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.

The technique described may be applicable to other fatal accidents, such as where cerebral arterial gas embolism is thought to have *occurred* subsequent to open or closed chest injury.

AUTOPSY TECHNIQUE

1. External Examination

   Look for suffusion of skin, mottling, petechiae, subcutaneous crepitus, bites or marks from marine animals and other injuries. Suffusion and petechiae may occur with fatal decompression illness and resemble the changes seen in crushed chest injury. Localised skin bruising and conjunctival haemorrhage may occur with barotrauma of descent. Crepitus may occur with lung or airway injury occurring either as a result of the initial accident or following resuscitation attempts, or may be evidence of cutaneous decompression disease.

2. Ophthalmoscopic examination

   This may be carried out with avoidance of direct contact and disposable cap. The fundi should be examined for exudates and haemorrhages and the retinal vessels examined for gas bubbles. Haemorrhage may also occur into either chamber of the eye, or beneath the conjunctivae.

3. Radiological examination

   Underpentrated films should be taken of the skull (antero-posterior and lateral), chest, abdomen, shoulders, hips, and knees. Gas may be seen in blood vessels, the gut, and soft tissues, or in the chest, pericardial and abdominal cavities. The upper humeri and femurs, lower femurs and tibiae should be examined for evidence of osteonecrosis. Other lung changes should be sought, particularly evidence of pulmonary congestion, oedema, and collapse, and the bones of skull, chest and limbs examined for fracture.

   Ultrasound examination may also be of value in detecting gas in the blood vessels or tissues of the limbs, but the author has had no experience with this procedure. Modern, small ultrasound units are easily transportable and may be used in the mortuary.

4. Examination of the brain

   The skull is opened before the other body cavities. The skin is first incised under water, and the scalp reflected. The body is then raised above water in the tank, and the calvarium opened with a vibrating saw, using careful control to keep the dura intact. The body is then submerged again, the calvarium removed and the dura incised under water. The brain is removed and inspected under water for bubbles in the cerebral circulation, then placed in formalin in a submerged container, remaining under water the entire time. In this way the brain is taken from the body and fixed without exposure to air. Any gas present in the cerebral circulation must have been present prior to the post-mortem.

   After fixation the brain is examined with serial sections, and blocks taken from standard areas[1] and areas of any macroscopic abnormality.

5. Examination of Thoracic Contents

   After the brain has been removed and placed in fixative, the tank is drained and refilled with fresh water. Any pneumothorax, pneumopericardium or pneumoperitoneum is needled under water, the gas pressure and volume measured, and a gas sample taken for analysis. The thorax is opened under water, using bone cutting forceps and taking care not to damage the lungs. The pericardial cavity and great vessels are incised and the presence of gas in the pericardium, pericardial vessels, pulmonary artery and aorta noted. The heart is then removed from the pericardial sac, and again opened under water, noting the presence of gas in any of the chambers. The body is then elevated and water drained from the thoracic and peritoneal cavities. The coronary arteries are dissected and standard blocks[1] taken from the myocardium after the entire heart has been weighed.

6. Examination of the lungs

   The lungs are removed together with the trachea and larynx taking care not to damage the pleural surfaces. They are then reinflated through a cuffed endotracheal tube with compressed air or oxygen to a pressure of 30cm of water, producing slight over-inflation. The reinflated lungs, with the tube in situ, are then placed beneath water using a stainless steel grid to obtain complete submersion. Air leaks to the pleural surfaces and into the pulmonary arteries or veins are demonstrated using this technique.

   The lungs are allowed to collapse and are again inflated, this time using 10% neutral formalin solution. Filled and submersed in formalin, they are fixed for 48 hours before blocks are taken from the pleural surfaces of each lobe and from the hilar areas of each lobe to include the segmental bronchi and associated branches of the pulmonary artery, and intervening pulmonary veins.2

7. Abdominal contents

   The bladder is punctured from the peritoneal surface using a 10ml syringe and 19 gauge needle, and urine withdrawn. This is transferred to a plain tube and tested for glucose, blood, and protein using a test strip method (Ames). Urine is also tested for the presence of fibrin degradation products using a latex suspension test (Wellcome). The presence of fibrin degradation products in the urine, in the absence of blood, is evidence of intravascular coagulation occurring prior to death, which, in the case of a diver, is most likely due to antemortem intravascular bubble formation. Gas bubbles should be looked for in the mesenteric vessels, and blebs of gas beneath serosal surfaces. All abdominal organs are weighed and blocks taken for histological examination.

