DEFINITIONS AND DESCRIPTIONS

Several definitions and multiple terminologies have appeared during the past half-century regarding the description of victims who suffer a fatal or near-fatal event from being submerged in water and other liquids. Some of the descriptors have had modifications placed on them and, furthermore, their meaning was somewhat lost when translated into some languages other than English. Because drowning is a global problem, in association with the World Congress on Drowning in Amsterdam, The Netherlands, on June 26 to 28, 2002, a group of experts was convened from multiple countries to develop a definition of “drowning” that would be applicable in multiple languages worldwide (1). Although unanimity may not have been present on every term discussed, there clearly was a consensus to simplify the terminology for international application. What follows is the consensus of that group with comment and, in some cases, slight modification representing the bias of the authors of this chapter.

THE DROWNING PROCESS

Drowning is the process resulting from primary respiratory impairment from submersion or immersion in a liquid medium. Implicit in this definition is that a liquid-to-air interface must be present at the entrance to the victim’s airway, thus precluding the possibility of the victim to breathe air. Although it is possible to suffer a drowning episode in multiple types of liquid, this chapter will be confined to the most common use of the terminology, namely drowning in water.

The drowning process is a continuum that begins when the victim’s airway is initially below the surface of the water. At this time, the victim first will voluntarily hold his or her breath. Some victims will swallow significant quantities of water during this time. This period of voluntary breath-holding, which has been found in human volunteers to last an average of 87 seconds at rest and shorter with exercise (2), is followed by an involuntary period of laryngospasm secondary to water in the oropharynx or at the level of the larynx acting as a foreign body (3). During this period of breath-holding and laryngospasm, the patient cannot breathe; therefore, oxygen is depleted and carbon dioxide is not eliminated. This results in the patient becoming hypercarbic, hypoxic, and acidotic (4).

As the levels of carbon dioxide increase in the blood and levels of oxygen decrease, respiratory efforts become very active but no exchange of air occurs because of the obstruction at the larynx. Victims who subsequently recover and recall this period frequently describe it as being quite terrifying and painful as they struggle, creating intense negative intrapleural pressure breathing against a closed glottis (5). As the patient’s arterial oxygen tension drops further, laryngospasm abates, and the patient then actively breathes water. Further evidence of the magnitude of negative pressure created during laryngospasm is the fact that the lungs of drowning victims frequently demonstrate significant hyperinflation at autopsy (6).

The amount of liquid a drowning victim inhales varies considerably (4). Studies comparing the biochemical changes occurring in humans after a drowning episode with those in experimental animals suggest that, while the volume of liquid actually inhaled varies, only 15% of persons who die in the water aspirate in excess of 22 mL/kg of water (7), and the percentage is considerably less in those who survive (4). Changes occur in the lung, body fluids, and electrolyte concentrations, which are dependent on both the composition and volume of the liquid aspirated (8–10).

RESUSCITATION

A victim can be rescued at any time during the drowning process and given the appropriate resuscitation measures, in which case the process is interrupted. The victim may recover with the initial resuscitation efforts or after subsequent therapy aimed at eliminating hypoxia, hypercarbia, and acidosis and restoring normal organ function. If the patient is not removed from the water, then circulatory arrest will occur, and, in the absence of effective resuscitative efforts, multiple organ dysfunction and death will result, primarily from tissue hypoxia.

Although the tolerance to hypoxia of various tissues is different, it should be noted that the brain is the organ most at risk for permanent detrimental changes from relative brief periods of hypoxia. Frequently, the question is asked, “How long can a person be submerged and still be rescued and resuscitated back to a normal life?” While, obviously, there are no controlled human studies on this subject, the limiting time factor is likely the duration that cerebral hypoxia can be tolerated before irreversible changes occur. Irreversible damage to brain tissue is reported to begin approximately 3 minutes after the PaO₂ falls below 30 mmHg under normothermic conditions in otherwise normal people (11). Such data suggest that if the victim is rescued and effective resuscitation efforts are applied within 3 minutes of the cessation of respiration (i.e., submersion in water), the vast majority of such victims should be able to be resuscitated and suffer no permanent brain damage. Further, because the period of voluntary breath-holding and laryngospasm is thought to last for approximately 1½ to 2 minutes (2,12), persons who are recovered within that time frame will likely not suffer lung damage secondary to the aspiration of liquid. Once the 3-minute time frame has been exceeded, although some normal survivors are reported, it becomes less likely that normal survival will result from resuscitation efforts. This time frame may be prolonged if...
hypothesis occurs rapidly because it decreases the cerebral requirement for oxygen (13,14).

Trained divers have been shown to be able to voluntarily hold their breath for much longer periods of time, approaching 4 to 5 minutes without complication (15). Persons who become hypothermic due to immersion or submersion in extremely cold water will rapidly develop hypothermia, which protects the brain by decreasing its oxygen requirement, and prolongs survival (13,16). In the latter case, seemingly miraculous recoveries of patients who have been submerged for over 20 minutes have been reported (17). It should be noted, however, that hypothermia is a two-edged sword; although it can protect the brain from oxygen deprivation, it also can cause death in the water secondary to its effect on the conduction system of the heart, resulting in circulatory arrest either by asystole or ventricular fibrillation (18,19).

The drowning process can be altered by the initiating event, such as if the victim suffers trauma, develops syncope, or unconsciousness, has a circulatory arrest either by asystole or ventricular fibrillation as the precipitating event, hyperventilates prior to breath-holding under water, or has a convulsive disorder that leads him or her to become incapacitated, thereby becoming submerged, or if the victim’s judgment and/or motor function is impaired by significant parenteral levels of depressant drugs, including alcohol. For example, in the victim who suffers a concussion from a blow to the head, subsequent recollection of the events is unlikely. If trauma results in a cervical fracture, disastrous damage to the spinal cord may occur acutely and, thus, motor function may be lost below that level. If the victim has a circulatory arrest either by asystole or ventricular fibrillation as a precipitating event, respiration will cease, and it is highly unlikely that significant amounts of water will be breathed into the lung given that active respiration is necessary for this to occur. If the victim hyperventilates prior to breath-holding under water, it has been shown that the breath-holding breaking point can be extended until the level of hypoxia is so severe that consciousness is lost, and thus the victim actively breathes in water (2,12). The effect of drug usage is variable, depending on the level of depression and the patient’s response. There is considerable variation in tolerance to depressant drugs and alcohol and their effects on performance and orientation. To better understand what to expect in each victim, the initiating event should be reported in every case if it is known.

