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REVIEW ARTICLE

Cold Water Immersion Syndrome and Whitewater Recreation Fatalities

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Sudden death during whitewater recreation often occurs through understandable mechanisms such as underwater entrapment or trauma, but poorly defined events are common, particularly in colder water. These uncharacterized tragedies are frequently called flush drownings by whitewater enthusiasts. We believe the condition referred to as cold water immersion syndrome may be responsible for some of these deaths. Given this assumption, the physiologic alterations contributing to cold water immersion syndrome are reviewed with an emphasis on those factors pertinent to flush drowning.

Keywords: drowning, kayaking, water sports

Introduction

Previous publications suggest whitewater sports are relatively safe compared with other adventure sports such as scuba or mountaineering.[1,2] Risk, however, is highly dependent on environmental conditions experienced during the activity; thus, such generalizations may be misleading. For instance, danger is greatly attenuated by avoiding the most lethal situations arising in each particular sport, such as deep diving or extreme altitude in mountaineering. Risk can also be reduced by understanding not only the modes of death associated with a given pursuit but the specific conditions preceding lethal accidents. An American Whitewater Association (AWA) study on nonmotorized craft between 1995 and 1998 suggests the greatest risk of death in whitewater sports involves entrapment in strainers (trees) and sieves, flood conditions, or exposure to large hydraulics.[1] The same study further recognizes the lethal impact of improper equipment or cold water conditions but considers them secondary causes.

A scenario referred to as "flush drowning" by whitewater enthusiasts involves fatalities that occur without continuous underwater entrapment or significant trauma, most commonly associated with high flows or cold water conditions. A review of accident data from the AWA suggests these flush drownings contribute significantly to the mortality burden in whitewater recreation.[3] Because flush drownings often defy medical explanation, accident reports commonly attribute the death to heart problems, hypothermia, or traditional drowning mechanisms. We propose the possibility that some flush drowning events, particularly those in colder water, are a manifestation of cold water immersion syndrome. In light of these assumptions, we provide a review of current concepts in cold water physiology as applied to understanding and preventing mortality in whitewater sports.

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Cold water immersion syndrome

Cold water immersion syndrome comprises the respiratory and autonomic physiologic responses after sudden immersion in cold water. The association between cold water and immersion mortality is largely anecdotal or inferential. Many cold water drownings happen among strong swimmers within feet of shore, suggesting some degree of incapacitation.[4] United States Coast Guard accident data suggest that a higher relative percentage of boating accidents during winter months result in death.[5] Likewise, Alaska has 10 times the overall national boating fatality rate, with a high percentage of whitewater deaths attributed to flush drowning.[3,6] Although the definition of cold water is highly variable, the physiologic changes in humans associated with both cold water

immersion syndrome and the human diving reflex begin below 21 to 25°C.[7,8] Recreational whitewater rivers in North America are frequently cold enough to induce cold water immersion syndrome; for example, United States Geologic Survey hydrological data for the Arkansas River drainage in Colorado suggest water temperatures in the warmest mountainous portions of the river only exceed 20°C briefly in the late summer.[9] Rivers in very hot climates may run well below 25°C, particularly rivers fed with discharges from the bottom of deep lakes. The AWA accident study suggests cold water is a contributing factor in one third of whitewater deaths, but the study defined cold water as under 10°C, well below the upper threshold for cold water immersion syndrome.[1]

Early concepts of cold water immersion syndrome were codified into 4 discrete stages by researchers at the University of Portsmouth in the 1980s.[10] Among these 4 divisions, stage 1 (cold shock) and stage 2 (swimming failure) are the most germane to whitewater accidents, although the third stage (hypothermia) or fourth stage (circumrescue collapse) can occasionally contribute. Adaptive responses to cold water immersion not addressed in this 4-stage model include but are not limited to cellular transcription of cold shock proteins, initiation of shivering thermogenesis, and increased metabolism.[7,11] The first stage of cold water immersion syndrome, the cold shock response, includes a group of reflexes lasting under 5 min and initiated by thermoreceptors sensing rapid skin cooling.[12,13] Water has a thermal conductivity 25 times and a volume-specific heat capacity over 3000 times that of air; subsequently, surface cooling is precipitous. The primary components of the cold shock reflex include an involuntary gasp, tachypnea, and peripheral vasoconstriction, the latter effect highlighting the presumed physiologic principle (ie, warmth preservation via central blood shunting). The magnitude of the cold shock response parallels the cutaneous cooling rate, and its termination is likely due to reflex baroreceptor responses or thermoreceptor habituation.[14] The second stage of cold water immersion syndrome, swimming failure, occurs within 30 min of water entry and likely affects many flush drowning deaths. The ability to self-rescue is an important whitewater survival skill, particularly on difficult or remote stretches of river where assistance may be impossible. Extremity cooling, hyperventilation-induced tetany, or shivering thermogenesis all contribute to coordination loss.[15,16] As tissue cools, nerve conduction diminishes, a process accelerated by muscle movement in a struggling swimmer.[7,17,18] Tactility loss combined with weakness impairs lifesaving tasks such as cutting an entangling rope or swimming to shore.

