The syndrome was looked at by Carl Edmonds in the early 1970s. He closely documented about 30 cases just to see what would happen. When he did blood gases on them and found that they were cyanotic, he thought he might try 100% oxygen. Not only did 100% oxygen reverse the dyspnoea and hypoxia but it also reversed the whole syndrome. If one gives these guys 100% oxygen, within about half an hour not only is the hypoxia gone but also the fever, the myalgia - the whole shooting match goes. I find it extremely interesting that oxygen should resolve the syndrome. Carl did some other things with the divers. Being a scientist he thought he would try to reproduce the syndrome and it was easily reproducible. If he gave them a leaky demand valve to breathe from, they would get the syndrome. He also found that if he then gave them 10% oxygen to breathe that would really bring the syndrome on. However, the divers were not really keen on this and neither was the Navy Ethics Committee, so he had to give that experiment away.

There are a lot of interesting features in the syndrome. Firstly, the divers would often present in groups. There would be no cases for a month or two and then all of a sudden there would be 10 or 15 in a period of 2 or 3 days. Why this should be I am not sure. The equipment leaked all the time and these men were aspirating sea water during their training all the time yet only occasionally would they get the syndrome. It suggests that there was something in the water that was giving it to them, either an infective agent or maybe it was an allergic reaction. The other interesting feature was that there seemed to be an individual susceptibility. Sometimes you would get the same diver who would present during his training with six successive cases of aspiration and yet other divers who had been diving alongside him and even using the same equipment and inhaling the same salt water did not get it at all.

I thought originally that this was perhaps confined just to Navy divers, but it is not. We now know that civilian and sports divers get the syndrome as well. We have seen it on a couple of occasions in surfers who have aspirated a bit of water after being dumped and that kind of thing. The patho-physiology of it is of great interest to me. I do not know much about it nor does anybody else either. There has not been much research on it. Maybe it is just part of a continuum between asymptomatic aspiration of a small amount of sea water and near-drowning. Maybe it is a sort of halfway point to near drowning. Maybe it is an allergic effect due to inhalation of material from sea water like plankton or sea water proteins of some description or other. Or maybe it is just a chemical pneumonitis due to inhalation of hypertonic saline.

I have got a sneaking feeling that this syndrome is a lot more common than we think it is. I have a feeling that there are divers and swimmers who are presenting to Casualty Departments and GP's surgeries and perhaps even Intensive Care Units complaining of symptoms and signs along these lines, who are being successfully treated with antibiotics. I would remind you that the salt water aspiration syndrome is self-limiting, it gets better whether you treat it or not but it gets better much quicker if you treat it with 100% oxygen.

THE CEREBRAL SEQUELAE OF DROWNING

Peter de Buse

One of the things that has concerned people for a long time when dealing with children who drown is the likely outcome. Now a number of children will be placed on ventilators for some time, with paralysis and thiopentone infusions which make it impossible to assess their state of consciousness during treatment. The parents are naturally very anxious to know what the outcome will be. I will mention briefly some of the cerebral sequelae to drowning and then outline the Royal Children's Hospital approach to the management of drowned children and then discuss the prediction of outcome in some of the children.

The initial problem is cardiac arrest, accompanied by apnoea. Hypoxia is not necessarily all that much of a neuronal insult. The idea that 3 or 4 minutes of hypoxia is the cause of the subsequent central nervous system deficit is very much in doubt. Animals which have been ventilated with gases of very low oxygen content but with their cerebral perfusion maintained have been judged to be normal after some 60 minutes of what we would consider to be hypoxia. Following hypoxia, very often because of the effect of the hypoxia on the myocardium together with the neuronal insults there is a problem of subsequent cerebral perfusion and oedema. The cranium is only some 5% greater in volume than the brain. If there is any oedema then the rigid skull makes sure that the intracranial pressure goes up. The effect of hypoxia and drowning on the cell, must be considered as well. The magnesium dependant ATPase system, and the sodium dependent ATPase system, result in an influx of sodium with an efflux of potassium and this is accompanied later by the influx of calcium ions into the cell. There is currently interest in the calcium blocking drugs in relation to hypoxic cerebral insults. Some people would suggest that the anoxic ischaemic insult leads to a massive influx of calcium ions into the cell, that mitochondrion uncoupling occurs at that stage and again because of the entry of calcium irons into the cell, there is an activity of phospholiphase and with the release of free fatty acids, particularly arachidonic acid and that this has an effect on the subsequent perfusion of the brain. In animal experiments there is a very good correlation between the arterial supply of the brain and the boundary zones at which neuronal damage occurs. Following the continuing insult that takes place after hypoxia these may be prostaglandin release and thromboxane release which with effects on platelets and small vessel activity may give rise to an increase in cerebro-vascular resistance. The calcium damaged cells, with the release of superoxide free radicals, may also be related to the increase in myofibrillar spasm.

