Reading assignment:
Aehlert Vol. 2: pp. 553-558

**KNOWLEDGE OBJECTIVES:**

Upon completion of the assigned text readings, class, and homework questions, each participant will independently do the following with at least an 80% degree of accuracy and no critical errors:

1. Define near-drowning.
2. Describe the pathophysiology of near-drowning.
3. Discuss the incidence and risk factors associated with drowning and near drowning.
4. Sequence the events that lead to drowning.
5. Differentiate a dry from a wet near drowning.
7. Describe the lack of significance of fresh versus saltwater immersion, as it relates to near-drowning.
8. Discuss the incidence of "wet" versus "dry" drownings and the differences in their management.
9. Identify the unique aspects of cold water immersion.
10. Explain the mechanism that triggers the Mammalian Diving Reflex and cite the advantages to the patient if this reflex is activated.
11. Discuss the complications and protective role of hypothermia in the context of near-drowning.
12. Correlate the abnormal findings in assessment with the clinical significance in the patient with near-drowning.
13. Identify the body systems that incur dysfunction/insult with drowning.
14. Differentiate among the various treatments and interventions in the management of near-drowning.
15. Integrate pathophysiological principles and assessment findings to formulate a field impression and implement a treatment plan for the near-drowning patient.
16. List the factors affecting survival and predicting a good outcome.
17. List the factors associated with unfavorable outcomes.
18. Organize assessment information into a report for ED personnel.
19. Generalize the physical principles of pressure known as Boyle's law and Henry's law and relate them to diving emergencies.
20. Differentiate diving injuries/accidents that occur on the surface, during descent, on the bottom, and during ascent.
22. Define compression illness (DCI).
23. Identify the various forms of DCI.
24. Identify the various conditions that may result from pulmonary over-pressure accidents.
25. Define nitrogen narcosis and apply "Martini's law" to the patient's presentation.
26. Discuss the etiology, pathophysiology, clinical presentation, and prehospital treatment for air emboli and decompression sickness (the bends).
27. List the factors to be obtained when getting the diving history or profile.
28. Correlate abnormal findings in assessment with their clinical significance in the patient with a diving related illness.
29. Describe the function of the Divers Alert Network (DAN) and how its members may aid in the management of diving-related illnesses.
30. Differentiate among the various treatments and interventions for the management of diving accidents.
31. Describe the specific function and benefit of hyperbaric oxygen therapy for the management of diving accidents.
32. Integrate pathophysiological and assessment findings to formulate a field impression and implement a management plan for the patient who has had a diving accident.
I. Overview
   A. Epidemiology: There are approximately 3,880 deaths in the U.S each year from fatal drowning and 5,789 were treated in EDs for non-fatal near drownings (CDC, 2012). It is considered the third most common cause of accidental death in children. About 90% of all submersion incidents occur within 10 yards of safety. Incidence increases dramatically during the warm weather months due to water-related activities. The elderly drown as a result of submersion in bathtubs.

   B. Definitions
      1. Immersion: To be covered in water or other fluid
      2. Submersion: The entire body, including the airway, is under water or other fluid
      3. Drowning: Process resulting in primary respiratory impairment from submersion/immersion in a liquid medium preventing the victim from breathing air. Conventional use narrows the definition to death by this process.
      4. Near-drowning: Survival, at least temporarily, after suffocation in liquid medium. Death did not occur or occurs more than 24 hours after submersion. Recommended that the term be dropped, but still in frequent use in clinical areas.
      5. Immersion syndrome: This is characterized by sudden cardiac arrest caused by massive vagal stimulation following sudden contact with very cold water. Because of the rapidity with which immersion syndrome can occur, victims are rarely rescued promptly. The majority drown.
      6. Post-immersion syndrome: Delayed deterioration of a previously asymptomatic or minimally symptomatic patient.
      7. Shallow Water Blackout (Hyperventilation drowning): Occurs when swimmers hyperventilate before going under water resulting in a lowered arterial carbon dioxide level thus reducing the stimulus to breathe. With increased muscular activity, arterial oxygen tension is lowered rapidly. Unconsciousness caused by progressive hypoxia will occur with drowning soon to follow.

II. Factors contributing to drowning (risk factors)
   A. Inability to swim
   B. Diving incident causing c-spine/head injury
   C. Alcohol/drug ingestion or intoxication: Be alert for S&S of alcohol use in victim or supervising adult
   D. Hypothermia
   E. Exhaustion
   F. Seizures
   G. Suicide
   H. Hyperventilation prior to underwater swimming
   I. Battery/abuse: Have a high index of suspicion for child abuse or neglect
   J. Lack of parental supervision complicated by improper barriers that fail to limit access to pools, lakes, bathrooms, or buckets of water

III. Sequence of events
   A. Something goes wrong - unexpected submersion: surprise/panic; dysrhythmic breathing or voluntary apnea for up to three minutes. May be complicated or precipitated by altered mentation (seizure/trauma/drugs/alcohol). Victim attempts to keep the head elevated above the water with deep inhalation antecedent to submersion and the instinctive downward movement of the arms. During this time, blood is shunted to brain and heart (mammalian diving reflex).
   B. Submersion with struggling. Victim holds his breath while struggling to raise himself. Apnea produces CO₂ retention/O₂ deprivation. Stimulant effects of the hypoxia ultimately overrides
the sedative effects of the hypercarbia resulting in CNS stimulation: tachycardia, hypotension, hypoxia, hypercarbia, acidosis.

