REVIEW

Drowning

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ABSTRACT

Approximately 500,000 deaths due to drowning are reported annually, 30,000 of which are reported in Europe. Because of the relatively low incidence of drowning victims at emergency departments, most emergency physicians do not routinely handle drowning victims. Although confusion regarding the classification and pathophysiology of drowning could be reduced by following the Utstein style consensus, the application of therapeutic modalities and, most important, the estimation of probable prognostic outcomes remain difficult for emergency physicians. This article presents an overview of the classification, pathophysiology, emergency-department treatment and prognostic outcomes of drowning accidents. *(Minerva Anestesiol 2012;78:69-77)*

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Drowning is a common cause of death worldwide, as about 500,000 deaths due to drowning are reported every year, and a gross underreporting of drowning deaths can be assumed.

According to the WHO, 30,000 persons drown every year in Europe, which translates into approximately 80 per day. Many cases of drowning could be avoided by proper preventive measures, such as parental surveillance, fencing of pools [1, 2] and proper swimming education. Socioeconomic factors seem to be associated with drowning deaths, as a higher number of children from poor families drown; however, the reasons for this association remain uncertain.[1-3]

For each death by drowning, at least three survivors are treated in emergency facilities, resulting in approximately 90,000 further visits at emergency departments in Europe.[4]

In Sweden, about 200 deaths and about 800-900 hospitalizations occur due to drowning every year.[5, 6] Drowning is approximately three times more common among men than women. Drowning is the most common cause of death among sportsmen and the third most common one in children.[7, 8] In this article, a comprehensive overview of pathophysiology and the clinical management of drowning victims is provided.

Classification and pathophysiology of drowning

In recent years, the definition of drowning has been discussed. The classical division into drowning in either salt- or freshwater and by primary (direct) or secondary drowning (by death within 24 hours) has been abandoned, because of confusion resulting from the various definitions. In 2003, the Utstein style consensus was reached, resulting in a simplification of the classification of drowning accidents. According to the consensus, a drowning accident happens with every incident in which the victim has been immersed or submerged, causing the surrounding fluid to block the airway and inhibit respiration. By this definition of drowning accidents, the victim might live or die.

Immersion is defined by the victim's body being immersed in water, but maintaining the airway above the surface of the water, while submersion occurs when the airway is underwater.

Furthermore, drowning accidents have been classified as witnessed or non-witnessed (similar to the classification of cardiorespiratory arrest), because of the prognostic value of this information.[9]

By this simplification of the classification of drowning accidents, factors of minor prognostic interest, such as the presence of saltwater or fresh water, were not considered. Prior studies concluded that drowning in fresh water resulted in osmotic intravascular hemolysis and massive hyponatremia, while drowning in saltwater resulted in osmotic pulmonary edema, hemoconcentration and hypernatremia. These theories were based on experiments performed on anesthetized animals, which were passively drowned.[10]

Today, the pathophysiology of drowning is defined differently: First, the victim's airway is under the surface of the fluid. Initially, the victim tries to hold its breath. Next, involuntary respiratory efforts evolve, starting with diaphragmatic contractions. Alternatively, fluid enters the pharynx through the nose. In any case, minor aspiration results, often causing reflex-mediated laryngospasm. Thus, respiration is not possible during this period, and hypoxia, hypercapnia and acidosis develop. When the laryngospasm ceases, the breath cannot be held voluntarily and major aspiration occurs. Often, large amounts of fluid are swallowed into the stomach during this period. As no further oxygen can be inspired, hypoxia rapidly develops. The arterial pO_2 declines by approximately 6 mmHg per minute. Next, unconsciousness develops. Finally, cerebral hypoxia results in a fatal outcome (Figure 1).

During the process of drowning, the victim may struggle or be unconscious from the beginning; in any case, hypoxia is the final result.[9, 11] If the victim does not manage to save itself, fatal drowning occurs.

