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Breath-Hold Diving Injuries — A Primer for Medical Providers

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Abstract

Breath-hold divers, also known as freedivers, are at risk of specific injuries that are unique from those of surface swimmers and compressed air divers. Using peer-reviewed scientific research and expert opinion, we created a guide for medical providers managing breath-hold diving injuries in the field. Hypoxia induced by prolonged apnea and increased oxygen uptake can result in an impaired mental state that can manifest as involuntary movements or full loss of consciousness. Negative pressure barotrauma secondary to airspace collapse can lead to edema and/or hemorrhage. Positive pressure barotrauma secondary to overexpansion of airspaces can result in gas embolism or air entry into tissues and organs. Inert gas loading into tissues from prolonged deep dives or repetitive shallow dives with short surface intervals can lead to decompression sickness. Inert gas narcosis at depth is commonly described as an altered state similar to that experienced by compressed air divers. Asymptomatic cardiac arrhythmias are common during apnea, normally reversing shortly after normal ventilation resumes. The methods of glossopharyngeal breathing (insufflation and exsufflation) can add to the risk of pulmonary overinflation barotrauma or loss of consciousness from decreased cardiac preload. This guide also includes information for medical providers who are tasked with providing medical support at an organized breath-hold diving event with a list of suggested equipment to facilitate diagnosis and treatment outside of the hospital setting.

Introduction

Breath-hold diving sports and activities have grown rapidly in the last few decades, both in the recreational and competitive realms. Athletes who participate in breath-hold diving sports may suffer unique injuries that are distinct from those of surface swimmers and compressed gas divers, although there is some overlap. This review is intended to guide medical providers caring for breath-hold divers suffering from injury and draws from published literature combined with expert opinion, as few clinical trials exist in this field.

Breath-hold diving encompasses a variety of underwater activities reliant upon holding your breath underwater. It is commonly referred to as freediving or skin diving while the competitive sport is often referred to as apnea or apnea diving. It is prominent in certain communities as a means of gathering food. Breath-hold diving competitive sports include Olympic synchronized swimming, as well as team sports such as underwater rugby or underwater hockey. The International Association for the Development of Apnea (AIDA) was established in 1992 and the Confédération Mondiale des Activités Subaquatiques (CMAS) started in 1995 to oversee competitions and certify freedivers for specific disciplines that focus on duration (static apnea), length (dynamic apnea), or depth (constant weight) while holding one's breath. Both recreational and competitive interest in the sport has rapidly gained popularity over the last few years. While there is no official record of the number of freedivers around the world, the number of athletes in AIDA competitions has increased more than thirteen-fold from 2000 to 2020 (1). In 2022, there were 249 official AIDA competition events.

Today, the Haenyeo of Korea, Ama of Japan, and fisherman of Tamil Nadu still practice breath-hold diving for sustenance, a tradition that has been passed down for many generations. Some, like the Ama, still use traditional materials of their ancestors while others, like modern Qatari pearl divers, have adopted modern freediving equipment and techniques. Freediving also has made it to Hollywood - the cast and crew of 2022's *Avatar: The Way of the Water* logged more than 250,000 breath-hold dives in the making of the film (2).

The increasing popularity of breath-hold diving activities parallels the increase in injuries and fatalities reported to the Divers Alert Network (DAN) (3). Of note, DAN includes snorkeling injuries in their breath-hold database even though this review article does not include snorkeling injuries or

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management (3–7). Given that many people engage in breath-hold diving activities without any formal instruction or certification, the number of accidents is likely underestimated.

The primary issues in breath-hold diving arise from the risk of hypoxia from extended durations of breath-holding and the effects of rapidly changing ambient pressure from the water column when descending to and ascending from depth (8,9). The common practices of glossopharyngeal insufflation "packing" and exsufflation "reverse packing" to increase lung volume above tidal volume and decrease it below residual volume, respectively, compound the effects of ambient pressure on lung tissues. This review article discusses various medical issues that breath-hold divers may experience during a diving session or competition. We include a list of suggested medical equipment along with a summary table of freediving injuries and their management.

Medical Issues in Breath-Hold Diving

Hypoxia

Pathophysiology

The primary cause of hypoxia is that the breath-hold duration is too long in relation to the metabolic consumption of oxygen. Hyperventilation, hypoxia of ascent, and a metabolic shift from carbohydrate to lipid metabolism has been shown to increase the risk of hypoxia.