Drowning episodes can lead to many possible outcomes. On the most basic level there are two outcomes: death or survival. Of the survivors there are, however, many outcomes: no residual damage, to minor neurologic difficulty to severe disability, bedridden and requiring continual nursing care.

### CLASSIFICATIONS

Numerous terms have been used to describe the episode of submersion and its sequelae. Many of these have fallen out of favor due to the confusion in meaning (Table 111.1). The terms drowning and near-drowning have been used for decades in an attempt to separate these outcomes (20). At the World Congress on Drowning, however, it became apparent that their meaning was not felt to be clear when translated into some languages (1). Furthermore, a victim could have no signs of spontaneous physiologic function and, therefore, be “drowned”; however, once resuscitative efforts were applied, they would respond positively and survive to varying degrees and, thus, the term applied to them would have to be changed to “near-drowned” (21). In addition, there is another group who do not die acutely, but die later of complications from their drowning episode. In this case, the question is, were they “near-drowned” or were they “drowned?”

The definition of “drowned” we believe to be fairly clear—namely, death secondary to undergoing the drowning episode. “Near-drowned” presents a significantly greater problem of understanding. We believe that the term “drowned” should be retained for both those who die acutely in the water and those who die later of consequences directly resulting from the submersion episode. However, we agree with the consensus of the World Congress members that “near-drowned” may lead to unnecessary confusion and, therefore, should be replaced by terminology such as “the victim survived the drowning episode” and then describe the ultimate condition of the victim.

Other terms that have appeared in the literature over the past few decades that we believe are confusing and should be abandoned are as follows.

### Dry versus Wet Drowning

Because all drowning occurs in liquid, by definition, they are all wet. This terminology has been used by some to categorize drowning victims into those who aspirate liquid into the lungs and those who do not. Frequently, it is not possible to determine at the scene of the accident whether the victim actually did aspirate water. This is particularly true when the quantity of water aspirated is small. Further, if evidence of fluid aspiration is not detected in the victim who dies or is discovered dead in the water, the diagnosis may be suspect (22,23). In these cases, one should look for other explanations such as acute
mechanical standstill of the heart, from asystole or ventricular fibrillation, or, for that matter, whether the victim was actually alive when he or she first became submerged.

**Active versus Passive versus Silent Drowning**

This terminology has been used by some to describe a situation when a precipitating event from another origin (e.g., syncope) causes a victim to be below the surface of the water, and then he/she drowns. On the other hand, some use this terminology to describe a victim who appears to be recovering from a drowning episode in the hospital and then develops adult respiratory distress syndrome. Not only is this terminology confusing but also, in the latter instance, a patient does not experience a second submersion or drowning episode, and therefore, this terminology should be abandoned.

**Secondary Drowning**

This terminology has been used by some to separate those victims who are observed to be struggling at the surface of the water from those who are first discovered when they are actually submerged and motionless. It has been shown with underwater cameras that even victims who were not seen to be in difficulty on the surface of the water by observers may have had unrecognized active motion while submerged. We believe, therefore, that these terms should be abandoned in favor of the terms “witnessed,” when the episode is witnessed from the onset of submersion/immersion to the time of rescue, or “unwitnessed,” when a body is found in the water without direct knowledge of how long ago the incident occurred.

**PATHOPHYSIOLOGY**

There have been extensive studies both in animals (7–9, 16, 20, 24–35) and in humans (4, 6, 22, 36–41) over the past century in an attempt to quantify the changes that occur as a result of a drowning episode. What has consistently been shown is that, acutely, drowning produces asphyxia (i.e., hypoxia, hypercarbia, and acidosis). The hypercarbia is due to absent or ineffective respiration, and is readily correctable when aggressive mechanical ventilation is instituted. The hypoxia that occurs initially is not as readily correctable and may be persistent for long periods of time (8–10). This hypoxia is first due to apnea, and then primarily to intrapulmonary shunting from alveoli that are perfused but not being ventilated, or not being ventilated adequately (33). The acidosis is mixed, and the respiratory component rapidly disappears with effective ventilation. The patient is, however, frequently left with significant metabolic acidosis due to anaerobic metabolism during the period of time that profound tissue hypoxia secondary to absent or ineffective respiration and cardiac output was present. The hallmark of this high anion gap metabolic acidosis is an increased level of serum lactic acid.

**Pulmonary**

While intrapulmonary shunting occurs after both freshwater and seawater aspiration, the etiology is different (Table 111.2) (13, 34, 42). In the case of freshwater, the aspirated water alters the surface tension properties of pulmonary surfactant. Thus, the alveoli become unstable and do not maintain their normal shape or patency, resulting in an increase in both absolute and relative intrapulmonary shunt (33, 34). Seawater does not change the surface tension properties of pulmonary surfactant but, because it is hypertonic, it pulls fluid from the circulation into the alveoli, disrupts the capillary–alveolar membrane, which leads to further permeability and thus producing obstruction to gas exchange at the alveolar level (42); bronchoconstriction also has been reported after aspiration of even small quantities of water (29).

Freshwater, being hypotonic, is absorbed very rapidly into the circulation and, because of the transient hypervolemia that occurs and the change in the surface tension properties of pulmonary surfactant, pulmonary edema results. The pulmonary edema is most commonly described as frothy or foamy and blood-tinged. This coloring is secondary to the presence of free plasma hemoglobin from the rupture of some red blood cells due to the absorption of hypotonic fluid into the circulation in the face of hypoxia (43). Pulmonary edema also occurs when sea water is aspirated, secondary to a semipermeable membrane effect because the seawater is hypertonic compared to plasma. Even though the etiology of the hypoxia is different between freshwater and seawater aspiration, the result of both is to increase intrapulmonary shunt, decreased compliance, and bronchospasm all of which requires aggressive therapy (13, 33, 42, 44, 45).