Concurrent pathology in drowning

Alcohol is often involved in drowning, and further drugs are more rarely involved.[12-14] Primary heart disease, arrhythmias or cerebrovascular accidents can result in drowning and, besides alcohol, are the most common concomitant factors in drowning.[13, 15] Less common causes of drowning include unconsciousness due to epilepsy, hypoglycemia or cerebral arterial gas embolisms in divers. Blackout after hyperventilation is not an unusual factor associated with drowning.

Spinal neck injury occurs when swimmers jump into the water or are overrun by boats. Most often, the clinical picture indicates a major trauma, and the overall incidence of spinal neck injury in drowning is relatively low (1/1000).[13, 16] Thus, routine stabilization of the neck can be recommended only when the victim is unconscious or when anamnesis raises suspicions of cranial or neck trauma.

Carbon-monoxide poisoning induced by exhaust from boat-motors is a relatively rare cause of drowning (Figure 2).[17]

Blackout

Primary underwater hypoxia can develop into blackout. Submerged swimmers and apnea-divers may lose consciousness after prolonged hyperventilation (more than three breaths) and subsequently drown. Respiration is controlled primarily by the level of arterial pCO_2 and, secondarily, by the level of arterial pO_2 in healthy individuals. In cases of hyperventilation, the level of pCO_2 is reduced without a significant increase in the level of pO_2. Upon submersion, the level of pO_2 is gradually metabolized, while the level of pCO_2 increases. If the level of pO_2 decreases below a specific level, cerebral hypoxia results in blackout. If the level of pCO_2 is still below the threshold required to trigger respiration while the level of pO_2 drops under the hypoxic level, the victim falls unconscious and drowns.

A further cause of drowning may be the dive response. The function of the dive response is still under discussion. It is triggered by the stimulation of pressure-sensitive and cold-sensitive receptors located around the mouth and nose. Pressure and cold applied to this region cause reflex-mediated bradyarrhythmia, centralization of the blood flow and contraction of the spleen,