Hyperventilation

Hyperventilation leads to one breathing out more CO_2 than metabolism produces. When done prior to the start of a breath-hold, it delays the body's natural urge to breathe, which is typically triggered by an increasing CO_2 level. Hyperventilation also adds more oxygen stores to the lungs and blood. The consequence of this is that the body has less innate warning that hypoxia is developing. Therefore, hyperventilation makes it easier to prolong a breath hold, but at the risk of exceeding the hypoxic threshold that leads to loss of consciousness (10). For experienced divers, this technique is beneficial in competition. However, hyperventilation is considered the main cause of death in water sport athletes and recreational swimmers practicing breath-hold unsupervised in a swimming pool. These submersion-related deaths are the primary reason breath-hold activities are banned in many swimming pools.

Hypoxia of Ascent

As a diver ascends from depth, the decreasing ambient pressure results in the partial pressure of oxygen in the lungs to decrease (11). This change is most notable in the last ten meters below the surface when the ambient pressure is halved, often resulting in a hypoxic loss of consciousness during the final stage of the ascent. Since this most often happens in the last few meters prior to surfacing, it is colloquially called a "shallow water blackout." This also can happen shortly after surfacing, termed a "surface blackout" as there is a delay from the re-initiation of ventilation to bring oxygenated blood to the brain. Many breath-hold divers practice "hook breathing" which is characterized by a rapid breath in, held while bearing down for a few seconds, followed by exhalation. This facilitates the rapid increase in oxygen to avoid a surface blackout (12). The competitive freediving discipline of constant weight no fins (CNF), where a diver swims down and up along a line without the assistance of fins, has the highest incidence of ascent blackout (13) suspected to be due to increased physical effort and a slower rate of ascent compared with when assisted with fins. Fins used can include the monofin which is shaped like a dolphin's tail and helps with legs kicking in unison while bifins are individual fins attached to each foot and help with legs alternating anterior and posterior. In competition, divers that require more time to swim down, turn at the bottom, and swim to the surface are more likely to experience a shallow water blackout (14).

Lipid Metabolism

Fasting prior to breath-holding can help athletes prolong a breath hold due to the slower accumulation of CO_2, at the risk of developing hypoxia due to significantly lower oxygen levels at the termination of a breath hold (15,16). When fasting is combined with light exercise, the breath hold duration also is increased. Fasting depletes glycogen and exercise with depleted glycogen stores shifts the body to lipid metabolism, using fat as fuel. Under these conditions, the body produces less CO_2 compared with a standard carbohydrate metabolism, which prolongs breath hold duration (17).

Presentation

One of the first visible signs of hypoxia is a loss of motor control (LMC) characterized by transient confusion, tonic-clonic jerks, or speech difficulty, commonly referred to as a "samba," or hypoxic fit. These fits involve mostly the antigravitational muscle groups (*i.e.*, quadriceps, erector spinae, etc.) in a pattern of tremor that is not chaotic nor aleatory, but rather resembles intentional tremor, and it has a specific pace and frequency between brief fits of atonia and recovery of muscle tone. This has been shown to develop when end-tidal PO_2 reaches ~20 mm Hg (18). LMCs are almost always followed by retrograde amnesia to the event (19). LMCs were the most common adverse event documented in the 2004 AIDA World Free Diving Championship (20).

Prolonged hypoxia can lead to loss of consciousness (LOC) with prolonged blank staring, eyes rolling backwards, or syncope (21) if the diver is unable to resume ventilation within the first few seconds, commonly referred to as a "blackout" in competition. Aspiration or drowning can occur if a loss of motor control or loss of consciousness occurs while the diver is submerged.

Diagnosis

Diagnosis of a hypoxic LMC or LOC is clinical. Underwater, the telltale signs are a change in swimming pattern or speed and/or a sudden release of air (19). On the surface, divers may be sputtering, apneic, and/or unconscious.

Field Management

If managed properly in the first few seconds, the diver may have a completely normal neurological exam and oxygen saturation by the time they present for a medical evaluation. While unconscious, divers underwater should have their airway closed to prevent aspiration. On the surface, supporting the airway above water and using both verbal and physical stimulation are first-line techniques to attempt to regain consciousness. Blackout management techniques are taught in all standardized freediving training courses.

