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John H. Pearn

Lady Cilento Children's Hospital, Queensland, AU, j.pearn@uq.edu.au

Richard C. Franklin

James Cook University

Amy E. Peden

Royal Life Saving Society-Australia

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Hypoxic Blackout: Diagnosis, Risks, and Prevention

John H. Pearn

Lady Cilento Children's Hospital and Royal Life Saving Society—Australia

Richard C. Franklin

James Cook University and Royal Life Saving Society—Australia

Amy E. Peden

Royal Life Saving Society—Australia

Hypoxic blackout, also called underwater blackout syndrome, is a distinct and preventable cause of drowning. The sudden and unexpected death of a young, fit swimmer or diver, almost always male, necessitates the consideration of a differential diagnosis which includes four syndromes—preexistent cardiac disease, electrical conduction abnormalities, epilepsy, and hypoxic blackout. The pathophysiology of hypoxic blackout (overriding the carbon dioxide sensor by presubmersion hyperventilation) may be induced by autonomic conflict between cold shock diving reflexes in certain predisposed individuals. Death occurs in both public and private swimming pools and in the sea, and case series include those training for underwater hockey, synchronized swimming, free diving, and playful submersion endurance challenges. The sole preventive stratagem is advocacy for awareness of risks, suitably targeted to “at risk” groups.

Keywords: aquatic risk management, fatal/nonfatal drowning, underwater swimming, water safety

Sudden unexpected death, in the context of recreational or domestic activities, is challenging to forensic (and later, coronial) analysis. “Natural” deaths while driving, the majority of which are caused by ischemic heart disease, comprise one example (Cheng & Whittington, 1998). Sudden death in athletes and rowers is another (Patel & Elliott, 2012). The phenomenon of sudden loss of consciousness in a sober and voluntary (discretionary) submerged swimmer is represented in all fatal drowning statistics.

All the authors are with Royal Life Saving Society—Australia, New South Wales, Australia. John Pearn is also with Lady Cilento Children's Hospital, South Brisbane, Queensland, Australia. Richard Franklin is also with the College of Public Health, Medical and Veterinary Sciences, James Cook University, Townsville, Queensland, Australia. Address author correspondence to John Pearn at j.pearn@uq.edu.au.

One preventable cause of drowning resulting from loss of consciousness is hypoxic blackout, a specific phenomenon and a distinct and preventable drowning syndrome. It has a high fatality rate. The syndrome typically involves young males under the age of 40 years (Australian National Coroners' Information System, 2015). The condition appears both in the literature (and in oral history) under a variety of other names, including "shallow water blackout," "underwater blackout," "underwater blackout syndrome," "sudden underwater blackout syndrome (SUBS)," "breath-holding blackout," and "free-diver blackout." The American Red Cross, USA Swimming, and the YMCA (USA) jointly use the term "hypoxic blackout" (American Red Cross, USA Swimming, & YMCA [USA] 2015). The consensus for a standard name is currently (2016) under review by the Medical Committee of the International Lifesaving Federation.

This paper reviews the specificity of this phenomenon and discusses its differential diagnosis, highlights the risks, and describes some preventive stratagems we have instituted as outreach advocacy through the Royal Life Saving Society—Australia (RLSSA). We highlight this preventive challenge as, in contradistinction to the falling rates of several other drowning syndromes, the death rate from hypoxic blackout has remained unchanged over recent decades.

Definition

We use the terms "underwater blackout syndrome" or "hypoxic blackout" as a modification of the definition originally proposed by Phillip Dickinson (Dickinson, 2014). We define the condition as "loss of consciousness in the underwater swimmer or diver, during an apnoea submersion preceded by hyperventilation, where alternative causes of unconsciousness have been excluded."

Differential Diagnosis

In the aquatic domain, the sudden and unexpected death of a swimmer, not in an involuntary drowning sequence, leads to the generation of a differential diagnosis of possible causes (Pearn, 2011). These include (a) preexistent organic cardiac disease, either coronary artery disease or cardiomyopathy; (b) preexistent cardiac electrical conduction abnormalities; (c) epilepsy; and (d) the phenomenon of hypoxic blackout or "shallow water blackout" (Dickinson, 2014). In rare cases, the differential diagnosis may also include homicide and suicide, particularly in those instances where an isolated drowning death has occurred in which the drowning episode was not observed. The diagnosis of drowning is one of the most difficult in the domain of forensic pathology and one that often has the most significant coronial—and sometimes legal—sequelae (Pearn, 2008).