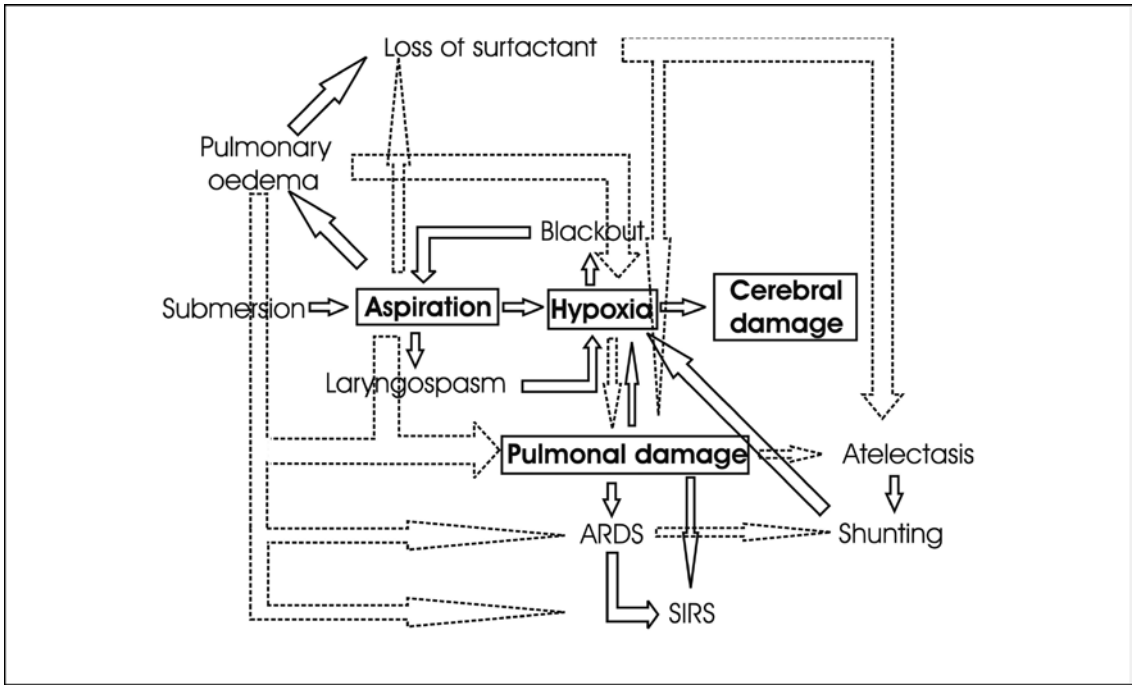

Figure 1.—Simplified scheme of pathophysiology in drowning. To the factors above, acidosis, hypercapnia, electrolyte disorders, arrhythmia, hypothermia and infection should be added. Cerebral and pulmonary damage, primarily due to hypoxia, are the limiting prognostic factors.

accompanied by a slight increase in hematocrit. Children are especially sensitive to these stimuli and might respond with direct asystole.

Hypothermia and drowning

In European waters, the victims of drowning accidents are predominantly hypothermic. Water carries approximately 25 times more warmth than air and possesses a higher warmth capacity, which means that more energy is required to increase water's temperature by one Kelvin. Because of the low temperatures observed in European waters, swimmers tend to lose large amounts of energy in relatively short time periods, depending on the temperature of the surrounding water and their bodies' insulating fat tissues, exposed surface areas, weights and levels of heat production.

Hypothermic cells exhibit a reduced metabolism, and cerebral hypothermia is assumed to protect against ischemia, which is used therapeutically for stroke patients and after cardiac arrest. The protective effect of hypothermia is higher if hypothermia develops before the onset of ischemia. On the other hand, enzymatic activity and the effectiveness of most biological functions (such as coagulation and immune defense) are impaired under conditions of hypothermia.[18] These effects may not be of primary interest in initial emergency situations, but can result in major complications during subsequent hospitalization.[19-22]

Children and elderly people tend to have a relatively large surface area in relation to their weight; therefore, they suffer hypothermia relatively quickly (Figure 3).

Prognosis

Factors influencing the prognosis of drowning victims

The results of drowning, thus, might be summarized as asphyxia, cerebral hypoxia and, often, hypothermia. Cerebral hypoxia is the lim-

Further factors in drowning accidents
Cardiogenic (heart attack, arrhythmia, stroke)
Intoxication (alcohol, others)
Hypothermia
Epilepsy
Hypoglycemia
Trauma (neck trauma in 0,5% of all cases, signs of trauma)
Attempted suicide
Attempted murder

Figure 2.—Concurrent disease and causes associated with drowning accidents. Most of the factors are associated with overweight status.

iting factor for the prognosis of the victim. As a variety of factors influence the development of cerebral hypoxia, the extension of the resulting cerebral damage is difficult to estimate during early evaluation of the victim. The answers to several questions can be of prognostic value: Was the drowning accident witnessed? This information allows an estimation of the duration of hypoxia. The victims of unwitnessed drowning accidents can be assumed to have being submerged for a longer period of time and to have suffered severe hypoxia. Which kind of fluid did the person drown in? What was the fluid's temperature? Various microbes can be expected to be present in certain fluids and might subsequently cause infections. For instance, after the tsunami of 2004, multiple cases of rare pulmonary infections have been reported in survivors of drowning accidents. Was the victim unconscious when rescued? The prognosis of the victim is improved if the victim was not unconscious or regained consciousness early. Was there cyanosis on initial presentation? This factor might be difficult to evaluate. Cyanosis indicates that the victims' oxygen reserve has been depleted and that the victim was submerged for a long period, resulting in prolonged hypoxia. However, peripheral vasoconstriction can result in peripheral cyanosis in swimmers in cold water, thus limiting the value of this information. Did the patient show any signs of life during the rescue? Consciousness, spontaneous respiration and stable circulation indicate a better outcome than unconsciousness and respiratory and circulatory arrest. Was CPR performed before the arrival of medical services? Early, effective CPR reduces the time of hypoxia and may maintain cerebral oxygenation. Thus, early CPR by bystanders can have a major influence on the prognosis.[23] Pulsoximetry cannot be trusted in treating hypothermic patients.