In the last couple of years the Royal Children's Hospital in Brisbane has admitted 38 drowned children. There is another series, with which I wish to compare our figures, from California, where the numbers are much the same. The age of drowning is very largely in the toddler age group. That is because of the number of domestic swimming pools in Brisbane and is perhaps related to the lack of fencing and safety measures in those pools. When the children are admitted initial evaluation is made. They can

be divided into groups whose general condition is good, the vital signs stable and who are neurologically normal, and by and large you can say that they are going to do well, whatever you do to them. In Group 2 the general condition is stable in terms of their cardio-vascular and other physiological signs, but they are neurologically abnormal and in Group 3 they are in or have recently had a cardiac arrest and are neurologically abnormal. The initial management of Group 1 is one of observation and minor fluid restriction only.

I will describe our approach to the situation in Groups 2 and 3. The children's condition on discovery, by that I mean when they were actually fished out of the water, was established by asking the older children or adults, who were concerned with their recovery, what they were like when rescued. Two of them were alert, though they had been underwater for some time. Two were considered to be blunted in their sensorium and 33 of them were comatose. The neurological condition on admission to the Royal Children's Hospital, that is the initial assessment when they came into the Casualty Department, is classified as awake (Group A) or they have some blunting of their sensorium (Group B), and in Group C they are comatose. Group C1 is when they are decorticate, in other words, they have some increase in tone but with a flexor attitude. Group C2 are decerebrate and in Group C3 they are flaccid or dead.

We would like to think that we did blood gases on most of the children but that is not so. Of the ones who had it done the moment they arrived some were not really very abnormal at all, and some are quite abnormal and still survived. Looking at the length of witnessed apnoea, from the time of discovery by the rescuers to the onset of spontaneous respiration, which may be just a gasp we can see that the majority of them have taken some sort of gasp within 5 minutes. But there are a few where there have been very prolonged periods before there has been any spontaneous effort at all, but is not necessarily associated with fatal outcome. Comparing the neurological status on admission with the period of witnessed apnoea shows that those children who had had prolonged periods of witnessed apnoea were in a rather worse neurological state but they do not correlate all that well.

The results of the experiments in animals that I mentioned earlier do not necessarily parallel the human state. So we have adopted the attitude that the damaged brain needs both oxygen and glucose and that we will try to minimize the needs of oxygen and glucose and will try to maximize the delivery. We have adopted the HYPER protocol that was made popular through the Pediatric Clinics of North America in 1979. We seek to normalize hyperhydration, hyper-ventilation, hyperpyrexia, hyperexcitibility and hyper-rigidity.

Most of the children who have been admitted have been hypothermic. There was only one who had a normal temperature. The average temperature has been about 35°C. That is probably related to the sea temperature and swimming pool temperatures in Brisbane which never, even in winter, are very low. We have not maintained hypothermia, as some people have felt should be done, because we feel that the effect of prolonged hypothermia below about 34° or so is very much bound up with suppression of the immune functions of the child. Our attitude has been to make no active efforts to warm the child but allow it to rewarm spontaneously. Extreme hyperthermia has sometimes been a problem and we have used chlorpromazine to deal with it.

We have maintained them at between one-third to half their normal fluid requirements. We aim at a serum osmolality of 305-310 in response to that fluid restriction. It is important when considering cerebral oedema and raised intracranial pressure to try to decide whether one is specifically trying to deal with oedematous tissues, and that probably is not all that easy to do, or whether one is non-specifically trying to lower intracranial pressure, by shrinking the blood volume and reducing brain water content, which is what we commonly do with diuretics and dehydration or whether one is attempting to prevent or mitigate the formation of oedema. Those people who give steroids in a dose of 0.1 mg per kg., four hourly, are presumably using that particular principle. Cerebral pressure monitoring has been part of our regime in the last few children that we have had. Our observations have been in keeping in with the work published from California. Other workers have shown that a cerebral perfusion pressure, that is the mean systolic blood pressure less the intra-cranial pressure, of less than 50 mm Hg was always accompanied by a poor outcome and the same was true for an intracranial pressure that was over 20.

We have used barbiturates. Our regime has used an ariaesthetic initial dose of 5 mg per kg and then between 1 and 4 mg per kg an hour. There are plenty of people who would use between 5 and 10 mg per kg an hour. It has been suggested that some of the neuronal effects of calcium influx may have been ameliorated by barbiturates. A recent report in the Annals of Emergency Medicine gave results of 10 dogs who were put into cardiac arrest and apnoea. During resuscitation, five of them received no calcium channel blockers and five of them were given an effective dose. The neurological status of the second group was definitely superior to that of the ones who were not given calcium channel blockers.

