

Hypothermia can be lethal, so reduction of heat loss as important in surviving immersion, by reducing the gradient between the body surface and its environment. We can do

this by eating as much as possible to improve our insulation, or by wearing a wet-suit. We can reduce the surface area by assuming a spheroid position if we are drifting in the water. Undue exercise and alcohol ingestion should both be avoided of course, to further minimise heat loss.

SALVAGE

Salvage of immersion victims really depends on skilled rescue and resuscitation and subsequent management. The ABC of resuscitation (Airway Breathing and Circulation) is well known to us all but once again worth reiterating. The maintenance of the airway is not necessarily so easy, and during the past decade, the Surf Life-saving Association of Australia was involved in 148 salt water rescues, of whom 48 victims died, with only one being from a patrolled beach (and he had a fractured cervical vertebra). Among the deaths, 41 had foreign material in the airway. This consisted of vomitus in 27, fluid in 12, loose dentures in 3, and sand in a further 3 subjects. Difficulty in keeping a clear airway was reported by the rescuers in half of those who died.

TABLE 2

48 deaths

Difficulty with airway		24
Due to:	Vomitus	20
	Water/Sand	2
	Clenched jaw	1
	Neck shape	1
	Edentulous subject	1

Heart-lung resuscitation was attempted in all but the one case. Among the 100 survivors, expired air resuscitation was performed alone in 39, and of these, vomiting occurred in 54%. Of the 15 who required cardiopulmonary resuscitation, 87 per cent vomited. Of the 57 who were breathing and had a pulse present at rescue, 40 per cent vomited. The application of mouth to mouth expired air resuscitation is not without its difficulties, and it is obvious from these figures that it can be a nauseating experience for the rescuer. In rescues in deep water, floatation devices of some sort are required, if more than one or two breaths are to be delivered to the patient, and for this reason alone buoyancy vests are essential for deep water activity.

Three inter-related phases of resuscitation exist as outlined by Goldin and Rivers. The first aid requires rescue, if the patient is not breathing the airway should be checked and cleared if necessary. It is probably worthwhile attempting to drain the lungs, especially of salt water, by gravity, then mouth to mouth respiration should be commenced immediately, as adequate cardiac rhythm may return following just this. The pulseless patient should have external cardiac massage commenced after three or four breaths, except the patient who has been supported by a life jacket in cold water for a long time, where marked hypothermia may be associated with extreme vaso-constrlction and apparent pulselessness. External cardiac massage in this situation may precipitate ventricular fibrillation. Rewarming may be all that is required.

The application of effective cardiac massage requires at least yearly practice. For a single operator 60 compressions per minute are required for an adequate cardiac output at above 25 per cent of normal. Two breaths are required for every 15 compressions. The lower half of the sternum, above the xiphoid cartilage should be compressed by the heel of the hand by 3 to 5 cm. Straight arms are required to prevent undue fatigue. With two operators, a rate of 60 compressions per minute must be maintained, with a breath being delivered after every five compressions. There should however, he no pause for the breaths to be given. It takes about three compressions to get up to the necessary 25 per cent of normal cardiac output. So if you stop after every five, you are not going to be very effective. Death should not be declared until a patient has been warmed, as resuscitations of over two hours have been rewarded with full neurological recovery, especially in cold patients. In the hospital first aid continues, to which is added the correction of acid-base disturbance. If the patient is not breathing, he should be ventilated with 100 per cent oxygen and intubated. Ventilation should continue with 100 per cent oxygen and positive end expiratory pressure.

Various methods for rewarming have been proposed and do depend on the facilities available. Surface rewarming by bath immersion is difficult to organise and may compound the problem by allowing flushes of cold peripheral blood to get back to the central core. Heated oxygen for ventilation helps to raise the body temperature and certainly is a little easier to apply, but it is not as effective as warm peritoneal lavage or better still cardiopulmonary bypass. If the patient is breathing, then progress over the subsequent few hours is monitored with chest x-rays, electrolytes and blood gases.

Modell of Florida and Conn of Toronto have written copiously with regard to the cerebral salvage of the near drowned victim and have suggested that patients should be assessed after immediate resuscitation and categorised as awake, blunted or comatose.

Category A

Patients who are alert and fully conscious need observation in case neurological, pulmonary or other deterioration occurs. History and examination and laboratory tests of blood gases and electrolytes and chest x-rays should be obtained as a base line. Twenty-four hours is usually a sufficient period for observation, although later complications, such as chest infection may occur.

Category B

Patients who are semi-conscious but rousable, with purposeful response to pain, normal pupil reaction and normal respiratory movements, require close observation, as for a head injury, and the tests should be as for Category A.

Therapy should be aimed at prevention of a raised intracranial pressure by the use of diuretics, restricting fluids to half daily maintenance and added oxygen and the maintenance of normothermia. They usually require a longer time in the intensive care unit because of pulmonary problems following resuscitation. They may well develop respiratory failure, requiring ventilation usually with positive end expiratory pressure (PEEP). They may progress into Category C, with neurological deterioration, but should all survive with normal neurological function.

