The pathophysiology of drowning

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Drowning, near drowning, pathophysiology

Abstract
The effects of drowning, both fatal and non-fatal, on the different major organ systems within the body, and the process of hypoxia are reviewed. Pulmonary fluid aspiration results in an increase in low ventilation-perfusion ratio areas and decreased compliance. However, in most victims variable quantities of the liquid medium are aspirated. It is believed that in at least 10% of drowned people laryngospasm occurs, but practical evidence conflicts with the theory that the closed larynx keeps water from entering the trachea. Widespread atelectasis occurs after surfactant is denatured, leading to large shunts and perfusion mismatch. Five to twenty five per cent of patients progress to life-threatening acute respiratory failure. The effects of immersion on the cardiovascular system are an increased venous return and a central shift of blood volume, but the cardiovascular system has been stated to show remarkable stability, and the changes in blood volume are not significant enough to be life threatening. There are differences in biochemical changes in the blood with inhalation of fresh water and sea water. However, these are minimal and not clinically significant. A severe lactic acidosis develops secondary to the hypoxia. The most common renal complication is oliguria due to acute tubular necrosis. The roles of the diving reflex and hypothermia in cerebral protection are still not well understood. The overall pathophysiological processes in drowning and near drowning are complex but ultimately are determined by the degree and duration of hypoxia.

Introduction

Drowning is the most common cause of death among divers, in both those using scuba and snorkelling equipment. Something usually happens to the diver initially to predispose them to drowning, for example, an out-of-air situation or, with breath-hold diving, loss of consciousness after having hyperventilated before descent, often referred to (incorrectly) as 'shallow water blackout'.

When someone does die from drowning, it is not simply a case of water flowing into their lungs. The effects of drowning and near drowning on the different major organ systems within the body, and the process of hypoxia will be reviewed. The effects of cold water immersion, the diving reflex and hypothermia and cerebral protection will also be discussed.

Respiratory response to immersion

With vertical submersion, there is an overall increase in the work of breathing of 65%, due to the pressure of water compressing the lower body. This causes displacement of blood into the pulmonary circulation, cephalic movement of the diaphragm and minor compression of the chest wall. This is generally not a problem in healthy people.[1]

With submersion of the head comes a voluntary apnoea. The 'break point' of this voluntary action is determined mainly by the arterial carbon dioxide level. When the break point is reached, an involuntary inspiration occurs.[2]

Pulmonary fluid aspiration destroys or dilutes surfactant (salt water versus fresh water near drowning), disrupts the alveolar capillary membrane, and damages the pneumocytes causing atelectasis. This results in an increase in low ventilation-perfusion (V/Q) ratio areas and decrease in compliance.[3] Aspiration of vomitus is common.[4] However, in most victims, variable quantities of the liquid medium are aspirated. After a latent period, which can extend to 48 hours, 5–25% of patients progress to life-threatening acute respiratory failure – the so-called 'secondary drowning syndrome'.

It is commonly believed that in at least 10% of drowned people laryngospasm occurs, thus occluding the airway. This is based on the finding that 10% of drowning victims at autopsy have little or no water in their lungs.[4] However, these are autopsy findings, not necessarily proving the absence of water in the lungs at the time of drowning. Fresh water is readily absorbed from the lungs and most dry lung autopsies were from fresh water victims.[6] Also, anaesthetists have been able to intubate an asphyxiated patient after agonal relaxation of the larynx in patients with laryngospasm. Hypoxia and hypercarbia are both factors leading to laryngeal relaxation. This practical evidence conflicts with the theory that the closed larynx keeps water from entering the trachea.

Associated with laryngospasm, there is a mucous outpouring in the bronchi and bronchospasm.[2] Both of these factors may help to prevent water entry with terminal relaxation. Laryngospasm occurs more commonly in adults than children and is believed to be facilitated by other factors, for example alcohol ingestion.

Once water enters the lungs, compliance falls and pulmonary vasoconstriction and pulmonary hypertension

occur. As described earlier, widespread atelectasis occurs after surfactant is denatured, leading to large V/Q mismatch. Drowning in salt water may differ from that in fresh water, in that pulmonary oedema is facilitated by the osmotic pull from salt water ions.[2] However, this effect can also happen due to foreign material in fresh water. Finally, after the period of involuntary aspiration, there is a secondary apnoea, followed by gasping and later respiratory arrest. Death ultimately ensues from apnoeic hypoxia.[6]

Cardiovascular response

The effects of immersion on the cardiovascular system are an increased venous return and a central shift of blood volume.[7] As a result, there is an increase in cardiac output of one to two thirds. The increase in central blood volume stimulates stretch receptors leading to a diuresis to counter the apparent increased blood volume.[8]