The third stage of cold water immersion syndrome, hypothermia, is often defined as a core temperature below 35°C, after which ataxia, shivering, dysarthria, apathy, or amnesia develops. As body temperature continues to decline, shivering thermogenesis fails, and potentially dangerous arrhythmias may occur.[7] Investigators have concluded that significant hypothermia is unlikely to develop until 30 min in a person immersed to the neck in 5°C water.[19–21] Although prolonged immersion can slowly decrease the core temperature, flush drowning victims are often recovered on a timescale, which makes hypothermia, at least in the traditional sense, an unlikely contributor to death. The fourth stage of cold water immersion syndrome, circumrescue collapse, is a multifactorial process related to hypovolemia, stress hormone reduction, and core afterdrop.[22,23] Theoretic explanations for circumrescue circulatory collapse include arrhythmias from sudden heart cooling (core afterdrop), hypovolemia secondary to cold diuresis, or the loss of hydrostatic cephalad blood displacement as upright victims are removed from the water.[7] The evidence pertaining to circumrescue collapse is derived loosely from observation of shipwreck victims and requires further development. Like hypothermia, circumrescue collapse requires prolonged immersion, and the effect on flush drowning death is likely negligible.

The cold shock response vs the diving reflex

The key stage of cold water immersion syndrome as it relates to flush drowning is the initial cold shock response initiated in water temperatures below 21 through 25°C.[7,8] The respiratory component of cold shock begins with an inhalational gasp (torso reflex) followed by pronounced tachypnea often exceeding 60 breaths·min^{-1}.[8,14,24–27] Although this initial gasp is often described as involuntary, variability among individuals exists, and these respiratory reflexes can be suppressed or modified.[27,28] Evidence also suggests the cold shock ventilatory response is more pronounced in colder water plateauing between 10 and 15°C.[8,25] Although tachypnea can be driven by a cortically generated sympathetic stress response, the rapid onset after cold water immersion suggests brainstem mediation.[14,24] Hyperventilation-induced cerebral vasoconstriction or alkalosis can theoretically induce syncope, arrhythmia, or muscle tetany. The combination of poor breath holding ability and tachypnea during the cold shock phase enhances the likelihood of aspiration in turbulent rivers where a swimmer must synchronize breathing.[29] Vascular features of the cold shock response include centrally mediated tachycardia and peripheral vasoconstriction, the latter effect enhanced by local skin cooling.[14] It is unclear whether the cold shock response directs adrenal catecholamine release. As with tachypnea, anticipation anxiety may contribute to tachycardia. Within seconds of

immersion, cold shock reflexes raise cardiac output, preload, left ventricular wall stress, myocardial oxygen consumption, and mean arterial blood pressure.[14,20,30] This hypersympathetic milieu might create an environment conducive to a variety of vascular catastrophes, particularly in persons with coronary or peripheral vessel disease.[31]

Cold water immersion is partly affected by the mammalian diving response, which consists of a series of adaptive reflexes after submersion. The physiologic purpose of the diving reflex is believed to be oxygen conservation, a quality evident in diving mammals, in which the response is most pronounced. The diving reflex encompasses bradycardia (cardiac parasympathetic control), expiratory apnea (respiratory control center), peripheral vasoconstriction (vasomotor control center), adrenal catecholamine release, and vascular splenic contraction.[32–34] Genetic, epigenetic, or environmental factors are likely important; a robust diving response is noted in only 15% of human adults, and exercise or breath-hold diving appears to enhance the effect.[12,24,34,35] Control mechanisms for the human diving reflex include surface cooling (thermoreceptors), apnea (pulmonary stretch receptors or chemoreceptors), blood pressure (atrial or vascular baroreceptors), and hydrostatic pressure (facial mechanoreceptors). Detection of surface cooling during the diving response is for practical purposes limited to trigeminal or vagal–pharyngeal distribution thermoreceptors, a notable difference from the cold shock response, in which thermal triggering feedback is obtained from the extremities and other cutaneous sites.[14,32,36]

