Decompression Sickness & Submersion Injuries

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Information does not go beyond ED care.

6. Etiology/pathophysiology

a. Definition, types, causes

i. Decompression sickness:

Nerve tissue starts to exhibit altered function at a depth of 150 meters of seawater. According to Boyles law, the gas in the lungs is affected by pressure. The volume of gas is compressed, and further gas cannot come in. The air in the lungs compress to half the surface volume when at 10 meters (33 feet) down, and to one third at a depth of 20 meters (66 feet) down. The lungs reach residual volume at a depth of 30 meters (98 feet). With farther descent, blood is shunted to the chest, and the abdominal viscera and diaphragm are pushed up high into the ribcage to compensate for further reductions in lung volume. When the diver is ascending back to the surface, the lungs expand to its original volume at the surface. Scuba divers breathe gas that is supplied at the ambient pressure of the surrounding water, which allows for normal lung volumes during the respiratory cycle. There is free exchange of gases between gases in the alveoli and the dissolved gasses in the bloodstream.

According to Henrys law, the partial pressures of the gases in the alveoli increase so the number of molecules of gases dissolved in the blood and in the body tissues increases. Nitrogen is poorly soluble in water and blood but is very soluble in lipids and neurological tissues. Therefore, the spinal cord and brain absorbs large amounts of nitrogen as the diver descends since the tissues contain a high lipid content. Increased nitrogen levels lead to nitrogen narcosis which causes altered cognition and leads to accidents. Narcotic effects begin to occur at 20 meters, and cognitive function is severely impaired at greater than 50 meters. This is cured by ascent, and it is avoided by replacing some of the nitrogen with less narcotic gas. Air is not used as a breathing gas for deep dives because of the risks of CNS oxygen toxicity, nitrogen narcosis and the increased effort of breathing the dense gas. Helium is used for dives deeper than 50 meters, and it replaces some of the nitrogen and some of the oxygen.

The increased pressure of the inert gas breathed causes tissues to absorb greater amounts of the dissolved gas than at the surface. During decompression, the tissues have an excess amount of dissolved gas that were taken up during compression and the extra gases must leave the tissues to return to the lungs. If the diver ascends slowly, then the ambient pressure is reduced gradually and the pressure of gases from the arterial and capillary blood decrease proportionately. Nitrogen diffuses out of the tissues and into capillary blood and carried in venous blood back to the lungs to diffuse in the alveoli. However, if decompression is too rapid, the gas that dissolved in the tissues will come out of solution to form bubbles in the tissue and venous blood. Definition: Decompression sickness occurs when nitrogen gases dissolved in the blood during high pressure situations are trapped as bubbles in the tissues during depressurization occluding blood flow and oxygenation.

SCUBA diving is the primary cause of decompression sickness, but it also occurs in those who work in conditions that have compressed air, like in tunnel construction. There are certain risk factors that put a diver at an increased risk for DCS. This includes age greater than 30, female sex, coldwater dives, dehydration, fatigue, seasickness, air travel after diving, lung

disease, obesity, prolonged or deep dives, rapid ascents, right to left shunts (patent foramen ovale), and tobacco or alcohol use.

1. Signs and symptoms:

The signs and symptoms of DCS usually present within 15 minutes to 12 hours after surfacing. Severe signs and symptoms that can be life threatening present within minutes of reaching the surface. The two most common types of DCS seen in scuba divers are identified by the systems affected and the onset of symptoms. Signs and symptoms occurring within the first 6 hours after surfacing can be either Type I or Type II. Type I decompression sickness is the milder version of decompression sickness, while Type II may be life-threatening since it mainly affects vital organ systems. Type I involves the epithelial and musculoskeletal system, which gives it the common name “the bends”. This presents as myalgia, arthralgia, pruritus, rash, or lymphadenopathy. The severity of pain in the joints and bones can cause the diver to continuously “bend” over. On the other hand, Type II involves the cardiopulmonary and neurologic system. This can present as dyspnea, chest pain, cough, vertigo, tinnitus, or confusion. If these symptoms present within 10 to 20 minutes of resurfacing, the patient will have sudden life-threatening symptoms. This includes neurologic signs such as stroke, ataxia, visual and speech disturbances, and spinal cord injuries. Also, this includes cardiopulmonary signs like myocardial infarction, dysrhythmias, or pulmonary embolism. The severity of DCS is determined by the direct tissue damage caused by the growing bubbles or the degree of indirect cell injury from the decreased oxygenation.

