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Drowning

Update 2009

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Over the past four decades, we have learned considerably more about the pathophysiology and treatment of drowning. This, coupled with increased emphasis in improvement in water safety and resuscitation, has produced a threefold decrease in the number of deaths, indexed to population, from drowning in the United States yearly. This review presents the current status of our knowledge of the epidemiology, the pathophysiology of drowning and its treatment, updates the definitions of drowning and the drowning process, and makes suggestions for further improvement in water safety.

DROWNING is the third leading cause of accidental death in the United States.‡ In 1970, there were 7,860 deaths from drowning in the United States.§ Based on data reported for 1984 through 1987, there were approximately 80,000 persons who suffered a drowning episode and survived each year[1] and nearly 6,000 who died from drowning.‡§ Worldwide, approximately 150,000 deaths per year are thought to occur from drowning.‖ Given the incidence of 1 death per 13 survivors of a drowning episode in the United States, this suggests that there are approximately 2 million survivors after a drowning event, annually, worldwide.

Nearly half of all persons who drown are under 20 yr of age; 35% are accomplished swimmers.[2] Factors contributing to drowning include unattended children at water sites, alcohol or other drug abuse in up to 50% of cases involving adolescents or adults,[3] limited swimming ability or exhaustion, trauma, risky behavior in the water, rough play, deliberate prolonged submersion, exacerbation of existing medical problems (*e.g.*, seizure disorder, cardiac disease, or syncope), and attempted suicide. While most drowning incidents occur in swimming pools (50%), these also occur in lakes, rivers, streams, storm drains (20%), and bathtubs (15%).[4] Some persons drown in hot tubs and swimming pools where the suction generated in drains is of sufficient force to trap them under water[5]; toddlers can become trapped in a bucket or toilet when they lean over to play in the water contained in these vessels.[4,6]‖

For approximately the past 45 yr, there has been increased interest in studying the pathophysiology of drowning. The expansion of this knowledge and increased sophistication in the critical care of drowned victims, increased emphasis on providing advanced emergency medical services, improving pool safety standards and lifeguard training, and cardiopulmonary resuscitation (CPR) training of the lay population have been instrumental in progressively reducing the death rate from drowning in the United States from 1970 to 2000. The death rate in 1970 was 3.87 deaths per 100,000 population, in 1980 it was 2.67, in 1990 it was 1.6, and in 2000 it was 1.24 deaths per 100,000 population (table 1). The latest data available for drowning deaths is 3,582 for the year 2005. Because population census is only published every 10 years, we cannot calculate the ratio of deaths per 100,000 population for this time period. However, if we assume the same annual rate of growth occurred in the United States population from 2000 to 2005 as occurred from 1990 to 2000, then the death rate from drowning dropped further in 2005 to 1.19 per 100,000 population.

One of us (Dr. Modell) has had the opportunity to review over 500 cases of death from drowning that resulted in litigation. Many of these deaths resulted from omissions of basic safety precautions such as absent or inadequate pool fencing, unattended young children at water sites, faulty pool design resulting in victims becoming trapped below the surface of the water, poor

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‡ Accident Facts. Chicago: National Safety Council, 1987.

§ Centers for Disease Control and Prevention: Fast stats: Deaths/mortality. Available at http://www.cdc.gov/nchs/fastats/deaths.htm. Accessed September 12, 2008.

‖ National Center for Health Statistics: Vital Statistics of the United States, 1990. Vol II: Mortality, Part A. Available at: http://www.cdc.gov/nchs/data/vsus/mort90_2a.pdf. Accessed September 12, 2008.

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Table 1. Deaths per 100,000 Population in the United States Every 10 Years from 1970 Through 2000

Year	Drowning Deaths*	US Population†	Deaths per 100,000 Population
1970	7,860	203,302,031	3.87
1980	6,043	226,545,805	2.67
1990	3,979	248,709,873	1.60
2000	3,482	281,421,906	1.24
2005	3,582	Not available	

* Centers for Disease Control and Prevention: Fast stats: Deaths/mortality. Available at http://www.cdc.gov/nchs/fastats/deaths.htm. Accessed September 12, 2008. † http://www.census.gov. Accessed September 12, 2008.

pool maintenance resulting in murky or cloudy water that obscured sight of submerged bodies, lifeguards being distracted by socializing with others and doing other chores such as manning admission booths and doing housekeeping chores while on lifeguard duty, and poorly trained lifeguards who did not recognize a person in trouble in the water or had not been properly trained in rescue and resuscitation techniques. Clearly, these are all correctable issues that would prevent avoidable drowning deaths with little additional effort. We anticipate that if pool and water safety standards are more strictly enforced, and as lifeguards continue to become better trained and adhere to important basic principles of surveillance, rescue, and resuscitation, the death rate will continue to decline.[7]

Finally, variability of existing definitions may make it difficult to analyze and interpret published studies, therefore, new definitions of "drowning" and "the drowning process" were proposed by a task force at the 2002 World Congress on Drowning.[8,9]

Definitions

There has been much confusion regarding the terminology used to describe persons who have drowned. *Dorland's Medical Dictionary* offers the following definition of drowning: "suffocation and death resulting from filling of the lungs with water or other substance or fluid so that gas exchange becomes impossible."[10] It is known, however, that victims of drowning frequently aspirate only relatively small quantities of water, and the lungs are seldom "filled with water."[2] Furthermore, drowning implies death, yet many victims are resuscitated and eventually recover. Thus, subdefinitions were proposed in 1971 to delineate drowning and near-drowning events with greater accuracy. To *drown without aspiration* is to die secondary to respiratory obstruction and asphyxia while submerged in water. To *drown with aspiration* is to die secondary to asphyxia resultant from aspiration of water while submerged. To *near-drown without aspiration* is to survive, at least initially, after asphyxia resultant from submersion in water. To *near-drown with aspiration* is to survive, at least initially, after submersion in and aspiration of water or another liquid media.[2]

At the World Congress on Drowning in Amsterdam, The Netherlands, in 2002, many international experts suggested that the above terminology was confusing. This is particularly true when someone is retrieved from the water in a state of asystole and successfully revived with CPR. According to the above definitions, the victim would be classified as "drowned" when retrieved, but after the CPR, the victim would be reclassified as "near-drowned." If the victim then went on to die, did they die secondary to complications from "drowning" or "near-drowning?" This led to the development of a new definition of "drowning" and the "drowning process" that was published in *Circulation* in 2003 as follows.[8]

Drowning

Drowning is a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium. Implicit in this definition is that a liquid/air interface is present at the entrance of the victim's airway, preventing the victim from breathing air. The victim may live or die after this process, but whatever the outcome, he or she has been involved in a drowning incident.