Vagal bradycardia and central vasoconstriction occur to a limited degree with either apnea or cold water face immersion alone, but the combination produces a robust response.[33,37–40] The primary diving response on–off switch is expiratory apnea, although its magnitude is graded by the rate of cooling, which reflects both water temperature and body surface area relative to mass.[41] The expiratory apnea trigger may be obstructive (submersion) or due to medullary inhibition after trigeminal cold water stimulation (Hering-Breuer inflation reflex).[32] Just as pulmonary stretch receptor activation during inhalation inhibits cardiac vagal motor neurons (eg, as in sinus arrhythmia), stretch receptor quiescence during prolonged apnea enhances vagal influences, particularly at the termination of breath holding.[36,38,39,42,43] Although thermoreceptors and mechanoreceptors initiate diving bradycardia in humans, it is sustained by blood gas alterations or baroreceptor stimulation.[33,34,44]

Unlike bradycardia, diving reflex peripheral vasoconstriction does not rely on pulmonary stretch receptors but is triggered by hypoxemia or hypercarbia detection at carotid or aortic chemoreceptors.[32,38,45–48] Unlike the cold shock reflex, adrenal catecholamine release is believed to contribute to sympathetic activation during the diving response. Diving mammals depress cardiac output during the diving response by maintaining a stable blood pressure, but humans tend to increase cardiac output, stroke volume, or blood pressure due to uncontrolled sympathetic influences.[32,33,49–52] This said, humans experiencing profound diving bradycardia may alternatively drop cardiac output, an outcome presumably more common in children (high surface area to body mass) or in deep water diving.[34] Even though trigeminal thermoreceptors are largely exposed with the use of standard whitewater gear, prolonged apnea is not typically experienced except in entrapment submersion; thus, the cause of flush drownings is likely dominated by cold shock rather than by diving reflex physiology.[53]

Cardiac arrhythmias and autonomic conflict

Early models of cold water immersion syndrome focused primarily on sympathetic responses, but recent research suggests sympathetic and parasympathetic coactivation (autonomic conflict) may be responsible for some cold water immersion deaths.[51] Although reciprocal activation between sympathetic (cold shock) and parasympathetic (diving response) systems is commonly adaptive (follow one another), simultaneous activation appears to be associated with ectopic beats or arrhythmias.[51,54,55] Cold water–induced rhythm disturbances are common, albeit frequently asymptomatic.[24,51,56] In most humans, head-out cold-water immersion results in sympathetically driven sinus tachycardia with variable ectopic beats and supraventricular or junctional arrhythmias.[27,56] These cold water immersion–induced arrythmias appear to be accentuated by parasympathetic stimulation resulting from facial submersion or breath holding.[54,57] Even vagally dominant diving bradycardia caused by isolated cold water facial immersion frequently is interrupted by supraventricular arrhythmias or premature beats.[24,51] In theory, atrioventricular blockade or sinus arrest due to profound parasympathetic dominance might result in syncope or sudden cardiac death, but these rhythms tend to be rapidly reversed by lung stretch receptor activation associated with breathing.[34,58–61] As such, a vagally produced arrest scenario is likelier during entrapment submersion than in flush drowning.

There is also evidence that sudden death surrounding emotional events such as a startle response, sudden awakening, or anger reactions involve some form of autonomic conflict.[55,62–65] Among the basic emotions, anger is most associated with ventricular fibrillation, presumably because it enhances sympathetic activity while

maintaining parasympathetic tone.[66] Although speculative, stressful emotional substrates during a river accident could add to the arrhythmogenic milieu in the setting of cold water immersion.[24,51]

Arrhythmias may also be driven by hyperventilation-induced coronary vasoconstriction, hypoxemia, cardiac ischemia from extreme hypertension, or heart distension from central blood shunting.[34,49,51] In addition, cold water immersion appears to delay QTi shortening or lengthening in response to tachycardia or bradycardia, respectively, potentially introducing another arrhythmogenic factor.[51,67,68]

To summarize, the relationship between the protean physiologic changes encountered during cold water immersion and sudden death is largely speculative, yet there are ample hypothetical reasons to suggest these reflexes may have dangerous consequences. Additional research is needed to define the complex interplay among cold water immersion syndrome, the diving reflex, and autonomic conflict as it relates to the pathology of cold water immersion death.