ii. Submersion Injuries (near drowning):

The World Health Organization defines drowning as “the process of experiencing respiratory impairment from submersion in liquid”. In the past, saying someone drowned meant that they died. The term “Near-drowning” is used to refer to a victim that survived drowning. There are many risk factors, but some include inability to swim, use of alcohol or drugs, trauma, seizures, hypothermia, stroke, and child neglect. Most of the victims are children younger than 5 years of age, or males between ages 15 and 25. Submersion injuries begin when there is swim failure, air hunger, and there is a struggle to keep the airway clear of water. When a person is submerged in a liquid medium, vital tissues will become hypoxic and acidotic. The breath-hold time following submersion will vary with factors that include water temperature, clothing worn, exercise life, training, aerobic fitness and more. Most drowning victims swallow water at the end of breath-holding which causes aspiration of water in the airways. A cough then occurs as an initial reflex response when water meets the lower respiratory tract. Transient laryngospasms happen due to the stimulation of the innervated mucosa of the oropharynx and larynx by the water. In most cases, the hypoxia stops laryngospasm, causing increased aspiration of water to occur. The hypoxemia then leads to loss of consciousness and apnea in seconds to minutes. Since the vital tissues become hypoxic and acidotic, cardiac dysrhythmias occur that progresses from tachycardia, bradycardia, pulseless electrical activity, and asystole. It is important to note that cardiac arrest happens because hypoxemia and not from ventricular dysrhythmias. Aspirated fluids lead to surfactant dysfunction, increased permeability of the alveolar-capillary membrane, decreased lung compliance and a ventilation/perfusion ratio mismatch resulting in respiratory complaints. Hypoxemia affects every organ system, but the major contribution to death is from cerebral hypoxia.

b. Wet vs. dry drowning

Laryngospasm is supposed to protect the airway by allowing the water to be swallowed and not aspirated. Unfortunately, most individuals will have laryngospasms terminated by the hypoxia. This causes aspiration of water into the lungs, which would be termed as "wet" drowning. Only a small percentage (<20%) of individuals develop tight laryngospasm that lasts even after cardiac arrest. These victims will swallow large quantities of water through the digestive tract, and not the respiratory tract. This is termed as "dry" drowning since aspiration of fluids did not occur.

c. Cold water drowning

The temperature of the water can significantly affect the outcomes of a drowning victim. Hypothermia is a protective mechanism that improves survival in prolonged submersion, and the process of water distress to cardiac arrest can go for up to an hour. Submersion in water with a temperature below 0 degrees Celsius will slow the progression of hypoxic brain injury. In cold water submersions, there is an antagonist response of the sympathetic nervous system and parasympathetic nervous system. The parasympathetic response is also known as "cold shock response", where there is a sudden gasp reflex to hyperventilation, vasoconstriction, and increased HR and BP. The other response experienced in the body is the activation of the parasympathetic nervous system also known as the "diving response". This response shunts blood to vital organs, causes bradycardia, reduces the electrical and metabolic activity of the brain, and reduces cerebral oxygen consumption. This prolongs the chances of survival since it delays cell death signaling. However, since there are antagonistic responses from the autonomic nervous system, the victim is more at risk for cardiac arrythmias which can cause sudden death.

d. Fresh water vs. Salt water

Immersion in either saltwater or freshwater is very important in non-fatal drowning because the aspiration of fluids can cause fluid imbalances in the body depending on the osmotic gradient. Freshwater is hypotonic and it leaks into the capillary bed and circulatory system. Also, freshwater is usually contaminated with chlorine, mud, or algae causing the breakdown of lung surfactant, fluid leaking and pulmonary edema. Aspiration of saltwater draws fluid into the alveoli because it is hypertonic. This impairs alveolar ventilation leading to hypoxia. The body tries to compensate by shunting blood to the lungs, but it leads to increased pulmonary pressures and a decline in respiratory status. The inadequate oxygenation of blood causes worsened hypoxemia resulting in cerebral injury, edema, and brain death.