After water enters the lungs of the 90% of drowning victims without 'dry' lungs at autopsy, the cardiovascular system has been stated to show remarkable stability.[2] Theoretically, with fresh water entering the lungs, the greater osmolality of blood draws water from the alveoli into the circulation. This leads to an increase in blood volume. Again, the greater osmolality intracellularly draws water into the cells and thus leads to haemolysis.[3] This process also leads to an increase in potassium. However, haemoglobin and haematocrit levels in victims drowned in fresh water are not significantly altered, so the theory may be incorrect. Salt water inhalation theoretically leads to osmotic flow of intravascular fluid into the alveoli, because salt water is hypertonic relative to serum, which would cause a decrease in blood volume.[9]

In both cases, the changes in blood volume are generally not significant enough to be life threatening. Although there are differences in biochemical changes in the blood with inhalation of fresh water and sea water, these are minimal and not clinically significant.[6]

A reduction in cardiac output may occur due to immersion diuresis and decreased myocardial function from hypoxia, induced electrolyte abnormalities and acidosis.[7,8] These cardiovascular changes and an increase in pulmonary vascular resistance are independent of the tonicity of the fluid aspirated and are a direct effect of hypoxia.[7]

Blood pressure changes during drowning are due to hypoxia, which initially causes marked sympathetic stimulation with tachycardia and an increase in systemic resistance, followed by myocardial failure and hypotension. Blood pressure changes may also be due to the development of arrhythmias. A variety of ECG changes are seen, varying from no change, ventricular fibrillation (VF) leading to arrest, and extreme bradycardia in cold water as hypothermia develops. Atrial and ventricular arrhythmias are a consequence of catecholamine excess, acidosis and hypoxia. In summary, any arrhythmia may develop.

Serum electrolytes are rarely abnormal, except for the acidosis and an elevated serum magnesium.[9] Near drowning in industrial fluid may, however, cause electrolyte abnormalities. Haemolysis and rhabdomyolysis may occur early or be a later complication of sepsis.[10] Hypermagnesaemia will depress myocardial function and cause peripheral vasodilation.

Hypoxia is the inadequate oxygenation of cells and is caused by apnoea as in the case of laryngeal spasm or inadequate delivery of oxygen to the lungs. A low partial pressure of oxygen is detected by the carotid and aortic bodies and the direct lack of oxygen to the respiratory centre in the brain also generates some of the following response. Hypoxia leads to vasodilatation and an increase in cardiac output.[4] Another feature of hypoxia is pulmonary hypertension. This is caused by constriction of the pulmonary artery as a response to the low partial pressure of oxygen. Subsequently, right ventricular afterload failure may develop.[7]

A severe lactic acidosis develops secondary to the hypoxia. Eventually, if hypoxia is severe the myocardium will fail. When there is a low level of oxygen and a high level of carbon dioxide in the blood, the cerebrovascular response is vasodilatation to increase cerebral blood flow. Eventually, an acidosis will develop in the brain, with subsequent cerebral oedema and raised intracranial pressure.[4] Severe hypoxia will affect brainstem centres and death will ultimately result after respiratory arrest.

In summary, there are a wide range of sometimes conflicting factors – immersion, aspiration, hypoxia, fluid and electrolyte shifts, sympathetically mediated cardiac rate and rhythm changes, the diving reflex and hypothermia – that may impact on the cardiovascular system.

Gastric and renal response

Swallowing of water occurs, particularly if the water is choppy, and is more common in colder water when swimming is more difficult due to impaired coordination. Vomiting often occurs and may lead to aspiration. The water that is swallowed also contributes to cooling of the body.[1]

The incidence of renal failure following near drowning is unknown but is certainly less than the frequency of lung, brain or heart injury. The most common complication is oliguria due to acute tubular necrosis (ATN). ATN in near drowning is thought to be a consequence of hypoxia, hypotension, haemolysis and rhabdomyolysis.[10]

Behaviour during drowning

Behaviour of drowning victims varies depending on the events leading to their drowning. A series of 100 fatalities reviewed by Edmonds, Walker and Scott[5] reported that over

half of the victims showed no change in behaviour. These 'quiet' drownings, when the victim does not struggle, are attributed to shallow water blackout, the effects of cold, alcohol/drug intoxication, or other medical conditions, for example arrhythmias or cerebral arterial gas embolism.

Animal studies have shown an initial struggle on submersion, sometimes with inhalation, followed by a suspension of movement. There is frequent swallowing and then a late, violent struggle. Finally, convulsive movements occur and exhalation.[4] Observations of humans drowning have shown the same sequence of events. Survivors of near-drowning episodes have reported a sense of panic, which is often described as a key factor in drowning but often cannot be observed by witnesses to the incident.

Diving reflex

The role of the diving reflex in drowning and near drowning is still not well understood. The reflex is mediated by stimulation of the ophthalmic division of the fifth cranial nerve.[1] It results in apnoea, peripheral vasoconstriction, shunting of blood to the cerebral and coronary circulations, and bradycardia.[2,4] The reflex occurs independently of chemoreceptor and baroreceptor influence.