8. Spinal cord

   The spinal cord is removed intact using a posterior incision and fixed intact, after incising the dura. Care is taken to avoid artefactual changes from kinking or twisting during removal. (There appears to be no practical way of removing the cord under water, and in any event the removal of the brain and thoracic viscera allows air to enter the vertebral circulation).

9. Further examination

   The scalp incision is extended inferiorly to allow removal of the mastoids. The middle ears are opened and examined, and cultures taken from both areas. Bone blocks from ears and mastoids should be retained and fixed in 10% formalin.

   The head and neck of both humeri, and both femurs, the lower ends of the femurs and upper tibiae are removed even in the absence of radiological abnormality. This tissue should be divided longitudinally using a vibrating or band saw and fixed as for the ear and mastoid blocks.

10. Interpretation of findings

    The post-mortem findings must be interpreted with a full knowledge of the dive, diver training and experience, water conditions, equipment and its operational condition, safety equipment, circumstances of the accident and resuscitation attempts. Details such as the brand and thickness of the wetsuit, type of weight belt release mechanism, total weights, brand of tank and regulator, and residual air supply all should be recorded.

    The cause of the death can only be assessed with a knowledge of these details and the post-mortem findings. As an example, bubble formation represents a physical rather than a physiological event, and bubble formation will occur after a body is removed from a decompression chamber or brought up from depth. Thus the presence of bubbles at post-mortem does not necessarily mean that the diver died from decompression illness, and their significance can only be assessed with a knowledge of the circumstances of the accident and resuscitation attempts. The reinflation of the lungs under water is a crucial step in the post-mortem diagnosis of cerebral arterial gas embolism and without this, the diagnosis is only one of surmise.

    Even if the pathologist himself does not wish to examine all the post-mortem material, samples should be collected for evaluation by others. Decompression sickness and other illnesses associated with diving have been identified clinically for over 100 years, but their pathogenesis is still poorly understood.

REFERENCES

1. Ludwig J. C*urrent methods of autopsy practice*. 2nd Ed. Philadelphia: WB Saunders, 1979.

2. Katzenstein AA and Askin FB. *Surgical pathology of non-neoplastic lung disease*. Philadelphia: WB Saunders, 1982: 3-5. (Bennington JL, ed. Major prob*lems pathology*. Vol 13).

DISCUSSION

Question

Do you have post-mortem figure on air embolism in people who have actually died in diving accidents or from chest trauma?

Dr John Hayman

I have found it difficult to evaluate air embolism. I have done a series of 12 post-mortems and it is only through doing that series of post-mortems that I have reached what I think is a satisfactory technique for performing them.

In three of those 12 cases I think death was due to cerebral air embolism, and in all these cases it was mainly circumstantial evidence. It was the nature of the dive and onset of symptoms that led to that conclusion. Only in the last case was there definite evidence. The victim was an SAS diver who was diving at 15 feet in the dark, his buddy surfaced for some reason. He was left in the dark and all of a sudden he shot to the surface from 15 feet, 5 metres, down. He was diving on one of the gas rigs and there were a lot of seals in the area and it was thought he was nudged by a seal in the dark which caused him to panic and come to the surface. He convulsed as soon as he got to the surface and was virtually dead when the rescue boat reached him. So on clinical grounds at least he was a case of cerebral air embolism. And at post-mortem in his case I was definitely able to demonstrate that there was this leak in the lungs. That is the only case where I can say definitely but there were two other cases earlier on that I thought were probably due to the same cause.

Question:

What are you looking at in the retina?

Dr John Hayman

I am looking for bubbles in the retina. I have found them in patients who have blunt chest trauma; I have not found them in any of the divers that I have looked at.

Question:

Do you routinely open the mastoid when you are doing a post-mortem.

Dr John Hayman

Yes, I should have gone into that in more detail.

Question:

And what have you found?

Dr John Hayman

Nothing. When I open the skull and then remove the brain, I then go on to look at the middle ear. The incision we use goes up around the side and exposes the mastoid on the way up. There is also the question of examining the spinal cord and examining the hips, knee and shoulder joints, which are the usual sites for articular bends. My feeling is that if I am dealing with a professional diver who may well have these changes then I should go ahead and so that full post-mortem. In a professional diver and certainly one of the abalone divers I would want to get as much tissue as possible and not necessarily to look at myself but to pass on to somebody else who would be interested in it.