C. Until unconsciousness, victim experiences air hunger and panic. Breakpoint is reached resulting in involuntary inspiratory and swallowing efforts. Water enters mouth, pharynx and stomach, stimulating laryngospasm and bronchospasm. Laryngospasm often prevents water from entering the lungs (dry drowning). If laryngospasm does not occur and water does enter lungs, it was referred to as a wet drowning.

D. Arterial hypoxemia, tachycardia, tissue hypoxia, and tissue acidosis worsens. In the lungs, ↑ peripheral airway resistance, reflex pulmonary vessel constriction, ↓ lung compliance, ↓ surfactant and fluid shifts across alveolar membranes.

E. Agitation ceases; Patient looses consciousness.

F. **Decompensation:** CNS stimulation with reflex gasping and inspiratory efforts results in further water aspiration and/or swallowed resulting in gastric distention and increased risk of vomiting. Patient experiences secondary emesis and secondary apnea.
   1. 85% - 90%: Laryngospasm aborted. Aspiration of water triggers vicious cycle of inflammatory events. Difficult to measure amount.
   2. 10% - 15%: Laryngospasm recurs resulting in anoxia, seizures and death without aspiration.

G. **Extinction of reflexes**

H. Neuronal dysfunction: EEG becomes flat, blood/brain barrier breaks down

I. Cardiac dysfunction: bradycardia, dysrhythmias, asystole

J. **Terminal phase**
   1. Somatic death within 1-60 minutes after submersion (median 3-10 minutes), depending on age, water temp, and degree of tissue hypoxia
   2. Brain death

### IV. Body system insults

#### A. Pulmonary system

1. **Hypoxia**, bronchospasm and pulmonary vasospasm, hypercapnia, and acidemia. Fluid aspiration produces alveolar/arterial $O_2$ difference ($V_a/Q$ mismatch). Consider the tonicity of the fluid, fluid contaminants, and the amount of fluid aspirated.
   
   2. "We have long considered water toxicity a significant cause of morbidity and ultimate mortality, and we still divide immersion into fresh (less than 0.2%/2000 ppm), 'natural salt water' (3.5%/35,000 ppm salinity), and 'salt-water pools' (0.2–0.6%/2000–6000 ppm salinity). Recent research has shown that most of the time, even with 'wet drowning,' not enough fluid enters the lungs to cause electrolyte problems from the water itself" (Krin, 2010).

3. **Hypotonic water drowning** (not a significant EMS consideration)
   Hypotonic water diffuses from alveoli to the vascular space which augments vascular volume and dilutes electrolytes in the blood.
   
   a. Blood cells may rupture releasing hemoglobin.
   
   b. Damages alveolar type II cells causing alterations in the production of surfactant, thickening of the alveolar walls with inflammatory cells, and hemorrhagic pneumonitis.
   
   c. Inactivation of surfactant alters alveolar surface tension which results in alveolar collapse and atelectasis with impaired gas exchange.

   d. Damaged capillaries allow plasma to leak into the alveoli resulting in multiple areas of atelectasis.
e. This leads to intrapulmonary shunting of blood, $V_a/Q$ mismatch and decreased lung compliance with decreased functional residual capacity. Result: clinical hypoxemia.

f. Pulmonary edema/ARDS - later sign (up to 24 hrs after event).

4. **Hypertonic drowning**
   a. May cause direct damage of alveolar capillary membrane.
   b. Hypertonic water pulls water from vascular spaces into alveoli - diluting lung surfactant.
   c. Alveolar flooding occurs; diffuse pulmonary edema - early sign.
   d. Contaminants in water such as sand, algae, chlorine and bacteria further cause additional lung injury.
   e. Aspiration of acidic gastric contents causes liquefaction necrosis, chemical pneumonia and additional cellular injury.
   f. Even an awake patient may be hypoxemic and refractory to oxygen therapy.