e. Signs and Symptoms

The signs and symptoms are determined by the amount of water aspirated, the reaction of the airways, and the hypoxia. Pulmonary insufficiency can develop rapidly, and is shown by an increased respiratory rate, and adventitious breath sounds upon auscultation. Neurologic injury can vary from subtle changes in level of consciousness to comatose. Dysrhythmias, such as sinus tachycardia, sinus bradycardia, PEA, and asystole can occur. ST segment changes in the ECG can show myocardial ischemia. Coldwater victims can have significant hypotension from

hypovolemia. The victim still might be breathing, coughing, and fully alert following incident, or they may not be breathing at all and unconscious. The most common laboratory abnormality is metabolic acidosis secondary to lactic acidosis.

f. Risk pulmonary edema

Fluid in the lungs, loss of surfactant, and increased capillary-alveolar permeability results in decreased lung compliance and ventilation/perfusion mismatch. Noncardiogenic pulmonary edema arises, alters the exchange of O2 and CO2 and predisposes the victim to acute respiratory distress syndrome. Upon pulmonary auscultation, rhonchi, rales, or wheezing may be present indicating respiratory distress. Increased respiratory rate and bloody froth from mouth is a big indication of pulmonary edema.

7. On-scene treatment

a. Decompression Sickness

If DCS is suspected, emergency services need to be contacted immediately, as well as the Divers Alert Network. DAN is an emergency hotline of dive medicine specialists. The basic on-scene treatment revolves around stabilization of airway, breathing, and circulation as needed. Administration of 100% oxygen is first treatment, even if adequate oxygen levels, because it helps flush residual inert gases from the tissues. Then, rapid transportation is crucial.

b. Submersion Injuries

The drowning chain of survival is a series of interventions that reduces the mortality with drowning. It is important to recognize distress and call for help as soon as it is recognized. Signs of distress include head low in the water, head tilted back with mouth open, hyperventilating, attempting to roll over on back, and more. Calling for help is key because it ensures early activation of professional rescue and medical services. The rescuer should provide flotation to the victim to stop the process of drowning. It is important for the rescuer to ensure self-safety and not become a victim themself. If the victim is unconscious and a few rescue breaths does cause a response, cardiac arrest should be assumed and transfer to land immediately. Once the victim is taken out of the water, a quick assessment needs to be done. The respiratory status can be done by placing ear next to the persons mouth and observing for chest rise. If the person is unconscious but breathing, then place them in a recovery position. If the person is unconscious and not breathing, their pulses need to be assessed. If pulseless and apneic, immediately begin CPR. Initially, you need to provide five initial ventilations followed by 30 chest compressions. These initial ventilations overcome the high lung resistance from fluid occluding the airways allowing oxygen to reach the alveoli. It is important that the patient is positioned on their back. Following this sequence, chest compressions need to be done at a ratio of two ventilations to 30 compressions. The rescuer needs to maintain a rate of 60 to 100 beats per minute, allowing for full chest rise between pushes, and ensure pressing down at least 2 inches. CPR should be continued until there are signs of life, rescuer exhaustion or medical support arrives at the scene. Use of defibrillators for cardiac arrest in drowning is low since the rhythm is usually PEA or asystole. Active efforts to expel water from the airway should be avoided as this impairs ventilation. If vomiting occurs, place in lateral position to decrease risk for aspiration. Once

EMS arrives, they will take over with CPR and ALS. Peripheral venous access may be initiated to ensure a good alternative route for drug administration. If ventilation and chest compression do not result in resuscitation, then IV epinephrine should be administered. Doses of 0.1mg/kg can be considered if routine dosage fails after initial 5 minutes of CPR. Once resuscitation is successful, an orogastric tube can be placed to reduce gastric distention and prevent further aspiration. Re-warming when hypothermia is present is also very important in the initial management phase. Overall, early call for help and early initiation of CPR increases survival rates and neurological outcomes.

i. When to use C-spine precautions

Studies show that there is a very low spinal injury incident of 0.5% associated with drowning. Cervical spine precautions should be taken for any drowning patient who is suspected and/or demonstrating signs of axial loading. Cervical spine precautions should be done for patients with a history of loss of consciousness secondary to trauma or at a high risk of having suffered axial loading. This can be suspected when there is a loss in motor and sensory function in the hands. Precautions include restricting spinal motion and maintaining the neck in neutral alignment. This can be done by turning the patient as a unit (logrolling) to prevent movement of the spine. It is important that a jaw thrust is preformed, there is no flexion or extension of the neck, and no head tilt or chin lift. Routine cervical spine precautions should not be done because it can impede initiation of proper respiratory interventions.