The Drowning Process

The drowning process is a continuum that begins when the victim's airway lies below the surface of the liquid, usually water, at which time the victim voluntarily holds his or her breath. Breath holding is usually followed by an involuntary period of laryngospasm secondary to the presence of liquid in the oropharynx or larynx.[11] During this period of breath holding and laryngospasm, the victim is unable to breathe gas. This results in oxygen being depleted and carbon dioxide not being eliminated. The victim then becomes hypercarbic, hypoxemic, and acidotic.[12] During this time, the victim will frequently swallow large quantities of water.[13] The victim's respiratory movements may become very active, but there is no exchange of air because of the obstruction at the level of the larynx. As the victim's arterial oxygen tension drops further, laryngospasm abates, and the victim actively breathes liquid, with the amount inhaled varying considerably from victim to victim. Changes occur in the lungs, body fluids, blood-gas tensions, acid-base balance, and electrolyte concentrations, which are dependent on the composition and volume of the liquid aspirated and duration of submersion."[12–15] A slightly shorter modification of "drowning" and the "drowning process" was also published by these same authors in the Handbook on Drowning in 2006.[9]

Surfactant washout, pulmonary hypertension, and shunting also contribute to development of hypoxemia.[16,17] Additional physiologic derangements, such as the cold shock response, may occur in victims immersed in cold water; water that is 10°C or colder has pronounced cardiovascular

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effects, including increased blood pressure and ectopic tachydysrhythmias. The response may also trigger a gasp reflex followed by hyperventilation, which can occur while the victim is underwater.[18]

A victim may be rescued at any time during the drowning process and may not require any intervention or may receive appropriate resuscitative measures, in which case the drowning process is interrupted. If the victim is not ventilated soon enough, or does not start to breathe spontaneously, circulatory arrest will ensue and, in the absence of effective resuscitative efforts, multiple organ dysfunction and death will result, primarily because of tissue hypoxia. The development of posthypoxic encephalopathy is the most common cause of death in hospitalized victims who have suffered from the drowning process.[19,20]

No two cases of drowning are identical. The type, temperature, and quantity of wate
and the victim's state of health bef
important.[21] Very cold water may
thermia, which decreases the vic
oxygen, thereby prolonging the pe
be submerged and still completely
other hand, significant hypothern
severe delays in myocardial cond
and cardiac arrest. In addition, immersion in cold (0° to 15°C) water increases minute ventilation and decreases maximal duration of breath holding. This may decrease the effectiveness of the "diving reflex" and increase the likelihood of drowning.[24–26]

Here is a nice review of Breath-holding

Pathophysiology

Pulmonary Changes and Acid-Base Balance

The pathophysiology of drowning has been studied extensively.[2,12,14,15,27,28] Alterations noted are primarily related to abnormal gas exchange induced by pulmonary injury causing severe hypoxemia and resultant cerebral hypoxia. Cerebral edema may follow from these processes.[29–31] Interestingly, at least in endothermic animals, hypoxia results in both a lowered set point of the thermo-neutral zone and vasodilation.[32] The result is hypothermia without shivering and, thus, a decrease in oxygen consumption of about 11% per degree Celsius. The sudden onset of hypothermia during a submersion episode prolongs the time one can be submerged and still survive after rescue and resuscitation without hypoxic brain damage.[33,34] One of us (Dr. Modell) has published a case report of a child who was underwater for 20 min during the coldest day of the year in northern Florida and survived after receiving CPR and postresuscitation intensive care.[33] His cerebral function returned to normal shortly after the event, and he is normal, by all accounts, 6 years later. Other such reports are available in the literature, including a child in Norway who was believed to be submerged for 22 min.[34]

Drowning without Aspiration. It had been estimated that approximately 10% of human drowning victims die without aspirating liquid,[35] *i.e.*, they die from hypoxic cardiac arrest during laryngospasm or breath-holding. Recently, this conclusion, based on Cot's studies[36] in the early 1900s and reported in the French literature, has been challenged.[37,38] Modell *et al.* reported a different interpretation of this work in 1999 and questioned whether drowning without aspiration actually occurs.[37] Lunetta *et al.* subsequently reviewed the results of autopsies of 578 persons who were presumed to have drowned; evidence of water was found in the lungs of 98.6% of victims studied.[38] They concluded that to be classified as having "drowned," the victim must have aspirated water. If the heart is stopped before the patient's airway becomes submerged in the water, *i.e.*, the victim is dead, water does not passively seep into the lungs. Thus, aspiration of water requires active ventilation while submerged. Therefore, persons who are found deceased in the water and do not demonstrate any evidence of water aspiration at autopsy should be considered as having probably died of some other cause.[38] An alternative, of course, could be a homicide victim whose body was then disposed of in the water.