B. **Cardiovascular system**: CV complications due to myocardial ischemia resulting from hypoxia, acidosis, hypothermia, and electrolyte disturbance. Clinical findings:
   1. Tachycardia
   2. Ventricular dysrhythmias: VF, bradycardia, or asystole
   3. Varying degrees of heart block
   4. Chest pain
   5. Decreased BP related to ↓ cardiac output due to ↓ contractility
   6. Fever, unless hypothermic
   7. Cyanosis or pallor

C. **Central nervous system**
   1. Five minutes of cerebral anoxia is sufficient to produce irreversible brain damage in warm water. Patient may present with AMS including irritability, restlessness, confusion and lethargy. Seizures, coma, or hyporeflexia may develop.
   2. Hypoxia and hypercarbia coupled with ventricular dysrhythmias and/or asystole promotes arterial hypotension which may cause an acute drop in cerebral perfusion pressure (CPP).
   3. A sudden drop in CPP leads to brain tissue hypoperfusion, cerebral anoxia, tissue acidosis and impaired neuronal metabolism.
   4. Ensuing neuronal cell damage is associated with loss of cell membrane integrity and extracellular leakage of fluid, and diffuse cerebral edema.
   5. Severe cerebral edema will produce ↑ ICP. Not common early; indicates a poor prognosis.
   6. Hypercapnia (high CO$_2$ levels) and vascular endothelial (inner lining) damage resulting from hypoxia disrupt intracerebral vasomotor auto-regulatory mechanisms and uncontrolled intracranial hypertension may develop contributing to further cerebral ischemia.
   7. **Ultimate insult**: brain herniation. Brain tissue is non-compressible. Based on Monroe-Kellie hypothesis, swollen brain tissue will encroach on vessels or subarachnoid space, further obstructing blood flow to tissues. Final insult = brain shift (sub-falx or transtentorial herniation).

D. **Renal system**
   1. Hypervolemia may cause hemolysis (blood cell destruction) and hemoglobinuria (hemoglobin in urine) which causes the urine to appear port-wine stained and can cause acute tubular necrosis (ATN). Hypoperfusion secondary to decreased cardiac output also contributes to ATN.
2. Oliguria (decreased urine output)
3. Proteinuria (protein in urine) – hospital finding
4. Increased BUN and creatinine

E. GI system
1. Dysfunction and distress is related to large volumes of swallowed fluids and hypoperfusion of GI organs.
2. About 50% vomit during resuscitation
3. Anorexia frequently develops.

V. Cold water immersion (CWI)
A. The sequences of the person drowning are generally the same as any other drowning. The physiologic changes are the same. There are, however, some special considerations that may change their prognosis.

B. Immersion hypothermia
1. There is a chain of events associated with prolonged exposure, shivering, and loss of body temperature caused by immersion in cold water.
2. In very cold water, hypothermia develops at very rapid rates and is enhanced by vigorous movement (may increase rate of heat loss by 50%). Water conducts heat 32 X faster than air.
3. Heat loss is high in the groin, neck, and lateral thorax. Peripheral vasoconstriction with decreased blood flow to limbs results in rapid muscle and nerve cooling. Limb strength, coordination, and response time deteriorate. HELP position is important.
4. The rate of cooling varies based on physical characteristics.
   a. Fast: patients who are tall, lean, or have thin skin.
   b. Slow: patients who are short, obese, or have thick skin.
5. Results are poorer if asphyxia and cooling occur simultaneously.
6. Victims who aspirate cold water cool faster than those who do not
7. Generally, the person experiences hypothermia, loses consciousness and then drowns (Titanic victims).
8. Cold provides protective powers against brain destruction for a limited time, but CNS dysfunction occurs as core temperature drops.
9. Mild to moderate hypothermia is characterized by impaired judgment and marked behavioral changes.
10. Metabolic rate is depressed in severe hypothermia. Brain tolerates hypoxia better in CWI than in warm water immersion.
11. Significant hypovolemia may result from diuresis due to central hypervolemia following peripheral vasoconstriction and the increased hydrostatic pressures on body tissues under water.
12. Unconsciousness develops when core temperature reaches approx. 32˚-33˚ C (89.6-91.4˚ F). Swimming ceases and drowning occurs. Brain death may occur in 10-15 minutes.
13. VF occurs at 28˚- 32˚ C (82.4˚ - 89.6˚ F). Asystole is likely at 22˚ C (61˚ F).

C. Mammalian diving reflex: greater relevance to cerebral recovery in children.
1. Cold water may enhance the chances for cerebral recovery after a near-drowning incident. In children and some adults, a diving reflex is triggered by immersing the face in cold water. This results in the following:
   a. A strong inspiratory gasp with cessation of breathing
b. A strong Vagal response with shunting of circulating blood from the skin to the heart and brain. Heart rate rapidly decreases to as low as 5-6 beats/minute mediated by vagal stimulation and peripheral vasoconstriction.

c. A small, sufficient supply of oxygen is very slowly circulated to the lungs, heart, and brain.

2. These patients may appear dead but may actually be alive. Begin resuscitation; leave the extremities cold with active rewarming of the thorax and transport.

