NEAR DROWNING

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We also wish to thank the workshop Committee and its chairman, Dr. Charles Lehner for their steadfastness and support in finding the workshop's very capable cochairmen, Drs. Chris Dueker and Steve Brown.

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INTRODUCTION

Christopher W. Dueker

We welcome you to the workshop on Near Drowning, which has been generously sponsored by the Undersea and Hyperbaric Medical Society (UHMS), Divers Alert Network (DAN), and The American Red Cross. This is the third workshop in a new series which includes an audience in addition to the panel of invited participants. We encourage your contributions, especially during the discussions which will follow the morning and afternoon sessions.

Controversy abounds in near drowning. Many older beliefs have been shown to be false, but they still have adherents. Partial truths get promulgated with increasing inaccuracy. And a lot remains unknown. There will not be complete agreement among the panelists. We shall try to resolve most of our differences during the discussions.

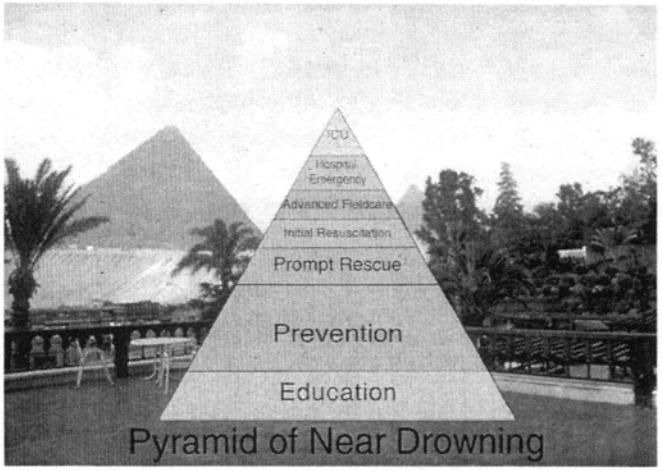
Why would the UHMS be interested in drowning and near drowning? Because of the significance of the problem. For any aquatic endeavor, drowning is the most common cause of death. DAN reports that about 63% of its fatalities are drownings. In the over-35 age group drowning occurs in about 29 percent of deaths in diving (1).

Unfortunately, any body found in the water tends to be called "drowning". Not all bodies found in the water died in the water. And not all deaths in the water are drownings.

In scuba accidents near drowning may be the result of many problems including exhaustion, bad air, medical illness, trauma, inert gas narcosis, cerebral air embolism, etc. Treatment may be required both for near drowning and the primary problem. Failure to treat properly in the right sequence may have fatal consequences. Further, in diving accidents there may be limited availability of emergency medical services and hospitals. Finally, unsatisfactory dilemmas occur, i.e., a mixed-gas scuba diver with seizures deep in a cave in rural Mexico.

According to "the burden of disease study", drowning ranks as the 20th most common cause of death worldwide (2). This puts drowning well behind heart disease and road accidents, but ahead of war injuries.

What is drowning? An asphyxial death from submersion in fluid, usually water. Some investigators would add to the definition, pulmonary aspiration of fluid. Death may be quick or may be delayed from the progressive complications of asphyxia. A near drowning is a severe survivable submersion accident. The injury may be so severe that death supervenes; this is no longer a near drowning. Persons who die after initially being considered to have nearly drowned should be classified as drownings.



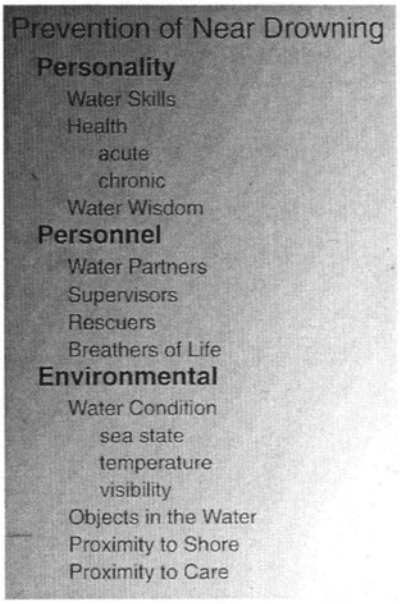


Illustration (*see* above) shows the pyramid of near drowning. Success in managing immersion accidents comes mainly from education and prevention. Unfortunately, our highest form of treatment—the ICU—has much less value.

Prevention of near drowning can be arbitrarily divided into the illustrated categories. For a scuba diver the most important consideration would be water wisdom, health, and proper equipment.

Another category for prevention concerns the personnel around the individual; for the scuba diver this would first be the buddy. Supervisors may or may not be present.

Lifeguards are rarely present.

Lifeguards are important in pools, lakes, and beaches. For a variety of reasons the lifeguard system frequently fails. Lifeguards should serve to prevent accidents, to make timely rescues, and to initiate resuscitation. For scuba divers, the buddy should be available for these jobs.

Environmental conditions affect accident prevention: water conditions, water temperature, visibility, animate and inanimate objects in the water, etc.

Preventing random access to swimming pools and other water sites has had enormous public health value.

In this workshop we cannot completely discuss near drownings. We shall examine mechanisms of submersion accidents and relate these to rescues, resuscitation, treatment, and outcome. When possible we shall emphasize scuba diving because it has been so neglected in the literature of near drowning.

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DROWNING SYNDROMES—THE MECHANISM

Carl Edmonds

The drowning syndromes are viewed as a continuum between the aspiration of a relatively small amount of water, causing symptoms and respiratory based signs, through to the near-drowning presentation in which there is loss of consciousness—but with survival. At the other end of the continuum are the fatal cases of drowning, rarely involving the gross hemodynamic and biochemical changes seen in some animal experiments. The behavior of the victims during the incident is reviewed, as are the experiments conducted on animals, with various types and quantities of aspirant, to model the physiology. These are compared with the adult human clinical case series. "Quiet" drownings are described and classified. The clinical features of near drowning are reviewed. Factors that influence survival are noted. The pathologic findings are discussed, with a critical approach to the concept of "dry" drowning, and some postulates on the findings of cranial hemorrhages. Lungs are the primary and dominant organ of involvement, and hypoxia is the major physiologic abnormality. A description of the salt water aspiration syndrome, including its development, clinical and laboratory findings as seen in scuba divers, is also reviewed. Finally, a brief review of the literature specific to scuba drownings is given.

Note: Excellent reviews of this subject are presented in references 1–6.

TERMINOLOGY

Drowning refers to the death of an air-breathing animal due to immersion in fluid.

Delayed drowning or *secondary drowning* occurs when the victim appears to recover from the incident, but then proceeds to die.

Near drowning (ND) refers to the loss of consciousness from the incident, but not leading to death.

The *aspiration syndrome* refers to the effects of aspiration of fluid into the lungs, but without loss of consciousness.

There is a continuum in the severity of symptoms and signs among aspiration, ND, and drowning. They can be incorporated together as *the drowning syndromes*, for a greater understanding of each. The continuum needs to be appreciated if a rational approach to the management of ND is to be made.

Post-immersion syndromes refer to the disorders that develop after immersion and subsequent rescue.

Other classifications have been proposed, based on the type and amount of fluid inhaled. These will be referred to later.

BEHAVIOR DURING DROWNING

Observation on experimental drownings (7) showed the typical behavior of *animals*—an immediate struggle for freedom, sometimes with an inhalation. Then there was suspension of movement, possible exhalation of a little air and frequent swallowing. Later, there was a violent struggle for freedom, followed by convulsive movements and the exhalation of air with spasmodic inspiratory effort, before death.

Observations of *human* drownings (8,9) parallel the animal experimentation, involving a panic reaction with violent struggling followed by automatic swimming movements. There may be a period of breath holding and swallowing of large amounts of water. Vomiting may occur, followed by gasping and aspiration of water. Blood stained froth develops in the airways and the patient convulses, and then dies.

More recently (10) the observations on children exposed to drown-proof training, as it is euphemistically called, has modified our knowledge of the normal response in human drownings. There is usually a failure of the infant to struggle. He holds his breath and makes automatic but ineffectual paddling-type movements as he sinks to the bottom.

Over the range of animals tested and observed, consciousness is usually lost within 3 min of submersion. For many years, drowning was considered a "fight for survival" (2), but this is now changing (3).

When a fully conscious human accidentally falls into the water, he fights to survive. In other circumstances drowning may proceed in a quiet and apparently unemotional manner. Examples of *quiet drownings* include:

1. Hyperventilation with breath-hold diving. Craig (11) reported eight cases of hyperventilation before breath-hold diving, which resulted in loss of consciousness due to the development of hypoxia. This developed before the blood carbon dioxide levels rose sufficiently to require surfacing to breathe. In these cases loss of consciousness occurred without any obvious warning, and the underwater swimmer then aspirated and drowned.
2. Hypothermia and/or cardiac arrhythmias leading to loss of function and drowning have been well described by Keatinge (12,13) and Conn and associates (14).
3. Drugs and alcohol increase the incidence of drowning (3,15,16) by impairing judgment and reducing the

struggle to survive. It is likely that nitrogen narcosis may have a similar effect in divers.

4. Diving problems may produce hypoxia. These include the dilution hypoxic effects with mixed gas breathing, ascent hypoxia, and carbon monoxide toxicity by the blockage of oxygen metabolism. They are all likely to cause loss of consciousness (17) without excess carbon dioxide accumulation, associated dyspnea, or distress.
5. Salt water aspiration. In animals, 2.2 ml of fresh water/kg body weight drops the Pa_{O_2} to approximately 60 mmHg within 3 min, or to 40 mmHg with sea water (18,19). A similar situation was observed clinically in the salt water aspiration syndrome of divers (20).
6. Other causes of unconsciousness leading to drowning have been described, e.g., some marine animal envenomations, coincidental medical illnesses, cerebral arterial gas embolism, etc.

PHYSIOLOGY

Animal Experiments

In the 1930s many animal experiments were conducted both in Europe and North America demonstrating that if an animal was immersed and drowned in water containing chemical traces or dyes, these would spread through the tracheobronchial tree to the alveoli surfaces (7,21,22). In the case of fresh water, this was also absorbed into the blood stream (21).

A consistent fall in arterial oxygen content was observed, followed by a rise in arterial carbon dioxide and sometimes ventricular fibrillation. (23,24)

Swann and his colleagues from Texas (25,26), in a series of accurate but misleading experiments, flooded animals' lungs with fresh or salt water, and demonstrated the significant differences between the two, attributable to osmotic pressures.

In both cases, flooding of the lungs produced a reduction in Pa_{O_2} and pH, with a rise in the Pa_{CO_2}. This was attributable to airway obstruction.

Because fresh water was osmotically much weaker than blood, it moved into the bloodstream and produced hemodilution—reducing most of the blood contents including proteins, sodium, chloride, etc.

The subsequent reduction in the osmotic pressure of the blood resulted in hemolysis and a liberation of both hemoglobin and potassium, with various metabolic and renal complications. Deaths were often cardiac in nature and due to ventricular fibrillation.