Breath-Holding. Craig[39,40] studied the breath-holding breaking point (the point at which a breath would be taken involuntarily) in human volunteers during simulated underwater swimming. He found that at rest, the breath-holding breaking point was 87 s; at that time, the partial pressures of carbon dioxide and oxygen in alveolar air (P_{AO_2} and P_{AO_2}) were 51 and 73 mmHg, respectively. After hyperventilation, breath-holding could be maintained for a full 146 s; at this time, the P_{ACO_2} increased to only 46 mmHg, whereas P_{AO_2} decreased to 58 mmHg. When exercise followed hyperventilation, the breath-holding break point was only 85 s. In this instance, while the P_{ACO_2} was only 49 mmHg, P_{AO_2} had dropped to 43 mmHg. Craig concluded that exercise, such as swimming, increased metabolically produced carbon dioxide, and the arterial carbon dioxide tension (P_{aCO_2}) was relatively low in his volunteers because stores had been depleted during hyperventilation; thus, the urge to breathe was delayed. With exercise after hyperventilation, the arterial oxygen tension (P_{aO_2}) decreased to levels incompatible with the maintenance of consciousness before the time the level of P_{aCO_2} had become unbearable. He surmised that loss of consciousness during underwater swimming was the result of cerebral hypoxia rather than hypercarbia. This work was sentinel in our understanding of the pathophysiology of drowning during underwater swimming and breath holding (or shallow water blackout).[41] In a canine model of asphyxia, Kristoffersen *et al.*[42] found that hypercarbia without hypoxia was not fatal but that the studied animals inevitably died when the P_{aO_2} decreased to between 10 to 15 mmHg.

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From data such as these, we know that hypoxia is the single most important abnormality in death resultant from submersion in water. While it is possible that the added effects of acidosis and hypercarbia may contribute secondarily to the terminal event, the primary phenomenon in these cases is hypoxemia. Reinstitution of adequate ventilation and oxygenation before the occurrence of circulatory arrest and irreversible nervous system damage results in complete and dramatic restoration of function. If, however, spontaneous ventilation begins while the patient is still submerged in water, aspiration occurs, resulting in a more complicated and persistent pathophysiology and a drowning incident, which requires additional treatment.

Drowning Effects on Blood-Gas Exchange. While multiple models have been devised to study the phenomena of drowning, all authors agree that hypoxia occurs immediately upon aspiration of the fluid. Profound alterations in arterial oxygenation may occur when as little as 1 to 2.2 ml/kg of water are aspirated into the lungs.[14,15,17,43] In persons rescued within 1.5 to 2 min of the onset of submersion in whom aspiration of water has not yet occurred, reinstitution of ventilation and circulation results in the immediate reversal of hypoxemia. However, when aspiration of water occurs, the hypoxemia is persistent. Modell *et al.*[12] showed that after instillation of 22 ml/kg of fresh water or normal saline solution into the tracheas of anesthetized dogs, significant arterial hypoxemia persisted even though the animals were breathing spontaneously and hyperventilating when the 60-min postaspiration blood samples were drawn. Hypoxemia also resulted when volumes as small as 2.2 ml/kg were instilled intratracheally and treatment was limited to restoring spontaneous ventilation and circulation.[13] In another study, when 11 ml/kg of fresh water or seawater was instilled into the trachea, and spontaneous ventilation was reestablished, Pa_{O_2} was reduced for at least 72 h after aspiration.[44]

A study of 91 consecutive drowning patients[13] analyzed arterial blood for Pa_{O_2}, Pa_{CO_2}, and pHa at various times after an accidental immersive event in either sea water, fresh water, or brackish water. In many of these cases, profound arterial hypoxemia was noted, with the Pa_{O_2} to inspired oxygen fraction (F_{IO_2}) ratio (Pa_{O_2}/F_{IO_2}) ranging from 30 to 585. Only one patient with a Pa_{O_2}/F_{IO_2} greater than 150 mmHg died; this individual was judged neurologically unsalvageable, and therapy was withdrawn. Two patients had Pa_{O_2} above 80 mmHg while breathing room air on admission to the ED after rescue. Both of these individuals were thought to be victims of drowning with little aspiration or may have been retrieved before aspiration occurred.[13] Although the Pa_{O_2} returns to normal within 48 h in some patients, others show persistent hypoxia for days and even weeks after an episode of drowning.[13,45,46]

Mechanism of the Pulmonary Effects Seen with Drowning. As early as 1933, Karpovich[47] suggested that water in the alveoli and conducting airways impaired ventilation by mechanical blockage during subsequent resuscitation. Since this initial work, other investigators demonstrated that aspiration of seawater leads to an increase in the volume of fluid within the air spaces of the lungs.[15,48] Halmagyi[17] showed that the weight of rat lungs increased threefold above the weight added by instilled seawater. Other studies[15,48] showed that attempts to drain the lungs of dogs by gravity or mechanical suction after aspiration of seawater results in more fluid being harvested than was initially instilled into the trachea. Indeed, in a canine model of massive seawater aspiration, where the subjects aspirated from 40 to 91 ml/kg of water, Redding *et al.*[48] noted that volumes drained from the lungs in excess of 14 to 33 ml/kg was associated with survival only when replacement of intravascular volume was provided. The increase in lung fluid is due to the hypertonicity of seawater, which pulls fluid from the circulation into the lung, resulting in hypovolemia.[15]

Conversely, after freshwater aspiration, the lungs of rats did not increase in weight.[17] When a sublethal amount of fresh water was aspirated by anesthetized dogs,[14,43,49] it was absorbed so rapidly that significant amounts of free water could not be recovered from the airway by gravity drainage after only 3 min.