D. **Clinical signs of hypothermia**

1. Cold skin
2. Unresponsiveness
3. Cyanosis
4. Apnea
5. Pulselessness
6. Fixed, dilated pupils

VI. **Three classifications of drowning patients**

A. Based on simplified, triage-based, neurologic classification system created by Conn et al. and Modell et al.

1. **A: Alert**, fully conscious at scene; 90% chance of survival w/o neuro deficits.
2. **B: Blunted**; patient who is obtunded but arousable at the scene. He responds purposely to pain and breathes on his own.
3. **C: Comatose** (not arousable, with abnormal respirations and inappropriate or absent response to pain); 34% die, 20% have neurological impairment. Patients may be classified in three stages. Each stage progressively lowers the chance of survival.
   a. Abnormal flexion (old decorticate)
   b. Abnormal extension (old decerebrate)
   c. Flaccid

B. **Morbidity**

1. Immediate CPR at the scene and continued in the hospital is necessary.
2. A cold heart is hard to start, and persistent, prolonged CPR is a key to the patient’s survival.
3. Hearts have resumed beating on their own after up to two hours of CPR.
4. Brain death should not be diagnosed within the first 24 hours. The pupils may remain fixed and dilated and the EEG may be completely flat, but patients have been known to resume cerebral activity after 24 hours and in two weeks be back to normal.

C. **Factors affecting survival and predicting a good outcome**

1. How long the patient was under water (usually unknown, estimate)
2. Water temperature (cold water best)
3. Duration and degree of hypothermia
4. Degree of water pollution/contamination
5. Duration of cardiac/respiratory arrest
6. How quickly and appropriately resuscitation is started
7. How quickly the patient exhibited spontaneous respirations. Within 15-30 minutes after rescue, <10% have cognitive dysfunction or spastic quadriplegia. Within 60-120 minutes or later, 50% - 80% show serious long-term neurological deficits.
8. Age and general health of the victim. Children have greater chance of survival.
9. GCS > 5 at two to six hours after rescue
10. First measured SpO₂: < 90% is a bad sign
D. Unfavorable outcomes foreseen with the following:

1. GCS < 3
2. Flaccidity
3. Sustained, increase in ICP
4. Apneic, no cardiac output
5. Fixed, dilated pupils
6. Intractable metabolic acidosis
7. Prolonged resuscitation
8. Gross pulmonary edema

VII. Prehospital assessment and management

A. All victims of drowning who require any form of resuscitation (including rescue breathing alone) should be transported to the hospital for evaluation and monitoring, even if they appear to be alert and demonstrate effective cardiorespiratory function at the scene (Class I, LOE C).

B. All persons submerged ≤ 1 hour should be resuscitated unless signs of obvious death.

C. Rescue and removal: Gain access to pt taking precautions not to put your safety at risk. "As with any EMS response, scene safety is paramount, because an injured or entrapped rescuer is just another victim. Various training programs provide appropriate training for rescue and other EMS personnel, and departments with known water hazards beyond backyard pools are encouraged to provide that training to responding personnel. Situations again vary from simple, shallow pools to the flat waters of a calm pond or lake, with the most challenging rescues occurring in the fast water of floods and wilderness whitewater streams or rivers. Rescue/recovery diver training, available from the Professional Association of Diving Instructors (PADI) and the National Association of Underwater Instructors (NAUI), as well as other sources of advanced scuba instruction, is useful for flat-water situations (slow currents as found in lakes, bays and larger, slow-moving rivers). Swiftwater rescue training is available from sources like the International Rescue Instructor's Association and Rescue 3 International, among others. Just as water entry techniques require training, rescue boat operators should also be trained, as there are significant hazards that even experienced 'flat water' boaters do not appreciate. If there is a significant search area to be covered, then coordination of air assets, primarily helicopters, is essential to reduce the time to find survivors. Just as structural entry firefighting requires intensive, realistic team training for personal safety, swiftwater rescue requires the same" (Krin, 2010).

D. Talk, reach, wade, throw, helo, row, go, tow. Rescue personnel should wear protective garments if water temp is < 70°. A safety line should be attached to the rescue swimmer. "Rescuer safety should include: talking the victim down from a panicked state to allow him to self-rescue; reaching for the victim with an appropriate pole, extension ladder or other device; throwing a lifeline with a ring or other buoyancy device attached; wading into shallow water to assist the victim in the last stages of rescue or to allow thrown devices to reach him; coordinating a helicopter rescue; using a boat or other rescue craft; and only going into the water as a last resort. Scene safety and the use of low- to high-risk rescue techniques cannot be emphasized too much."

"Trained and qualified personnel directly involved in the rescue who might end up in the water should have available and be wearing protective equipment (as a minimum, protective technical rescue headgear; gloves; a wet, dry or other exposure suit to prevent rescuer hypothermia; and an appropriate personal flotation device). Class I and II PFDs are designed for use in rough water and will inherently turn an unresponsive victim face up. Class III PFDs are the most common type in use on the water, as they are required for recreational boating and are used by the USCG Auxiliary. Some Type III PFDs are 'speed-rated' for use by water skiers and PWC users. Type IV PFDs are 'throwable,' such as ring or horseshoe-style devices. Type V buoyant work vests, often used by waterborne workers (tugboat deck hands or bridge workers) are less desirable, as they have no inherent face-up capability. They do, however, provide good 'workability.' Other
appropriate equipment as required by the situation, common sense and unit operating procedures should be available and used’ (Krin, 2010).