Dr Carl Edmonds

I would like to go on record as saying that I have slung off at pathologists for the last 15 years and I now withdraw those comments.

Dr John Hayman

I think that your slinging off at me at least would be firmly justified 15 years ago when I first went to Sale. I think I have only just acquired this experience. I would like to think that I am now much better at doing post-mortems on divers than I was 15 years ago.

ABALONE DIVING IN NSW, INCLUDING A CASE REPORT

John McKee

My first interest in abalone divers occurred some twenty-five years ago, after I had spearfished for some years, then undertaken a Scuba course, and then observed the activities, from a medical point of view, of New South Wales Far South Coast abalone divers.

In those days, it was a rather frightening observation in many ways, because of their rather extraordinary dive profiles and diving habits, which if adopted by we amateur divers would almost certainly have led to our demise.

In 1962 the first diving and medical text book; written by Stanley Miles, became available in Australia, and during my four or five years overseas I studied that book, and on my return I found that things really had not changed very much in regard to the way abalone divers behaved in the water in New South Wales.

In the early 1970s, as some of you may be aware, abalone reached a very high price, and it produced a very good income for many professional divers, varying between $500 and $1000 per week. At that time, some 10 to 15 years ago, I found that school leavers were taking up abalone diving, as a means of obtaining an income, and many of them had no training, and some tended to drink excessively at night before diving, and also take drugs. Unfortunately, in my area, when the abalone were fished out or they went too deep, some of these young men would then have a one week course in using a chainsaw, and they would then become tree fellers, and regrettably some of them came back to me as surgical patients!

In recent years regulations have come into the abalone diving industry, and in New South Wales there are now some 60 professional divers, of which 20 are on the North Coast. Those 20 tend to dive in shallow and surge conditions, whereas the 40 in my region, between Narooma and the Victorian border, dive much deeper, anywhere between 1 metre and 40 metres, although the average would be between 13 and 25 metres.

In southern New South Wales the average abalone diver, weather permitting, does three dives per day, each to around 22 to 25 metres. These usually last for one hour, following each descent, and then the diver brings up his abalone bag. After a brief rest, he then goes down again. He basically does three deep dives, of approximately one hour each, of 2025 metres, and then the final, usually fourth dive, the so-called “shallow dive”, tends to be between 8-10 metres. It is that latter dive, which for some unknown reason, seems to prevent the abalone diver from developing one of the most serious complications of diving, decompression sickness.

Abalone diving may be hard work, and in fact it is estimated that a diver who is working at about 20 metres, in reasonably heavy conditions, perhaps in one hour does as much physical work, as does a labourer doing manual work at atmospheric pressure, on the land for seven hours. The abalone diver in our area may be wearing a 20-30 lb weight belt, he may be dragging behind him an abalone bag of varying size and weight, he will have a hookah connection to the surface supply air, of between 75 to 100 metres length, and this often has to be dragged through a current of 2 to 3 knots.

Abalone divers suffer from numerous medical conditions, diving related, similar to those which may afflict amateur divers. One of the most troublesome ones is the so-called ear drum “scarring” or perforation, related to changes in pressure. The abalone diver makes frequent ascents through the water, often from great depths, bringing back bags of abalone, and as he is constantly in the water over many hours, he probably has inflamed and rather waterlogged ears. Every now and again he is subjected to an emergency free ascent, when his hose gets cut by a propeller, or the hose bursts.

Another complication of abalone diving is decompression sickness, but we have been rather fortunate in our area. Over the last eight years there have been about 15 cases of decompression sickness, of which several have been severe. All of those in recent years have survived, but until about 1975 there were a number of deaths on both sides of the Victorian border.

From a subjective point of view, abalone divers consider that they acquire a partial deafness defect, due to prolonged ear drum exposure in water and it is probably reasonable to say that in our area some 80% of the divers are afflicted in this way. Probably all veteran divers have some permanent form of ear problem, as a result of their diving. From the subjective point of view, I am told by friends of mine who are abalone divers, that as time passes, they notice that the screeching of the kids and cats is no longer an annoyance as it used to be. From the objective point . of view of their families however, when Dad comes home from a tiring day at diving, he comes into relax and turns on the hi-fi or the TV,, adjusts the volume, and then the whole family, including the cat, leave the room!

A further problem is dysbaric osteonecrosis, bone infarction from exposure to pressure, and this condition does