Because seawater is hypertonic, it is relatively easy to understand why pulmonary edema occurs in patients who have suffered a drowning event in seawater. Pulmonary surfactant extracted from the lungs of animals that died after total immersion in fresh water exhibit abnormal surface-tension properties, thus promoting alveolar instability and collapse.[16] Because alveoli deficient in surfactant are unstable and become permeable to fluid, this is at least one factor contributing to the pulmonary edema occurring after freshwater aspiration. Another factor that may be involved is the transient hypervolemia that occurs with freshwater aspiration.[14,29] In contrast, during total immersion in isotonic saline or in seawater, although some surfactant may be washed from the lungs, significant quantities of normal surfactant material remain, so that surfactant extracted at autopsy displays surface-tension properties compatible with normal function. The pulmonary edema that occurs after seawater aspiration, on the other hand, is probably due primarily to an osmotic gradient across the alveolar capillary membrane, resulting in fluid-filled but perfused alveoli.[16]

Whether the drowning event occurs in fresh water or sea water, the end result is pulmonary edema, a decrease in pulmonary compliance, and an increase in the ventilation/perfusion mismatch. Immediately after aspiration of either liquid, a large alveolar-arterial oxygen gradient is seen, whether the subject is breathing room air or

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100% oxygen. This suggests that the hypoxia seen in drowning victims is due to ventilation/perfusion mismatch, which spans the spectrum from absolute intrapulmonary shunt to a simple imbalance in the ventilation/perfusion ratio. In a detailed and, for its time, rather elaborate investigation, Colebatch and Halmagyi[43] studied the effects of fluid aspiration on pulmonary mechanics in an ovine model. Animals were divided into groups of between three and seven, and either fresh water (1 or 3 ml/kg) or sea water (1 or 2.5 ml/kg) was instilled intratracheally. Compliance of the lung was noted to decrease by as much as 66% within 5 min of liquid instillation; elastic work of breathing increased five- to nine-fold, and airways resistance increased two- to eight-fold. The administration of atropine (intravenous) or isoproterenol (intravenous or inhaled) lessened the degree of compliance alteration noted.[50]

During the recovery phase, after aspiration of either fresh water or sea water, significant hypoxia may be seen when breathing room air, even after the alveolar-arterial oxygen gradient while breathing 100% oxygen has returned to normal.[44,45] This finding suggests that areas of nonuniform ventilation/perfusion or diffusion alteration cause hypoxia even after absolute intrapulmonary shunt is no longer clinically significant. A number of changes that occur after aspiration of fluid may also contribute to this delayed defect. These include organization of a proteinaceous exudate, injury to the alveolar-capillary membrane, or secondary infection of the lung.

A variety of microscopic findings have been reported after fluid aspiration. After instillation of small quantities of fresh water (0.1 ml/100 mg body weight), rat lungs did not show changes when studied by electron microscopy. After aspiration of the same quantity of seawater, however, increased lung weight and intraalveolar hemorrhage were observed.[17] When rat lungs were perfused with fresh water *via* the trachea, findings included a widening of the alveolar septa, collapse of capillaries, decrease in the number of red blood cells, enlargement of the endothelial and septal nuclei, swelling of the mitochondria, and obliteration of cell outlines. These changes can all be attributed, presumably, to the rapid absorption of large quantities of fresh water from the alveoli. When rat lungs were perfused intratracheally with sea water, the changes noted were less marked, and the septal and endothelial nuclei were small and dark. Red blood cells were seen in the capillaries and, although there was irregular folding of cell walls, the overall architecture of the lung was preserved.[51]

In humans and animals, another frequent finding on autopsy after death by submersion is hyperexpansion of the lungs, resembling acute emphysema.[30] Miloslavich[52] attributed these changes to rupture of alveoli due to wide fluctuations in airway pressure during violent ventilatory efforts against a closed glottis or resulting from airway obstruction from water during submersion; the precise mechanism remains unclear. If the patient survives for at least 12 h after drowning, only to die later, the lungs will frequently show evidence of pneumonia, abscesses, mechanical injury, and deposition of alveolar hyaline material through the third day after the drowning event.[23] These findings are not surprising in light of Fuller's report that, in 70% of humans autopsied after drowning, evidence of having aspirated material other than water–vomitus, mud, sand, or algae–was found. Despite the above findings, Butt *et al.*[46] were unable to demonstrate any consistent pattern of abnormality in pulmonary function or arterial oxygenation of patients who were studied after recovery from a drowning episode. It appears that, whatever the acute changes, clinical pulmonary abnormalities are usually completely reversible after recovery from a drowning episode.

Intrapulmonary shunting and Pa_{O_2} can be improved after drowning by the appropriate application of positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) to the airway.[53–56] Early studies on the utilization of newer modalities of therapy such as nitric oxide[57] appear to be promising in the treatment of some other causes of severe pulmonary edema–at least as far as improving the physiologic parameters–but randomized, prospective clinical studies have not yet been reported in the treatment of patients who have drown.

Changes in Blood Volume and Serum Electrolytes

After the aspiration of hypotonic or hypertonic fluid, changes other than those affecting the pulmonary parenchyma were historically thought to be of considerable importance.[27] To produce a significant change in blood volume requires the aspiration of a large volume of fluid. If more than 11 ml/kg of hypotonic fluid is aspirated, the blood volume will increase in direct proportion to the amount aspirated.[14,53] If the patient is successfully resuscitated, this absorbed fluid is rapidly redistributed and hypovolemia may occur within 1 h.[35] When a significant quantity of hypertonic sea water is aspirated, significant hypovolemia may rapidly ensue.[15] When the aspiration of significant amounts of either hypotonic or hypertonic fluid are suspected, effective circulating blood volume must be followed by measuring the patient's central venous pressure, pulse waveform or stroke volume variability, pulmonary artery occlusion pressure, right ventricular end diastolic volume, or transesophageal echocardiography to assist in the determination of necessary treatment. However, most drowning victims do not aspirate enough fluid to cause life-threatening changes in blood volume.