E. Remove pt from water as quickly as possible. Patient should be kept in a horizontal position if at all possible. Cold-induced hypovolemia, cold myocardium, and impaired reflexes may result in significant hypotension. If hypothermic, appropriate rewarming should be done concurrent with resuscitation.

F. “The reported incidence of cervical spine injury in drowning victims is low (AHA, 2010). Unnecessary C-spine immobilization can impede adequate opening of the airway and delay delivery of rescue breaths. Routine stabilization of the cervical spine in the absence of circumstances that suggest a spine injury is not recommended.”

C-spine stabilization is unnecessary unless history of diving into shallow areas, use of a water slide, high-powered personal watercraft (PWC), boating crash; whitewater incidents, where the victim may be ejected from a canoe, raft or kayak; signs of injury, or signs of alcohol intoxication. Consider possibility of other trauma if associated with crush injuries from ejection into whitewater or from personal watercraft, lacerations from propellers or broken glass/torn metal, and impaled objects from whitewater or personal watercraft incidents. Persons who fall or jump from high bridges (over 10 meters) are also at high risk for blunt and spinal trauma (Krin, 2010).

If found face down w/ possible S&S of injury:
1. Stabilize head and neck; turn patient face up.
2. Open airway and begin ventilation as soon as face up.
3. Float a buoyant spine board under the patient.
4. Remove patient from water on the board and begin CPR.

G. Patient requires very gentle handling; the cold heart is very prone to fibrillating.

H. Assess and resuscitate ABCs

1. Category A: Awake and alert
   a. Symptomatic therapy; careful ongoing monitoring and supportive care.
   b. Focus on 3 critical areas: pulmonary, cardiac, and neurologic.
   c. Respiratory assessment: Evaluate skin color, character of breath sounds, respiratory effort including work of breathing, respiratory pattern, use of accessory muscles, and the type & amount of pulmonary secretions. Monitor pulse oximetry. Look for signs of irritability or restlessness as an early sign of hypoxia. Apply C-PAP if hypoxic.
   d. Cardiovascular assessment: Evaluate heart rate and rhythm, BP, quality and character of peripheral pulses, temperature of extremities, color of mucus membranes and nailbeds, and skin color.
   e. Neurologic assessment: Level of consciousness - arousal and awareness, cognitive ability, presence of seizure activity, degree of motor and pupillary responses.

2. Category B: Altered mental status; purposeful response to pain
   a. ABCs same as above plus the following:
   b. Careful observation of airway/ventilatory status: Noisy ventilations, copious secretions; retractions, grunting, nasal flaring, stridor, use of accessory muscles. No need to clear airway of aspirated water by any means other than suction. Oxygenate before and after suctioning. Abdominal thrusts are contraindicated. Use only if airway is obstructed with matter.
   c. Assess for signs of worsening hypoxia: Restlessness and confusion, incoordination, loss of judgment, lethargy, coma, substernal chest pain, widening pulse pressure, dysrythmias, tachypnea, increased WOB, and central cyanosis.
If awake with adequate ventilatory drive: **CPAP** is the mainstay of management. Start at 60% FiO₂ and 5 cm PEEP if available. The decreased functional reserve (residual) capacity can be partially reversed with positive pressure breathing. Don’t apply strap of CPAP mask due to risk of vomiting w/ aspiration. Hold mask in place. If inadequate ventilations, assist with BVM. Treat pulmonary edema if present.

d. They probably have an alteration in fluid volume and electrolyte imbalance related to aspiration of water.

(1) **Signs of overhydration and hypovolemia**: JVD, tachycardia, bounding pulse, increased BP, dyspnea, hypertension, crackles, edema, pallor, moist, turgid skin. IV TKO.

(2) Do not give fluid challenges if crackles already present or full bounding pulses. If evidence of circulatory insufficiency: large bore IV and treat shock.

e. **Careful observation of neuro status.** Observe for and report S&S of increased ICP: AMS, irritability, nausea and vomiting, posturing, seizures, changes in motor function, respiratory pattern, pupil response, ocular movements, and vital signs (Cushing’s response - ↑ SBP; ↓ HR; ↓ RR).

f. If patient responds to initial resuscitation: Cut off and remove wet clothing; cover with warm blankets if mild to moderate hypothermia. If severe hypothermia, leave extremities exposed, and warm only thorax/core.