After the sudden unexpected death of a swimmer, the specific diagnosis is critical for three specific reasons. First, if a (preexistent) genetic cardiac conduction abnormality was present, other individual family members (particularly first-degree relatives) may be identified and prophylactic medications provided. Such include the long QT_c syndrome and other rarer genetic channelopathies. Second, the misdiagnosis of epilepsy has significance for all concerned. It has been shown that one third of a group of unrelated subjects with the long QT_c syndrome were

initially misdiagnosed as having an epileptic seizure disorder, resulting in years of inappropriate treatments (MacCormick et al., 2009). Third, the forensic and coronial sequelae of such sudden aquatic deaths always have potential legal sequelae including tort action.

One cause of aquatic misadventure and of this syndrome, hypoxic blackout, is totally preventable. The risk of incorrect diagnosis can be reduced by careful history taking and the reporting of observed events that surrounded the sudden loss of consciousness in the water. Aquatic misadventure in previously fit and asymptomatic swimmers typically occurs during racing competition, training for aquatic sports that necessitate prolonged breath-holding, and endurance underwater swimming and diving (Albertella et al., 2011). In the context of cardiac causes, whether organic or malconductive, the swimmer typically will be seen on the surface to have stopped swimming and may manifest transient peculiar behavior for several seconds. By contrast, in the case of hypoxic blackout, the victim may be seen hyperventilating preimmersion, does not surface, and subsequently is found unconscious (or dead) on the bottom of the pool or sea. Accounts of bystanders as first-person observers, and by lifeguards, swimming teachers, or relatives (in the case of children or adolescent victims), or companions—such accounts may be of compelling diagnostic significance in coronial investigations and in emergency room discussions during the resuscitation and life support of survivors.

Pathophysiology

Traditional beliefs about the pathophysiology of hypoxic blackout are well understood. Rising plasma carbon dioxide levels are sensed by receptors in the medulla, which in turn send electrical impulses to the conscious areas of the frontal cortex. These induce an irresistible impulse to surface and ultimately to inspire. This is the moment of “inspiration break point,” whether head-out or not. In the voluntary (discretionary) submerged swimmer or diver, the atavistic, irresistible urge to surface occurs before falling plasma oxygen levels cause unconsciousness. If a swimmer unwisely performs extensive presubmersion hyperventilation, the pre-dive carbon dioxide levels fall dramatically, and the subsequent CO₂ rise during submersion may not be sufficient to trigger the compulsive instinct to surface before consciousness is lost, this latter due to falling plasma (and hence brain) oxygen levels. The inspiration break point then occurs in the unconscious submerged victim, and the drowning sequence inexorably follows.

During both voluntary and involuntary submersion, whether following hyperventilation or not, two sequential autonomic reflexes occur in all marine and terrestrial mammals, including humans. The first is the “cold shock response,” in which the afferent cold thermoreceptors induce immediate tachycardia, a respiratory gasp, hyperventilation, peripheral vasoconstriction, and hypertension (Tipton, 1989). These physiological responses are followed within 30 s or so (slower in humans than in diving mammals; Lin, 1988) by a parasympathetic reflex, called the diving response. This protective reflex is strongest in children and young adults. The diving reflex comprises initial bradycardia, peripheral vasoconstriction, decreased cardiac output, increased arterial blood pressure, and a redistribution of oxygen perfusion by shunting from cutaneous and splanchnic vasculatures to cerebral and cardiac

vessels. Splenic contraction may be extreme (Espersen, Frandsen, Lorentzen, Kanstrup, & Christensen, 2002).

Professor Michael Tipton recently has proposed that in certain vulnerable individuals, these two “antagonistic” autonomic reflexes may result in cardiac dysrhythmias (Shattock & Tipton, 2012), and specifically that presubmersion hyperventilation might trigger such “autonomic conflict.” This persuasive hypothesis includes the predictions that individuals who might be vulnerable to such autonomic conflict include those with preexisting organic cardiac pathology or electrical conduction channelopathies (Tipton, 2015).