Similarly, serum electrolyte concentrations can change after drowning, and the degree to which they change is dependent on the amount and type of fluid aspirated. Experimental work in a canine model has shown that significant persistent changes in serum electrolyte concentrations did not occur with aspiration of 22 ml/kg or

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less of either fresh water[14] or sea water.[15] Aspiration of volumes of water in excess of this amount will result in extracellular electrolyte concentrations that reflect the resultant hypervolemia and hypovolemia that occur with fresh water and sea water, respectively. However, aspiration of this large amount of water is highly unlikely in humans who survive the drowning process, being found in only 15% of humans who die in the water from drowning.[58] This explains why life-threatening changes in serum electrolyte concentrations have seldom been reported in freshwater and seawater drowning victims. Thus, each patient's initial intravenous fluid therapy should be 0.9% saline solution, and serum electrolyte concentrations should be evaluated before any specific corrective electrolyte therapy is started; hypotonic solutions should be avoided. There is rarely any need for emergent treatment secondary to electrolyte abnormalities unless very large volumes of water are aspirated or the drowning event occurs in an extremely concentrated liquid medium, such as the Dead Sea (Na^+, K^+, Cl^-, Ca^{2+}, and Mg^{2+} are 3 to 36 times as concentrated in this body of water as in the Mediterranean Sea[59]).

Effects on Hemoglobin and Hematocrit

If large volumes of fresh water are aspirated in the presence of hypoxemia, hemolysis of red blood cells can occur, causing plasma hemoglobin and serum potassium levels to increase. The hemolysis is secondary not only to hypotonicity, but it also requires the presence of profound hypoxemia.[60] When animals received an intravenous infusion of distilled water (44 ml/kg, a sufficient volume to cause severe extracellular electrolyte changes), they showed an elevated plasma hemoglobin only when the water infusion was associated with a concomitant hypoxic event (due to an occluded endotracheal tube). When the same volume of water was instilled intratracheally, profound hypoxia resulted and hemolysis occurred.

In a canine model of drowning, chlorinated or unchlorinated distilled water or saline at a volume of 22 ml/kg was instilled into the animals' tracheas.[12] Gross hemolysis was observed in 9 of 10 animals having aspirated distilled water, but it was observed in none of those having aspirated saline. Significant changes in hemoglobin and hematocrit values are rarely found in human drowning victims,[13] lending further support to the theory that, although humans aspirate water during the drowning process, those who survive drowning, as a rule, do not aspirate large amounts of fluid. When a sufficiently large volume of water is aspirated and hemolysis does occur, serious bleeding disorders such as disseminated intravascular coagulation may be seen.[61] Only 1 of 91 consecutive drowning patients reported by Modell *et al.*[13] demonstrated a plasma hemoglobin level of at least 500 mg/100 ml; this individual later died of massive lung consolidation.

Effects on Cardiovascular and Renal Systems

A wide range of electrocardiographic changes have been observed in experimental studies of both freshwater and seawater drowning.[62] Rarely, however, is a specific therapy required, because these changes usually abate when adequate oxygenation has been reestablished. Death from ventricular fibrillation after freshwater aspiration is rare, but it can occur if very large amounts of fluid are aspirated.[27,53] In one canine study in which 22 ml/kg saline or distilled water was instilled intratracheally, bigeminy occurred in 9 of 15 dogs, and T-wave elevation was noted in 6 of 15.[12] Karch[63] reported finding significant cardiac pathologic changes 29 min after either a freshwater or saltwater (6 ml/kg intratracheal instillation) drowning episode. Light microscopy showed focal areas of disruption of the normal pattern of striation and, on occasion, of the intercalated disks and hypereosinophilia of myocytes. On electron microscopy, the damaged myocytes showed hypercontraction with swelling and an increased lucency of the mitochondria; also seen in the myocyte nuclei was chromatin clumping and crenation. These changes were not noted in the one control animal. Although the clinical relevance of these findings is uncertain, they are of interest to the further study of cardiac abnormalities after drowning.

The sudden onset of a lethal cardiac arrhythmia has been postulated as a potential event to precipitate submersion and drowning. Also, it has been proposed that a long QT syndrome might be responsible for some drowning episodes.[64,65] However, this can rarely be proven because persons with a documented long QT syndrome may have a normal electrocardiogram at other times.[66] Sometimes an abnormality may be found with the performance of a molecular autopsy, but these are rarely done in a forensic autopsy, which is what is usually performed in a suspected drowning victim. Further, Lunetta *et al.* looked for genetic mutations in 63 drowning victims and failed to document a single case of long QT syndrome founder mutations in KCNQ1 (KVLQT1) and KCNH2 (HERG) genes.[38,67] Although long QT syndrome must be considered when someone suddenly and unexpectedly dies in the water,[68] its occurrence is likely rare and difficult to confirm in the absence of a positive family history, a positive electrocardiogram, or documentation of founder gene mutation at autopsy.

Renal function is usually unimpaired during a drowning episode; however albuminuria,[31] cylindruria,[30] hemoglobinuria,[31,35,69–72] oliguria,[35] acute tubular necrosis,[31] and anuria[35] have been noted on occasion. Several etiologies for renal dysfunction have been implicated, including myoglobinuria due to muscle trauma, lactic acidosis, hypoxemia or hypoperfusion, and hemolysis with subsequent hemoglobinemia.[2] Although these are not common problems, all should be considered possi-

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ble threats to renal function and treated accordingly when indicated.

Neurologic Effects

Most drowning victims suffer a period of unconsciousness secondary to cerebral hypoxia.[53] Frequent neurologic assessment using the Glasgow Coma Scale score should be performed. Both Conn *et al.*[19] and Modell *et al.*[73] found that patients who are awake and oriented upon arrival to the emergency department (ED) survive without neurologic sequelae if treatment of their pulmonary problem is successful. Between 90%[19] and 100%[73] of patients who arrived in the ED with a blunted mental status (that is, stuporous but capable of being aroused, with purposeful movements to pain) survived without neurologic residua. Patients who presented to the ED in a coma, however, had much worse outcomes; 44%[19] to 55%[73] recovered completely, but 10%[73] to 23%[19] had severe persistent neurologic sequelae, with the highest incidence occurring in pediatric drowning cases. Approximately 34%[19,73] of comatose patients died after presentation. In children who were comatose on arrival to the ED, survival with normal brain function approximated 44%; 39% died, and approximately 17% survived with incapacitating brain damage.[19,73] The implication of these data are that the healthy hearts of children, as opposed to those of adults who may have cardiac pathology secondary to the aging process, will respond to CPR even after a significant period of hypoxemia results in persistent brain damage.