3. **Category C: Comatose/cardiac arrest state**

a. Many comatose patients present with overhydration, fever, excitability, and hyper-rigidity which negatively affect neurological recovery. Aggressive interventions as above plus the following:

b. **If unresponsive and ineffective ventilations with a pulse**: Ventilate using BLS airways and BVM. They have a potential for impaired gas exchange related to bronchospasm, increased mucus production, aspiration, pulmonary edema and hypercapnia. Pts usually respond after a few ventilations. Consider need for intubation if patient does not respond to initial bag and mask ventilations.

c. If unresponsive, apneic and pulseless: CPR using traditional A-B-C approach due to hypoxic nature of arrest. CPR should be started on pulseless drowning victims unless it is known that they have been under water for more than one hour. Do not delay CPR to take a temperature. **Do not pronounce dead (confirm triple zero) at scene** unless submerged for over 1 hour. Death should not be pronounced until the patient is rewarmed to at least 90˚ F and fails to respond to resuscitative efforts. Rx per appropriate SOP.

d. Vomiting is common in those who require compressions & ventilations; prepare suction.

e. Remove wet clothing; dry patient as possible and protect against additional environmental exposure while providing initial BLS therapies– especially the chest before applying pads and defibrillating pt.

f. If pt is cold: refer to HYPOTERMIA SOP. “Given the lack of human evidence and relatively small number of animal investigations, the recommendation for administration or withholding of medications is not clear. It may be reasonable to consider administration of a vasopressor during cardiac arrest according to the standard ACLS algorithm, concurrent with rewarming strategies (Class IIb- AHA, 2010).

g. Monitor vital signs including temp q. 5 minutes while unstable if cold.
I. **ED report** - convey the following information to ED personnel:

1. Precipitating event
2. Type and temperature of the water
3. Amount and type of water contamination
4. Degree and duration of immersion/submersion
5. How and when the patient was rescued
6. Time to first resuscitation efforts (duration of untreated cardiac arrest)
7. Initial response to treatment
8. Adequacy and type of ventilation
9. Measurement and production of blood flow during chest compressions (demonstrated by capnography), and return of spontaneous circulation
10. Patient condition on arrival at the emergency department.

VIII. **Diving emergencies**

A. Scuba diving is a very popular recreational sport. Scuba diving emergencies are fairly uncommon but are more prevalent among inexperienced divers. Scuba diving incidents can occur on the surface, in shallow water, or at any depth (Bledsoe, 2006).

B. Reefs that attract snorkelers and scuba divers can be found in the Caribbean, northern South America, Australia, Indopacific, Hawaii, southern California and down into Mexico on the Pacific coast. A reef is any water environment in which one or more organisms are living together and are dependent on each other for survival. Coral can't survive without fish and fish can't survive without coral. People often like to swim, snorkel, and scuba in these last unknown frontiers without appreciation of the lurking dangers.

C. **Protective clothing:** Wet suits are generally constructed of neoprene which insulates and protects the body. People typically only wear these suits if the water is colder than 65°F. Otherwise, they wear a "second skin" which is constructed of 1/8th inch spandex or Lycra. It is basically a swim suit that covers the whole body. They don't insulate and might protect against small hazards like Portuguese Man-O-War (*physa*), but not hard coral or anything that would cause a major abrasion or sting. Without proper protection, swimmers get too close to coral, sponges, and marine life which can produce local injuries via stings, poisons from chemoreceptors, spines, and sharp edges. What looks the prettiest, with bright colors, is often the most dangerous.

D. **Physical principles of pressure:** Water is an incompressible, colorless liquid. Humans at sea level live in an atmosphere of air weighing and exerting an atmosphere of 14.7 pounds per square inch or 760 mmHg. This pressure is called 1 atmosphere absolute or one "ata" and will vary within the environment. At the surface, body fluids have absorbed an amount of nitrogen equal to the partial pressure of nitrogen in air at atmospheric pressure. Body cells do not use nitrogen, it is simply dissolved in blood as an inert gas under normal circumstances.

E. **Gas Laws pertinent to diving**

1. **Boyle's law:** The volume of gas is inversely proportional to its pressure if the temperature is kept constant. Example: doubling the pressure of a gas mixture will decrease its volume by one half. Therefore, 1 liter of fluid at sea level would be compressed to 500 mL at 33 feet below the surface (2 ata) and 250 mL at 66 ft below the surface (3 ata). At sea level, average lungs are under 14.7 lbs of pressure. At depth of 99 feet with same amount of air present, lungs are 1/4th normal size as air is compressed. If additional air enters lungs at depth, it will expand upon ascent causing expansion injury. Never hold your breath while scuba diving.

2. **Henry's law:** At a constant temperature, the solubility of any gas in a liquid is almost directly proportional to the pressure the gas exerts on the liquid. Since the body is primarily made up of water, gases that are inhaled will be dissolved in the body in proportion to their partial pressures. Gas molecules will be absorbed into a given quantity of liquid until a condition of equilibrium is reached where the gas in the liquid reaches a value equal to the partial pressure of the gas. At 33 ft below the
surface, the quantity of oxygen and nitrogen dissolved in the tissues will be twice that at sea level. A diver's body can become saturated with nitrogen if he or she breathes gases long enough under pressure. As the pressure is gradually reduced, the gases in the solution can escape with no noticeable effects. During controlled ascent, dissolved gases escape through respiration or else nitrogen bubbles can form within the body. The ascending diver who comes to the surface too rapidly, not adhering to safety measures, is at risk of becoming a veritable "living" bottle of nitrogen soda.

