by cold. They are much more the risks of cold induced errors due to clumsiness in muscles, clumsiness in thinking, inappropriate thoughts, and if the person does go unconscious from cold, drowning through loss of protective reflexes.

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THE DROWNED LUNG

V Callanan

I am going to talk about some of the pathophysiology of the drowned lung. This was not always the part of drowning that received much emphasis. In the 40's and 50's more emphasis was placed on changes in serum electrolytes and blood volumes. In the 60's and 70's the lung problems and the acidosis associated with it became a subject of research. In the last 10 years or so lung problems and cardio-vascular functions, in particular, have become prominent and in more recent years, brain preservation has become a widespread topic in this area.

I intend confining myself to talking about cardio-pulmonary aspects of near drowning. I will exclude victims of dry drowning, that is those who have laryngeal spasm and do not actually have water entering the lung. So I am talking about patients who get water in their lungs. Water in the lung quickly produces profound hypoxia, hypercarbia and a profound metabolic acidosis. By that time the patient reaches hospital, some of this may be reversed slightly by cardiopulmonary resuscitation. The hypercarbia may be reversed, however the profound hypoxia and metabolic acidosis will always be present. Usually the patients present with all of this triad unless the patient has recovered sufficiently to restore his cardio-pulmonary function almost to normal.

By the time the patient reaches hospital it really doesn't matter whether the patient drowned in salt or fresh water because by that time the syndromes are fairly similar. In fresh water aspiration the patient will finish up with surfactant damage and the adult respiratory distress syndrome (ARDS). If they drown in salt water, for various reasons, they finish up with the same syndrome. If they happen to aspirate their stomach contents then the same thing can happen. I would like to highlight the importance

of the aspiration of stomach content or vomiting in the near drowned victims because, as you are aware nausea and vomiting is very common in patients who become hypoxic and that is the problem with the patient that is near drowned. Figures taken from the Surf Life Saving Association show how common vomiting and regurgitation is in patients who need resuscitation. In those needing cardiopulmonary resuscitation, the experience was that 90% of the victims had either vomiting or regurgitation and overall difficulties with the airway occurred in 50% of these patients. However, whatever the exact mechanism is, the patient finishes up with the adult respiratory stress syndrome.

Large amounts of water may be ingested. That, with the water aspirated and perhaps some fluid resuscitation along the way, leads to a hyperhydration syndrome with an expanded blood volume. This, combined with alveolar and capillary damage, leads to excess amounts of water in the lung and to pulmonary oedema, which is the basic problem in the near-drowned victim. When a person presents to hospital the pulmonary care of the patient must be the initial concern. I do not want to dwell on these various aspects because they are reasonably well known. The techniques that are needed in the pulmonary care are airway control, which depends predominantly on the level of conscious of the patient, oxygen therapy for hypoxia, positive end expired pressure (PEEP) with or without intermittent positive pressure ventilation, depending again on the cerebral status and respiratory status of the patient. These techniques and the initial state of the patient are assessed using clinical examination, blood gases, chest x-ray and in the more severely affected patient invasive haemodynamic monitoring. The cardiovascular care of the patient, which is always done in conjunction with the pulmonary care, is firstly to optimize the patient's cardiac function. That means optimizing the blood volume, perhaps using bicarbonate for the severe acidosis if that is thought to be depressing cardiac function, and the use of a vasopressor and again the assessment is done using techniques with which most people here will be familiar. Other therapies which may be necessary are the use of bronchodilators if bronchospasm is present to a significant degree using either aminophylline or a 82 stimulant. These may have other beneficial effects. Steroids have been used by some people. There is, however, no evidence that they make any difference to the outcome of the patient or to the magnitude of the pulmonary oedema. Antibiotics are in the same class as steroids. They are not necessary unless the patient happened to drown in a sewer.

If humans get water in the lungs profound hypoxia occurs. One of the main reasons for this is that the shunt through the lung can be as high as 70% of the cardiac output. This means that 70% of the blood, as it goes through the lungs, is not being oxygenated. In the patient with anything but a mild degree of hypoxia, then the use of positive end expired pressure produces a dramatic result in these patients. Now whether this is applied as continuous positive airway pressure (CPAP) via a mask in the patient who is conscious, and able to generate adequate respiratory volumes, or it is CPAP via an endotracheal tube in a patient whose level of consciousness is not good but who can still maintain, although cerebration is impaired, adequate ventilatory volumes, or whether intubation, intermittent positive pressure ventilation and PEEP is needed, does not matter. PEEP always has a dramatic effect. Some patients seem to need intermittent positive pressure ventilation especially to open the collapsed alveolar ducts and alveoli that have filled with water. When a significant amount of this occurs, the lungs become very stiff and the amount of work necessary to generate the pressures to initially open these airways in a particularly ill patient cannot be generated by the patient, so often intermittent positive pressure is needed. Sometimes only for a short time in the initial stages and then as improvement occurs, the patients can manage to ventilate themselves if they are assisted with the positive end expired pressure.

PEEP does not decrease the amount of water in the lung but probably expands the alveoli to the point where the amount of water is made a thinner layer so that gas exchange can occur across the alveolar capillary membrane. PEEP is good stuff but unfortunately it is a two-edged sword. The good edge is that it increases the functional residual capacity, in other words, it expands the lung. It certainly decreases the shunt and so allows oxygenation of blood going through the lungs to increase and therefore increases the partial pressure of oxygen in the arterial blood. However, it does have some unwanted effects. The first being an increase in physiological dead space, which means that the patient needs to move, or have moved for him, larger volumes of ventilation to eliminate carbon dioxide adequately. In most cases that is not a significant clinical problem. However the other detrimental effect of a decreased cardiac output certainly is. Whether the patient gets the decreased cardiac output from ARDS or some other cause or from near drowning the problem is the same.

Let us look at exactly why this occurs and what we might do about it. The most probable cause for the decreased cardiac output is the decreased pre-load in the heart. I would ask you to keep in mind what pre-load is. Starling, in his well known articles, did not mention preload as pressures but referred to fibre length, and fibre length is better reflected by an end diastolic volume in whichever heart chamber you are referring to, rather than pressure. While in the healthy heart volume and pressure might be linearly related, in any patient who is ill the compliance, or stiffness, of the chamber can vary enormously and therefore the pressure that one measures may not necessarily accurately reflect the pre-load of the heart. What are the mechanisms by which the pre-load is decreased? The one which we have all been familiar with for a long time, is increased mean intra-thoracic pressure, or increased pleural pressure, decreasing venous return therefore decreasing the cardiac output. There are some other mechanisms which may be important, whether the patient has ARDS or is having PEEP for near drowning or whatever cause. One is the fact that the lung volume, increased by the PEEP, squeezes the heart, and so the ventricle does not relax as well, which means that it has effectively become more stiff. At the same time PEEP and the ARDS cause an increased right ventricular after-load due to the increase in pulmonary vascular resistance, both from the disease process and the extra pressure that PEEP will apply. These can induce a degree of right heart failure with dilation of the right side of the heart. This can shift the ventricular septum across, pushing it into the left ventricle and changing the functional characteristics of the left ventricle causing

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.