Attempts to "resuscitate" the brain in pediatric drowning cases have been largely unsuccessful. Three studies[74–76] involving 75 severe pediatric drowning incidents found that, despite aggressive cerebral resuscitation attempts, which included fluid restriction, intracranial pressure (ICP) monitoring, intravenous mannitol for elevated ICP, hyperventilation, neuromuscular blockade, barbiturates and, in some of the children, steroids and induced hypothermia, only 12 (16%) survived neurologically intact; the remainder either died, remained in a persistent vegetative state or, in one case, survived with mild to moderate mental retardation.

Conn *et al.*[19] attempted to use hypothermia, barbiturate coma, neuromuscular blockers, hyperventilation, and dehydration to enhance neurologic outcome; this treatment regimen was termed "HYPER therapy." Although they initially thought that there was significant improvement in the number of normal outcomes in some subgroups of drowned patients, a comparable series presented by Modell *et al.*[73] found similar results without using HYPER therapy. In a follow-up study to the work by Conn *et al.*, Bohn *et al.*[77] from the same institution were unable to substantiate the former's findings.

Other groups also have suggested that the use of multimodality therapy for brain protection is of little use. Allman *et al.*[78] studied 66 children between April 1979 and September 1984. Each patient required full CPR and had an initial Glasgow Coma Scale score of 3 in the ED. Each patient's Glasgow Coma Scale score was reclassified according to the results of a repeat neurologic examination upon arrival to the pediatric intensive care unit. The results showed 16 patients (24%) with apparently intact survival, 17 patients (26%) with vegetative survival, and 33 who died (50%). None of the patients who arrived in the intensive care unit with a Glasgow Coma Scale score of 3 survived neurologically intact. Fifty-five patients were also treated with ICP monitoring and aggressive therapy directed at control of ICP. Despite adequate control of ICP and maintenance of cerebral perfusion pressure, only eight of the monitored patients (14%) survived intact, 12 (21%) survived in a vegetative state, and 35 patients died. The authors suggested that, although aggressive ED resuscitation of pediatric drowning victims was justified, cerebral resuscitation measures needed to be subjected to critical prospective evaluation before their value can be substantiated.

Although there are no data evaluating the effects of hypertonic saline resuscitation in drowning victims, animal studies have suggested that these solutions help prevent elevation of ICP postinjury (global ischemia and focal cryogenic injury). Some,[79,80] but not all,[81,82] studies attempting to evaluate the differential effect of hypertonic *versus* isotonic/hypotonic fluid to resuscitate animals from hemorrhage[83,84] or the effect of hemodilution[80] without brain injury have noted that ICP was lower in the experimental subjects (rabbits, dogs, and pigs) in which hypertonic solutions were used. In one study[83] in which animals suffered a focal cryogenic brain injury, cerebral blood flow was higher and ICP lower in animals infused with hypertonic sodium lactate (500 mOsm/L) as compared with lactated Ringer solution (270 mOsm/L). These studies have not been performed in a model of drowning.

Two animal studies evaluating outcome in complete[84] or incomplete[85] cerebral ischemia suggest that fluid replacement with solutions containing glucose results in a worse neurologic outcome than does replacement with a crystalloid. Ashwal *et al.*[86] found that elevated blood glucose levels and/or reduced cerebral blood flow measured by the stable xenon technique correlated with a worsened outcome – the former by 68% and the latter by 50% – the precise mechanism for this has not been elucidated. Others note that hyperglycemia is detected on admission in about 33% of stroke patients,[87] and that persistent hyperglycemia (blood glucose level greater than 200 mg/dL) in the first 24 h after stroke independently predicted ischemic stroke infarct volume expansion and poor neurologic outcome.[88] In rat models of global ischemic injury, mitogen-activated protein kinases[89]– the family of which has been proposed as a major mediator producing cellular damage in diabetes and ischemia–and superoxide[90] are noted to be in-

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creased in the presence of hyperglycemia. Although these data may be mechanistically incomplete, the clinical data are compelling enough that we aggressively control glucose–keeping it between 100 and 140 mg/dL–with care taken to avoid hypoglycemia and its attendant morbidity.

In a retrospective study of 93 patients who suffered drowning events, Nichter and Everett[91] showed that none of the patients who required cardiotonic drugs to obtain a perfusing cardiac rhythm survived with intact neurologic status. However, all patients who had reactive pupils on arrival to the ED survived neurologically intact. In another retrospective study of 55 patients, Biggart and Bohn[22] showed that, all other things being equal, prolonged in-hospital resuscitation and aggressive treatment of nonhypothermic drowning victims resulted in an increased number of survivors in a vegetative state. When accidental hypothermia occurred at the time of the drowning episode, a better outcome usually resulted.[33] Another study suggests that those children who show significant neurologic improvement within hours after successful CPR usually recover; those who do not are more likely to have a poor outcome.[23]

Despite these rather dismal data, in a study of 121 drowned children, of whom 51 were in the C_3 stage of coma (no response to pain, fixed/dilated pupils, absence of spontaneous respiration, hypotension, and poor perfusion as described by Conn *et al.* and Modell *et al.*[19,73]), Nussbaum[92] found that with aggressive resuscitation as noted above, 19 of 51 (37%) had complete recovery, 14 of 51 (27%) had significant brain damage, and 18 of 51 (35%) died. The correlates of outcome in the C3 group were submersion time, mean ICP, and mean cerebral perfusion pressure. Although the report of Nussbaum[92] is encouraging, the fact that aggressive therapy is so often unsuccessful suggests that the cerebral insult is related not to ICP *per se* but rather to the duration of the ischemic injury. The mechanisms for this type of injury have been recently reviewed.[93]