When, however, the animals' lungs were flooded with sea water—which has a higher osmotic concentration than blood—water was drawn from the bloodstream into the lungs, producing a pulmonary edema and hemoconcentration. This caused an increase in the hematocrit, blood proteins, and electrolytes.

For many years physicians attempted to correct these presumed electrolyte, metabolic, and cardiac abnormalities in human drownings, but their cases did not seem to conform to the animal model.

Earlier workers had shown that in dogs that drowned there was still large volumes of air in the lungs (7), as there is in humans. This is unlike the Texan model.

Colebatch and Halmagyi (27–32), working in Australia in 1961, produced an animal model more relevant as regards clinical management of patients, by aspiration of only 1–3 ml/kg body weight (27–32). By using these smaller volumes they demonstrated the dominance of arterial hypoxia, largely independent of the amount of fluid inhaled. Pulmonary hypertension, vagal inhibition, and reduced compliance were also noted.

Sea water aspirant was usually associated with significant pulmonary edema, but the fresh water aspirant was often absorbed from the lungs within 2–3 min.

Subsequent animal experiments, using intermediate volumes of aspirant, by Medell and colleagues (18,19,33) verified the importance of the shunting of blood to be the predominant factor causing persistent arterial hypoxemia. It is due to perfusion of blood through non-ventilated areas of lung.

The effects of different types of aspirant on lung surfactant were described (33). Fresh water appears to destroy the surfactant, whereas salt water dilutes and washes it out.

Human Series

Clinical series described by Fuller (34,35) in 1963, Griffin (36) in 1966, and Medell et al. in 1976 (4) illustrated the considerable differences between near-drowning humans and animals with flooded lungs.

Human cases did, however, reflect the animal experiments in which smaller amounts of aspirant were administered.

They all stressed that the initial organ involved is the lung. The physiologic consequence is hypoxemia. The major damaging effects are on the lung and brain. Significant electrolyte disturbances and cardiac arrhythmias are not frequent.

The initial symptoms are respiratory, then come the effects of hypoxia causing subsequent pulmonary and cerebral damage. Aspiration of vomitus, following the ingestion of sea water, is frequent in the early and the resuscitation phases.

Investigations reveal: Hypoxemia; a variable arterial CO_2; acidosis; the effects of reduced pulmonary compliance; patchy and variable consolidation in the lung x-rays; and a polymorphonuclear leukocytosis. Others depend on other organ involvement.

An excellent series of pediatric drownings have been

described (10) and much work has been done on the relevance of hypothermia to drowning, which will not be dealt with here.

Survival From Near Drowning

In human drownings, deterioration after initial resuscitation is frequently recorded which influences management.

The likely causes for delayed deaths include the extensive and progressive lung damage, cerebral hypoxia, secondary infections (usually of the lungs), renal failure, and iatrogenic factors.

Factors that negatively influence survival have been well documented by Medell (3):

- prolonged immersion;
- delay in effective cardiopulmonary resuscitation;
- severe metabolic acidosis (pH < 7.1);
- asystole on admission to hospital;
- fixed dilated pupils; and
- a low Glasgow coma score (< 5).

Nevertheless, none of these predictors are infallible and survival with normal cerebral function has been reported with all of the above factors. A flat EEG may be reversed.

Claims of survival after extended duration underwater without any supplementary air have been used to encourage rescuers to persevere with resuscitation efforts.

Cases have been reported in which people have been submerged for between 15 and 45 min (37–43) and have survived without neurologic sequelae. Many other cases have not been reported. Two such cases with which I have been involved were submerged for 15-20 min.

The explanations given for such prolonged durations of survival are as follows:

- Hypothermia (10,12,41–43) is protective and develops very rapidly with aspiration of water. In swimmers and divers, hypothermia may well develop even before the incident.
- The "diving reflex" (10,43) is a possible, but a contentious, explanation. Within seconds of submersion the diving reflex may be triggered by sensory stimulation of the trigeminal nerve, reflex, or voluntary inhibition of the respiratory center in the medulla. This results in a bradycardia and shunting of the blood to the brain and coronary circulations. It is independent of baroreceptor or chemoreceptor imputs. The diving reflex is more intense in the frightened or startled animal, compared to those which dive or submerge voluntarily, but it is not known whether this is applicable to humans. Typical water temperatures above 20° do not inhibit the diving reflex, but progressively lower temperatures augment it.
- Respiratory gas exchange in the lungs can continue after submersion. With or without the effects of laryngospasm there is often some liters of air remaining within the lungs, allowing for exchange of respiratory gases. Whether fluid enters the lungs in an unconscious victim will depend on many factors, including the spatial orientation of the body. A dependent position of the nose and mouth, facing downwards, will not be conducive to fluid replacement of the air in the lungs. Increased pressure (depth) might increase the availability of oxygen uptake through Henry's Law. In a comatose state, with low oxygen utilization and the effects of hypothermia and the diving reflex, a retained respiratory gas volume might add considerably to the survival time, although it is not often mentioned in the drowning literature.
- Gas exchange between the pulmonary blood and the aspirated fluid might have a marginal effect on prolonging life.

Despite the fact that spectacular and successful rescue can be achieved after prolonged submersion, it is more frequent that this is not so. Many victims lose consciousness and die after only a few minutes of submersion.

Pathology (10,44–46)

In recreational scuba deaths (17) drowning is the commonest cause, but is usually a secondary effect, with the primary cause leading to loss of consciousness.

Other accidents (dysbaric, medical illnesses, trauma, etc.), occurring while immersed or submerged, are likely to result in the secondary complication of drowning, with all its pathologic sequelae.

Drowning often complicates the interpretation of the diving accident and produces a combined pathology.

Various aspects of drowning pathology need to be addressed.

"Dry" Drowning

Many references have been made to the possibility of drowning without any aspiration of fluid. In 1931, Cot (47) stated that 10% of victims of drowning do not aspirate water. They die from acute asphyxia while submerged, now attributed to reflex laryngospasm.

Virtually every review of drowning over the rest of this century refers to this belief, without question, although the incidence is often extended to 20%. It does conflict with the other animal work of the 1930s, but was given support by Pearn (10) in a fascinating philosophical review of pathophysiology, in 1985. Pearn, who is an eminent pediatrician, admitted that it is not so frequent in childrens' drowning.

In a recent review by Medell in 1993 (3), the concept is reiterated. Three references are given. Two (16, 44) refer to autopsy findings; the third to one of Medell's own papers(4), but perusal of that paper reveals little supporting evidence.

He states that by the time near-drowning/aspiration victims had reached the hospital, 10 of the 81 had a PaO_2 of 80 mmHg or greater. No information was available as to whether those cases were fresh or salt water victims, but the majority of the total number were in fresh water.

As has been stated earlier, fresh water is absorbed very rapidly from the lungs, and therefore autopsy findings, and indeed the PaO_2, cannot really be used to imply (let alone prove) the absence of an aspirant. Especially is this so when these investigations are performed some time after the event. Thus, there is good reason to question the literature on "dry" drowning.

Reference to the anesthetic literature is also informative. Miller's *Textbook of Anaesthesia* (48) with its associated references (49–51) defines and describes laryngospasm as the exaggerated and prolonged response of a protective glottic closure reflex. There is no airflow and the true vocal cords cannot be seen. The development of hypoxia and hypercarbia remove the effect. Thus laryngospasm eventually ceases spontaneously as hypoxia and hypercarbia develop.

The indications from the anesthetic literature imply that laryngospasm will not, by itself, be continued until death. The glottic closure will relax before death, allowing the passage of gases and fluids into the lungs.

In the absence of more definite information, it would probably be worthwhile presuming that all near-drowning or drowning victims have aspirated, and base one's first aid and management on this presumption. "Dry drowning" could well be an artifact of fluid absorption from the lungs, or death from other causes. This author has never witnessed a case among divers in salt water.

Autopsy Observations (10,44–46)

Only a brief mention will be made of this, as the theory and the practice of drowning autopsies are surprisingly contentious for such a common disorder.

Frequently there are coincidental signs of immersion, marine animal injury, or resuscitation damage. The stomach may contain swallowed fluid.

It was stated (45) that autopsy examination of 118 consecutive drowning cases suggested that 85% aspirate 22 ml of fluid or less per kg body weight. It was therefore considered unlikely that drowning victims die acutely of electrolyte imbalance and/or ventricular fibrillation. Death is more likely to be secondary to asphyxia.

The respiratory findings are congested voluminous lungs and in the airways; frothy hemorrhagic sputum (especially in salt water cases), vomitus, foreign bodies, and particulate matter. Respiratory infections, abscesses, etc., are not infrequent if death is delayed. Otherwise there is little typical to describe macroscopically.

The pulmonary changes at autopsy reflect not only the activities during the time of the incident, but also those of resuscitation and the changes in the lung fluids between the time of rescue and the time of death. Many such factors may influence the final macroscopic result.

Histologic changes may demonstrate toxic effects both of chemicals and the specific aspirant. The surfactant changes, including denaturation, can progress even after apparent clinical improvement. The epithelial and endothelial changes, with detachment of the basilar membrane and cellular disruption have been described elsewhere (10) and are beyond this author's area of expertise.

Usually, death is due to a progressive or irreversible pulmonary damage associated with the drowning per se, and there are obvious reasons for this. They include progressive surfactant damage despite rescue, pneumonitis from the aspirant or vomitus, infections, etc. Pulmonary oxygen toxicity, associated with resuscitation attempts, may also be present.

The major effects on the neurologic system are those of hypoxic brain damage and subsequent cerebral edema with raised intracranial pressure. In the event of delayed drowning deaths the relevant organs are damaged, either lungs, brain, or kidneys.

Neither the Gettler chloride tests nor the specific gravity of serum can be relied upon to establish a diagnosis of death by drowning.

Identification and comparison of environmental and systemic diatoms have been recommended, but are complex and not frequently performed. It does not prove drowning, merely the aspiration of water while the circulation is still functioning.

Conventional pathology teaching claimed that mastoid and middle ear hemorrhages were indicative of drowning. As we explained in 1976 (46) these hemorrhages are the sequel of barotrauma, not drowning. The acceptance of this explanation by diving pathologists has encouraged us to extend the concept a little further.

Autopsies on drowning cases who have submerged while still alive, although unconscious, may develop other cranial hemorrhages which are sometimes interpreted as a cause of the accident. Meningeal hemorrhages, both dural and arachnoid, are frequently observed. These are usually not very extensive and are quite different from the brain hemorrhages of arterial gas embolism or decompression sickness.

They are probably derived from the hemorrhages of descent sinus barotrauma, which ruptures into the cranial cavity when the enclosed and compressed gas expands as the body is surfaced.