The diving reflex is more pronounced in infants than in adults, enhanced by cold and anxiety, and is modified by the equipment being used by the diver.[1] For example, a diver wearing a drysuit with only the face exposed will have a more profound response than a diver without the drysuit. Fifteen per cent of people are said to show a profound diving response.[1] The diving reflex may be a significant part of why some children have been able to survive with minimal or no functional loss after prolonged periods of immersion.

Immersion in cold water

Upon immersion in cold water, there is often an initial involuntary gasp. In most cases, this appears to be controlled by the diver until the head is again clear of the surface. This may be followed by uncontrolled hyperventilation.[2] Due to the initial large inspiration, tidal breathing occurs close to total lung capacity, thus a feeling of dyspnoea occurs. The maximum breath-hold time in cold water becomes only about 10 seconds. Respiratory drive increases in water colder than 25°C, and is maximal at 10°C.[1]

It is easy to imagine in colder water, particularly if it is choppy, that aspiration will occur, as the swimmer is breathing a lot harder and is unable to hold their breath for very long. Aspiration is also more likely to occur when core body temperature has fallen to about 34°C. Impaired neuromuscular function results with cold peripheries when limb temperature is about 28°C, and with a core temperature of between 33°C and 35°C.[1]

Vasoconstriction occurs in cold water, leading to both increased arterial and venous blood pressures. This centralises blood volume, and may double cardiac output. Urine output increases by up to a third of that seen in thermoneutral water.[1] However, vasoconstriction of the scalp is less than elsewhere and so more heat is lost from the head.

Shivering occurs to produce heat but this function ceases at about 33°C. Ventricular fibrillation occurs at about 28°C core temperature and is either mediated vagally or from excessive catecholamine release. Asystole occurs between 24°C and 26°C.[1]

Hypometabolism occurs with decreased temperatures. There is 6–7% decrease in cerebral blood flow for each degree of temperature loss. Loss of consciousness occurs at about 30°C core temperature, and cerebral activity ceases at 22°C.[1] Children are affected more by cold and cool down quicker than adults, as their body surface area to mass ratio is greater and they have less fat to insulate them.

Hypothermia and cerebral protection

The true mechanism of cerebral protection by cold is unknown. It is thought to work with the mammalian diving reflex, which shunts blood to the brain after peripheral vasoconstriction, and hypometabolism due to the cold. Theoretically, it is not possible physiologically to cool an immersed person quickly enough to prevent hypoxic brain damage from occurring.[1]

The rate of change in core temperature is dependent on the water temperature, movement of the water against the skin, body surface area to mass ratio, insulation (for example fat, wetsuit or drysuit), peripheral circulation, and conditions affecting the person (for example alcohol or injury). Even with these conditions 'optimised' it is still not thought possible to cool a body rapidly enough to afford cerebral protection.[1]

Conclusions

Drowning eventually leads to death via respiratory arrest resulting from hypoxia, but before that the effects on the body involve all the major organ systems. The effects on each contribute to the responses and effects on the other systems, making the pathophysiological process quite complicated. Conditions such as cold and the diving reflex also compound the physiology of drowning. However, much has yet to be learnt about the process and of what value cold and the diving reflex may have in the survival or treatment of near-drowned patients.

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Causes of drowning in divers

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Abstract
Drowning is the endpoint of many diving fatalities for a variety of reasons. Most divers are not well enough trained and should be encouraged to undertake more training beyond basic recreational open water diving. Unexpected equipment failures may sometimes occur. How to cope with these problems is not always adequately taught during initial training. A quarter of equipment failure incidents result in morbidity, yet a surprising number of divers have poorly maintained or serviced equipment. Running out of air is one of the most common serious diving incidents, which suggests that there is something wrong with how divers are trained. Air integrated gauges/computers, most of which have audible warnings, are useful tools for gas supply management and should be utilised more than at present. Wrist mounted displays are more readily seen than dangling consoles. The Cave Diving Association of Australia training and certification system is an example of successful self-regulation in recreational diving. Medical conditions may predispose divers to a diving incident. Increasingly, risk recognition and management is being emphasised in the medical assessment of diving candidates.

Introduction

Prevention is the best method of stopping divers drowning. Only fish are fit to dive, so humans are at risk every time they enter the water. Drowning is the endpoint of many diving fatalities for a variety of reasons. The following is an experienced scuba diver/general practitioner's personal view of this subject. I will comment on aspects of prevention, diving equipment, diving practice and medical problems that I consider relevant.

Prevention

How might we reduce the number of divers drowning? My belief is that most divers are not well enough trained in general, and the preponderance of untrained or inexperienced divers in the fatality statistics bear this out.[1,2] We should be encouraging divers to undertake more training and particularly for some of the more specialised aspects of diving such as wreck diving or cave diving. We should encourage people who want to do anything more than basic recreational open water diving to obtain the appropriate training. It really is worthwhile doing extra training, and there are many training modules available these days.