A panel of experts was convened to discuss the subject of brain resuscitation in the drowning victim in conjunction with the World Congress on Drowning in 2002. Although they noted that there have been no specific controlled studies performed in drowning victims that would conclusively dictate a specific approach to therapy, they did make the following consensus recommendation. "The highest priority is restoration of spontaneous circulation, subsequent to this continuous monitoring of core and/or brain (tympanic) temperatures is mandatory in the ED and intensive care unit and to the extent possible in the prehospital setting. Drowning victims with restoration of adequate spontaneous circulation who remain comatose should not be actively warmed to temperature values above 32°–34°C. If core temperature exceeds 34°C, hypothermia (32°–34°C) should be achieved as soon as possible and sustained for 12 to 24 h. Hyperthermia should be prevented at all times in the acute recovery period. There is insufficient evidence to support the use of any neuroresuscitative pharmacologic therapy. Seizures should be appropriately treated. Blood glucose concentrations should be frequently monitored and normal glycemic values maintained. Although there is insufficient evidence to support a specific target Pa_{O_2} or oxygen saturation during and after resuscitation, hypoxemia should be avoided. Hypotension should also be avoided. Research is needed to evaluate specific efficacy of neuroresuscitative therapies in drowning victims."[94] This recommendation is compatible with the renewed interest in the use of "therapeutic hypothermia" in treating victims of cardiac arrest from a variety of causes.[95]

Treatment

Prehospital Treatment. Most episodes of drowning occur away from the hospital, and the initial prehospital therapy clearly affects patient outcome. The central issues that the rescuer must be concerned with when encountering a drowned victim are: Is the victim breathing? Does the victim have a pulse? Has a cervical spine injury occurred?

The main objective of out-of-hospital therapy is to restore normal ventilation and circulation and to normalize gas exchange, acid-base status, and the circulation as rapidly as possible. Thus, CPR must be initiated as soon as the rescuer encounters a drowned victim. If the victim is apneic, mouth-to-mouth resuscitation must be initiated immediately. Ideally, the procedure is begun in the water if it can be done without putting the rescuer in danger. During mouth-to-mouth resuscitation, care must be taken to maximize forward movement of the jaw rather than hyperextension of the cervical spine, because cervical injury may be present. This point is especially important in drowning cases thought to be related to diving.[53] The importance of maintaining an adequate airway cannot be overemphasized. The drowning victim may have swallowed large amounts of water before losing consciousness; therefore, gastric distension from inadequate mouth-to-mouth ventilation and/or respiratory obstruction may cause the patient to regurgitate and aspirate stomach contents, with resultant aspiration pneumonitis.[96] When the rescuer is able, the victim must be evaluated carefully for the presence of a pulse. Severe bradycardia due to hypothermia, vasoconstriction, and/or hypoxia may make the arterial pulse difficult to palpate. If there is any question about the existence of a pulse, closed chest cardiac compression must be initiated, and artificial ventilation continued until further help arrives.

As soon as more extensively trained individuals and equipment are available, other therapeutic modalities should be considered. Ventilation with a bag-valve-mask device using 100% oxygen should be initiated as soon as available. CPAP will improve matching of ventilation to

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perfusion; therefore, it should be initiated as soon as appropriate equipment is available. However, because CPAP will increase the mean intrathoracic pressure, its effect on circulation should be monitored closely. In the unconscious or severely hypoxic patient or in a patient in whom the airway cannot be protected for some other reason, an endotracheal tube should be placed for airway protection and to facilitate mechanical ventilation and oxygenation. If that is not possible, a laryngeal mask airway or other emergency airway adjunctive measure, such as a King Tube, Combi-Tube, or cricothyrotomy, may be used. In addition, a large-bore intravenous catheter should be placed for fluid and drug therapy. Fluid therapy is initiated with 0.9% saline solution. Drug therapy should be given as needed (*e.g.*, epinephrine, atropine, bicarbonate, *etc*).

The subdiaphragmatic thrust (Heimlich) maneuver has been recommended by Heimlich for use rather than initial CPR in the drowned victim.[97,98] Previous work has shown that water exiting the mouth after drowning comes from the stomach, rather than the lungs.[99] A thorough review of publications and testimony by the Institute of Medicine in 1994 concluded that use of the Heimlich maneuver is inappropriate in treating drowning victims unless a foreign object is obstructing the airway.[100] Use of this technique also may result in delay of effective CPR or in regurgitation and aspiration of gastric contents, with consequences as serious as aspiration pneumonitis, respiratory failure, and death.[100–102]

Automatic electrical defibrillation devices are now available to suggest when the patient may be in ventricular fibrillation and require an electric shock to terminate fibrillation. The rescuer should be cautioned, however, that just because an electric complex, which resembles a reasonable QRS complex, is seen on the electrocardiogram screen, it does not mean that an effective cardiac output is present, eliminating the need to continue cardiac compression.

Finally, no matter how well the patient appears, they must be taken to a hospital for evaluation, as the initial appearance of the drowned patient may be misleading. Transport to the hospital should be carried out with basic monitoring, including, at a minimum, pulse, blood pressure, respiratory rate and pattern, electrocardiography, and pulse oximetry. Oxygen (100%) should be administered *en route* until oxyhemoglobin analysis by pulse oximetry demonstrates that it can be reduced safely with maintenance of hemoglobin/oxygen saturations in the mid 90s to high 90s.

Hospital Treatment. Initial hospital care should emphasize pulmonary support, with treatment individualized to patient status. As soon as possible, arterial blood gas values should be obtained to evaluate the adequacy of ventilation, acid-base status, and pulmonary gas exchange.[53] Supplemental oxygen administration should be continued while obtaining data from arterial blood gas measurements or a pulse oximeter to evaluate for the presence of hypoxemia. Awake, alert, and cooperative patients do not require tracheal intubation unless their pulmonary pathology does not respond favorably to enriching inhaled gas with oxygen, applying a CPAP mask, or both.[53] All comatose patients require tracheal intubation; those with a blunted mental status must be evaluated individually to determine what degree of airway support is necessary.