In the traditional view of posthyperventilation hypoxic blackout, the balance between the two chemical sensors (carbon dioxide and oxygen) varies according to familial or individual differences and to varying adaptation to hypoxic training. These variations in turn imply that predictive risks cannot be ascertained (Ferretti & Costa, 2003). What is certain is that presubmersion hyperventilation carries definite risks although their quantification remains unknown at present.

Equally uncertain is what effect, and potentially what extra risk, presubmersion hyperventilation has upon those with clinically silent genetic channelopathies. At least one in every 2,500 members of the general Western population has the long QT_c syndrome. Among this group, the risk of unexpected misadventure is high among young males (Goldenberg et al., 2008), the same group known to be vulnerable to hypoxic blackout, sometimes mislabeled as “shallow water blackout.”

Prevention

The risk of presubmersion hyperventilation has been known since 1961 (Craig, 1961) and has been reiterated and reinforced in both the adult and pediatric literature since that time (Craig, 1973). Our constant monitoring of the causes of all fatal drownings in Australia (RLSSA, 2015) has shown, nevertheless, that in contrast to other drowning rates that have continually been reduced, the rate of hypoxic blackout fatalities has not fallen.

Advocacy to reduce deaths from hyperventilation-induced hypoxic blackout faces two challenges. The first is that in the absence of exposure denominators, there exists no current quantifiable risk. The second is that in the face of uncertainty about those at specific risk, it is both prudent and necessary to make the entire swimming and diving population aware of such risks. Specifically, in the case of children and vulnerable adults, it is appropriate to advocate strongly against the practice of significant presubmersion hyperventilation.

Adventure literature, especially boys’ adventure novels, is replete with heroic feats of endurance free diving (Forester, 1964). In the past, many schools have included events of endurance underwater swimming as part of school swimming carnivals. One example was an event known as the “Long Dive”—won as an older adolescent by Valentine McDowall (1881–1957), later a pioneer pediatric radiologist in Australia (Pearn, 2014), with an underwater swim of 159 feet 6 inches (49 m; McDowall, 2010). As the syndrome of hypoxic blackout almost entirely affects boys and young male adults (Craig, 1973; Australian National Coroners’ Information System, 2015 [*Cumulative Register*, 2015]), this offers the opportunity for a more targeted intervention.

Other targeted at-risk populations are free divers and spear fishers; those training to compete in activities such as underwater hockey, synchronized swimming, and free diving, most commonly performed in public pools; and those recreating in home swimming pools and hyperventilation activities, either as endurance challenges or occasionally as pranks. Different drowning syndromes necessitate specific preventive responses. Of the four options available (i.e., heightened awareness through public education, better ergonomic design, safety legislation, and improved rescue and resuscitation), only increased advocacy to promote an awareness of this specific risk is available for this syndrome of hypoxic blackout. Albeit the least effective preventive stratagem, we believe that there is no place for a defeatist ethos in this context. Targeted awareness campaigns have been effective in reducing the risk of surf drownings and some types of boating misadventure. Similar to the preventive options to reduce infant bathtub drownings and deaths of rock fishers, the challenge is great.

In 2015, several international bodies revitalized the preventive advocacy to reduce risk of hypoxic blackout. The Quebec Lifesaving Society (Société de Sauvetage du Québec) published brochures for private pool owners that specified “establish the rules: no diving head first, no running around the pool, no breath-holding games” (Quebec Lifesaving Society, 2015). The tripartite policy of the American Red Cross, USA Swimming, and the YMCA of the USA condemned “the practices of hyperventilation preceding underwater swimming and extended breath-holding in the water as dangerous and potentially deadly activities” (American Red Cross, USA Swimming, & YMCA [USA] 2015).

The RLSSA has produced literature for members of the aquatic industries to highlight these same risks. RLSSA also teaches lifeguards and facility staff to discourage the practice of presubmersion hyperventilation at public pools, where half the fatalities from hypoxic blackout occur. We believe that increased advocacy will help reduce this otherwise preventable cause of drowning.

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