Prolonged syncope may require rescue breaths or supportive ventilation to recover. Medical personnel should make sure

the airway is open and patent, as trismus may prevent effective ventilation. If rescue breaths are ineffective, ventilation with a bag valve mask attached to 100% O_2 is preferred. Adequate chest rise and auscultation of bilateral breath sounds are used to confirm effective ventilation. A pulse oximeter may be used to monitor recovery, but finger oximetry is often delayed from central oxygenation (22). Peripheral vasoconstriction from freediving also may limit the utility of a finger oximeter. Organized competitions typically staff medical providers trained in airway management and cardiopulmonary resuscitation.

Barotrauma of Descent and Reverse Packing

Pathophysiology

Descent

As a diver descends, the increased ambient pressure compresses the air spaces according to Boyle's law. Air within the mask, nasopharynx, paranasal sinuses, and middle ear must be repeatedly equalized on descent. On average, diving to 30 to 50 m (8,23) will compress the lung to residual volume. Over 1 L of blood (24) can shift into the thorax and cause pulmonary vascular engorgement to accommodate for this lung collapse at depth (25). Other mechanisms that compensate for lung air volume decrease include diaphragmatic inflection upward and thoracic cage compression inward. When the pulmonary capillary pressure exceeds oncotic pressure, leakage of fluid into the airways causes pulmonary edema.

Shear (or tensile) stress from negative intrapulmonary pressure, diaphragmatic contractions (26), and/or movement may cause rupture of the alveolocapillary membrane and subsequent alveolar hemorrhage. This is colloquially called a "squeeze" or "lung squeeze". Disruption of small capillaries lining the airspaces also may cause bleeding in the sinuses. In the ears, a significant pressure gradient across the tympanic membrane can cause rupture. Negative pressure within the mask also can lead to subconjunctival hemorrhage (27).

Reverse Packing

Glossopharyngeal exsufflation, also known as "reverse packing," is the opposite of packing in that one uses the glossopharyngeal musculature as a piston to draw up air from the lungs into the oropharyngeal cavity. This technique is used to facilitate equalization at depths where the lungs have shrunken beyond residual volume. When done after a full exhalation on the surface, this maneuver allows one to decrease their residual volume by up to 0.4 L (28). Divers may practice using this technique to simulate lung collapse at a depth deeper than where they are performing their training dives. The main risk with reverse packing is putting negative pressure on the air spaces, which can lead to the development of edema and/or localized tissue damage similar to a squeeze.

Presentation

The presentation of a squeeze varies depending on which airspace is affected. A middle ear squeeze resulting in tympanic membrane rupture presents as bleeding, hearing loss, pain, and/or vertigo (9). A mask squeeze may range from a mild subconjunctival hemorrhage to significant periorbital ecchymosis. A sinus squeeze may manifest as a headache, sinus discomfort, and/or epistaxis. A tracheal or laryngeal squeeze may result in transient voice alteration and which is commonly attributed to subglottic swelling. Divers also may develop a productive cough from excess mucus development or soreness in the sternum or ribs from the strain on the thoracic cage. In a lung squeeze, divers may present with cough, dyspnea, chest pain, and/or hemoptysis. Although the bleeding appears to be self-limited, the amount of blood and fluid in the lung may affect normal respiratory function and lead to dyspnea.

Diagnosis

The diagnosis of a squeeze can be difficult due to subtle symptoms and is suspected to be under-reported. Auscultation may demonstrate rales or crackles. Pulse oximetry >95% within 30 minutes of surfacing has a negative predictive value of 100% for lung injury (29). Ultrasound may demonstrate lung comets and areas of atelectasis (30). This may be nonspecific as asymptomatic lung comets are present in 85% of competitive divers (31) and 33% of competitive spearfishermen (32) after immersion. Of note, asymptomatic decreases in pulse oximetry and increases in extravascular lung fluid may be present for up to 2.5 h after a dive using reverse packing (33).