Extensive studies of serum electrolyte concentrations after drowning have shown that only 15% of victims who die in the water aspirate more than 22 mL/kg of water. In patients who survive, the percentage is much less and, thus, significant changes in serum electrolyte concentrations that require treatment are rarely observed (7), with the only exception being, perhaps, victims of drowning in the Dead Sea (46).

The treatment of the respiratory lesion requires providing mechanical ventilatory support in a fashion that will restore an adequate functional residual capacity and keep the alveoli open during all phases of the respiratory cycle, thus

<table>
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<tr>
<th><strong>TABLE 111.2 Freshwater versus Seawater Effects on Pulmonary Physiology during Aspiration</strong></th>
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<tbody>
<tr>
<td><strong>Freshwater Aspiration</strong></td>
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<tr>
<td>Intrapulmonary shunting</td>
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<tr>
<td>Surface tension</td>
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<tr>
<td>Fluid movement</td>
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<td>Capillary–alveolar membrane</td>
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<tr>
<td>Pulmonary edema</td>
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<tr>
<td>Bronchoconstriction</td>
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<td>Compliance</td>
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decreasing the intrapulmonary shunt. Obviously, if foreign material such as sand, silt, or plant life is aspirated into the lung, it may produce obstruction, and it should be removed via bronchoscopy.

**Cardiovascular**

The cardiovascular changes that occur during a drowning episode can best be ascribed to inadequate oxygenation. Although fatal dysrhythmia, such as ventricular fibrillation, is rarely documented in human drowning victims, ventricular fibrillation can occur with profound hypoxia, especially if very significant changes in serum potassium and serum sodium result from the movement of fluid and rupture of red blood cells. Usually the sequence of cardiac dysrhythmias progresses from tachycardia to bradycardia, then pulseless electrical activity and finally asystole (42,47). Although a wide variety of cardiac dysrhythmias have been reported (20), particularly in animal models, rarely do they require specific therapy other than improving oxygenation, correcting severe metabolic acidosis, fluid resuscitation, and correcting temperature (13,42). More common problems are profound hypoxia and the leak of fluid into the lung as pulmonary edema, resulting in a relative hypovolemia in the patient. It has been shown by multiple investigators that to treat this hypovolemia, it may be necessary to infuse significant amounts of intravenous fluid to maintain an adequate effective circulating blood volume, even in the face of pulmonary edema (27,45). Without such therapy, even though the arterial oxygen tension might have improved with mechanical ventilatory support, the delivery of oxygen to the tissues remains compromised and incompatible with supplying adequate tissue oxygenation (45).

**Neurologic**

The neurologic injury that occurs during drowning, like cardiovascular complications, can also be attributed to hypoxia and its risk is similar to patients who require cardiopulmonary resuscitation (CPR) from other etiologies (13). When cerebral blood flow drops to less than 20 mL/100g/min, cerebral metabolic oxygen demands exceed oxygen supply, and neuronal ischemia will begin to occur and permanent injury can occur within 4 to 10 minutes to the hippocampus, basal ganglia, and cerebral cortex (48,49). As previously discussed, hypothermia commonly occurs with the drowning incident and, if this is the case, it may have neuroprotective qualities and decrease the amount of neurologic injury expected to occur for the length of period with decreased oxygen delivery; this occurs due to the decrease in cerebral metabolic rate at lower temperatures as, for each degree of Celsius below 37°C, the cerebral metabolic rate decreases by 5% to 7%. In addition to the primary injury at the time of the event, there is also secondary injury and delayed neuronal cell death (49). There are complex cellular signals and responses to both hypoxia and to reperfusion, which may lead to subsequent pro-death signaling (49).

**Renal**

Detrimental changes in renal function are rarely seen in persons recovering from near-drowning. However, when present, they likely are the result of inadequate perfusion and oxygenation rather than anything specifically related to the drowning episode per se. Some have emphasized the need for the kidneys to clear free plasma hemoglobin after freshwater drowning; however, significant levels of free plasma hemoglobin have rarely been reported in such patients. This is likely due to the fact that for red blood cells to rupture and release enough hemoglobin into the plasma during a drowning episode to require specific therapy for its clearance, it requires transfer of substantial volumes of free water into the circulation in the face of hypoxia (43); as stated above, this rarely occurs.

**INITIAL RESCUE AND RESUSCITATION**

To ensure survival after a drowning episode, it is imperative that one never lose sight of the fact that time is of the essence. The longer a person is without the ability to breathe air, the more profound are the hypoxia and permanent damage to vital tissues. Thus, those who are entrusted with guarding swimming facilities must never lose sight of the fact that continual vigilance is required to recognize a victim in distress, and that the victim must be removed from the water and resuscitative measures begun in a timely fashion. Frequently, bodies are discovered motionless in a pool, without anyone in attendance being able to pinpoint the length of time that the victim was submerged. If an individual is not noted to be making purposeful movements for more than 10 seconds, rescue attempts should be initiated. The individual responsible for safety at the pool should always be in proper attire and in position to initiate such a rescue and complete it within 20 seconds of the recognition of the problem.

While removing the victim from the water, care should be taken to avoid complicating neck injuries when they are suspected. Routine stabilization of the neck is unnecessary unless the circumstances leading to the drowning episode suggest that trauma was likely (50). These circumstances include a history of diving, use of a water slide, signs of injury, or evidence of alcohol intoxication. If neck injury is suspected, gentle immobilization of the head should be accomplished, securing it in a neutral position. However, if the neck appears to be obviously deformed and the patient has pain with neck movement, the neck should be immobilized in the existing position.