SALT WATER ASPIRATION SYNDROME (17,20)

A common diving illness in the Royal Australian Navy in the 1960s was the salt water aspiration syndrome. It is called "salt water fever" by the Australian abalone divers. Its importance lay not only in the light it shed on (ND) cases, but also because it was often confused with other diving or infectious diseases. This condition, which is due to the aspiration of small amounts of salt water during diving, may occur because of inexperience, while buddy-breathing training, or due to a faulty regulator. At that time navy regulators did not have purge valves. Novices were trained in buddy-breathing during their first dive, in the open ocean. This led to a frequent aspiration of sea water. In other cases the aspiration occurred on the surface, after the diver had removed his regulator.

Experienced divers often developed it following a fast towed search, while abalone divers used inadequate surface supply equipment available with "leaky regulators". Their term "salt water fever" indicated that they were well aware of the cause.

Other water users to present with a similar disorder, but possibly not as frequent, are snorkellers, surfers, and helicopter rescuees.

A prospective survey was carried out on 30 consecutive cases that presented for treatment, their symptomatology was documented, and investigations performed. Subsequently, "volunteers" were encouraged to aspirate sea water through doctored demand valves. The clinical and laboratory manifestations they developed were consistent with those in the clinical series.

The following observations were made on the clinical cases:

Immediate symptoms: On specific interrogation a history of aspiration was given in 27 (90%). Often this was not causally associated by the novice diver with the subsequent events. Over 90% noted an immediate postdive cough, with or without sputum. It was usually suppressed during the dive. Only in the more serious cases was the sputum bloodstained, frothy, and copious.

Subsequent Symptoms:

Symptoms	%
Rigors, tremors, or shivering	87
Anorexia, nausea, or vomiting	80
Hot or cold sensations	77
Dyspnea	73
Cough	67
Sputum	67
Headaches	67
Malaise	53
Generalized aches	33

Respiratory symptoms: There was often a delay of 1–2 h before dyspnea, cough, sputum, and retrosternal discomfort on inspiration were noted. In the mild cases, respiratory symptoms persisted for only an hour or so, while in the more severe cases they continued for days. The respiratory rate roughly paralleled the degree of dyspnea. Respiratory stimulants appeared to aggravate the dyspnea and tachypnea.

Physical examination of the chest in about half the cases revealed crepitations or occasional rhonchi, either generalized or local. Rarely, they were high pitched and similar to that of obstructive airways disease. Signs usually disappeared within the first 24 h.

Administration of 100% oxygen was reliably effective in relieving respiratory symptoms and removing cyanosis when this was present.

X-ray of the chest revealed areas of patchy consolidation, or a definite increase in respiratory markings, in about half the cases. These usually cleared within 24 h, but remained longer in severely affected cases. X-rays taken after the incident and repeated within a few hours sometimes showed a variation of the site of the radiologic abnormality.

Expiratory spirometry performed repeatedly over the first 6 h, showed an average drop of 0.7 liters in both $FEV_{1.0}$ and VC measurements. These usually reverted to baseline levels soon after this time, although they could persist in a lesser form for up to 24 h. Even those patients who had no respiratory symptoms demonstrated a reduction in lung volumes. Arterial blood gases, when performed, revealed oxygen tensions of 40–75 mmHg with low or normal carbon dioxide tensions.

Generalized symptoms: The patient complained of being feverish in most cases. Malaise was the next most prominent feature. Headaches and generalized aches through the limbs, abdomen, back, and chest were important in some cases, but usually not dominant. Anorexia was common and unexpected in this group.

In some there was an impairment of consciousness, including a transitory mild confusion (three cases), syncope with loss of consciousness on standing (two cases).

The feverish symptoms were interesting, and are also seen in near-drowning cases. Shivering, similar in some cases to a rigor, and in some cases to generalized fasciculation, was a characteristic feature in the colder months. It was precipitated or aggravated by exposure to cold, exercise, or breathing 10% oxygen (a research procedure, not recommended clinically). It was relieved by administration of 100% oxygen. It occurred especially in those exposed to cold because of duration and depth of dive, clothing worn, and environmental conditions during the dive and subsequently.

The association of shivering with hypoxia and cold has been described by others (52). The shivering occurs concurrently with the pyrexia, which also takes an hour or two

to develop.

Pyrexia was able to be verified in half the cases, up to 40°C (mean 38.1°C, SD 0.6), and the pulse rate was elevated (mean 102/min, SD 21) over the first 6 h.

Some patients realized that relief from these symptoms may be obtained by either hot-water baths or showers, or lying still in a very warm bed.

These systemic signs and symptoms also usually reverted to normal within 6 h, and rarely persisted beyond 24 h, unless the case was of considerable severity.

Hemoglobin, hematocrit, ESR, and electrolytes remained normal. The white cell count was usually normal, although a mild leukocytosis (not in excess of 20,000/mm^3) was noted in a few cases, with a moderate polymorphonuclear increase and a shift to the left.

Lactic dehydrogenase estimations revealed a mild rise in some cases. X-ray and lung volume changes are described above.

A subsequent investigation into the causes of recreational scuba diving deaths (53,54) revealed that water aspiration was part of the sequence leading to death in 37% of the cases, often a consequence of equipment or technique problems. In these cases "leaking regulators" were either observed and commented on by the victim beforehand or demonstrated during the diving investigation. Although there was often a fault in the actual regulator, with a failure of valve seating, the degree of leaking was frequently demonstrated to increase with the volume of air being required (e.g., with exertion, swimming against currents, panic, etc.) and/or with a diminished line pressure to the second stage. Salt water aspiration often formed a vicious circle with panic and exhaustion.

Hypoxia from salt water aspiration, as could be expected, aggravated the problems of fatigue and exhaustion and was a precursor to loss of consciousness (with or without dyspnea) in both near-drowning and drowning cases.

DISCUSSION

There is no distinct division in the initial presentation among aspiration, near-drowning, and drowning cases. Aspiration syndromes merge with ND, often the intensity of the symptoms and the degree of consciousness, depending on various circumstances, the activity of the victim, and the administration of oxygen.

Near-drowning cases sometimes die hours or days later, thereby being re-classified as secondary or delayed drowning.

Some of the apparently "drown" victims, because of adequate CPR and good intensive care management, surprisingly recover without serious sequelae.

In a prelude to the 1997 UHMS Workshop on Drowning and Diving, the Chairman made the following statement in the pre-workshop correspondence: "As you know, the drowning literature ignores diving whilst the diving literature ignores drowning." (Dueker C, personal communication, 1997.)

It is paradoxical that drowning, which causes more than 80 times the number of deaths in recreational divers than either decompression sickness or contaminated air, does not rate more than a paragraph or two in some diving medical texts.

Nevertheless, of the major seminal reviews (1,5,6,) presented on this subject, most have been by diving physicians. In reviewing the literature on drowning, the only papers that I could find that specifically relate any of the drowning syndromes to scuba diving, was my own one on the salt water aspiration syndrome (20), and one with an anecdotal review followed by a case report (55). One can only assume that it is too common a disorder to excite much academic interest.

In general, apart from the treatment of the near drowned, which is frequently reviewed, very little critical thought or assessment has been directed toward the literature that is available, and most reviews are merely a rehash of previous presentations. Even more uncommon is the addressing of the drowning problem with scuba deaths and accidents (56). It is the task of the UHMS workshop to redress this.

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DOES THE CARDIOVASCULAR DIVING RESPONSE HAVE A PROTECTIVE EFFECT IN NEAR-DROWNING INCIDENTS?

Claes E. Lundgren

The classical cardiovascular diving response is very well developed in natural divers such as the duck and the seal but present in virtually all species in which it has been studied, from the chicken to man. Its components are shown in Table I borrowed from Wolf (1). The dive response is most pronounced when breath holding is combined with stimulation of facial cold receptors of the trigeminus nerve. The biological value of this reaction is presumably to divert blood flow from hypoxia-tolerant tissues such as muscle, skin, and abdominal viscera and provide more of the oxygen in the blood and the lungs to the most hypoxia-sensitive organs, the brain and the heart.

The value of the diving response in near-drowning situations in humans is obviously not amenable to experimental testing. However, it has been suggested to explain, at least partly, the survivability of amazingly long periods of submersion in cold water, ranging from 38 min (2) in adults to 65 min in children (3). While there would be no information about physiologic reactions, such as the diving response during the submersion phase in these cases, the victim is typically hypothermic when recovered and brought to treatment. Hypothermia is not part of the diving response, but the hypothermia-induced suppression of metabolism without doubt plays a role for survival.

The relative importance of the two events, diving response and hypothermia, depends on the temporal interplay as pointed out by Gooden in an excellent review article (4). If the diving response is effective in making the body economize with the oxygen stores, the maximal breath-holding time should be longer the more pronounced the diving response, that is, it should be longer in cool water than in tepid water. Indeed, recent evidence points to an enhanced breath-holding endurance when the diving response is elicited during cooling of the face (5). However, this gain in breath-holding time was only observed in trained breath-hold divers (5).

Paradoxically, not only did this positive relationship between breath-hold duration and diving response not hold up in untrained volunteers, who held their breath maximally when totally submersed in water of different temperatures, but the breath-hold duration was 25.6% longer in thermoneutral water than in cold water (6). The reduction in heart rate as shown in Fig. 1 was 28% in 32°C and 26% in 20°C water compared to controls in thermoneutral air (28°C) (6). Yet, the breath-hold duration was by far the lowest in 20°C water—down by 55%. We ascribed this to the powerful respiratory drive initiated by cold stimulation of skin receptors, and more importantly, to the increase in metabolism by about 250% that occurred in our subjects when submersed for a relatively long period in 20°C water. Surprisingly, breath-holding time in thermoneutral water increased by about 25%. This is probably due to the improvement in the body CO_2 storing capacity that happens during immersion, as we have shown in rebreathing experiments (Fig. 2) in our laboratory (7). However, survival in the apneic person depends greatly on oxygen availability and usage. With the lung volume at functional residual capacity, the total oxygen stores of a 70-kg adult amount to about 1,500 ml (cf 8). If these were all usable, which is clearly not the case, the stores would, at an oxygen consuming rate of 300 ml/min, last 5 min.

Yet, a complete depletion of the oxygen stores obviously does not occur during a 5-min resting breath hold even in the absence of face cooling, as shown in Fig. 3. These recordings were made in elite breath-hold divers and in matched controls. They show that the breath-hold divers had a distinctly lowered oxygen consumption, based on alveolar gas analysis, compared to the resting apneic condition and compared to the breath-holding controls. The divers held their breath for 4–5 min with oximeter readings still indicating between 40 and 75% saturation (9). The fully developed diving response is probably connected with even more substantial changes in how the oxygen stores are used. Large volumes of tissue, including muscles and the splanchnic area, are subject to vasoconstriction and reduced blood flow. In our laboratory, we have, for instance, recorded reductions in forearm blood flow of 58% during breath holding combined with application of cool water to the face (10). This intense vasoconstriction of the diving response increases blood pressure to very high levels in some persons. We have recorded diastolic blood pressures of more than 150 mmHg in breath-hold divers performing fully submersed simulated dives to 50 m in the hyperbaric chamber (11). The slowing of the heart rate was in these cases clearly secondary to the increase in blood pressure. Coupled with this is a lowering of cardiac output exemplified in Fig 4.