F. Injuries/accidents generally occur at one of the following four stages:

1. **Injuries on the surface**
   a. Entanglement of lines or in kelp (sea grass) fields
   b. Current shifts and undertows
   c. Cold water immersion with shivering or blackouts
   d. Boats
   e. Improper water entry with scuba equipment - tank is forced upward and hits them in the head. Facial bruises if mask is not held.

2. **Descent problems:**
   a. Gasses are compressible and tissues are not. Sudden increase of pressure on body (barotrauma or the "squeeze") - may cause severe pain.
   b. Usual areas affected: lungs, sinus cavities, middle ear, teeth and area surrounded by diving mask. Higher risk with *sinus infections or clogged eustachian tubes*.
   c. Causes middle ear pain, ringing in the ears (tinnitus), dizziness, hearing loss, rupture of the ear drum (extreme), frontal headaches, or pain beneath the eyes in the maxillary sinuses.
   d. Diver who continues to complain of pain after return to the surface should be taken to a hospital.
   e. If ear drum ruptures, may lose balance or become disoriented and shoot to the surface, thereby sustaining ascent problems.

3. **Injuries on the bottom (or at depth)**
   a. "Rapture of the deep" or nitrogen narcosis occurs due to increased nitrogen affecting cerebral function. As the diver descends, increased nitrogen is forced into the blood. At a depth of about 150 feet there may be noticeable effects as the high partial pressure of N₂ affects the CNS. The person appears intoxicated, taking unnecessary risks. The state of lightheadedness may begin as early as 33 ft (2 atmospheres - or double atmospheric pressures at the surface). Generally the narcotic symptoms are first noticed at 100 ft. beginning with mental confusion and impaired judgment. Any task requiring calculation, judgment or manual skill will reveal these impairments.

   (1) **"Martini's Law"**: under usual conditions, the effect induced by N₂ intoxication at 70-100 feet is comparable to the effect of 1 dry martini on an empty stomach. At 50 feet increments thereafter, the effect is one additional martini for every 50 feet of descent. These symptoms are not affected by time at depth as much as by depth alone as pressure causes the gas solubility. (Most safety standards advocate against diving below 100 feet for this reason - not to mention the fact that you won't see anything below 100 feet. Photo vision extends to about 75-80 feet in clear, non-turbid water.)

   (2) **Individual susceptibility to nitrogen narcosis**: As there is an individual tolerance to alcohol, there is great deal of difference in susceptibility to N₂ narcosis.
3 stages of illness

(a) Stage 1: primary nerve centers are affected. Mild, pleasant drunkenness. Diver swims in complete bliss.

(b) Stage 2: secondary nerve centers affected. Inhibitions are released and person may swim off to race a fish, or perhaps even believe they are a fish.

(c) Stage 3: lower nerve centers affected. Diver is completely relaxed and lapses into total submission to the point of losing sense of self-preservation.

Danger: Diver may panic - ↑oxygen consumption and ↑carbon dioxide production

Oxygen poisoning: Breathing high partial pressures of oxygen over extended periods of time can produce dangerous effects (14-24 hours can cause serious lung irritation). Oxygen poisoning is a convulsive reaction caused by breathing compressed $O_2$ in a depth pressure range of about 2 atmospheres. Sport divers should ALWAYS have their tanks filled with compressed air instead of oxygen.

Ascent problems: most of the serious injuries associated with diving.

(a) Ascent vertigo: After a deep scuba dive the diver's middle ear spaces may not depressurize consistently with the rate of ascent. The inner ear balance mechanism senses this pressure imbalance and reacts spontaneously giving the diver a sudden wave of vertigo. This phenomenon, though startling, has no serious consequence if the diver slows his ascent giving the sluggish middle ear spaces enough time to depressurize normally.

(b) Pulmonary overpressure: pneumothorax, pneumomediastinum, sub-q emphysema and air embolism

(1) May occur if a diver holds his breath during ascent.

(2) Air inside lungs expands while external pressure on the chest decreases.

(3) Rapid expansion causes alveoli and small pulmonary vessels to explode (barotrauma).

(4) This results in pneumothorax, pneumomediastinum, or air emboli.

(5) Divers are taught to purse lip exhale all the way up in an emergency ascent. Otherwise, they have charts that explain how long ascents must take based on the depth and duration of the dive.

(6) Signs and symptoms of air emboli

(a) Any neurologic deficits during or immediately following ascent: assume air embolism

(b) Sharp, tearing pain

(c) Paralysis (hemiplegia) and/or coma

(d) Cardiac and/or pulmonary collapse

(e) Unequal pupils

(f) Wide pulse pressure

(g) Frothy sputum in nose and mouth

(h) Dyspnea; subcutaneous emphysema

(7) Treatment

(a) Remove patient from water; try to make them calm

(b) Support ABCs, position head-down L side. $O_2$ 15 L/NRM

(c) Auscultate lungs for evidence of pneumothorax
c. **Decompression sickness** (Caisson Disease or the "Bends")

(1) Caused by a rapid reduction of air pressure while ascending to the surface following exposure to compressed air. Nitrogen bubbles form in the blood and body tissues. Symptoms present when a diver, exposed to a depth of 33 feet or greater for a sufficient time for the tissues to be saturated with nitrogen, ascends too rapidly. Decompression sickness can also occur after a safe dive if one goes up in an unpressurized airplane or climbs a mountain in a car to too high an altitude within 24 hours of diving.