If the victim is apneic, the airway should rapidly be cleared of foreign material, a patent airway secured, and mouth-to-mouth resuscitation started immediately. It is preferable to begin artificial ventilation in the water if it can be accomplished without jeopardizing the safety of the rescuer. Starting resuscitation efforts while in water increases the chances of a good outcome by threefold compared to waiting until a victim is brought to land (51). In-water resuscitation efforts are, therefore, ventilation alone due to the inability to perform chest compressions (50). It should be remembered that not all victims are in a state of cardiac arrest when the rescue attempt begins. They may be in a state of vasoconstriction or have a significant bradycardia, in which case, if effective ventilation is started, the myocardium will be re-oxygenated, and increased cardiac activity will result in improved tissue perfusion. Upon removing the victim from the water, he or she should rapidly be assessed for the presence of both spontaneous respiration and cardiac activity. In the absence of these, the airway should be inspected rapidly to ensure that there is no mechanical obstruction, and artificial respiration and cardiac...
compression should be instituted without delay. Although, chest compression alone—without artificial respiration—is an alternate resuscitation method that has been proposed for victims of dysrhythmic cardiac arrest, it must be emphasized that these recommendations do not apply to the drowning victim because the pathophysiologic lesion in the lungs requires active attempts at re-inflation and stabilization of the alveoli. Therefore, cardiac arrest following drowning is more likely due to asphyxia, and thus, immediate provision of ventilation is recommended (50). Resuscitation efforts should follow the traditional airway-breathing-circulation (ABC) sequence, which starts with 5 initial rescue breaths, then 30 chest compressions, then 2 rescue breaths, then 30 chest compressions, and repeated until return of spontaneous circulation occurs. Five initial breaths are recommended instead of two due to water in the airways in difficulty performing the initial breaths (50,52,53).

PATIENT TRANSPORT AND EMERGENCY MEDICAL SERVICES

Neither equipment nor properly trained personnel are usually available at the site to provide advanced cardiac life support, including endotracheal intubation, intravenous access, drug therapy, and electrical defibrillation. However, these measures should be instituted when indicated and when the proper equipment and properly trained personnel are available. It is crucial that someone other than the individual rescuing and resuscitating the patient contact emergency medical services (EMS) as rapidly as possible so that they can respond in a timely fashion and perform advanced cardiac life-support treatment on the victim.

Whenever a drowning victim has to be transported to a location or facility such as a hospital emergency room, it is important that a call be made promptly to inform the emergency room personnel of the exact circumstances, type of treatment instituted, and condition of the patient en route so that they will be prepared to accept the patient and render appropriate therapy immediately upon arrival.

When moving a critically ill drowning victim, it is imperative to remember the fragility of such patients because they can decompensate in a matter of a few seconds or minutes if appropriate therapy is withdrawn. Examples of such situations are movement (a) from the scene to the EMS vehicle, (b) from the EMS vehicle to the hospital emergency department, or (c) from the hospital emergency department to other hospital locations for testing, such as radiology, or for treatment, such as the intensive care unit (ICU). Thus, every attempt should be made to continue essential therapy at all times.

Treatment in the Emergency Department

In the emergency department, a thorough evaluation of the patient should be performed, keeping in mind that the most serious problems that require immediate therapy are pulmonary insufficiency and cardiovascular instability, which result in inadequate delivery of oxygen to vital tissues. If the victim is responding fully, does not require respiratory or cardiovascular support, and has a normal oxyhemoglobin saturation while breathing room air, it is unlikely that the victim has aspirated a significant amount of water, and observation may be all that is necessary. At the other extreme is the patient who is still unconscious and requires extensive pulmonary and cardiovascular support in an attempt to normalize vital signs and produce adequate cardiac output and tissue oxygenation. Thus, a cookbook-type treatment that would apply to every victim cannot be prescribed. However, the treating physician should keep in mind that increased intrapulmonary shunt and poorly matched ventilation-to-perfusion ratios are the rule rather than the exception for the victim who has aspirated a significant quantity of water.

Therapy must be aimed at improving ventilation-to-perfusion ratios and restoring adequate residual lung volume to optimally oxygenate the blood. A relative hypovolemia frequently is present due to fluid shifts between the lung and the circulation. These can be accentuated by the increase in mean intrathoracic pressure that occurs with mechanical ventilatory support. Thus, evaluation of effective circulating blood volume and replenishment of intravascular fluid volume to physiologic levels is important as a primary concern.

If the patient is a victim of seawater drowning and has aspirated sufficient water to produce hypernatremia, we might be better advised to use an agent such as tris-(hydroxymethyl) aminomethane (Tris) buffer, 2-amino-2-hydroxyl-1,3-propanediol (THAM) to avoid compounding the hypernatremia. However, once again, it should be noted that the quantity of water aspirated is seldom sufficient to produce such significant changes in serum electrolyte concentrations, except perhaps when the drowning occurs in water of extreme hypersalinity such as the Dead Sea (46).

Changes in serum electrolyte concentrations and hemoglobin and hematocrit of sufficient magnitude to justify specific therapy are rare, as are alterations in renal function other than those that might be expected in the hypovolemic, hypoxic, or markedly acidic patient.

The patient's level of consciousness on admission to the emergency room has been shown to markedly influence outcome (54,55). The most important consideration here is to provide adequate oxygenation and perfusion and to avoid producing increased intracranial pressure if possible. Treatments aimed specifically at preservation of cerebral function have not been shown to be particularly beneficial to date (49,55,56).

If the patient requires diagnostic testing in a distant location such as the radiology department, it is imperative that adequate personnel and equipment accompany the patient to ensure that optimum therapy is not interrupted at any time during transport or when performing the procedure. Likewise, transportation to the ICU should be done with a “full team approach.” Should optimum therapy be interrupted during any of these time periods, adverse consequences should be anticipated.

Drowning episodes in cold water may produce significant hypothermia. There are several methods of rewarming that have been recommended including, but not necessarily limited to, heating blankets, warmed intravenous fluids, warmed humidification of breathing circuits, gastric lavage, and cardiopulmonary bypass. The method used should be tailored to the resources available and the condition of the patient. It must be remembered, however, that rewarming peripheral tissues before the patient's circulation is capable of supplying adequate amounts of oxygenated blood can compound the situation and increase the degree of metabolic acidosis.
IN-HOSPITAL THERAPY:
POSTRESUSCITATION CARE

Expert intensive care is vital to survival once optimal prehospital and emergency department management have been performed. Hemodynamic instability after cardiac arrest, respiratory insufficiency, and severe neurologic impairment are all criteria for admission to the ICU. The administrative structure of the hospital’s critical care service dictates the setting to which the patient is admitted. A recent attempt to classify survivors of drowning based on the severity of symptoms on a scale of 1 to 6 recommends ICU admission for all pediatric patients requiring high concentrations of oxygen, with or without the need for invasive ventilation (57).