The power output of the heart may reduce its oxygen

Table 1: *Features of the Dive Reflex (Wolf 1978)*

Inhibitions of somatic innervation to diaphragm and other respiratory muscles	Apnea
Excitation of vagal nerves to heart, suppression of SA pacemaker	Bradycardia Junctional rhythm Reduced contractile force and stroke volume
Inhibition of β-adrenergic nerves to heart	Reinforcement of above
Excitation of α-adrenergic nerves to visceral, cutaneous, and muscle arteries	Reduction in interruption of peripheral blood flow, elevation of diastolic pressure
O_2 sparing through anaerobic metabolism	Temporary accumulation of lactate, CO_2, and potassium, and lowering of blood pH

consumption by as much as 42% when the dive response is elicited during exercise, as shown by Bjertnaes et al. (12). If we assume that the dive response completely reserved the blood and lung oxygen stores for the heart and the brain, we arrive at the following:

Oxygen stores (total)	
Lungs (at FRC)	370 ml
Arterial blood	280 ml
Venous blood	600 ml
	1,250 ml

and let us, for the sake of this discussion, assume that two-thirds of this oxygen or 825 ml can be used before hypoxic damage occurs,

Oxygen usage	
Heart (58% of normal)	15 ml/min
Brain	50 ml/min
Total	65 ml/min

which would yield a duration of the O_2 stores of 12.7 min. Now, this obviously neglects the fact that some oxygen usage must occur in other tissues. However, we have evidence that in persons with a marked diving response, such as elite breath-hold divers, there is a pronounced shift to anaerobic metabolism. In a 70-m dive performed by Enzo Majorca, the lactic acid accumulation measured upon surfacing was equivalent to an oxygen volume of 1,133 ml, and this is despite the fact that the alveolar oxygen tensions upon surfacing still were in the 30 mmHg range which, while low, certainly does not threaten the diver with death from brain hypoxia (9).

However, it would be difficult to imagine that the diving response, if acting alone, would allow a longer grace period, in terms of preventing permanent hypoxic brain injury, than about 10 or 15 min. So the remaining 45 min

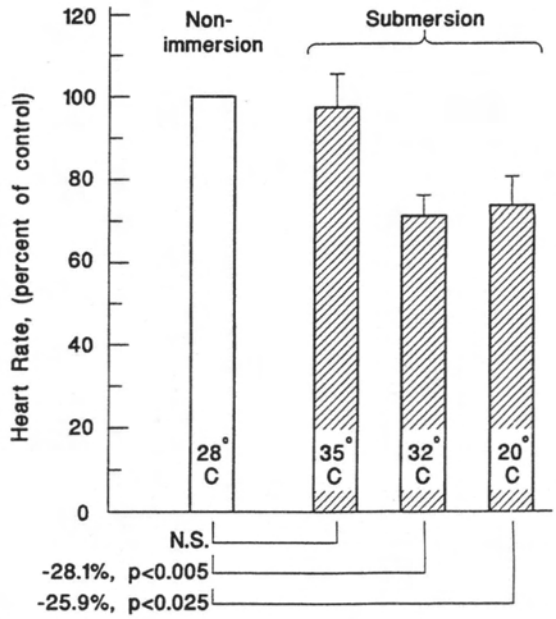

*Fig.*1. Heart rates 30 s into breath holds during submersion in water at 35°, 32°, and 20°C expressed as percent of heart rate during breath holding under controlled conditions, i.e., non-immersion in ambient air at a temperature of 28°C. Values are means ± SE of two series of breath holds in each of five subjects; statistics by paired comparisons and *t* test. *With permission from* (5).

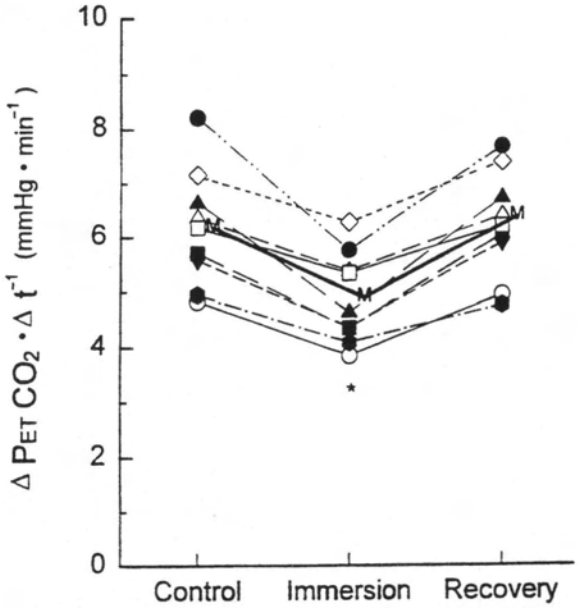

Fig. 2. Rate of rise of end-tidal carbon dioxide pressure ($\Delta P_{ET_{CO_2}} \bullet \Delta t^{-1}$) under conditions of non-immersion, head-out immersion, and non-immersion recovery. The $P_{ET_{CO_2}}$ build-up was significantly slower during immersion (*$P < 0.05$, Bonferroni *t* test). With permission from (6).

of survival most probably can be ascribed to hypothermia. A case, based on a literature review, has been made by Dr. Gooden for the diving response allowing enough cooling-down time (assuming sufficiently cold water) for hypother-

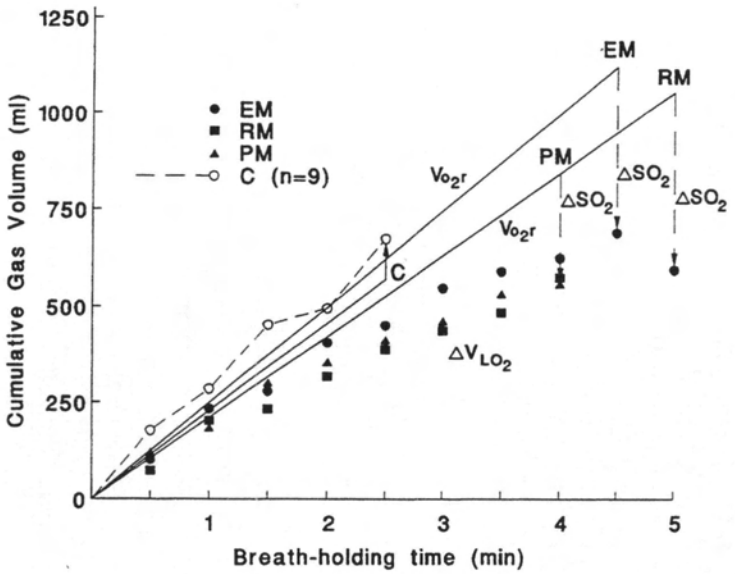

Fig. 3. Cumulative volume of oxygen taken up from the lung during non-immersed breath holds performed by three highly trained divers (EM, PM, RM) and matched non-diver control subjects (C). *Solid lines* represent cumulative oxygen consumption under eupnea. *Arrows* and ΔSO_2 represent changes in gas stores. *With permission from* (8).

mia and protective hypometabolism to be established. The rate of cooling in infants being cooled in the operating room in preparation for surgery, according to one study (13), was to reduce rectal temperature by 2.5 °C in the first 10 min. This would account for a reduction in metabolism of about 25% or by 33% after 15 min., i.e., not fast enough. However, one could imagine that the complete submersion in cold water could enhance the cooling rate further. It is noteworthy that fear potentiates the diving response in humans (14), is often more pronounced in diving animals during forced diving than during spontaneous diving, and fear is certainly part of the drowning scenario. The diving response is likely to play a crucial role in the survival of drowning incidents, although current information does not allow us to account fully for how nature deals with the threat of hypoxic damage after the diving response is established but before metabolism is sufficiently suppressed by hypothermia.

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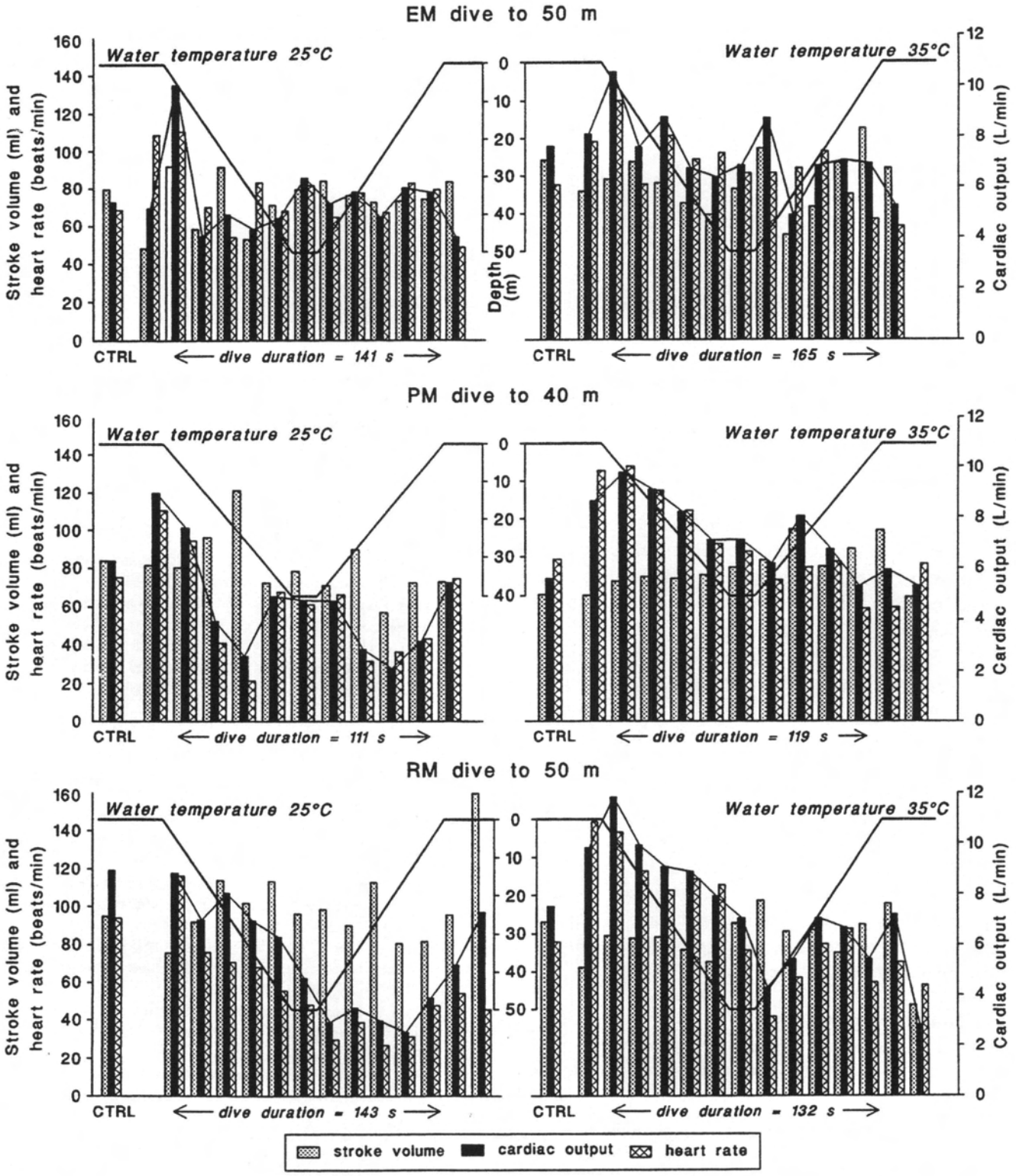

Fig. 4. Stroke volume, cardiac output, and depth vs. time during breath-hold dives to 50 m by three highly trained divers (EM, PM, RM). Measurements before and after dives were averaged over 10-s intervals. Water temperature was either thermoneutral (35° C) or cool (25°C). *CTRL* = control. *Note* that the circulatory dive response was generally more pronounced in cool water than in thermoneutral. *With permission from* (10).