In-hospital radiographs may show patchy airspace disease (34,35) and CT may show areas of alveolar hemorrhage (36,37) ground-glass opacities, and/or pleural effusion (38). Nasal endoscopy may reveal hemorrhagic fluid in the airways adjacent to affected sinuses (39). Nasopharyngeal laryngoscopy has demonstrated hemoptysis originating from below the vocal cords (28). Bronchoscopy may demonstrate diffuse airway inflammation (40) or bleeding from the bronchus (30).

Management

Squeezes are often self-limited (26) so many divers do not seek medical attention. Treatment is supportive and focuses on symptom management and bleeding control. If available, 100% oxygen can be given in cases of lung squeeze resulting in hypoxia or respiratory distress. Divers with hemoptysis may have alveolar damage and/or rupture, therefore positive pressure should be used carefully to avoid introducing pressurized air into the pleural cavity, mediastinum, or arterial circulation. Divers with persistent symptoms should be referred for hospital evaluation. Vasodilators, bronchodilators, and diuretics have been used on divers presenting to emergency departments (41).

Barotrauma of Ascent and Pulmonary Overinflation Syndrome

Pathophysiology

Ascent

As a diver ascends from depth, the decreasing ambient pressure causes expansion of air spaces according to Boyle's law. In breath-hold diving, there should only be a minimal change in the amount of air secondary to gas uptake from descent to ascent. However, redistribution of air throughout the dive secondary to body positioning, equalization techniques (42), and/or increased pressure from strenuous work (43) may allow for airspace rupture and subsequent release of air into the bloodstream, adjacent organs, and/or tissue spaces. This is similar to the pathophysiology of arterial gas embolism in compressed air diving (44). The change in volume is most prominent in the last few meters of a dive, prompting some divers to exhale as they near the surface.

Table 1.
Medical Supplies for a Breath-hold Diving Event.

General	Incident report forms, pens Gloves, surgical masks, face shields Hand sanitizer Trash bags Cots/chairs Portable litter
Airway/ Respiratory	Pulse Oximeter Stethoscope Oxygen tank, regulator, tubing, mask with demand valve Nonrebreather mask BVM w/PEEP valve, tubing, and mask Nasopharyngeal airway, oral airway, supraglottic airway Suction device with suction catheters and tubing Large-bore needle for decompression (ThoraVent) Albuterol MDI with spacer CPAP machine with mask and tubing
Cardiovascular	Portable rhythm strip (KardiaMobile) AED BP cuff Ultrasound with cardiac probe and ultrasound gel Aspirin Nitroglycerin paste or SL tabs
Hemorrhage control	Tourniquets, Sharpies QuikClot, Surgicell, or hemostatic dressing Tranexamic acid Gauze, absorbent pads, roll bandages, medical tape
Wound care	Irrigation solution Alcohol, Iodine, betadine, chloraprep Tweezers, Scalpel #11 Leukotape, moleskin, blister pads Steri-strips, Dermabond, bandages, Tegaderm Coban wrap, ace wrap, sports tape SAM splint Sutures with needle drivers and forceps, sharps container Silver nitrate or cautery Skin stapler Polysporin or bacitracin
Analgesia	Cetacaine, benzocaine, lidocaine, bupivacaine Ethyl chloride, LET, EMLA Ketamine 18 to 27 gauge needles 5 to 10 mL syringes Sharps container Ibuprofen, acetaminophen Lidocaine patches, diclofenac gel, camphor, menthol
Musculoskeletal	Triangular bandages SAM splints ACE wrap, Coban Medical tape
Environmental	Thermometer Reflective emergency blanket Instant heat and cold packs Aloe vera, burn gel Vinegar EpiPen, antihistamine tabs/cream, steroid tabs/cream Scopolamine, ondansetron, meclizine, alcohol pads
GI/GYN	Urine pregnancy test Tampons, pads Ondansetron Loperamide Azithromycin, levofloxacin
HEENT	Fluorescein strips, tetracaine, blue penlight Artificial tears Otoscope, ear speculum Ofloxacin (ophthalmic and otic) Oxymetazoline Nose clip or RhinoRockets Fluticasone nasal spray Pseudoephedrine tablets Tongue depressors Amoxicillin-clavulanate
Endocrine	Glucometer, test strips Oral glucose paste or honey

Packing

Glossopharyngeal insufflation, also known as "lung packing," is a technique where after maximal inspiration, one uses the glossopharyngeal musculature as a piston pump to force additional air into the lungs. This maneuver allows some divers to increase their lung capacity by up to an additional 4 L (45). Divers may desire to increase the total volume of air in the lung prior to a dive to delay the onset of hypoxia (46) and increase intrapulmonary volume and pressure to accomplish equalization maneuvers at greater depths. This increased volume and pressure combats the ambient pressure of the water column at depth and has been postulated to be protective against the development of pulmonary edema and/or alveolar hemorrhage (47). However, packing also predisposes divers to alveolar rupture from over-pressurization of the lungs, or "overpacking" (48).

