a decreased compliance. If you have a decreased end diastolic volume for the same filling pressure that is usually interpreted as a decrease in contractility and it gives a decrease in the left ventricular stroke volume. The problem that occurs from this mechanism, is that it is difficult to know how to treat it. Another mechanism which has been postulated to decrease the cardiac output is a humeral one which has been shown in cross-circulation experiments to occur, although its exact significance in any particular patient and exactly what the mediators of this humeral response are, have yet to be elucidated. So it is a complex problem.

How best to increase the cardiac output, given that you have a problem? There are three paths and they effect the determinants of the cardiac output. As we know, one is by affecting pre-load, and I have mentioned that. The second is by increasing contractility by the use of a vasopressor and the third would be to increase forward flow by decreasing the after-load of the ventricle. PEEP should be considered in the overall context as not curing the disease that is going on, but to pinch an orthopaedic term, as a splint to maintain the lungs in the position of function until recovery slowly occurs.

The message I want you to remember is that no matter how good the care once the patient reaches hospital RESUSCITATION AT THE SITE OF DROWNING IS THE MOST IMPORTANT DETERMINANT OF ULTIMATE PULMONARY AND CEREBRAL SURVIVAL.

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SALT WATER ASPIRATION SYNDROME

Bart McKenzie

Navy divers have very strong macho image. When they are under training, it is very important to submit to peer group pressure and live up to this macho image. They do not complain about minor problems. When they are under training if they get some fractures in their toes or perforated eardrums, they just keep on diving as long as they can put up with the pain and vertigo. But there was one thing that really used to stop these divers in their tracks, and that was the salt water aspiration syndrome.

This was a syndrome that we often used to see in Navy divers. They would present with an acute febrile illness. It was short lived and was characterised by a high fever, by myalgia and by respiratory distress. The typical scenario was that the divers would present to the Royal Australian Navy School of Underwater Medicine which was closely associated with the Diving School, with a constellation of symptoms. The main feature was that they felt very ill and they looked very ill, often they were cyanotic. They complained of fever, rigors, malaise and dyspnoea. They usually presented in the evenings after a day's diving and often after night diving as well. They were usually breathing fast. They had some respiratory distress. They had creps and rhonchi in their chests and occasionally they were blue. They usually had a high fever, a temperature of 39° or so and they had a tachycardia. Chest x-rays showed that they had patchy pulmonary infiltrates, which looked a bit like pulmonary oedema but it could pass for respiratory tract infection as well. There was a cotton wool appearance around the lungs. When we did their blood gases they were invariably hypoxic, sometimes with oxygen tensions down to about 40 mm Hg or so and they had a slight hypocapnia as well.

They invariably gave a history of aspiration of sea water. The Navy had some fairly obsolete equipment up until about five years ago, and the demand valves that they used almost invariably leaked. Most of the divers, most of the time, were inhaling a fine aerosol of water through the leaky demand valve. Another source of aspiration of water was from buddy breathing. Buddy breathing is when the divers share a demand valve. They pass it from one to the other. When one diver takes it out of his mouth and hands it over to the other diver it usually fills up with water. This is not a great problem if you have got modern equipment, because you just exhale into the demand valve and that exhausts the water out through an exhale valve. But the Navy divers' equipment had a rather large dead space. It needed about a lung and a half full of air exhaled into the demand valve in order to clear it. Modern equipment has a purge button and if you have not got enough air in your lungs to clear the demand valve you press the button and it blasts compressed air into the demand valve and blows the water out. But the Navy equipment did not have purge buttons. So buddy breathing was a fruitful source of water aspiration. There were other ways of aspirating water as well in Navy diving training but buddy breathing was the most common one. At night in Sydney Harbour the only thing you can see is the luminous dial on your watch, everything else is done by feel and by guess work. So buddy breathing at night was another fruitful source of aspiration of sea water.

Salt water aspiration is not near-drowning or even near, near-drowning. The amount of sea water that was being aspirated was only about just 10 mls or even less. So, I am not talking about a drowning syndrome, this is something quite different. The divers would aspirate water for one reason or another, and then continue their diving. They were not incapacitated. Often they would have a history of a bit of a cough after the dive. They might even cough up a little bit of blood, and then they would be OK. They would carry on with their normal diving activities without any problem. There was a latent period before they developed the full blown syndrome. This latent period was usually about one hour to two hours, but it had a range. Sometimes if they were really bad they would present almost immediately and the outer limit was about 15 hours. Then they would present with the syndrome myalgia, shortness of breath, cyanosis and all the rest of it.

Before the salt water aspiration syndrome was described, they would present complaining of this syndrome, and the doctors thought that the divers either had an acute respiratory tract infection or viraemia or even pneumonia. The whole clinical pattern certainly fits that sort of thing. So the doctors, quite reasonably, would fill them up full of antibiotics and put them to bed. By the next morning they were completely well, absolutely asymptomatic. However without being given antibiotics, they still got better in the same time. This was a bit perplexing.

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