Establishing the prognosis of drowning victims

The prognosis of drowning victims is highly variable, ranging from full recovery to severe neurological handicap or death. The fatal outcome is related to cerebral hypoxia, acute respiratory distress syndrome (ARDS), multiple organ failure secondary to hypoxia and sepsis caused by aspiration or nosocomial infections.[9]

Several algorithms have been developed to provide an adequate estimation of the final outcome as early as the time of presentation at the emergency room. The most difficult prognosis to estimate is for the unconscious drowning victim. Even though they provide a certain prognostic reference, algorithms and models are not absolutely accurate. Although assessment of patients in these cases can commonly distinguish between a favorable and pessimistic prognosis, a certain portion of the patients judged to have a poor prognosis on the first evaluation can recover without any sequelae if treated aggressively, and these patients cannot be detected in the early phase.[11, 19, 20, 21, 24-26]

For children and adults drowning victims, different algorithms are employed

Children presenting at the emergency department who are fully conscious and have no need for advanced therapy, CPR or respiratory support have an excellent prognosis and can be discharged after a few hours of observation.[27, 28] If, on arrival of the ambulance, children present with GCS > 8, they have a nearby 100% chance of uneventful recovery, dropping to 80% with a fortunate outcome on GCS of 4-8, while a GCS of 3 is associated with an 80% rate of mortality or severe handicap. The pediatric risk of mortality score (PRISM-score)

might be employed to estimate the general prognoses of drowned children. The score is based on 14 physiological parameters and has been employed in pediatric intensive care in various versions since 1988.[29, 30]

In two separate studies, children presenting a PRISM/score below 8 could be discharged after several hours of observation, and children with a score below 16 (associated with a 16% mortality rate in intensive care) survived without any handicap. A score above 24 (associated with a risk of mortality of 42%) resulted in massive cerebral handicap or death in 100% of cases. A grey-zone score between 17 and 23 (16-42% mortality) indicated a poor outcome (PO) in 33% of cases.[29, 30]

A favorable outcome is often achieved in unconscious children being submerged for fewer than 5 minutes. Children younger than 4 years often have a better prognosis than older children. If CPR is begun after more than 7 minutes, if the patient responds to CPR after more than 10 minutes, if, upon arrival at the emergency room, cyanosis and cardiopulmonary arrest persist and if the initial blood pH is below 7.10, then the final prognosis is often poor. Hypothermia below 35 °C is commonly associated with prolonged submersion and, thus, hypoxia, but might prevent neurologic damage and can only be considered an unreliable indicator regarding the final prognosis. Common neurological predictors of the final outcome are a GCS of 3 (80% PO), Conn C (66% PO), and dilated, unreactive pupils (100% PO). In hypothermic patients, these signs must be considered with reservations, as there are reports of cases presenting with GCS 3, Conn C and unreactive pupils achieving a full recovery.

Certain authors regard gender as a risk factor in drowning, and more males than females drown; however, several studies could not indicate that the gender of a drowning victim exerts any influence on the final outcome.[5, 13, 28]

Importantly, no scoring system yet devised can predict a drowning victims' prognosis with total confidence.