Respiratory Support

Although the degree of intrapulmonary shunting after drowning is variable from one patient to the next, if the patient is breathing adequately to clear carbon dioxide, the single most important method of treatment in reversing hypoxemia is the application of continuous positive airway pressure (CPAP). The amount of CPAP applied must be individualized because the degree of atelectasis, the amount of pulmonary edema, and the magnitude of the intrapulmonary shunt varies between patients. In great measure, this will depend on the type and quantity of the water aspirated. Although the mechanism for producing the intrapulmonary shunt is different between freshwater and seawater (34), Lee (58) found no statistically significant difference between the PaO2/FiO2 ratio in patients after the two types of aspiration.

The pathophysiologic mechanism involved in freshwater drowning is lowering of the sodium concentration in the alveolus, thus changing the surface tension characteristics of pulmonary surfactant (34). The alteration in the surface tension properties of pulmonary surfactant increases alveolar surface tension upon compression of the surfactant layer and results in alveolar volume loss. Also, pulmonary capillaries become more permeable, resulting in an increase in interstitial lung water that eventually compresses alveoli and promotes volume loss and causes pulmonary edema. Based on the severity of the acute respiratory derangement, this “abnormal surfactant state” has been termed mild, moderate or severe acute respiratory distress syndrome (ARDS) (39).

ARDS represent a final common pathway that accompanies a number of physiologic insults that may occur after drowning, including respiratory obstruction, aspiration of water or gastric contents, and global hypoxemia from cardiovascular insufficiency or cardiac arrest. Unfortunately, ARDS often can be clinically and radiologically confused with acute pulmonary edema from left ventricular dysfunction or fluid overload of different etiologies.

Both CPAP and positive end-expiratory pressure (PEEP) have the capability to restore lung volume and improve oxygenation in many patients with decreased lung volume, especially functional residual capacity. However, there are some differences in their function. By definition, CPAP means that airway pressure remains positive during all phases of the respiratory cycle. With PEEP, during the inspiratory phase of a spontaneous breath, circuit pressures drop to zero or become negative as a result of a vigorous inspiratory effort by the patient. Because PEEP with spontaneous ventilation increases the work of breathing, it may increase pressure gradients between the pulmonary vasculature and the alveoli, thereby leading to more pulmonary edema. Also, it does not forcibly inflate alveoli with abnormal surfactant after freshwater drowning (33). Thus, CPAP is more beneficial than PEEP for spontaneously breathing drowning victims (44,60).

Both CPAP and PEEP increase expiratory pressure; thus, air is trapped within the lungs during the expiratory phase of respiration. This results in an increase in residual lung volume in many patients with ARDS. As alveolar units re-expand, intrapulmonary shunt decreases, and improvement is seen in oxygenation and compliance. The increase in compliance decreases the work of breathing (61). The degree of lung volume restoration roughly correlates with the improvement in oxygenation. As lung volume increases toward normal, gas exchange continues to improve. It has been shown, however, that while the above beneficial effect is found with CPAP in many victims of both freshwater and seawater drowning (60,62), unless mechanical breaths are added, PEEP does not improve the ventilation-to-perfusion ratio after freshwater drowning (33,44,60). Additionally, in some freshwater drowning victims, CPAP alone does not produce an adequate response, and hence, mechanical breaths should be added (44).

When ARDS develops and oxygen desaturation occurs, an FiO2 of 1.0 is initially recommended to attempt to restore adequate oxygenation. Increased work of breathing, severe hypoxemia, and hypercapnia are all indications for instituting mechanical ventilation. Ordinarily, CPAP is titrated to achieve an oxygen saturation greater than 95%, with the lowest possible inspired oxygen (FiO2) levels down to an FiO2 of 0.4 or less. We routinely increase CPAP at the bedside in increments of 3 to 5 cm H2O in an attempt to achieve an oxygen saturation of 95%, and subsequently, the FiO2 is gradually decreased to reach a PaO2/FiO2 of greater than 300 mmHg. Increased dead-space ventilation and decreased preload are the two most important adverse effects that can limit the use of CPAP. Once adequate PaO2/FiO2 has been achieved, CPAP can slowly be weaned based on improvement of patient lung compliance and general clinical conditions.

Mechanical Ventilation

CPAP therapy alone is not sufficient in the case of the patient who is apneic, hypventilating, or hypercarbic, or shows little to no improvement in ventilation-to-perfusion matching while breathing spontaneously. In these patients, mechanical ventilatory breaths must also be provided. In general, mechanical ventilation in patients with ALI or ARDS can be applied either noninvasively or invasively (i.e., face mask vs. endotracheal tube, respectively). Noninvasive ventilation is reserved for milder cases of ARDS or pulmonary edema when the patient is awake, cooperative, triggering spontaneous ventilation, and has his/her swallowing and protective laryngeal reflexes intact. Although successful experience with noninvasive positive pressure ventilation (NPPV) for patients with respiratory failure other than from chronic obstructive pulmonary disease (COPD) is growing (63), potential complications include gastric distention, nasal congestion, regurgitation and aspiration of stomach contents, nasal bridge ulceration, and eye irritation (64). Several modes
of mechanical ventilation and adjunct therapies are available; while not specifically used in drowning, their use has proven valuable in the ventilatory support of any patient with ARDS. A list of the most commonly used forms in drowning victims follows. Invasive mechanical ventilation modes for patients with ARDS have been discussed in Chapters 104 and 109. The same principles apply to patients whose cause for ARDS is drowning.