MYTHS IN NEAR DROWNING

Christopher W. Dueker

The world of near drowning abounds in myths and misconceptions. I have selected some of the more prominent and some of my favorites.

THE MEDIUM

Dr. Edmonds has discussed the issue of fresh vs. salt water immersion. The medium essentially does not greatly influence the outcome of the accident. Different things happen sometimes; sometimes, they don't. Treatment focuses on correcting hypoxia. Outcome depends on the promptness and efficacy of this treatment.

LARYNGOSPASM

Tight closure of the laryngeal vocal cords helps protect the trachea and lungs from aspiration of foreign material. This involuntary laryngeal spasm (or laryngospasm) occurs when water enters the throat. During the spastic closure, air cannot pass through the larynx.

Anesthesiologists frequently encounter laryngospasm in their patients, especially during the induction or emergence from general anesthesia. In deep anesthesia, the vocal cords are relaxed and cannot protect the airway. During profound laryngospasm, hypoxia may occur. Positive airway pressure by mask or airway manipulation may circumvent the spasm. In severe cases it may be necessary to inject muscle relaxants and place an endotracheal tube through the vocal cords. Profound hypoxia will cause the laryngospasm to relax or "break". Most anesthesiologist are taught that laryngeal spasm thus cures itself and that the patient will again breathe.

If breathing resumes in the controlled setting of the operating room, reoxygenation quickly occurs and the problem will usually be resolved; however, in an immersion the breath will be of water and the outcome is not good.

The drowning literature frequently claims that some deaths occur during laryngeal spasm without water ever entering the trachea. This assertion contradicts the experience of anesthesiologists who would expect at least an agonal breath which would permit water aspiration. Further, at death the vocal cords relax and water might passively enter the trachea.

This is not to say that there are not serious problems with laryngeal spasm; and, in truth, death may follow laryngospasm. These would be examples of deaths occurring in water which are not actually drownings.

During laryngospasm, cardiac dysrhythmias may occur. In surgical laryngeal spasm these presumably are caused by hypoxia. In near drowning there would also be confounding influences that make dysrhythmias not uncommon in breath-hold immersion.

A partially anesthetized patient or an immersion victim may attempt to breathe through their involuntarily closed vocal cords. Breathing against a closed glottis causes negative intrathoracic pressure. This negative pressure may cause pulmonary edema. The edema worsens the hypoxia. The effects of pulmonary edema persist even after relief of the laryngeal spasm.

This type of pulmonary edema would be common in the "classic near drowning" in which the victim struggles and makes vigorous breathing efforts against a closed glottis. If the victim is not promptly rescued and treated, the pulmonary edema might cause serious damage.

The finding of "dry lungs" at autopsy has been used to support the theory of drowning without aspiration during laryngospasm. In most cases, however, a death associated with laryngospasm would result in wet lungs because of pulmonary edema.

Why, then, do writers continue to pontificate "dry drownings" or drownings from laryngospasm? Many times one person quotes another person who has misquoted a person who has misread an old article based on misleading experimentation (1).

There is no good experimental foundation to support drowning in laryngeal spasm. Many of the oldest works (subsequently quoted by following generations) are based on supposition, not experimentation. A fairly recent article supported "dry drownings" by referring to work done on weighing lungs of drowning victims (2). This project definitely did not provide a method to determine fatal laryngeal spasm.

Laryngeal spasm is a prominent factor in many near drownings. With prompt rescue the lungs may be protected from water. This would improve likelihood of a prompt recovery. However, pulmonary edema may have developed during the immersion. Efforts have been made to determine how many near drownings occur without water aspiration. These efforts based on blood gases and electrolyte measurements are not well founded (3).

Deaths during laryngospasm are not drownings. Laryngeal spasm may cause problems as well as be protective.

ROLE OF THE ABDOMINAL THRUST IN RESUSCITATION

The abdominal thrust (Heimlich maneuver) has become the accepted method for primary treatment of airway obstruction caused by foreign objects. Heimlich and his cohorts have proposed the maneuver's use as a first step in resuscitation of immersion victims. However, resuscitation guidelines from the American Heart Association and the American Red Cross advise use of this abdominal thrust only when other techniques fail (4).

Heimlich errs in believing that mechanical obstruction of the airway is common in near drowning. Almost always properly performed rescue breathing will be effective in inflating the lungs. Rarely is there mechanical obstruction (usually aspirated material in the throat) and the abdominal thrusts may be effective as a secondary technique for these.

The Heimlich maneuver is not benign. Using it delays rescue breathing and cardiac compression, and may worsen the hypoxic injury. Abdominal thrusting can force swallowed water from the stomach up into the throat. This water may then pass through the relaxed vocal cords into the lungs. Finally, the Heimlich maneuver may cause mechanical injury.

The scientific literature has established the limited value of the Heimlich maneuver in near drowning. The latest definitive report was published by the Institute of Medicine in 1995, which verified previous reports of the maneuver's drawbacks (5). Unfortunately, the recreational diving industry still sometimes touts the Heimlich maneuver.

COLD AND PROLONGED IMMERSION

Lundgren and Edmonds have previously discussed cold water in near drowning. Hypothermic reduction in metabolic rate has been considered as at least a partial explanation for survival after prolonged immersion.

Profound hypothermia does reduce oxygen consumption. However, mild or moderate cooling increases metabolism. Sterba and Lundgren (6) showed that on sudden immersion in cold water their subjects had a rise in metabolic rate. (6) This would impede breath holding and not be protective. It is quite likely that immersion in very cold water would be fatal before hypothermia could be achieved.

Scuba divers, and most other divers, use exposure suits. Core temperatures of recreational divers are usually stable. They may feel cold without a decrease in central temperature. If they nearly drown and are left in the water in their rubber suits, they will not get cold enough to be in a protective range before they die from hypoxia. For most divers, cold is more a problem than a protection.

This is true for most other aquatic activities. Hypothermia kills victims of boating accidents whose life vests keep them from drowning.

An early protective step in immersion is breath holding (voluntary and involuntary). Cold water stimulates breathing and makes breath holding difficult. Sterba and Lundgren (6) showed that despite a strong cardiovascular dive response, breath holding was not enhanced in cold water. Children have also been shown to have shortened breath holding when suddenly immersed in cold water. (7)

Cold water impairs breath holding. Cooling increases oxygen consumption. Hypothermia is dangerous. Scuba divers rarely become profoundly cold. These truths must be considered against the possible protective value of hypothermia in immersion accidents.

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DISCUSSION

Dr. Hamilton: You mentioned pulmonary edema from struggling to breathe against a closed glottis. It seems to me, I may be wrong about this, but that in autopsies of divers that have had their breathing gas shut off—had nothing to breathe—they find lung petechiae. Would this be the same sort of thing?

Dr. Dueker: Well, they are different things, but I would think that the mechanism would be similar.

Dr. Lundgren: I want to emphasize a little more what perhaps everybody is aware of. We do have a problem here when on the one hand we talk about what cold exposure does to the body and, on the other, what the diving re-

sponse does, in that the cold response has been studied in people who do not have the diving response elicited.

For the sake of discussion, I'd like to say that what we'd really need is information about what happens with the reaction to cold when you have profound hypoxia. That's not the case in the clinic.

Could you envision a different response to cold when, during a breath hold, you have a rapidly developing, severe hypoxia, a centralization of blood volume, et cetera, et cetera. I don't know the answer to that. And the other thing is, of course, that these long-term survivals have never, to the best of my knowledge, been observed in divers wearing a wet suit and only having a small area of skin exposed to the cold water. Long-term survivals all occur in people who fall in. They probably haven't even had time to fully develop a metabolic drive before they become unconscious. What happens then with the shivering? It really is a complex situation.

Dr. Dueker: Oh, it is very complicated and that's true, and that's what I'm wanting people to take away with them, that it is very complicated. It's not a simplistic thing.

But you're right about the scuba divers, and since we're emphasizing divers here rather than small children who are virtually naked when they go in the water, it's important to recognize that. The scuba diver is not going to have that cold insult.

Dr. Chimiak: I also agree there are complicated interactions between the pathologic processes and the physiologic events that lead to pulmonary edema. Your discussion of laryngospasm resulting from minimal amounts of water is indeed seen clinically as well as during aggressive pool and ascent-tower training.

Also, considerable intrapulmonary negative pressure is generated by young, healthy people challenged with an obstructed airway such as laryngospasm. Furthermore, pulmonary edema has been precipitated in clinical situations by laryngospasm. Pulmonary edema has also been seen in well-intentioned but misguided overhydration of immersed, healthy swimmers.

The combination of laryngospasm, coupled with increased central fluid volumes from immersion, breathing resistance imposed by the UBA, and perhaps elevated sympathetic stimulation may lead to significant pulmonary edema.

Dr. Neuman: Chris, only because you sort of encouraged this. A couple of disjointed comments. One, you're to be congratulated. This whole business of myths of drowning and near drowning has truly achieved lore, and some of the lore, of course, is the laryngospasm business and there are a variety of others. So let me bring up some more lore that might be useful if you decide to discuss.

The issue of how many angels dance on the head of a pin was not meant to be flip, but actually was a theological argument that took place in the middle ages all of the time. [*Editor's note*: Dr. Neuman had previously used this as an analogy for the significance of Dr. Deuker's insistence that cardiac deaths during laryngospasm are not drowning.]. And the reason I bring that point up is because for the scuba diver—and now I'm talking about the scuba diver—our interest in drowning and near drowning tends to be because we want to prevent these accidents from happening, and the problem with drowning for a scuba diver is that it is the final common pathway of a variety of things that happen in water.

And so the issue, if somebody has a seizure and drowns, is did he die of the drowning or did he die of the seizure? For the purpose of dive safety, you would probably say that you want to eliminate those folks who are likely to have seizures in the water to get to the bottom of your prevention and education kind of thing. And this goes along with a whole variety of things that cause near drowning or drowning in scuba divers.