In adults, a relatively simple scoring system was developed by Brazilian researchers after studying more than 2,000 drowning victims.[31]

In this system, the patients were divided into six groups. Group 1 consisted of patients without any impairment of respiration, circulation and consciousness and with excellent prognoses. The patients of group 2 exhibited rhonchi at the base of the lungs without any further findings and had excellent prognoses (mortality rate of 0.6%). The patients of group 3 exhibited signs of pulmonary edema, but no hypotension, and had good prognoses (mortality rate of 5.2%). Pulmonary edema and hypotension were associated with a mortality rate of 19.2% in group 4. Unconsciousness and respiratory arrest without cardiac arrest (group 5) was associated with a mortality rate of 44%, and cardiopulmonary arrest (group 6) was associated with a 93% mortality rate (Figure 4). Several victims of drowning accidents have been reported to recover without persistent neurological handicaps after long-term rehabilitation, even after presenting prolonged neurological symptoms during hospitalization. Thus, even cases initially considered to indicate a poor probable outcome should be treated aggressively, considering the full spectrum of therapeutic options. This notion especially applies to children, in whom, even after prolonged submersion, a positive outcome may be achieved.[23] Long-term coma and slow recovery is not unusual in these patients, and the duration of the coma cannot be taken as an absolute prognostic factor.[21, 24, 32] Because of these uncertainties, deciding when to end the primary treatment and proceed to further therapy in intensive care is difficult. No clear advice can be given regarding this matter.[26]

At the emergency room, the victim should be re-warmed to a core temperature of at least 35 °C and prove to be unresponsive to massive inotropic and volume support before one should consider aborting further therapy. The presence of acute respiratory distress syndrome (ARDS) at the emergency department might be considered an aggravating but treatable factor requiring the full spectrum of intensive care. A potassium level above 10 mmol/L might be considered to be a poor prognostic sign, but therapeutic decisions should never be made based on a laboratory value alone. Radiological detection of diffuse cerebral edema and resulting brainstem incarcera-

	Light	Moderate	Severe
Core temperature	32-35°C	28-32°C	<28°C
Circulation	Tachycardia Vasoconstriction	Bradycardia Atrial arrhythmia EKG-changes (J-wave, prolonged QT-time)	Ventriculary arrhythmia Asystole PEA
Respiration	Tachypnea Hyperventilation	Bradypnea Hyperventilation	Apnea Pulmonary oedema
Neurology	Apathy Ataxia Hyperreflexia Impaired speech Impaired cognition	Impaired consciousness Hyporeflexia Dilated pupills	Coma Unreactive pupills
Muskulary symptoms	Shivering	Musculary rigor	Rhabdomyolysis
Gastrointestinal symptoms	Ileus	Pancreatitis erosive gastritis	Pancreatitis erosive gastritis
Genitourinary symptoms	Cold diuresis Bladder atoni	-------	-------

Figure 3.—Grades of hypothermia (light, moderate, severe) and symptoms.

tion can rarely be stated at the emergency room, limiting the practical value of this information in this setting.

Clearly, patients presenting with definite signs of death, such as rigor mortis, decomposition or signs of trauma incompatible with life should not be carried to the emergency department.

Therapeutic measures in drowning accidents

After rescuing the victim from the water, circulatory support and oxygenation of the patient are the first and most important measures. The final prognosis is directly related to an early therapy and CPR, as required.[33]

In unconscious patients, this therapy is achieved by the securing of the airway and by applying the principles of CPR, similar to the steps employed in cardiac arrest. No major difference exists between the resuscitation of patients in cardiopulmonary arrest due to cardiac affection and the resuscitation of drowning victims, both regarding basic and advanced measures, such as inotropics. However, as the victims of drowning accidents often have swallowed large quantities of fluids into the stomach, the risk for vomiting and, thus, aspiration is increased. The placement of a gastric tube after intubation, therefore, might be considered, but this measure should never delay more important resuscitation measures.

The rescue operations should be performed cautiously in hypothermic patients to avoid sudden redistribution of cold blood to the central circulation, which could result in ventricular fibrillation.

Hypothermia influences the process of resuscitation, as well (Figure 3). On one hand, after the initial response to outer cold with grossly ele-

vated loss of energy and hypermetabolism, overall metabolism and oxygen demand are reduced once hypothermia has developed. Accordingly, in the hypothermic phase, tissue survival can be achieved by lower amounts of oxygen than usual, so that a residual centralized circulation and strongly reduced respiration can be sufficient to preserve vital organs for a limited amount of time while the periphery becomes hypoxic, acidotic and hyperkalemic. Unfortunately, re-warming of the patient will result in the decentralization of the circulation, as peripheral vessels re-open, which can result in sudden inflow of cold peripheral acidotic and hyperkalemic blood into the central circulation and can trigger ventricular fibrillation, known as after-drop and previously described during the Napoleonic campaigns.

