ILS POLICY STATEMENT N° 12

SHALLOW WATER BLACKOUT

BACKGROUND

Shallow water blackout, also known as shallow water drowning or death following hyperventilation with apnoea diving (breath hold diving), is a loss of consciousness and sudden unexplained death in the water. Apnoea diving is used in many water based activities including underwater hockey, big wave surfing, spear fishing, synchronised swimming, underwater rugby, competitive free diving and by recreational swimmers attempting to swim a distance underwater.

The definition of shallow water drowning is very variable with numerous terms describing the same phenomena. There are also various phenomena described using the same terms! As a result of no standard definition or classification, records of incidence are vague. In a survey of Australian snorkeling deaths between 1987 and 1998 20% (12 of the total 60 deaths) of the deaths were attributed to hypoxia from breath holding and hyperventilation.[i] The possible physiological mechanisms resulting in shallow water blackout will be discussed below. There are many incidents that result in sudden unexplained death in water that are not considered shallow water blackout and hence not discussed. They include:

- Non-swimmer, weak swimmer or injured swimmer who has attempted to remain at the surface of the water but has become submerged.
- Seizure related disorders, including atypical epileptic seizures, which may have no noticeable motor component.
- Trauma related incidents where a swimmer is unconscious due trauma prior to entering the water or becomes unconscious due to collision upon entering the water (e.g. submerged object, or water surface tension if entry from a significant height, “tomb stoning”).
- Diving response: Apnoea, marked peripheral vasoconstriction and profound bradycardia, seen with facial immersion in cold water[ii].
- “Cold shock” response: This is seen when the body is immersed in cold water[1]. Initial rapid inhalation “gasp”, uncontrolled hyperventilation then occurs and subsequent swim failure due to asynchrony of ventilation to swim stroke. It is associated with immediate intense reflex vasoconstriction, increase in heart rate, increase cardiac output and hence rise in arterial and venous blood pressure. Myocardial workload is increased and additionally in those with existing ischaemic heart disease can produce a fatal cardiovascular event (arrhythmia and/or infarction).[iii]
- Divers using self contained underwater breathing apparatus (SCUBA) with re-breathing circuits. Case reports of the CO_2 scrubbers functioning poorly have resulted in hypercapnia and associated narcosis. This has also been termed “shallow water blackout”.[iv,v]

Many of these conditions will have no findings on post mortem and cause of death will be based upon eye witness accounts which, without background knowledge or skills, may prove inaccurate.

[1] Cold water responses are seen at temperatures below $25^{O}C$[1], according to the Federation Internationale de Natation (FINA) rules water temperature during swimming competitions shall be $25\text{-}28^{O}C$.[15]

Physiological Explanations of the Phenomenon of shallow water Blackout.

1. Hypoxia associated with Hypocarbia

Craig (1976) described 58 cases of loss of consciousness during underwater swimming. He suggested this was due to hyperventilation reducing the arterial partial pressure of carbon dioxide ($PaCO_2$), thus removing the respiratory drive. In the interim the oxygen content of the blood is decreased, due to consumption by the tissues, to a point at which the brain no longer functions. As unconsciousness develops muscle tone is reduced allowing any gas volume above the expiratory reserve volume to be passively exhaled and the drowning process to commence. In the majority (but not all) of the survivors (n=34) they had an urge to breath but had loss of consciousness without warning. Unlike a sudden cessation of brain perfusion, as seen in a primary cardiac arrest, the oxygen content of the tissues, blood and lungs is significantly diminished and so the onset of hypoxic brain injury is quicker. As a consequence the rescuer will have a reduced window of opportunity to resuscitate the casualty.[vi]

The partial pressure of alveoli CO_2 ($PACO_2$) at the breaking point of a breath hold is the same during exercise as it is at rest.[vii] Pendergast *et al* (2006) showed that the ventilatory response to CO_2 is variable between individuals and that respiratory muscle training can normalise the respiratory CO_2 sensitivity for individuals with high or low sensitivity. They hypothesised this was due to modulation of the effector side of the reflexive respiratory control i.e. the motor response to chemoreceptor stimulation.[4]