Nonbarotraumatic symptoms of packing can include syncope due to compression of the mediastinum due to the increase in lung volume, which subsequently decreases the preload to the heart and results in decreased cardiac output (49). Additionally, air entering the arterial system via rupture can travel to the cerebral circulation and block perfusion to the brain parenchyma (50).

Presentation

The timing of symptoms usually presents between 0 and 60 min. Common presentations in the chest include chest pain, swelling, and/or dyspnea (51), correlating with pneumomediastinum, subcutaneous emphysema, and/or pneumothorax. Of note, the pneumomediastinum may be asymptomatic (52). If the sinuses are affected, air may be found in the periorbital region (42). If the air travels through the circulation to the spine or brain, neurological deficits may present. Neurological complaints vary from mild dizziness and headache to focal weakness, confusion,

or loss of consciousness (44). Transient neurological deficits may resolve within 30 minutes (50,53), which is characteristic of an arterial gas embolism (AGE). Typically, thoracic complaints precede neurologic complaints.

Diagnosis

Diagnosis is primarily clinical and focuses on visual inspection for areas of swelling, palpation of the skin for any crepitus, and auscultation of breath sounds. Pulse oximetry and lung ultrasound can be helpful adjuncts in diagnosing a ventilation deficit or areas of lung collapse. Neurological symptoms in the absence of cardiopulmonary complaints should prompt investigation into a possible airspace rupture. If available, pulse oximetry and ultrasound can be helpful adjuncts in diagnosing lung or cardiac injury.

In-hospital chest X-ray may show lung collapse or subcutaneous air while CT is the imaging modality of choice when looking for smaller areas of free air.

Management

Management of barotrauma in the field should focus on airway protection and close evaluation for neurological injury. If available, 100% oxygen should be given. Divers with neurological symptoms suggesting AGE should be considered for recompression. A hyperbaric chamber may not be available, and as such in-water recompression has been used in the past (54,55).

Prognosis

When chamber recompression is used, neurologic symptoms can resolve within 24 h (44). Of note, CT findings of pneumomediastinum have shown resolution within 2 to 3 d (48,56) without recompression. Periorbital air was found to resolve upon two-week follow-up (42) without recompression.

Decompression Illness

Pathophysiology

Repetitive Dives

In addition to arterial gas embolism caused by barotrauma, repetitive shallow dives with short surface intervals may allow for nitrogen buildup in tissues like decompression sickness (DCS) in compressed-air diving (57–59). It also has been suggested that nanobubbles on the arterial side of the vasculature could grow and embolize upon ascent (60) even in the absence of a patent foramen ovale (61). It is theorized that vessel walls in distal arteries are so thin as to allow nitrogen diffusion from surrounding tissue into the artery, creating a nanobubble that increases in size with subsequent dives (62,63). The bubble is then thought to preferentially embolize to the spinal and cerebral vasculature on ascent, resulting in an arterial gas embolism (57). These symptoms are sometimes referred to as "Taravana Syndrome," which translates to "to fall crazily" from observations made by Polynesian divers (64).

Deep Dives

Long deep dives over 100 m (65) also allow for potential gas buildup and diffusion. It is postulated that intrapulmonary arteriovenous anastomoses allow for right-to-left shunting of venous gas emboli that develop at depth (66), and that hypoxia, hypercapnia, and exercise during a dive all contribute to the opening of these shunts (67) which then allow gas bubbles to enter the arterial bloodstream. These circulating bubbles have been implicated in the cause of endothelial damage to the blood-brain-barrier and resultant reversible encephalopathy (68). Additionally, profound cerebral hypotension at depth secondary to decreased cardiac output from increased afterload could result in low perfusion in terminal cerebral vessels leading to infarct (59).