Spontaneous breathing with CPAP added is preferred if the patient can support a sufficient degree of minute ventilation with intermittent mandatory ventilation added to clear carbon dioxide. If this arrangement does not provide adequate ventilation, the patient is fully supported with controlled mechanical ventilation and PEEP. Pressure-support ventilation (starting at 10 cm H_2O) may be used to overcome work imposed by the endotracheal tube and breathing apparatus to spontaneous breathing in adult patients.[103,104]

The CPAP or PEEP is titrated to maintain adequate oxyhemoglobin saturation ($\geq$ 95%) while using the lowest possible F_{IO_2}. We attempt to keep the P_{aO_2}/F_{IO_2} ratio above 300 and the F_{IO_2} below 0.5; a F_{IO_2} below this level is generally considered not to put the patient at risk for oxygen toxicity. Several controlled studies have shown the effectiveness of CPAP with either mechanical or spontaneous ventilation to treat the hypoxemia associated with drowning.[53,55] Because the work of breathing with CPAP is less than with PEEP, we recommend the former for spontaneously breathing patients. In addition, Bergquist *et al.*[56] found that adding mechanical breaths *via* the use of intermittent mandatory ventilation to CPAP in otherwise spontaneously breathing animals that aspirated fresh water further decreased intrapulmonary shunting.

Invasive hemodynamic monitoring is not required in most patients. However, if there is concern as to the adequacy of intravascular volume, invasive monitoring may be appropriate. While cardiac output may decrease when mechanical ventilation, PEEP, and CPAP are used, it will usually improve with fluid loading,[35] and prolonged inotropic support of the circulation is rarely required.

In general, steroids have been shown to be ineffective in treating the pulmonary lesion of drowning and may, in fact, worsen outcome by interfering with the normal healing process.[13,105,106] Antibiotics are appropriate when and if the patient develops signs of infection or in special cases, such as submersion in a grossly contaminated body of water. If the medium in which the individual suffered their drowning event was heavily contaminated, it is appropriate to use broad-spectrum antibiotics before obtaining bacterial cultures. Otherwise, antibiotic therapy should be guided by the results of bacterial cultures of tracheal secretions. Bronchospasm may be seen in these patients and

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may be treated with either a metered dose inhaler or nebulized albuterol.

Pulmonary edema is a common finding after drowning. The application of CPAP or PEEP will recruit collapsed alveoli, better match ventilation to perfusion and thereby facilitate oxygenation while the underlying pulmonary injury recovers. Breaking the ventilator circuit to suction or provide nebulized treatments after CPAP or PEEP have been optimized can lead to a rapid and rather dramatic recurrence of pulmonary edema and hypoxemia. Thus, interruption of the ventilator circuit should be minimized or avoided if possible.

Despite controversial data regarding the treatment of ICP, it is reasonable to consider placing an ICP monitoring device in comatose drowning victims as soon as possible after arrival at the intensive care unit. Although mild hyperventilation (Pa_{CO_2} approximately 30 mmHg) has been used empirically to reduce ICP in patients with cerebral edema without an ICP monitor,[107] we suggest the use of an ICP monitoring device if hyperventilation were to be considered in a comatose drowning victim. If ICP is elevated (20 mmHg or greater), hyperventilation to achieve a Pa_{CO_2} of 25 to 30 mmHg may be used in an attempt to decrease cerebral blood flow and thereby decrease the ICP while simultaneously keeping the cerebral perfusion pressure (defined as the mean arterial pressure – ICP) at 60 to 70 mmHg. It should be noted, however, that hyperventilation *per se* has not been proven to specifically reduce ICP in comatose drowning victims. A mannitol bolus, 0.25 g/kg ideal body weight, may be used to decrease ICP if hyperventilation alone does not produce the desired result. However, the damage to the brain in drowning is probably the result of severe hypoxia at the time of the injury as opposed to late elevated ICP; elevated ICP in these patients may well be nothing more than a marker of damage already done.

Although there are some extremely interesting data on the use of brain tissue oxygen monitoring in traumatic brain injury,[108] no studies of which we are aware have used this technique in drowning victims.

The importance of prehospital care of drowning victims is now understood, and we are now more adept at providing it than we have ever been in the past. Patients who receive early, effective basic cardiopulmonary life support have an increased chance for normal survival. We are able to treat the pulmonary and cardiovascular complications of drowning quite successfully in most cases. However, despite years of investigation, the issue of neurologic outcome has remained a stumbling block that we have been unable to surmount. It appears that, in the absence of hypothermia that may have occurred during the drowning episode, aggressive brain resuscitation using current techniques is of questionable value in terms of returning the patient to normal neurologic function. Some suggest, however, that maintaining mild hypothermia (32–34°C) may be indicated for 12–24 h after a drowning episode.[94,95] Although there appear to be no "magic bullets" on the horizon, multiple agents – including free-radical scavengers, calcium channel blockers, prostacyclin/thromboxane manipulations, blockers of excitatory neuropeptides, and investigation of phenytoin have been studied after cardiac arrest.[109,110]

Prevention

The most important things we can do today to prevent drowning deaths and a persistent vegetative state as a result of drowning are to implement standards that govern safe pool design, enforce rules that require adequate (at least 5-ft-high with self-closing latches) perimeter fencing around pools,[111] instruct lifeguards in the latest methods of waterfront surveillance and resuscitation techniques, educate the public in prudent consumption of alcoholic beverages in boating, pool, harbor, marina, and beach areas, impose severe sanctions against boat drivers who are intoxicated, post signs in areas of dangerous underwater tow, encourage people to learn to swim and never swim alone, and instruct larger numbers of the general population in basic cardiac life support.

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