During an apnoea dive oxygen is taken from the lungs and utilised in the tissues. This causes a decrease in the PAO_2 but due to co-operative binding with haemoglobin a drop in total oxygen content is delayed, and the PAO_2 will increase as intrathoracic pressure increases. The $PACO_2$ will remain at a relatively static level for the duration of the dive. This is due to the increase in intra thoracic pressure due to hydrostatic pressure increase from the surrounding water with subsequent reduction in lung volume, (Boyles law), consumption of oxygen and the relationship between $PACO_2$ and the partial pressure of arterial CO_2 ($PaCO_2$). Carbon dioxide passes into the alveoli by passive diffusion which relies on a pressure gradient between the delivering blood vessel and the alveolus. (Normally $PACO_2$ is less than $PaCO_2$ and so CO_2 is released to the atmosphere upon expiration.) As the $PACO_2$ increases it will reach equilibrium with the $PaCO_2$. If depth increases CO_2 may even pass back into the blood from the lungs. On ascent the lungs will expand decreasing the partial pressure of CO_2, (and hence $PaCO_2$ and so less respiratory drive), and decrease the partial pressure of O_2 in the alveoli (PAO_2.) This results in a hypoxic gas mixture in the lungs. The effect of hydrostatic pressure on alveoli gases is usually only seen with dives beyond a depth of 5 metres. [viii] Shallow water blackout incidents are commonly reported involving dives to depths far less than this, and the effect of depth cannot therefore wholly account for the phenomenon of shallow water blackout.

Reduced CBF in hypocarbia

Shift in oxyHb dissociation curve to left impairing tissue 02 delivery with hypocarbia

Inc capacity for c02 carriage in deoxygenated blood via haldane effect limiting C02 rise

The central and peripheral respiratory chemoreceptors in the medulla oblongata and Aortic/Carotid bodies respectively respond to changes in hydrogen ion concentration ($[H^+]$), This in turn is proportional to the $PaCO_2$. Hence a rise in $PaCO_2$ will cause a proportional rise in $[H^+]$ this stimulates the chemoreceptors causing an increase in ventilation – or the urge to breathe if apnoeic. During exercise an oxygen debt will occur and CO_2 will be produced stimulating the body to breathe. If hyperventilation has occurred prior to diving, lowering, total body CO_2, hypoxia will cause a loss of consciousness before a sufficient level of CO_2 is produced to develop the urge to breathe. Of note in

many of the case studies there is a preoccupation with a goal (competing against colleague, longer time submerged etc.) and it is speculated this may alter the interpretation of physiological warnings such as the urge to breathe.[ix,x]

2. Alteration in Free Calcium Ions.

Central chemoreceptors exist which respond to low levels of oxygen tension. The action of hyperventilation removes CO_2 resulting in an increased serum pH. This decreases the unbound portion of calcium ions. As calcium is involved in both muscle contraction and neurotransmitter signal cascade, this will result in reduced transmission of neurotransmitters and loss of motor control. If a swimmer is submerged, loss of motor control will result in swim failure and a drowning event.

3. The "Samba" Phenomenon

Competitive free divers describe a phenomenon known as "*Samba".* This presents as a loss of motor control and associated bilateral fine tremor with head bobbing post static breath holding. The suggested mechanism is hypoxaemia at GABAnergic[xi]and cholinergic[xii] neurotransmitters causing a reduced signal production. These neurotransmitters are associated with signal transmission in the central nervous system and muscle innervations. In addition to reduced unbound calcium they may provide an explanation as to why the oxygen receptors do not respond appropriately to prevent loss of consciousness and reduced motor activity underwater.

4. Air embolism

During an apnoea dive, attempts to breathe against a closed glottis to reduce the urge to ventilate can cause an increased intrathoracic pressure and subsequent air embolism (demonstrated at post mortem in the brain and right ventricle)[xiii] leading to cardiovascular compromise and death.

STATEMENT

1. Prevention is better than cure. While it is appreciated that many free divers will continue to advocate hyperventilation as a method of extending breath hold dive time it should actively be discouraged due to the potentially fatal consequences. Any rescuer who observes an individual performing a breath hold dive should have a low threshold for initiating rescue if the swimmer demonstrates signs of swim failure or ceases activity whilst submersed in the water.

2. Swimming underwater whilst breath holding is safer if effective physical activity continues for the duration the swimmer is under water, hyperventilation has not been performed prior to entry, no attempt to breath against a closed glottis occurs and the water is less than 5m deep.

3. In the case of unconsciousness or apparent cardiac arrest following recovery of a casualty from a breath hold dive the resuscitation protocols appropriate to the level of training of the rescuer should be commenced. This may include ventilation with supplemental oxygen, chest compressions and the use of an automated external defibrillator. If advanced life support is available consideration should be given to ensuring a definitive airway, Intravenous drug administration (including reversible metabolic disturbances e.g. hypocalcaemia[xiv]) and transfer to an appropriate critical care facility.

4. Shallow water blackout as a cause of death is a difficult diagnosis to make with certainty. They are many other causes of death associated with submersion in water that have similar presentations.

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