ii. Respiratory support – CPAP, PEEP

When cardiopulmonary arrest is present and advanced CPR is initiated using a bag-valve-mask ventilation with high flow oxygen until a definitive airway can be initiated. Initial bag-valve may be ineffective because of the fluid in alveoli, so multiple ventilations may be required. An oropharyngeal or nasopharyngeal airway is enough during the initial management. However, an advanced airway needs to be placed as soon as possible and is required for a patient with prolonged periods of apnea. This assists with oxygenation and ventilation and prevents further aspiration of stomach contents. If there is spontaneous ventilation, but oxygenation is compromised then the goal is to keep oxygen saturation above 92% by administering oxygen via face mask at a rate of 15L/min.

8. ED treatment

a. Priority definitive ED treatment

i. Decompression Sickness: Tx- meds, decompression chamber, NI's

Upon arrival of the DCS patient, the nurse needs to ensure administration of 100% oxygen, hydration using isotonic fluids to reduce hemoconcentration, and administer pain medications such as NSAIDs if needed. The DAN emergency hotline has physicians specialized in dive medicine, and it needs to be called, if not so already. They will provide triage assistance, consultation on the DCS patient and assist with transport to an appropriate hyperbaric facility. The goal treatment is to improve oxygenation by increasing nitrogen elimination. The gold standard of care for decompression sickness is recompression in a hyperbaric chamber. Hyperbaric therapy utilizes high pressure oxygen response and elevates the concentration of

oxygen in hemoglobin and plasma. The main mechanism of hyperbaric oxygen therapy is that the solubility under pressure causes an increase in the diffusion gradient allowing for a deeper delivery into tissues. In other words, it helps in correcting hypoxic conditions by increasing oxygen delivery. The increased oxygen pressures have a positive effect of healing of inflammatory and microcirculatory disorders caused by ischemia. The individual is placed in a closed chamber where they will breathe pure oxygen. The pressure inside the chamber and duration is adjusted depending on the pathological conditions of the patient. Hyperbaric oxygen recompression is usually given at a pressure of 250-300 kPa for 2-5 hours to relieve symptoms. Most patients respond well to only a single treatment, but more than one treatment can be prescribed if signs and symptoms persist. The diagnostic confirmation is if the symptoms are released by compression. Patients with mild signs and symptoms may not need recompression therapy and can be managed with supplemental oxygen and analgesic administration.

ii. Submersion Injuries

As soon as the drowning patient arrives to the ED, the airway needs to be assessed and secured, oxygen needs to be provided, core temperature is assessed and ventilation assisting as needed. Pulse oximetry, capnography, and chest x-ray should be performed as soon as possible. Arterial blood gas levels, blood glucose and electrolyte levels are important to be obtained because it will determine how care should be provided. If hypoxemia, acidosis, or hypercarbia is detected, then aggressive treatment, such as endotracheal intubation, is necessary. If hypothermic, administer warmed isotonic IV fluids and provide warm blankets. Address any associated injuries, such as cervical spine injury if present. If the patient has a Glasgow Coma Scale (GCS) of greater than 13 and an oxygen saturation of greater than 95%, then the patient is at low risk for complications. Low risk patients are observed for around 4 to 6 hours and if their vital signs remain normal, they are discharged home. Those with a GCS of less than 13 should be maintained on supplemental oxygen or respiratory support as needed. Higher risk patients are generally admitted to the ICU. Continuous cardiac monitoring, pulse oximetry, temperature monitoring and frequent reassessments should be performed for all patients. If the patient is in cardiopulmonary arrest or asystole upon arrival to ED, may have to discontinue resuscitation efforts.