Nitric Oxide

Inhaled nitric oxide (NO) appears to act selectively on the pulmonary vascular bed and only in those areas associated with adequate ventilation, locally reversing hypoxic pulmonary vasoconstriction and increasing oxygenation. However, outcome in terms of mortality or number of days alive and off mechanical ventilation between patients treated with NO and those not treated has not changed when the effect of NO is studied in a prospective randomized fashion (63). Nevertheless, reducing the level of mechanical ventilatory support or FiO2 needed to achieve adequate oxygenation is a potential benefit that could reduce barotrauma and the side effects of treatment. There are two case reports published in which NO was used in drowning victims: one in a 16-year-old boy in which both oxygenation and pulmonary hypertension improved with no untoward cardiovascular events (66), and the second a 21-year-old man in which both NO and prone positioning were used which lead to improvement in oxygenation and extubation within 5 days (67).

Prose Positioning

Rotation of patients from supine to prone may cause rapid improvement in oxygenation that may last for up to 12 hours (68). With this maneuver, there is a relatively high risk of inadvertent extubation and removal of invasive monitors; nonetheless, oxygenation improves mainly because the nondependent dorsal portion of the lung has a higher air-to-tissue ratio (69). Obviously, the risks and benefits need to be considered before using this technique in any specific patient.

Bronchodilator Therapy

Small airway closure has been shown to occur even with aspiration of relatively small amounts of water (26). Thus, bronchodilator therapy should be considered in patients when bronchospasm is thought to be present.

Surfactant

ARDS from drowning involves both quantitative (seawater) and qualitative (freshwater) alterations in lung surfactant (34,70). Although the use of exogenous surfactant has been shown to lower mortality in neonates with respiratory distress syndrome (71) and a few successful case reports in drowned children have been reported (72–74), this effect in adults has been disappointing, and its prohibitive cost makes its use infrequent.

Prophylactic Antibiotics

Prophylactic antibiotics are not needed in most drowning victims, and the use of broad-spectrum antibiotics may enhance the emergence of resistant organisms, as well as the potential for Clostridium difficile infection. Pneumonia is often misdiagnosed due to the consolidation which appears on radiographs due to water in the alveoli (13). It has been shown only about 12% of patients rescued from a drowning episode developed pneumonia and required antibiotic treatment (75). An exception represents survival from drowning in heavily contaminated water such as stagnant ponds or public spas, where Pseudomonas species are endemic. Our initial choice in this situation is usually a fourth-generation cephalosporin with broad gram-negative coverage. In other patients, antibiotics are not recommended unless the patient develops evidence of infection, in which case cultures and sensitivities will guide the choice of antibiotics to be given.

Cardiovascular Support

By the time a drowning victim reaches the ICU, cardiac dysrhythmias are rarely a problem. If witnessed in the emergency department or the ICU, the most common cause of arrhythmias is severe hypoxia, and providing adequate ventilation and oxygenation will usually restore a normal rhythm. If not, drug therapy or, in the case of severe ventricular arrhythmias, electrical intervention is appropriate. Hypotension may require initial pharmacologic support, but it should be remembered that the hypotension seen in drowning victims is predominantly due to fluid shifts resulting in hypovolemia (27,43). This hypovolemia may be accentuated when mechanical ventilatory techniques that increase mean intrathoracic pressure are used (45).

Experimental studies have shown that, whereas mechanical ventilation and CPAP will decrease intrapulmonary shunt and increase PaO2, because of the detrimental effect on cardiac output, tissue perfusion is compromised. In one study, attempting to increase oxygen delivery by use of vasopressors and inotropes was not productive, but fluid administration to increase blood volume resulted in an increased cardiac output and oxygen delivery (45).

Precise fluid replacement is dependent on an accurate assessment of effective circulating blood volume. To this end, monitoring the patient with a pulmonary artery catheter or transesophageal echocardiography is extremely helpful.

Temperature Management

As previously mentioned, many drowning victims are hypothermic upon rescue and upon presentation to the hospital. Deciding to keep a patient hypothermic or induce hypothermia is largely based on extrapolation of studies of therapeutic hypothermia in cardiac arrest and asphyxia (49). In cardiac arrest there have been multiple studies showing improvement in neurologic outcome with therapeutic hypothermia. In two landmark, randomized controlled trials, moderate hypothermia, 32° to 34°C, was induced in patients after return of spontaneous circulation after ventricular fibrillation arrest for 12 to 24 hours and was compared to standard temperature care and showed improvement in neurologic outcome in the mild hypothermia arm (76,77). International guidelines began recommending therapeutic hypothermia in comatose patients after cardiac arrest in 2003 and this then extended to patients with in-hospital arrest and arrest from initially nonshockable rhythms. In 2013, another landmark multicenter, randomized study compared targeted temperature therapy of 33°C versus normothermia at 36°C in patients suffering out-of-hospital arrest presumed of cardiac etiology, not finding a difference in outcomes between the two arms, and both had improved neurologic outcomes compared to previous studies (78,79).
This has since raised the question if the therapeutic benefits of targeted temperature therapy are in avoiding fever rather than in hypothermia itself, but clearly establishes temperature should be controlled (79).

Therapeutic hypothermia in asphyxia has been studied almost primarily in neonates but also in children. A Cochrane review of eight randomized controlled trials and two other subsequent meta-analyses of neonates with intrapartum asphyxia and moderate-to-severe encephalopathy found hypothermia had a beneficial effect on mortality and major neurodevelopmental disability at 18 months (80–82).

Therapeutic hypothermia has only been sparsely studied in drowning itself. More than two decades ago, two small retrospective studies examined deep, prolonged hypothermia with and without barbiturate therapy and found only an increase in survival of patients in persistent vegetative state (49,56,83). Two cases of drowning associated with cardiopulmonary arrest and requirement of extracorporeal membrane oxygenation were reported in which a hypothermic state was maintained for 6 days; both survived without neurologic sequelae (84). In contrast to the previous studies of deep hypothermia, mild hypothermia and targeted temperature therapy, as have been studied in cardiac arrest, should be a point for future investigation in drowning victims.

**Central Nervous System Support**

The two most important factors influencing morbidity and mortality in victims surviving drowning are severe respiratory insufficiency and permanent neurologic impairment secondary to cerebral hypoxia (Fig. 111.1). Despite improvement in emergency and intensive pulmonary and cardiovascular care, neurologic outcome in drowning patients is directly related to the initial duration of hypoxia from the onset of submersion until effective CPR is provided. The Glasgow coma scale (GCS) score mirrors this relationship during the first few hours after submersion. As in other neurologic injury, care is often separated into neuromonitoring and neuroprotective strategies; we discuss both below.