Category C

Patients who are comatose, are further sub-divided by Conn into:

C1: Decorticate, with flexor response to pain and Cheyne Stokes respiration.

C2: Decerebrate, with extensor response to pain and hyperventilation.

C3: Flaccid, with no response to pain and either apnoeic or have cluster breathing.

These patients have a generally worsening prognosis, with regards to survival and normality of central nervous function.

Therapy is aimed at preventing or relieving raised intra-cranial pressure which might cause further damage to neurones which have survived the initial hypoxic insult. Pulmonary oedema and hypoxaemia is usual in these cases and requires diuretics, hyperventilation with PEEP, and a high inspired oxygen concentration to reduce the inevitable cerebral hyperaemia and consequent cerebral oedema.

Conn's concept of "hyper" therapy is based on the findings in the comatose patients of hyperhydration, hyperventilation, hyperpyrexia, hyperexcitability and hyperrigidity.

Hyperhydration is controlled by using Frusemide, 1/2 to 1 mg per kilogram, repeated until an adequate diuresis occurs. Fluid restriction to 1/3 daily maintenance is started and fluid balance is monitored with ECG, central venous pressure, blood pressure and urine output being charted.

Hyperventilation, here nasotracheal intubation and a volume cycled respirator is required. The arterial PCO_2 is maintained at about 30mm Hg to reduce cerebral vasodilation, although this is a very transient effect. A high arterial PO_2 level should favour the diffusion of oxygen through oedematous areas of the brain and so is aimed for. Conn risks pulmonary oxygen toxicity by aiming for an arterial PO2 level of about 150mm Hg. 5-10 mm of positive end expiratory pressure is usually employed to prevent a fall in the functional residual capacity of the lung and atelectasis without raising the cerebral venous pressure. Cardiovascular stabilisation, with the correction of metabolic acidosis, arrhythmias and effective volume repletion may be required to allow hyperventilation to take place and dopamine or dobutamine may also be required to improve cardiac output, without the infusion of large volumes of fluid which would worsen both pulmonary and cerebral oedema.

Hyperpyrexia commonly follows drowning and should be controlled. Active cooling to 30°C is recommended by Conn to reduce cerebral oxygen requirements and intracranial pressure. The core temperature must be monitored, and because of the suppression of normal immune responses, daily bacterial cultures from blood, trachea and urine are required. White cell counts and platelet levels may also indicate infection. Prophylactic antibiotics are probably not indicated.

Hyperexcitability is prone to raise the intracranial pressure but barbiturates have been used recently with a beneficial effect on neurones. Conn recommends Phenobarb 50mg/kg on the first day in three divided doses and 25mg/kg daily subsequently for three days. He also used methyl prednisolone, 1mg/kg daily, and he believes this helps to lower the intracranial pressure.

Hyperrigidity, both lowering of the head and tracheal suction, may raise the intracranial pressure and so should be limited or avoided. Muscle relaxants help to reduce the reaction to tracheal suctioning and also reduce straining against the respirator and facilitate adequate ventilation. Monitoring of the intracranial pressure using a Richmond screw or ventricular catheter is also called for, and so limit this technique to a unit geared for this. Conn's results before hypertherapy were rather dismal. He had a high incidence of central nervous damage at 42 per cent and had 21 deaths. 21 per cent of the deaths were in groups C1 and C2. Since introducing the hypertherapy, deaths have not occurred in these groups and the central nervous system damage has been reduced to 9 per cent. Since they introduced all the factors mentioned at the same time, it is difficult to know whether one or all aspects are in fact essential. Modell has very similar figures without resorting to deliberate hypothermia or barbiturate therapy.

It is to be hoped that we shall soon know which of the above measures are in fact essential to improve results in the managements of near drowning victims.

References for this article are available from Dr John Doncaster, whose address is 220 Noble Street, Newtown VIC 3220.

AUTOPSY METHOD FOR INVESTIGATION OF FATAL DIVING ACCIDENTS

John Hayman

INTRODUCTION

Fatal diving accidents should be investigated with a thoroughness at least equal to that involved in the investigation of fatal aircraft accidents. As with such aircraft accidents, the investigation requires a meticulous post-mortem examination of the body using several special techniques.

A complete autopsy protocol is given in this article. It is appreciated that such a protocol may not be applicable to every diving accident; for example, there may be no indication to remove the femurs from a sports diver, although these should be examined as a routine in every deceased professional diver. In general, the pathologist should collect all appropriate material, for even if he does not intend to examine it himself, it should be available for examination by others.

The autopsy technique involves submersing the body using a special autopsy tray and hydraulic lift, within a water filled stainless steel tank (Atherton AE and Sons, Melbourne). If this apparatus is not available, it may still be possible to detect small intravascular gas bubbles by opening the head beneath water, and opening the chest by using a local water seal. It is assumed that the pathologist is familiar with normal autopsy procedure. Details of such procedures are readily available.[1] In recording the post-mortem, negative as well as positive findings must be noted, and photographs taken of any abnormality.