The whole issue, then, of the pathologist who says well, he went into the water alive, he came out dead and he's wet, he drowned, obviates the entire problem of the confusion of aspiration with all of these other things.

Aside from the fact that you picked on orthopedists, let me pick on pathologists for a moment. Most of them have never seen somebody die. They are very used to seeing dead bodies and they probably have as much experience in seeing alive bodies as you and I do, but for those of us who have spent some time in the intensive care unit and in the emergency room and watched people die, we realize when they have a sudden cardiac death, for example, they breathe for 5 or 10 seconds after that death. Now this is going to confuse a pathologist because there's going to be water in the lungs under those circumstances, but what was the cause of death?

So these are things that we need to think about. And then finally, just as an amusement point for everybody in this room —to remind you of how careful you have to be with animal models. The supposition from Swan's work of course, when they dumped the fresh water in the animal's lungs, was that as the hemodilution and hemolysis took place it was the release of potassium—they did watch potassium go up and hence ventricular fibrillation and death. And the supposition, of course, was that the hemolysis caused the hyperkalemia. Please remember that the major intercellular cation in the dog erythrocyte is not potassium, but sodium. And so as a result of that—couldn't have happened that way. Undoubtedly hypoxemia, acido-

sis, all of those things were much more involved.

But again, you're to be congratulated on dealing with these myths. The quiet drownings are probably really medical underlying issues, and from our perspective, I think one of the things that we should all take away from this conversation is trying to look to the causes of drowning, as you said when you drew that pyramid.

Dr. Dueker: Thanks very much. If we can look more at the causes we can get more information about scuba incidents. I do think that will enhance the objective of prevention, which is definitely worthwhile.

Now I've got a real treat because as has been mentioned, and I'm sure most people here are aware, there's virtually nothing written specifically about scuba and near drowning and drowning. Carl Edmonds is going to present the work that he has been working on for quite a bit of time, trying to reduce that lack in our knowledge base.

DROWNING WITH SCUBA

Carl Edmonds

BACKGROUND

A normally functioning human, with adequate equipment in a congenial ocean environment, is protected from drowning as he carries his own personal life support with him–his own air supply. Drowning would only eventuate when there is diver error, failure of the equipment to supply air, or hazardous environmental influences. Nevertheless, the commonest ultimate cause of death in recreational scuba divers is drowning. Factual information that clarifies the causes and management could be of value in preventing further fatal outcomes.

Previous surveys (1–5) illustrated the importance of drowning as the ultimate cause in 74–82% of recreational scuba diving fatalities. It contrasted with the rarity of the more conventionally accepted diving diseases, decompression sickness and gas contamination, which accounted for less than 1% each. Those surveys also demonstrated the demographic similarity of the United States and Australia recreational diving deaths. This observation is understandable as they have a similar socioeconomic standing, are controlled by the same diver instructor organizations, and use the same diving equipment.

Of note in the more detailed surveys (3–5) was the high frequency of multiple contributing factors to each death. Drowning tended to obscure those preceding factors. The drowning sequelae and drowning pathology were the result of the environment in which the accident occurred, not the initiating or primary cause of death. For example, loss of consciousness when engaging in terrestrial activities is unlikely to cause death. It would do so more frequently if the victim was diving under water.

The three major manifestations of inhalation of water in the scuba diving situation are:

- Aspiration syndromes (causing symptoms and signs).
- Near drowning (producing unconsciousness).
- Drowning (causing death).

The aspiration of sea water causing clinical features in scuba divers, while retaining consciousness, has been dealt with previously (6). Sometimes this progresses into the other manifestations of near drowning and drowning (3,6). Specific attention to both the scuba drowning deaths and the "nearly drowned" have been poorly documented in the literature. Some texts on diving medicine hardly mention it. For this reason the following retrospective analyses were undertaken and compared.

SURVEYS

Drowning Survey (Fatalities)

The last 100 scuba deaths attributable to drowning in the Australian Dive Fatality Series (Project Stickybeak) were reviewed. The following criteria were required for admission:

- Compressed air diving equipment had to be worn by the victim in the water, with the intent of diving.
- Exclusion of all military, large commercial, or helium diving activities.
- At least three of the four following sources of detailed information:
  - A Coroner's Inquest of Inquiry (full transcript including witness declarations and cross-examination).
  - Autopsy findings (anatomy, histology, and toxicology in detail).
  - Official Government (Navy, Water Police, etc.) assessment of equipment functioning and in-water trials. This included gas analysis of scuba tank compressed air.
  - Detailed written accounts of witnesses (buddies, other divers, boatmen or bystanders, rescuers).

Cases complying with the above criteria and designated as drowning in Project Stickybeak were included. These mainly covered a period over the last decade, although it had to be extended to obtain some data from the preceding decade, to as far back as the early 1980s.

NEAR-DROWNINGS AND ASPIRATION SURVEY (SURVIVORS)

Diving organization, both national and international, which had previously promoted themselves as collecting diving accident questionnaires, were approached for assistance.

With one exception, they could give no information or supplied data with insufficient detail to be of any value. PADI (Australia) supplied valuable data on 12 divers, but too late to include in this presentation. It will be coded as part of an ongoing study of this condition.

And so a new survey was carried out, using various message boards and websites and organizations on the Internet, during the first 3 months of 1997. We guaranteed anonymity to the respondents, although the names of the respondents were given in all but one case.

A questionnaire entitled "Have you nearly drowned?"

was submitted to websites that were designed for divers. To increase compliance, the questionnaire was kept to two pages, but it was designed to supply most of the information we required. If the diver wished, there was the option of supplying more information.

Forty-eight replies were coded, but others were either not comprehensive enough to include or not pertinent. Only those that completed the questionnaire were used.

DEMOGRAPHICS

Population—Slide 1 (*see* end of paper for all slides)

The average age of the survivor group was slightly older than the fatalities, probably reflecting the fact that survivors, by definition, have added some time to their post-incident life span. Of the 100 fatalities, 89% were male and 11% female; of the 48 survivors, 52% were male and 48% female. During this period the female frequency among divers varied from 25 to 35% depending on the years involved and authority sought. Compared to the diving population at the time, it appears that males are over represented in the scuba drowning cases, as they are in almost all other forms of drowning (6). The surprise was that females appeared to be over represented in the "survivor" series. Whether females had more accidents, or whether they only reported them more frequently, could not be deduced. However, it does appear that the female accidents result in fewer deaths. The gender response on the Internet (survivor) survey also was a surprise. We had presumed, in common with most other marketing groups, that the Internet would be dominated by males.

PERSONAL FACTORS

Training—Slide 2

In the fatalities, a high 38% had either no known formal qualification. This group were approximately equally divided between:

a) those in whom the documentation was inadequate;
b) those without training, but who were experimenting with scuba under their own or their friends' cognizance;
c) those who were engaged in introductory dives, brief resort courses, or "dive experiences" with a recognized commercial organization.

A surprising number were under formal training at the time, comprising 8% of the fatalities and 15% of the survivors.

Experience—Slide 3

This did not entirely correlate with training. In both the fatality and survivor series, the divers were equally represented among inexperienced (<5 dives), novice divers (usually 5–20 dives), and experienced divers—one third each. Of the fatalities, over one half were experiencing diving situations to which they had not been previously exposed, whereas one third had previous experience of the conditions in which they died. The others were unable to be assessed. The buddy or dive leader appeared to be considerably more experienced than the diver in most cases, possibly explaining why the diver died and the buddy survived. The survivor questionnaires were not appropriate for us to compare the relative experience of the divers to undertake the eventful dive.

Victim's Behavior—Slide 4

In those fatalities that were observed (to achieve 100 cases we had to extend the survey beyond the last 100, because of the number of "solo" divers), over a third were noted to have either a panic response or rapid/abnormal movements. The survivors reported these sensations in over one half. The increased incidence of panic and rapid or abnormal movements in the surviving group could well be attributed to the fact that this is a reported sensation, whereas the fatality figure represented the observed behavior. Panic could well have been experienced but not necessarily observed in the latter group. Over half the fatalities showed no change in their behavior, with loss of consciousness (LOC) being the first objective warning in a third. LOC was the first manifestation noted in a quarter of the survivors. Of interest was the absence of panic in many of the cases, during the development of the incident, even though it is a frequent initiator of other diving deaths (1–3,6). Drowning scuba divers frequently drown quietly, possibly because of the effects of previous aspiration (hypoxia), depth (narcosis), or training ("don't panic", like the "drown proofing" babies). Of the total fatalities, 21% requested assistance in the form of an air supply. It was difficult to judge the success of this in many instances, because although they were sometimes reported as successful, the subsequent events would indicate otherwise. A request for a supplementary air supply was made by twice as many fatalities as survivors. This may bring into question the value of relying on a buddy responding to such a request. Alternatively, as we will see later, it is more frequent for the buddy to offer an emergency air supply, and this may be appreciated. Occasionally there was the apocryphal underwater tussle for a single regulator. When the low-on-air (LOA) diver went for an air supply, he more frequently sought the companion's primary reg, than the octopus.

Medical Disorders—Slide 5

This is a contentious area, not only regarding the incidence of medical disorders but also their significance. Authors differ in their assessments of this (3,6,7) and none

are free of prejudice. Project Stickybeak is the most comprehensive data base of diving fatalities available. It is compiled by Doug Walker, in Australia, and has been running for 25 years. Unfortunately, any medical history obtained from these records is inevitably an underestimate. In one analysis originally based on such data (3), when an attempt was made to search for and complete the medical history, in less than a half of the cases could this be achieved. Even then, it was often not up to date. A complete medical history, or even the routine diving medical history and examination forms, was not usually available. A statistical axiom is that absence of evidence is not evidence of absence. Questioning dead divers is not productive. Survivors, completing questionnaires, probably supply more accurate assessments. To avoid controversy, in this survey no attempt has been made to draw conclusions regarding the correlation between past illnesses and subsequent drowning.

ENVIRONMENTAL FACTORS

Water Conditions—Slide 6

The adverse influences of water conditions were expected. Probably the only surprise is the frequency with which drowning occurs in calm waters in over half the cases. Strong tidal currents were slightly more frequent in the fatality group.

Fresh / Sea Water—Slide 7

Most of the accidents occurred in the ocean without obvious differences between the fatality and survivor groups. The extra difficulty of performing rescues in cave diving is obvious.