**NEUROPROTECTION STRATEGIES**

Unfortunately, there is a lack of evidence to guide any neuroprotective strategies specifically for the drowning victim (49). Much of the research regarding drowning victims has focused more on the pulmonary and cardiovascular systems. Attempting to study different neuroprotective strategies in drowning victims is difficult, if not impossible, given the rarity and heterogeneity of the events (49). It is also unknown if there are any differences in cerebral injury from saltwater versus freshwater drowning (49). Temperature management and therapeutic hypothermia, previously discussed, have possible benefits if extrapolated to the drowned patient. Barbiturates have been studied and were specifically part of the HYPER (hypothermia, hyperventilation, steroids, dehydration, barbiturate coma, and neuromuscular blockade) therapy, introduced in the late 1970s to control intracranial pressure postdrowning; barbiturates had mixed results in outcomes (85–87). Barbiturates then were specifically studied in the pediatric population, which showed no improvement in outcome and the drugs...
have since fallen out of favor (88). Magnesium therapy, allopurinol, and erythropoietin have all been studied in neonatal asphyxia and hypoxic-ischemic encephalopathy (49). A recent review and meta-analysis of magnesium for neuroprotection in hypoxic-ischemic encephalopathy reviewed five studies, finding improvement in short-term outcomes and only a trend toward improved mortality (89). Allopurinol has been extensively studied in hypoxic-ischemic encephalopathy due to its inhibition of xanthine oxidase, which may reduce delayed cell death (90). A recent Cochrane review did not show a statistically significant difference in the risk of death or severe neurodevelopmental disability (90). A large ongoing multicenter trial, the ALLO-trial, is studying antenatal allopurinol during fetal hypoxia (91). Erythropoietin has been shown to have neuroprotective qualities by modulating antioxidant enzyme activity and the genetic expression of apoptosis; numerous trials show promise in hypoxic-ischemic encephalopathy in newborns (49).

**NEUROMONITORING**

No single neuromonitoring modality is considered the gold standard, and the use of multimodality monitoring is recommended in this population. Severity of illness can be estimated by the quality of resuscitative efforts on the scene, patient age, submersion time, temperature, initial pH, hyperglycemia, and presenting neurologic examination (92). The following discussion includes evidence from patients suffering from anoxia secondary to drowning or near-drowning or cardiac arrest; studies reviewed include the adult and pediatric population. It is important to consider the use of hypothermia after anoxic event and patient age, both of which may impact neuroprognostication (93).

The pediatric population is of considerable concern because the increased neuroplasticity of their immature neurologic system makes recovery more difficult to predict. This can be extrapolated to an extent to young adults, making prognostication difficult in this population. Nonetheless, it is important to keep in mind that no one modality is the gold standard, and multiple modalities should be utilized to provide a clearer picture of the damage, prognostication, and to guide resuscitation.

**NEUROLOGIC EXAMINATION**

The neurologic examination is an important monitor for secondary injury, and has less of an impact with prognostication in hypoxic–ischemic encephalopathy (92). However, it is important to realize that the neurologic examination can be clouded by the use of neuromuscular blockade, sedatives, and narcotics; in these circumstances, the physical examination can be unreliable. We recommend allowing more than five-drug half-lives (depending on the patient’s ability to metabolize the medications) prior to predicting neurologic outcomes via neurologic examination.

Several grading scales can be used to classify patients, the most frequently being the GCS, pupillary response, corneal reflex, and motor responses (92–94). Patients who present awake have better prognosis than those who present comatose. A GCS score of less than 5 combined with no papillary response at presentation, 24 and 48 hours is associated with poor outcome (92). The American Academy of Neurology states clinical findings that predict poor outcome in anoxia after cardiac arrest include myoclonus, status epilepticus (SE) within the first 24 hours, absence of pupillary responses on days 1 to 3, absent corneal reflexes within 1 to 3 days, and absent or extensor motor responses after 3 days (94,95). One study evaluating pediatric patients with hypoxia-ischemia found a GCS score of less than 5 after 24 hours was always associated with poor outcome (96). These results are further echoed in patients treated with hypothermia after arrest (97). It should be emphasized that the neurologic examination, especially motor response, is an unreliable predictor of neurologic outcome when used alone and should be correlated with clinical circumstance and other neuromonitoring modalities (97).

**INTRACRANIAL PRESSURE MONITORING**

Hypoxic injury leading to cell death and subsequent cerebral edema makes ICP monitoring a logical choice for monitoring and prognostication in this population. Poor outcomes have been associated with sustained ICPs above 20 mmHg and CPP below 50 mmHg (98). Regardless of the pathophysiology of elevated ICP with cerebral anoxia, the risks of invasive ICP monitoring may outweigh the benefits, and currently is not in the mainstream of monitoring modalities. Earlier studies on ICP monitoring in this population showed mixed results and unreliable prediction of outcome with CPP just below normal (99).

**SOMATOSENSORY EVOKED POTENTIALS**

The use of somatosensory evoked potentials (SSEPs) has been validated as an alternative method of confirming brain death in the United States. SSEPs can also be used for prognostication in patients with anoxic encephalopathy, with the absence of bilateral cortical responses at 24 hours after injury closely correlating with poor prognosis (95). More definitively, absent cortical responses with SSEPs at 1 week have been shown to have 100% positive predictive value for poor outcome after cardiac arrest (95,100). These results are more predictive when combined with serum levels of neuron-specific enolase greater than or equal to 33 μg/L (95). A systematic review comparing SSEPs to clinical examination, including pupil and motor responses, to predict poor and favorable outcomes in pediatric TBI showed that SSEPs provided better reliability in predicting favorable and poor outcomes. In addition, SSEPs showed good reliability with the use of NMB and sedation when compared to the clinical examination (100). Similar findings are reported in a study of anoxic encephalopathy after cardiac arrest in adults (101). Absent bilateral median nerve SSEPs along with pupillary response, corneal reflexes, and motor response after 72 hours after arrest correlated to poor outcome (101). In another study of adults after cardiac arrest, bilateral absence of SSEPs was always predictive of an unfavorable prognosis, defined as death or persistent vegetative state (96).
Electroencephalography

In patients with hypoxic encephalopathy due to cardiac arrest, EEG findings of SE or suppression correlated with nonsurvivability, and nonreactive EEG correlated highly with in-hospital mortality (93). EEG may thus have both prognostic and therapeutic utility. While evidence of SE increases mortality and morbidity, its recognition and management may improve outcome (97).