1. Respiratory support – CPAP, PEEP

An oropharyngeal or nasopharyngeal airway is enough during the initial management. However, an advanced airway needs to be placed as soon as possible and is required for a patient with prolonged periods of apnea. This assists with oxygenation and ventilation and prevents further aspiration of stomach contents. Positive end expiratory pressure (PEEP) should be added initially at a level of +5 and should be increased in increments if needed and possible. High levels of PEEP improve oxygenation by assisting with the resorption of fluid from the alveoli to the vasculature. The PEEP should be used until the desired P:F ratio of greater than 250 is achieved. PEEP should be maintained unchanged for at least 48 hours prior to weaning. Premature ventilatory weaning may cause the return of pulmonary edema. As mentioned, spontaneous ventilation, yet persistent hypoxemia can be treated with noninvasive O2 measures like continuous positive airway pressure (CPAP). The goal is to maintain oxygen saturation above 92%. CPAP can also be used as a weaning strategy from mechanical ventilation.

9. Role of the ED nurse

a. Decompression Sickness

The role of the ED nurse is to maintain adequate airway, breathing and circulation. The nurse needs to be quick in providing supplemental oxygen, ensuring a patent IV, and administering fluids. The initial evaluation is crucial so that the nurse can determine the degree of severity. If the patient is conscious, the nurse should ask about the onset, duration, and progression of symptoms. The nurse needs to determine the dive profile and gas mix, and a detailed neurological exam. The duration of dive, maximum depth, breathing gas mixture, decompression stops, altitude exposure after fiving and the number of recent dives should be assessed. DCS should be suspected if related symptoms occur after a decrease in blood pressure within 24 hours of diving. Also, education on ways of prevention, and to stay away from high altitudes currently prior to discharge is crucial to prevent further injuries.

b. Submersion Injuries

The role of the ED nurse with a drowning patient is to first maintain adequate airway, breathing, circulation and assess for possible disability. It is important that the ED nurse rapidly obtains the necessary values and diagnostics to determine further treatment. Also, a patent IV needs to be initiated to administer warm IV fluids, or for other emergent administrations. The nurse needs to understand the extent of hypoxia that occurred and what body of water the submersion injury happened to anticipate plan of care. Stabilization of the patient's ABC's needs to be the nurse's #1 priority. The nurse also needs to perform a focused respiratory, cardiac, neurological, and skin assessment is always first. After the patient is stabilized, the nurse needs to further assess the drowning incident to determine if the individual was drowning accidentally, or if it was intentional. Intentional deaths in newborns are not uncommon, so the nurse needs to assess the maternal factors. If it was a suicide patient that survived, the nurse needs to ensure suicide precautions are implemented. This includes a one-to-one sitter, no sharp objects, paper gown, removal of cords from the room, etc. The nurse also needs to provide education about how to prevent this from happening in the future.

10. Discharge/ prevention instructions

a. Scuba diving risks and prevention

SCUBA divers face potential danger of decompression sickness with every time they dive because of the changes in pressure and physics of the gases. However, it is a rare case and preventative measures can be taken. Educate SCUBA divers to be aware of any preexisting cardiac and pulmonary disease and to contact their primary care provider prior to diving. They can reduce their risk by being well-rested, hydrated and avoiding alcohol before diving. The most important preventative measure is a gradual ascent back to surface and the duration limited. Also, educate that they are not able to fly, use hot tubs, or saunas within 24 hours of surfacing.

b. Risks of flying after diving

Exposure to reduced barometric pressure within a short time frame of diving increases a person's risk of decompression sickness. The diver needs to remain at sea level for enough time

to allow for elimination of excess nitrogen from tissue. Flying too soon can cause nitrogen to come out of solution and form bubbles in the tissues ultimately resulting in decompression sickness. For dives that required decompression stops, evidence shows that the diver should wait about 18 to 24 hours before flying.

a. Submersion injuries prevention

Prevention education needs to be specific to the patient and their situation. Since children are at highest risk, the nurse needs to educate the caregiver on adequate supervision as the primary principle of prevention in this age group. If the caregiver is unable to provide sufficient supervision, then consider enrolling child in daycare, or other care facilities. Other measures such as fencing around the pools can significantly reduce the rates of drowning in children. For general age groups, educate on water safety and appropriate flotation-gear if unsure how to swim. Educate males about the consequences of risk-taking behaviors and to refrain from doing so. Educate family and caregivers to learn how to perform CPR. As nurses we need to prevent drowning from occurring and educating on being safe in and around water.

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