In our series all those children who are awake or had minor blunting of their sensorium did well and the children who did badly were predominantly in the decerebrate or flaccid group. The Californians had a rather more scattered group in the comatose group and they had rather more deaths and with a rather larger number with persistent deficit. There is a very wide variation between the various reported series. Of course some of them come from places where the water is cold and some where the water is quite warm, but it is still very, very difficult to predict the outcome of children. Very few factors are significant for prediction. Age and sex are not significant. pH on presentation does not seem to have all that much bearing on survival, nor does the temperature on presentation. If the child was virtually dead on presentation, that was very strongly significant. So was the presence of fixed dilated pupils. Although it is always put forward that if they have been fished out pretty quickly, you can say to the parents that the child is going to be alright, that is not necessarily true at all.

Other things are not significant either. In our hospital, we have taken the time to the first gasp being below 20 minutes as a predictor. I have my doubts about that being a very valuable predictive factor. We elected to ventilate one, a child who at the initial evaluation was on the border line between being blunted in sensorium and being comatose. The child had a cardiac arrest from its endotracheal tube becoming blocked during the middle of the night. That child subsequently was a really very grossly physically handicapped and mentally damaged child. I wonder if we had not intubated her and had left her alone, whether she would have been alright.

Dr Peter de Buse is the Director of Intensive Care at the Royal Children's Hospital, Brisbane.

DISCUSSION

Chairman, Dr C Acott
I would like to comment on Dr Knight's introductory remarks. People who come from the higher latitudes and far south seem to think that it is always warmer up here. I did. When we had a conference here in July everybody came up in their shorts and shirts and they sat around the fires all night. The water temperature at which people can maintain their body temperature in water, is 72°F which is about 24°C. The water temperature at Yeppoon in July is 21.5°C. The map issued by the Department of Transport in one of their safety education articles shows Townsville as being the latitude at which people can maintain their body temperature in the water without aids such as extra clothing in winter, for 12 hours. That means in practical terms that if you fall into the drink just before or just after sundown, which is about the most likely time, and if you get found after daylight next day which is about when you can hope to be found, if you are lucky, you are likely to be either dead or fairly close to it in this latitude in winter time.

Dr J Knight
I would not disagree at all. It is a fallacy, and a widely believed one, that warm water does not induce hypothermia. There are people in this room who would undoubtedly survive much longer than others. It depends on your insulation and your heat output. There are obvious survival differences in shape. This is shown in fell walkers, people out getting hypothermic from exposure to wet windy conditions, as well as in people falling in the water. It really means that you have got to choose your genes and your cook very well if you are going to spend a long time in the water unexpectedly.

Dr Vic Callanan
How much water do you need to aspirate to get the salt water aspiration syndrome? I suspect that if they are obtaining their water in a nebulised form, and these people are in the water for some time, they could get into their lung quite considerable amounts of water. I wonder how much hyperhydration may play a part in this giving them combined cerebral and pulmonary effects.

Dr B McKenzie
I do not know how much water you need to inhale. We have seen cases of aspiration syndrome where people have developed it after one inhalation event. With buddy breathing for instance, they have received a demand valve full of water and they have inhaled a bit of it. The demand valve does not hold much water even if you inhale the whole lot. No research has been done on this so we do not know, but I suspect that the volume of water can be quite small to provoke the syndrome although it can be large as well.

Dr J Knight
As somebody who has had this syndrome using Navy equipment, buddy breathing, it is not much water. We were passing the regulators flowing, with them upside down so the water would not get in. We had a lecture about salt water aspiration syndrome that morning and we did our buddy breathing in the afternoon. We did occasionally fail to have it flowing. I shivered all night and came down next morning and announced that I had the salt water aspiration syndrome. Funnily enough two other doctors who had been to the same lecture turned up and said "I had a bloody awful night, I was shivering all night, I feel sore as anything. I wonder what is the matter with me?"

Dr A Holloway
Did the Navy do water samples for bacterial count and did you do microscopy of what they coughed up, looking for things like E coli in water and algae in the lungs? Around the coast of Southern Africa, chills rigors and infection in near-drowned children correlated with currents, sewerage flow and bacterial colonisation of water.

Dr B McKenzie
I would believe the contamination theory, especially in Sydney because they dump raw sewage into the water. It is probably on the cards. We never analysed the water. Very little research has been done into it, we just do not know. I was thinking of doing a project making some artificial sea water or ultrafiltering sea water and getting people to breathe that and see if that provoked the syndrome. Sometimes they had eosinophils in their sputum but we did not do cultures on their sputum. The thing against the bacterial contamination theory is that we have seen it in other parts of the world and in other parts of Australia where there is clean water. If it was just confined to Sydney I would say that it was sewage inhalation.