Depth of Incident—Slide 8

As in previous surveys (1,3), over half the problems in the fatality cases were observed on the surface, although this frequently was related to the fact that the diver no longer had an adequate air supply to remain under water. In referring to depth, we are measuring the commencement of the aspiration/drowning incident, not necessarily the original problem. Thus a diver who had been diving excessively deep (related to their experience), used most of the air supply and then panicked and ascended, might then not show any evidence of aspiration until he reaches the surface. Approximately half the fatalities in this survey occurred while on the surface or on the way to the surface. Another 20% occurred in the first 9 m, and the rest were distributed over the remaining depths. This implies that just reaching the surface is not enough. Successful rescue then requires the victim to remain there. The survivors, probably because of the ability to supply a more detailed and specific history, indicated a greater incident depth. They probably more accurately represented the depth at which the incident developed, as opposed to the depth at which the incident was noted by others. Nevertheless, almost two thirds occurred in the top 10 m. In the fatality and the survivor groups, the dive was the deepest of their diving career in 26 and 33%, respectively. In almost half the "inexperienced" and "novice" divers the depth was beyond that which had previously been undertaken, suggesting that these groups are especially susceptible to the various problems associated with depth (panic, air consumption, visibility, narcosis, and logistic difficulty with rescue). This demonstrates that it is not so much the environment that is the problem, but the diver's experience of that environment. The danger of "diving deeper" without extra prudence and supervision is apparent. Any dive deeper than that previously experienced should be classified and treated as a "deep dive", irrespective of the actual depth.

Visibility—Slide 9

Visibility was usually acceptable, but seemed to be more frequently adverse in the fatalities, compared to the survivors.

Adverse Environments—Slide 10

The cases, in general, demonstrated the adverse effects of various environments, especially with tidal currents, white (rough) water, poor visibility, and deeper diving than previously experienced. There was not a great deal of difference between the two groups, except in the higher incidence of strong tidal currents, night diving, and cave diving in the fatalities. The figures, however, were small. If such observations are valid, then they may reflect either the effect on the victim or the problems with rescue, produced by such environments.

EQUIPMENT—**Slide 11**

In most fatalities the equipment showed no structural abnormality, and there were only significant or serious *faults* contributing to the fatality in 20%. This corresponded with the reported incidence by the survivors. Minor faults observed probably would not have contributed significantly to the deaths. Equipment faults were most frequently found with buoyancy compensators and regulators (both first and second stages). The incidence of equipment misuse was more frequent but more difficult to ascertain in the fatality series, and depends on one's definition. *Misuse* of equipment included the use of excessive weights or the failure to carry equipment that could have been instrumental in survival (e.g., buoyancy compensator, contents gauge, snorkel, etc.). Difficulties in using buoyancy compensators were also frequent. The incidence of equipment misuse would be much higher if one included a compromised air supply. Of equal interest was the failure to utilize equipment to ensure

buoyancy following the incident. These are dealt with under Diving Technique.

DIVING TECHNIQUE

Various diving techniques seemed to contribute to the drowning incidents, or influenced rescue and survival. They include a compromised air supply, buoyancy factors, buddy rescue, and resuscitation attempts.

Air Supply—Slide 12

In 60% of the fatalities either an out-of-air (OOA) or LOA situation had developed. There was insufficient air in the tank for either continuing the dive or returning to safety, underwater. In the survivors there were fewer incidents of compromised air supply, but it was still very frequent. The survivors tended to be more likely to have air in their tanks to cope with an emergency. The failure to use the available contents gauge, in both groups, is a source of concern, which could sometimes be attributed to the conditions placing other stress on the diver (depth, anxiety, tidal current, deepest dive ever, etc.). In many more cases there appeared to be a voluntary decision to dive until the tank was near reserve or "ran out". One surprising feature was the failure in both groups (8 and 13%) to turn on the tap of the scuba tank. Thus, even though there was plenty of air in the tank, it was unavailable other than to sometimes allow a rapid descent to 10 or so m. Only then was the diver aware that further air was not available. In none of these cases was there a buddy check of equip ment—breathing from it near the water surface and checking the equipment before descent. In a smaller number of cases there was a failure to adequately turn the tap back on, after previously checking the tank pressure, resulting in a partial restriction of the air supply—which became obvious only later in the dive or at depth.

BUOYANCY FACTORS

Buoyancy was frequently a vital factor in reaching the surface, and in remaining there as an unconscious diver, and being found, rescued, and resuscitated in time. The three major influences on this are the buoyancy compensators (BCs), weights, and the companion (buddy) diver practice.

Buoyancy Compensators (BC)—Slide 13

In the survivor group the BC was inflated by the victim or rescuer in twice as many cases as in the fatality group. This figure is even more relevant when the delay in producing buoyancy in the fatality group is considered (*see* below).

Weights—Slide 14

Although in 30% of the fatality cases the weights were ditched, in practice this was not as valuable as it sounds. In most of the instances in which the rescuer ditched the weights, the victim was probably no longer salvageable because of the delay (*see* below). The survivor group not only ditched the weights more frequently, but more often it was done by the victims themselves, and when it was done by the rescuer, it was usually performed early in the incident.

Buoyancy Action by Victim—Slide 15

Fatality and survivor groups seem to differ in that the latter divers did tend to perform an action that resulted in them achieving positive buoyancy during and following the incident. An interesting observation was made that when the victim and buddy were both in difficulty, usually based on a LOA/OOA situation, in the ensuing scenario, irrespective of whose problem developed first, the overweighted diver tended to be the one that died, and the buoyant diver the one that survived. The ratio of the two were 6 to 1, in the 14 instances. All this gives support to the current Instructor agencies emphasis on buoyancy training, although one could argue for its inclusion in the introductory courses more than the advanced courses.

COMPANION DIVER PRACTICE, RESCUE, AND RESUSCITATION

In most cases of significant aspiration of water, rescue depends on rapid action being undertaken by either the victim or the companion (buddy) diver. Once a diver gets into difficulty and is unable to carry out safety actions by himself, he is heavily reliant upon his buddy or dive leader. The fatality and survivor populations were so different in this respect, that we had to separate them for presentation.

FATALITIES

Buddy Experience—Slide 16

In the fatality group, less than half the victims had an experienced buddy available to assist them. When there was no buddy available, his experience was not applicable

Buddy Diver/Group Practice—Slide 17

In 21% of the fatalities, the dive was a solo one. In 38% the diver had separated from his buddy and in 12% from the group, before the serious incident. Thus the voluntary separation developed in 50% of the cases before the fatality occurred. It was initiated in 31% because the victim could not continue (usually due to a LOA situation). The victim then attempted to return alone, essentially making it a solo

dive in over half the total fatalities. The diver was separated from his buddy or the group during the actual incident, and often by the incident, in 21% of cases. But in almost half of these cases the separation was produced because the diver was following his buddy or the group. The others occurred during the "rescue". Thus, separation was elected voluntarily in 71% of cases, making early rescue and resuscitation improbable, by virtue of the diving practice. Another 9% were swimming behind their companion(s), and thus the victim was not visible to the "buddy" at the time of the incident, due to the diving practice. In fact, 80% of the victims did not have a genuine buddy by virtue of their elected diving practice. In less than 1 in 10 deaths was there a continued contact with the buddy or group during and following the incident. The fatalities seemed to flagrantly disregard the "buddy" system, as did their companions, the organization which conducted the dive, or the "dive leader". Group diving conferred little value because the "leader" often had insufficient contact with individual divers to be classified as a buddy, and the responsibility of others was not clear, especially toward the last of the "followers".

"Rescuer" Action—Slide 18

As the buddy system was essentially not used in the fatality group, the commonest response to the accident by the other divers was that no early attempt was made to rescue the victim. The second commonest response was that an attempt was made, but failed. This is understandable when one appreciates that no one knew where the victim actually was. A smaller number of rescuers actually found the victim and attempted a rescue, with some initial response by the victim. In a quarter of the cases there was no actual search for the victim until after the planned dive had been completed, and it was realized that the victim had not returned. In these cases there was a search for the body, which failed in most cases. In a number of the cases referred to in this section, it was not the original buddy or group diver that undertook the attempted rescue or search. Sometimes it was other divers who were coincidentally in the same area. In other instances, coincidental observers, from shore or boat, were aware of the victim's distress and undertook the rescue attempt.

Body Search—Slide 19

A formal search for the diver's body, undertaken separately and usually well after the dive, was successful in a third of the cases, with another third successful during future attempts. In a very small number the body was found coincidentally and in only 3% was it never found.

Resuscitation—Slide 20

Resuscitation was not a feasible option for most of these cases, who were obviously dead or showed no response to the rescuers attempts in 9 out of 10 cases. This is explained by the excessive delay in the rescue in most cases

Delay in Rescue—Slide 21

In only 20% was the diver rescued within 5 min of the probable incident time, and thereby having a real chance of successful resuscitation. In another 12% the diver was recovered within 6–15 min, and theoretically there was a slight chance of recovery with these divers had the rescue facilities been ideal and had fortune smiled brightly.

Autopsy—Slide 22

In 10% either the body was not available, or an adequate autopsy was not performed. In the remaining 90% the autopsies were either routine or conducted specifically by a diving pathologist.

SURVIVORS

In the surviving group most were rescued by their companion. Some form of artificial respiration or CPR was required in 29% of the cases. Oxygen was available and used, usually in a free-flow system, in 52% of cases.

Rescuers Behavior—Slide 23

No specific data are available on the buddy divers assisting the survivors, other than the subjective assessment as to whether the survivor believed the buddy to be of much value. The buddy was immediately available to the survivor in 71%, was considered to be of assistance in 58%, supplied an independent air source in 15%, inflated the BC in 25%, ditched the weight belt in 25%, and attempted buddy breathing in 4%. In 52% the diver surfaced under the buddy's control. The attitude toward buddy diving practice with the survival group appeared to be very different from those in the fatality group. The high figure for oxygen utilization must represent a more sophisticated and organized diving activity, which may also be related to a more conscientious attention to responsible buddy behavior. The axiom is that to successfully rescue an incapacitated diver one must know where he is and reach him quickly. This implies some form of buddy responsibility. Once reached, the buddy divers seemed to be of considerable value, implying good training in this aspect of diver safety. In recent years there has been a promotion of solo diving and reliance upon oneself, as compared to buddy diving practices. Denying or repudiating the hard-learned lessons of the past is fashionable and innova-

tive, implying a diving expertise and an avant garde approach, as well as ensuring an audience. In diving medicine it is also easier than acquiring practical experience or doing the hard data collection. In this instance, as in others, these "experts" may well be misleading both their contemporaries and the diving trainees.

CONCLUSION—Slide 24

There are many lessons to relearn from this survey, as well as from the diving medical experience of the past, to reduce the likelihood of drowning with scuba. They can be summarized as follows.

1. *Personal factors*: Ensure both medical and physical fitness, so that there is no increased likelihood of physical impairment or loss of consciousness, or difficulty in handling unexpected environmental stresses.

2. *Experience*: Ensure adequate experience of the likely dive conditions (dive under the supervision of a more experience diver, when extending your dive profile.).

3. *Equipment*: Although faults with diving equipment are inevitable, they are a less frequent contributor to drowning than misuse of equipment. The latter includes the practice of overweighting the diver, and his over reliance upon the buoyancy compensator. Failure to possess appropriate equipment is a danger, but not as much as the failure to use the equipment and thereby permitting a compromised air supply to develop.

4. *Environment*: Hazardous diving conditions should be avoided, using extreme caution with tidal currents, rough water, poor visibility, enclosed areas, and excessive depths.