Presentation

The symptoms of decompression illness (DCI) present between 0 and 120 min and usually start mild and may be transient and improve or may persist and worsen over time (66,69). Common symptoms include headache, dizziness/vertigo, nausea, numbness, weakness, visual disturbances, seizure (61), loss of consciousness, hearing loss, euphoria, trouble concentrating, and/or sudden death (70).

Diagnosis

Frequent and thorough neurological evaluation (71) is the cornerstone of diagnosis and monitoring in the field. Persistent symptoms should prompt evacuation for further evaluation.

In-hospital CT imaging may show areas of hypodensity (61) consistent with cerebral infarcts or arterial bubbles (72). MRI may show areas of hyperintensity in large vessels as well as watershed areas (59). Areas of infarct have been found to involve the cerebral cortex, subcortex, basal ganglia, brainstem, and cerebellum (66). Areas of partial hemorrhage may be seen as well (61). MRI perfusion studies may demonstrate vasogenic edema (68). Contrast-enhanced transcranial Doppler ultrasound can detect bubbles in the arterial circulation (73).

Management

One hundred percent oxygen is the mainstay of treatment. Some competitions offer oxygen breathing at depth as a preventative measure after deep dives (57). Recompression should be considered for divers with persistent or debilitating symptoms. Recompression in a hyperbaric chamber is preferred over in-water recompression due to challenges with patient monitoring and reassessment. An expedited diagnosis leads to rapid evacuation and chamber recompression, increasing chances of a favorable outcome.

Prognosis

Most symptoms resolve after hyperbaric treatment, and are often diagnosed as either decompression sickness or arterial gas embolism (74). MRI findings have shown to attenuate within 2 to 3 wk after hyperbaric treatment (75) that may completely resolve within 1 month of hyperbaric treatment (76).

Arrhythmia

Pathophysiology

Diving Response

The mammalian diving response is characterized by peripheral vasoconstriction, increased blood pressure, and bradycardia. This bradycardia is vagally mediated and triggered by apnea. This response can be further stimulated by face cooling, most commonly by submersion in cold water (77,78). However, cold water immersion also induces a cold shock response, characterized by gasping and sympathetically mediated tachycardia. This autonomic conflict predisposes cold

water divers to arrhythmias (79). Once the breath hold is terminated, the arrhythmia disappears.

Packing

Divers who pack may develop syncope from decreased venous return due to increased intrathoracic pressure, known as a "packing blackout" (49,80). Monitoring has demonstrated a sudden drop in heart rate and blood pressure (81) without hypoxia. Electrocardiogram recordings have shown junctional bradycardia below 10 bpm (82). This type of syncope usually prompts the diver to release the breath hold which then allows for normal venous return and restoration of normal cardiac activity. If this occurs underwater, there is a risk of aspiration or drowning.

Diagnosis

The symptoms of arrhythmias manifest because of inadequate cardiac output and brain hypoperfusion. Diving partners or safety divers may witness an aspiration event underwater initiated by a sudden release of air. On the surface, divers may exhibit involuntary movements, near-syncope, and/or loss of consciousness.

If available, portable electrocardiogram devices can aid in the detection of an arrhythmia. Toward the end of a maximal breath hold, single-lead electrocardiogram recordings demonstrated junctional rhythms, second-degree heart block, and sinus bradycardia (83). Other recordings have found ectopic beats, ventricular tachycardia, junctional escape beats, bigeminy, and intermittent bundle branch blocks (8).

Management

Most arrhythmias in breath-hold diving are self-limited and terminate concurrently with the end of a breath hold. However, if electrocardiograph evidence of abnormal heart rate or rhythm persists, divers should be sent in for further workup and evaluation.

Inert Gas Narcosis

Pathophysiology

As a diver descends, the partial pressures of each gas in the breath of air being held increase according to Dalton's law. Nitrogen is narcotic at high partial pressures and can exert a negative effect on a diver's ability to complete simple tasks and affect one's awareness. This is commonly seen in SCUBA divers once they exceed depths of 30 to 35 m, and almost immediately resolves with a minor change in depth (84).

Breath-hold divers can experience a similar syndrome once they exceed depths 70 to 90 m (23). It is still debated whether this is solely the effect of nitrogen or confounded by high partial pressures of carbon dioxide and oxygen and/or extremely high ambient pressure from the water column (85). Freedivers who descend to much deeper depths and surface within minutes may, due to their rapid rate of ascent, briefly continue to feel symptomatic on the surface.