Hypothermia triggers arrhythmia, thus hampering CPR. Arrythmia may result in ventricular fibrillation that is impossible to defibrillate into sinus-rhythm in gravely hypothermic patients. Thus, continuing CPR until a core temperature of 34 °C is reached is recommended if defibrillation proves unsuccessful after three shocks. Furthermore, severe hypothermia results in reduced compliance of the victims' tissues, making CPR and mechanical ventilation more difficult. In such cases, devices such as LUCAS, which perform external thoracic compressions, can prove useful, as they allow prolonged CPR. Extracorporeal circulation allows both circulatory support and active re-warming of the patient. The accepted rule for hypothermic patients, "no one is dead before he is warm and dead," also applies to hypothermic drowning victims. Re-warming should be performed cautiously at a maximal rate of 1-2 ºC/h and should never exceed the 37 ºC-limit, as hyperthermia increases post-anoxic brain damage.[22]

After successful primary therapy, a secondary deterioration occurs in about 5% of drowning victims in the following hours and days. This deterioration is attributed to pulmonary damage caused by anoxia and aspiration, which result in epithelial lesions and the loss of surfactant and cause alveolar collapse, atelectasis, pulmonary shunting and increased susceptibility to infection. The final result is of these symptoms is ARDS.[34, 35]

Therapy in these cases should start with non-invasive (CPAP, BiPAP) respiratory support. In severe cases, invasive support by intubation and artificial respiration or ECMO might be necessary. Antibiotic therapy should be empirical and based on signs of infection or of severe contamination.[32] The application of exogenous surfactant has been adopted in small studies in children and in adults in rare occasions; however, no clear evidence to recommend this highly expensive therapy in drowning victims exists.[36] The cost for a single therapy in one adult with bilateral total atelectasis might be estimated to be approximately 60,000 €, and repeated doses are generally required.

In this phase of treatment, CO_2 levels should be monitored closely to maintain normocapnia. While hypercapnia results in cerebral vasodilation and increased ICP, hypocapnia results in cerebral vasoconstriction and concurrent localized cerebral hypoxia, which aggravates hypoxic brain damage.[26]

Hyperglycemia should be closely controlled and adequately treated; however, care must be taken not to induce hypoglycemia instead, as both conditions have been shown to impair the outcome of the critically ill.

Organ ischemia causes a systemic inflammatory response (SIRS). As a result, circulation is hyperdynamic with both peripheral and central (pulmonary) capillary leakage. Thus, drowning victims are hypovolemic, because of changes in the distribution of the plasma volume. Fluid resuscitation by crystalloids may be required (Figures 5, 6).

In patients suffering prolonged comas, brain death might be stated according to actual routines after 24-72 hours. The long-term absence of corneal reflexes, papillary light reflexes, motor responses to painful stimuli, repetitive EEG, magnetic resonance imaging (MRI) and MRI-spectroscopy, brainstem and cortex-evoked potentials, such as the N20 and laboratory samples, such as protein S-100 or neuron-specific enolase are modalities employed to detect the extension of cerebral damage. However, the results of the methods are associated with the long-term outcome in varying degrees, resulting in a maximum specificity of only 82% for the most advanced investigations.[4, 26, 37, 38]