Prevention of cold water immersion syndrome

Because environmental factors are fixed, methods to avoid cold water immersion syndrome must be directed toward adequate equipment and preparation. Both physiologic and psychological conditioning appear to modify breath hold ability, shivering, panic reactions, or even autonomic balance after cold water immersion.[51,53] Cold water–induced rhythm abnormalities are more common in aerobically unfit persons, possibly indicating poor autonomic balance.[49,56,69] Even though immediate responses to cold water immersion are largely mediated by the brainstem, cerebral modification implied by preimmersion tachypnea or tachycardia suggests cognitive preparation may be useful in attenuating cold shock breathing patterns.[24] Physical adaptation is also possible; studies show repeat brief exposure to cold water diminishes the respiratory components of cold shock by up to 30%.[27,28,70,71] The ability to control breathing during intermittent submersion in turbulent whitewater is an important survival skill for whitewater boaters. Adaptive cardiovascular responses after repeated cold water exposure include altered chemoreceptor sensitivity, reduced adrenal catecholamine release, and enhanced parasympathetic activity.[32,72–75] Serial cold water immersion may also lower baseline body temperature, reduce the sensation of cold, delay shivering, and reduce the rate of temperature decline during future exposures.[76–78] Physiologic adaptation may affect survival; for example, controlled breathing may prevent aspiration, and delaying shivering might allow a larger window for self-rescue. Attenuation of the cold shock response by acclimatization persists 2 wk after the cessation of the adaptive behavior.[77]

Cold water immersion physiology is affected by the duration of exposure, water temperature, body movement, body fat, facial submersion, and protective clothing.[77] Although insulating fat may limit the cold shock response, for practical purposes thermal protection during whitewater activities is best accomplished with a drysuit or wetsuit. An insulating cap or hood under a whitewater helmet may further attenuate the effects of immersion. The AWA recommends protective insulation when the water temperature is less than 16°C or when the combined air and water temperature is less than 49°C, although very high air or low water temperatures likely invalidate the use of combined temperature as a guide. River safety requires situational awareness of environmental conditions, some of which may not be obvious. For instance, water temperature at the launch point for the Grand Canyon is typically under 14°C, whereas the air temperature can exceed 38°C. Lastly, predisposing factors may play a role in lethal physiologic responses after sudden immersion in cold water. Cardiomyopathy or coronary artery disease are known risks for exertion-provoked death. Furthermore, unexplained drownings as a whole are believed to often have causal associations with channelopathies such as hereditary long QT syndromes.[79–81] It is not hard to imagine drug-induced QTi lengthening may have similar implications.

Limitations

The research on cold water physiology primarily comprises small case studies, expert opinion, or observational data. As such, this review should serve to develop concepts and encourage further investigation. Many aspects of cold water immersion syndrome remain controversial or unexplained. For instance, it is unclear why some humans seem well adapted to entering cold water. Future research should be directed toward defining specific or combined mortality factors or predispositions associated with sudden cold water immersion deaths as well as methods of attenuating or eliminating these factors.

Conclusion

Mitigating risk in whitewater sports requires an understanding of the applicable injury mechanisms. Entrapment submersion fatalities occur by conspicuous means and can be limited by anticipating or portaging hazards. In contrast, the mechanisms behind flush drownings are unclear. Of the potential factors contributing to flush drowning, cold water immersion syndrome or the corollary concept of autonomic conflict are compelling candidates. If cold

water immersion syndrome is associated with flush drowning death, understanding the concepts of cold shock and swimming failure may alleviate some risk in swift water activities. Specific cold water effects that may contribute to flush drowning include interruption in breath holding ability, extreme hypertension, arrhythmia, or coordination loss. The physiologic effects of sudden cold water immersion may be attenuated by mental preparation, habituation, or, most importantly, proper equipment. Poor aerobic fitness, cardiovascular disease, or channelopathies may be risk factors for poor outcomes after cold water immersion, and those at highest risk may consider avoidance the best prevention. At a minimum, strong consideration should be given to the use of a drysuit or wetsuit, particularly where water is coldest. Furthermore, a discussion of the effects of cold water and mitigation techniques and screening of high-risk participants should be offered to commercial rafting passengers. Boaters who fall into cold water should try to control breathing and position themselves to keep the head up and facing downstream to anticipate submersion. Experienced whitewater boaters always tell their passengers to "rig and dress for a swim."

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