Presentation

The symptoms of narcosis include difficulty concentrating, confusion, numbness, and/or retrograde amnesia (85). Some divers have reported hallucinations, altered time perception, and blurred vision (23).

Diagnosis

This syndrome is often self-diagnosed retrospectively after the diver has returned to normal mentation. Symptoms persisting beyond several minutes should prompt evaluation for DCI.

Management

Inert gas narcosis is self-limiting, and therefore underreported and understudied. Close monitoring with frequent neurological re-evaluation will help medical professionals differentiate

Table 2.
Freediving Injuries and Management.

	Causes/Risk Factors	Diagnosis	Treatment
Hypoxia	Hyperventilation Ascent Fasting	Loss of motor control "samba" Syncope "blackout"	Airway protection while underwater Stimulation Rescue breaths Assisted ventilation
Barotrauma of descent "squeeze"	Inadequate equalization Contractions Reverse packing	Airspace bleeding Cough, dyspnea Lung auscultation, pulse oximetry, lung ultrasound	Bleeding control Oxygen Positive pressure ventilation
Barotrauma of ascent Pulmonary overinflation Arterial gas embolism	No presurface exhalation Body positioning Packing	Pain, dyspnea, neurological deficits Lung auscultation, pulse oximetry, lung ultrasound	Oxygen Recompression
DCI Decompression sickness Arterial gas embolism	Repetitive dives Deep dives Patent foramen ovale	Neurological deficits Syncope	Oxygen Recompression
Arrhythmias	Breath hold Cold water Packing	Involuntary movements Syncope Electrocardiogram	Self-limited
Inert gas narcosis	Deep dives	Altered mentation, amnesia	Self-limited

narcosis from more dangerous disease processes, such as an arterial gas embolism.

Medical Service Planning

There is no universal standard for medical care at breath-hold diving events, although larger events usually have an on-site medical director, several medical staff, and safety divers trained in basic airway management and the provision of oxygen. The location of each event and its remoteness will dictate the extent to which medical crews should be prepared to treat potential patients as well as indications for evacuation. Collaboration with the closest hyperbaric chamber, emergency transport and evacuation services, and hospital is essential.

Translation services should be available for providers to properly communicate with patients. Staff fluent in the local language(s) should be assigned to communicate with local emergency personnel. Divers should be encouraged to carry medical insurance and complete preevent health screening. Any diagnostics performed or treatment rendered should be documented, and hand-off between medical providers should include a summary of such.

Medical supplies and equipment will vary depending on the experience level of medical providers as well as the anticipated injuries from the event. CMAS mandates a minimum kit of airway supplies, oxygen, suction, and an AED (86). AIDA mandates a minimum kit of oxygen, a first aid kit, and a spine board (87). We have included our list of recommended medical supplies based on category of injury (Table 1). Written guidelines and protocols, with hands-on instruction and training with the medical kit, should be available to all medical staff. We have included a summary table of freediving injuries and their immediate management considerations (Table 2).

Safety

Breath-hold diving alone is not recommended. The buddy system of "one up, one down" ensures there is always a lookout on the surface to intervene in case of an incident underwater. Even after a diver surfaces, their buddy should stay at the surface for an additional 20 s to ensure the first does not have a delayed blackout before initiating their own dive (88). Safety divers are a mainstay of competition freediving but are notably lacking in other breath-hold diving disciplines. Therefore, buddy groups are responsible for the safety of their fellow divers.

Conclusion

Increased participation in both recreational and competitive breath-hold diving has led to a steady increase in injuries. Breath-hold divers are susceptible to hypoxia, barotrauma, arrhythmias, inert gas loading, along with injuries typical of a marine environment. While many injuries are immediately obvious, others require thorough evaluation and frequent monitoring, with consideration for evacuation in certain cases. Medical personnel at breath-hold diving events and competitions may only have minimal training in basic life support and not all breath-hold divers have completed a formal training course in diving safety. We believe this primer will be a helpful reference for any providers caring for patients suffering an injury from breath-hold diving.

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation, and statement that results of the present study do not constitute endorsement by ACSM.

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