(2) **General factors predisposing to decompression sickness**

(a) Cold water dives
(b) Diving in rough water
(c) Strenuous diving conditions
(d) Hx of previous decompression incident
(e) Overstaying time at depth
(f) Dive of 80 feet or greater
(g) Rapid ascent; panic, inexperience, unfamiliarity with equipment
(h) Heavy exercise before or after dive to the point of muscle soreness
(i) Flying after diving (24 hour wait is recommended)
(j) Driving to high altitude after dive

(3) **Individual factors**

(a) Age: Older individuals
(b) Obesity
(c) Fatigue; lack of sleep before dive
(d) Alcohol consumption before or after dive
(e) History of medical problems

(4) **Effects can be direct or indirect**

(a) **Direct**

(i) **Intravascular**: Blood flow is decreased causing ischemia or infarct.
(ii) **Extravascular**: Tissues displaced, resulting in pressure on the tissues.
(iii) **Audiovestibular**: Air diffuses into the vestibular system, causing vertigo.

(b) **Indirect**

(i) Platelet aggregation/intravascular coagulation
(ii) Electrolyte imbalances
(iii) Lipid (fat) emboli released

(5) **Types of decompression sickness**

(a) **Type I** (the bends)

(i) Severe abdominal or joint pain that causes patient to "bend" over caused by expansion of gases present in the joint space.
(ii) Pruritus (itching)
(iii) Rash, spotted pallor, or cyanosis, pitting edema
(iv) Cyanosis
(b) **Type II:** All symptoms of type I plus:
   (i) Paresthesias (numbness/tingling)
   (ii) Dizziness/vertigo
   (iii) Nausea
   (iv) Auditory disturbances
   (v) Vestibular (balance) disturbances
   (vi) Paralysis
   (vii) Headache
   (viii) Dyspnea (the chokes are extremely serious)
   (ix) Chest pain
   (x) Loss of consciousness
   (xi) Hemoptysis; frothy, reddish sputum
   (xii) Fatigue (early)
   (xiii) Urinary dysfunction
   (xiv) Back pain
   (xv) Priapism
   (xvi) Dysarthria (difficult speech)

(6) **Treatment:** same as air embolus
   (a) Assess/support ABCs
   (b) Begin CPR if pulseless
   (c) \( O_2 \) 12-15 L/NRM or BVM; unconscious diver should be intubated
   (d) Left lateral Trendelenburg position (10-15˚) if possible.
   (e) Protect from excessive heat, cold, wetness, or noxious fumes
   (f) IV NS
   (g) Anticipate need for benzodiazepine if patient seizes
   (h) Transport to nearest hospital with hyperbaric chamber

G. **General diving history or profile**

1. Time at which symptoms occurred; S&S more than 36 hours after a dive would not be decompression sickness.
2. Type of breathing apparatus used
3. Type of (hypothermia) protective garment worn
4. Parameters of the dive:
   a. Depth
   b. Number of dives
   c. Duration of dive
5. Aircraft travel following the dive
6. Rate of ascent
7. Associated panic forcing rapid ascent
8. Experience of the diver, i.e., student vs. pro
9. PMH
   a. Old injuries
   b. Previous history of decompression sickness
   c. Use of medications
   d. Use of alcohol
References


Homework questions:

1. Define each of the following:
   - Drowning
   - Immersion syndrome:
     - Shallow-water blackout

2. List the risk factors associated with drowning.

3. Sequence the events that lead to drowning.

4. Identify the body systems that incur dysfunction/insult with submersion:

5. Explain the mechanism that triggers the Mammalian Diving Reflex

6. What are the advantages to the patient if this reflex is activated?

7. What are the immediate effects of cold water immersion?
8. List the factors affecting survival and predicting a good outcome in a near-drowning situation.

9. List the factors associated with unfavorable outcomes.

10. Prioritize the prehospital assessment and management of drowning victims including airway access; assisted ventilations, oxygen delivery devices, the order of CPR priorities, and drug therapy depending on their presentation.

   Awake at scene:

   Blunted at scene but respond to pain:

   Comatose at scene:

11. Under what circumstances should the c-spine be protected in a drowning victim?

12. What are the assessment findings that must be reported to ED personnel?
13. Generalize the principles of pressure known as Boyle’s law

   Henry’s law.

14. List diving injuries/accidents that occur:

   On the surface

   During descent

   On the bottom

   During ascent

15. Define nitrogen narcosis (rapture of the deep) and apply "Martini’s law" to the patient's presentation.

16. What is the etiology, pathophysiology, clinical presentation, and prehospital treatment for Air emboli
Decompression sickness (the bends).

17. List the factors to be obtained and reported when getting the diving history or profile.