Renal Support

Albuminuria, hemoglobinuria, oliguria, and anuria, while rare, have all been described in drowning victims secondary to acute tubular necrosis from hypoxemia, rhabdomyolysis, or both. Hypothermia leads to reduced blood flow to the skin and muscle, preserving core temperature and central organ perfusion. The pathophysiology of acute rhabdomyolysis is probably secondary to tissue hypoxia from acute vessel constriction, due to the competitive need for heat conservation; skeletal myolysis and increased circulating myoglobin will result. Acute renal failure may be aggravated by acute tubular necrosis secondary to hemodynamic instability.

Acute tubular necrosis and rhabdomyolysis require early and vigorous treatment directed at correcting hypovolemia, improving oxygenation, and enhancing heme protein elimination. Volume replacement therapy aims to restore normal blood flow and enhance renal oxygen supply; the medullary ascending limb of Henle loop is most vulnerable to hypoxic injury. Invasive monitoring may be necessary to provide adequate intravascular volume. The window of opportunity for restoration of intravascular volume and volume expansion is likely within 6 hours or less of the acute event.

If rhabdomyolysis is present, enhancing the elimination of heme protein helps to limit tubular damage. Intravenous fluids should be increased to 400 mL/hr and may need to be as high as 1,000 mL/hr with a goal of 3 mL/kg/hr of urine output (102). Systemic alkalinization of the urine with sodium bicarbonate increases the solubility and, therefore, the elimination of heme protein (102,103). A urine pH between 6.5 and 8 produces a myoglobin solubility of around 80% and is a reasonable goal. Caution should be taken with administration of sodium bicarbonate as it may worsen hypocalcemia associated with rhabdomyolysis (102). However, in a patient with low urine output, massive doses of sodium bicarbonate may be associated with volume overload secondary to an acute increase in intravascular osmolarity (102,104). In these cases, when the hemodynamic goal is mild hypervolemia, the weak diuretic acetazolamide may be a valid alternative. Acetazolamide increases the excretion of bicarbonate in urine as a result of the inhibition of the enzyme carbonic anhydrase. However, diuretics, particularly in patients on significant ventilatory support, may adversely affect venous filling and cardiac output. Mannitol, an osmotic diuretic, has also been used in rhabdomyolysis due to the increased urinary flow, excretion of nephrotoxins, its ability to pull fluid from the injured muscles due to its osmotic properties, and its free-radical scavenging properties (102).

Manipulating the renal output by means of significantly altering the effective circulating blood volume in drowning victims frequently has a detrimental effect on pulmonary and cardiovascular function. Therefore, a fine-tuned balancing act is frequently required to not adversely affect one organ system while treating another.

Other Concerns

Severe metabolic acidosis from low systemic oxygen delivery and resulting anaerobic metabolism should be corrected. We recommend correction of the base deficit with bicarbonate or acetate solutions to maintain a pH no lower than 7.20. Mechanical ventilation is adjusted frequently with the help of arterial blood gas determinations to maintain a PaCO₂ between 35 and 40 mmHg. Lactic acid levels are checked frequently for a few hours after resuscitation. In fact, while base deficit and single absolute levels of lactic acidosis do not necessarily correlate with the development of multiple organ failure and survival, the rate of lactic acid clearance does (105). Because significant electrolyte abnormalities requiring specific therapy are rarely observed in the drowning victim, normal saline is used as replacement fluid. Isotonic solution also provides less chance of aggravating cerebral edema.

Prevention

An awareness of the hidden dangers of recreational activities in and around water, and close supervision of infants, children, and adolescents are the secrets to preventing a significant number of drowning incidents. Swimming pools should be enclosed by security fences to prevent small children from entering the water inadvertently or unsupervised. By identifying age-related drowning risks, communities can reduce drowning rates. Effective CPR and water safety skills should be encouraged in the community, particularly for parents with small children who own home pools. Furthermore, children who can swim should never do so alone or without adult supervision. Everyone participating in water sports should wear an approved personal flotation device. Adolescents need to be taught to swim and informed about the dangers of alcohol and other drug consumption during water sport activities. Between 13 and 19 years of age, risk-taking behavior increases significantly in boys; therefore, extra counseling is warranted. Alcohol should never be consumed, regardless of age, while swimming or engaging in water sports. Swimming with a partner is particularly important for individuals with medical conditions that may abruptly alter their level of consciousness, such as seizure disorders, cardiac disease, and several metabolic diseases. Emergency gear for rescuing and resuscitating drowning victims should be readily available at the poolside. The specific gear required may vary with the size, access, and ownership of the facility.

The community expects the government to enforce safety rules, promote health education through medical and nonmedical personnel, and punish individuals who transgress basic safety rules and regulations. Despite recent advances in CPR and more sophisticated intensive care medicine, drowning victims with poor GCS scores have a high likelihood of living in a vegetative state as a result of the initial injury. When this occurs, making life or death decisions regarding withdrawal
of life support by relatives and health professionals represents a significant stressful event. At the time of this writing, prevention is still the most fundamental way to limit neurologic disasters from drowning.

Key Points

- Because cardiac arrest following drowning is more likely due to asphyxia, immediate provision of ventilation is recommended and not chest compressions alone.
- Drowning in both freshwater and seawater produces intrapulmonary shunting, although from differing mechanisms.
- The single most important method of treatment in reversing hypoxemia is the application of CPAP.
- Prophylactic antibiotics are not needed in most cases.
- Targeted temperature management and hypothermia have been shown to improve neurologic outcomes in anoxic injury due to cardiac arrest and may be helpful in drowning victims.

References