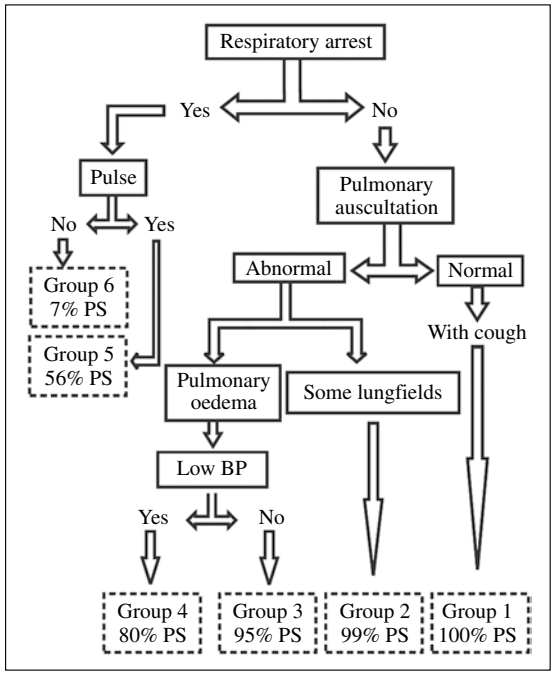

Figure 4.—Stratification into six groups of the total risk for adult drowning victims according to clinical presentations. PS: percentage of survivors.[31]

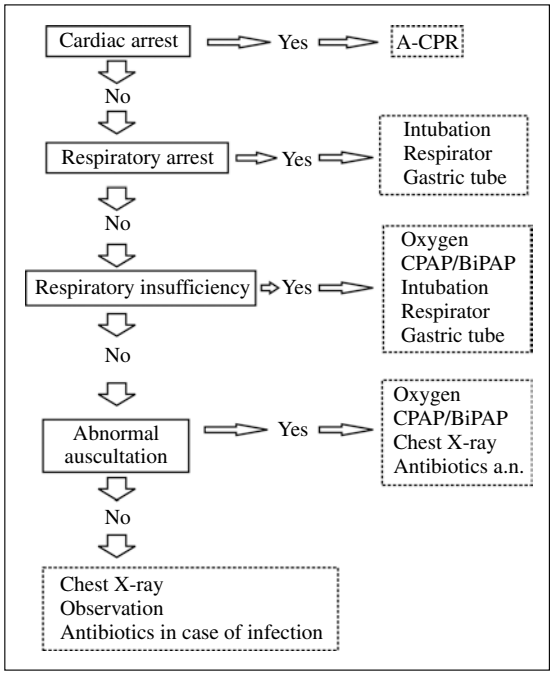

Figure 5.—Therapeutic flow scheme for drowning victims. Fluid substitution according to vital parameters is not included in the schema. The treatment of hypothermia is achieved by adequate measures.

Respiratory insufficiency	Circolatory insufficiency	Hypotermia
Oxygen	CPR	Warm blankets
Ventilation by mask	Advanced CPR	Warm oral fluids
CPAP, BiPAP	Volume substitution	Warm infusions
Laryngeal mask	Inotropic drugs	Warm air (external)
Intubation and artificial ventilation	Mechanical devices (LUCAS)	Warm air (respirator)
Heart-lung machine	Aortic balloon pump	Warm peritoneal lavage
ECMO	Heart-lung machine	Warm pleural lavage
Therapy with surfactant		Heart-lung machine

Figure 6.—Therapeutic options for respiratory and circulatory insufficiency and hypothermia in drowning victims.

Complications of intensive care are common in victims of drowning accidents, consisting mainly of ARDS, coagulopathy and sepsis. Atypical infections due to aspiration may manifest long after drowning incidents.

Conclusions

Drowning occurs if a victim's breathing is impaired because of surrounding fluid blocking the airway. Asphyxia resulting in cerebral and pulmonary damage is the most important pathological factor in drowning accidents, and cerebral and pulmonary lesions determine the final outcome. Hypothermia is often associated with drowning and might be assumed to improve the final outcome. After careful rescue, aggressive early therapy is directly related to the final prognosis. In unconscious drowning victims, the final outcome is difficult to determine at the emergency room; thus, extensive measures at intensive care might prove beneficial. Prolonged advanced therapy should be considered before making the decision to abort further treatment.

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