5. *Neutral buoyancy (dive)*: Ensure neutral buoyancy while diving. This implies not being overweighted and thus not being too dependent on the buoyancy compensator.

6. *Air supply*: An inadequate supply of air for unexpected demands and emergencies may convert a problematical situation into a dangerous one. It also forces the diver to experience surface situations that are worrying and conducive to anxiety, fatigue, unpleasant decision making, and salt water aspiration. Equipment failure is not as common a cause of LOA/OOA as failure to use the contents gauge and/or a decision to breath the tank down to near reserve pressure.

7. *Buddy diving*: Use traditional buddy diving practice—two divers swimming together. Solo diving, for the whole or part of the dive, is much more likely to result in an unsatisfactory outcome in the event of diving problems. The divers who are committed to the traditional buddy diving practices are likely to survive the more serious of the drowning syndromes.

8. *Positive buoyancy (with problems)*: Positive buoyancy is frequently required if problems develop. Failure to remove the weight belt during a diving incident continues to be the major omission, and must reflect on training standards. In most situations, unbuckling and then ditching (if necessary) the weight belt is the most reliable course of action once a problem becomes evident. Buoyancy compensators cause problems in some emergency situations, and not infrequently will fail to provide the buoyancy required. They are of great value in many cases, but are not to be relied on.

9. *Buddy communication*: If feasible, inform the buddy before ascent. If correct buddy diving practice is being done, he will automatically accompany the injured or vulnerable diver.

10. *Rescue*: Employ the rescue, water retrievals, first aid facilities (including oxygen), and MEDEVAC systems that were planned before the dive.

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1 SCUBA POPULATIONS - DROWNING Syndromes

	FATALITIES	SURVIVORS
N=	100	48
• M/F	89 %, 11 %	52 %, 48 %
• AGE	32 (SD IO)	35 (SD 8.7)

2 TRAINING

	FATALITIES	SURVIVORS
• NIL	38 %*	4 %
• CURRENT	8 %	15 %
• COMPLETED	54 %	81%

* NIL, UNKNOWN OR BRIEF
(INTRODUCTORY OR RESORT COURSES)

3 EXPERIENCE

	FATALITIES	SURVIVORS
• NIL OR SLIGHT	37 %	31 %
• NOVICE	30 %	35 %
• YES	27 %	29 %
• VERY	6 %	4 %

re. THE DIVE
NO = 56%
YES = 32%

4 VICTIM'S BEHAVIOUR

	FATALITIES (OBSERVED)	SURVIVORS (REPORTED
• PANIC	21 %	27 %
• RAPID/ABNORMAL	16 %	31 %
• N.A.D.	63 %	42 %
LOC	33 %	25 %
AIR REQUEST	21 %	10 %

5 MEDICAL DISORDER

	FATALITIES*	SURVIVORS
• ASTHMA	10 %	19 %
• CVS	6 %	2 %
• DRUGS	10 %	8 %
• VERY UNFIT	5 %	4 %
• PANIC	7 %	8 %

*BUT OFTEN NOT QUESTIONED
*? SIGNIFICANCE

6 WATER CONDITIONS

	FATALITIES	SURVIVORS
• CALM	56 %	60 %
• DETERIORATED	4 %	
• MOD ROUGH	25 %	40 %
• VERY ROUGH	15 %	
CURRENT	46 %	31 %
STRONG	9 %	

7 SITE

	FATALITIES	SURVIVORS
• SEA WATER	93 %	98 %
–SHELTERED	9%	
• FRESH WATER	7 %	2 %
–CAVES	2%	

8 DEPTH OF INCIDENT

	FATALITIES	SURVIVORS
• SURFACE	51 %	15 %
+ ASCENT	-	17 %
• 1 - 9 M	20 %	33 %
• 10 - 18 M	10 %	15 %
• 19 - 30 M	10 %	13 %
• 31 - 45 M	3 %	6 %
• 46 - 60 M	6 %	2 %
• DEEPEST	26 %	33 %

9 VISIBILITY

	FATALITIES	SURVIVORS
• GOOD	51 %	
• FAIR	23 %	
• POOR	26 %	18 %
DETERIORATED	4%	
NIGHT	5 %	

10 ENVIRONMENTS

	FATALITIES	SURVIVORS
• CURRENTS	55 %	31 %
• ROUGH WHITE WATER	44 %	41 %
• VISIBILITY POOR	26 %	18 %
- NIGHT	5%	-
• COLD	14 %	12 %
• DEPTH > EXPERIENCE	26 %	33 %

11 EQUIPMENT

	FATALITIES	SURVIVORS
FAULTS MAJOR	20 %	18 %
MINOR	11 %	-
MISUSE	43 %	38 %
• OVERWEIGHTED	25 %	27 %
• OMITTED EQUIP	12 %	8 %

12 AIR SUPPLY

	FATALITIES	SURVIVORS
• OOA	49 %	27 %
• LOA	11 %	8 %
• 1/4 - 1/2 TANK	11 %	20 %
• > 1/2 TANK	29 %	45 %

13 B.C.

• NOT INFLATED	52 %	31 %
• FAILED TO INFLATE	5 %	8 %
• PREVIOUS INFLATION	12 %	
	= 69 %	=41 %
• VICTIM OPERATED	15 %	35 %
• BUDDY OPERATED	16 %	25 %
	= 31%	=60 %

14 WEIGHTS

	FATALITIES	SURVIVORS
• NOT WORN	1%	6%
• NOT DITCHED	66%	48%
• ENTANGLED	3%	2%
• VICTIM DITCHED	10%	19%
• RESCUER DITCHED	20%	25%

15 BUOYANCY ACTION (VICTIM)

	FATALITIES	SURVIVORS
• B.C. INFLATE	16 %	35 %
• WEIGHTS DITCHED	10 %	19 %

16 BUDDY EXPERIENCE

• NIL OR ?	12 %
• NOVICE	20 %
• EXPERIENCED	33 %
• INSTRUCTOR	8 %
• N/A	27 %

17 BUDDY/GROUP TECHNIQUE

SOLO DIVE	21 %
SEPARATED FROM	
BUDDY 38%, GROUP 12%	50 %
(SEPARATED AFTER A PROBLEM = 31 %)	
SEPARATED BY INCIDENT	21 %
BUDDY ASSIST THRO'OUT	8 %

18 "RESCUER" ACTION

• NO RESCUE ATTEMPT		- 31%
• ATTEMPT + FAILURE		
BUDDY/GROUP/OTHER		- 24%
• ATTEMPT + INITIAL SUCCESS		
BUDDY/GROUP/OTHER		- 17%
POST DIVE SEARCH		
SUCCESS	8%	
FAILURE	18%	26%

19 BODY SEARCH

- NOT REQUIRED 26 %
- SUCCESSFUL 34 %
- OTHERS SUCCESSFUL 30 %
- FOUND COINCIDENTALLY 7 %
- NEVER FOUND 3 %

20 RESUSCITATION - DROWNINGS

- NO, OBVIOUSLY DEAD 44 %
- FAILED TO RESPOND 47 %

TOTAL = 91 %

- RESPONDED INITIALLY 7 %
- INEFFECTUAL 2 %

21 RECOVERY DELAY

- < 5 MIN 20 % -REAL CHANCE
- 5 - 15 MIN 12 % - 32%?CHANCE
- 16 - 60 MIN 19 %
- HOURS 36 %
- DAYS 5 %
- WEEKS 5 %
- NEVER 3 %

22 AUTOPSY

- NO BODY 3 %
- AUTOPSY DENIED 4 %
- INADEQUATE AUTOPSY 3 % - 10 %
- ROUTINE 72 %
- DIVING AUTOPSY 18 %

INTERESTING OBS

HAEMORRHAGES - cerebral GI

23 BUDDY VALUE

IMMEDIATELY AVAILABLE	71 %
"OF ASSISTANCE"	58 %
DITCHED WEIGHTS	25 %
INFLATED B C	25 %
INDEPENDENT AIR SOURCE	15 %
ATTEMPT BUDDY BREATHING	4 %
SURFACE UNDER BUDDY CONTROL	52%

24 DROWNING PREVENTION

1. PERSONAL fitness
2. EXPERIENCE
3. EQUIPMENT
4. ENVIRONMENTS
5. BUOYANCY -dive neutral
6. AIR supply
7. BUDDY diving

DIVE

8. BUOYANCY - positive
   Unbuckle WTs - Inflate BC
9. INFORM buddy, if feasible.
10. RESCUE, First aid and Medevac

PROBLEM

DISCUSSION

Dr. Hamilton: That's great Carl, it was worth coming here just for that. One thing we have learned in the last 10 years, and I look at this from the point of decompression, is the introduction of the safety stop. In a lot of cases we teach divers to make a stop 10 to 20 feet below the surface for 3 minutes. If the truth be told, the benefit of this is not so much that it provides a decompression stop that prevents bubble formation, but that it forces the diver to have buoyancy control; and this is something I think we're finding is working very well. So that in a way is a little bit new—it's about 30 years old.

Dr. Edmonds: I agree fully with that and I also agree with the fact that it's being used for the wrong reason, but it's an excellent result.

Dr. Hattori: You know, we have a base of about one million divers in Monterey over 20 years that I gathered statistics on during 1970 and 1990, and your stats really bring out almost the same thing that I found. The only difference was the survivors and the fatalities were about even on the Monterey peninsula, but I think that's because the majority of dive sites are within a mile of the hospital or any of the fire department rescue units on the peninsula. Up until 1990, most of the diving was done off the shore, and not on a boat where it takes a half an hour to get somebody to shore.

But the rest of the statistics go along with everything that I found. And another thing, some of the things that were taught in a rescue courses were not folllowed. When two people—or one person—gets in trouble the buddy says okay, let's go to shore and he goes ahead of the diver in trouble, and then when he gets to shore he looks back and finds the diver floating in the water face down, unconscious.

And the buoyancy problem is the same thing. We had a brother and sister diving in the peninsula. The sister panicked and eventually drowned. But the brother forgot, dropped his weight belt but didn't drop hers; and then when she got washed back off a rock into the water and went under, he had dropped his belt so he couldn't get down to her. But overall your statistics are just like what I have.

Dr. Edmonds: Well, actually I've never met you before, but I'm delighted to do so because I've always been impressed with your data. However, as you were saying in the last case, that's one of the reasons why I think unbuckling the weight belt as opposed to dropping it, is the more valid procedure.

Dr. Hattori: Yes, you've got to get it away from the body. Last year I had a lady who we eventually found out had a coronary and died, but the buddy had an awful time keeping her above water, and he claimed he did loosen her weight belt, but he forgot one of the things that they teach in rescue is that you just don't unbuckle it, you take it away from the body and drop it. Yes, when they eventually got her up on the boat, they found the weighted belt caught on the strap that held the tank. All the patients that we've had—I've had 80 near drowning and drowning cases—the ones who lost consciousness, you can never get a story from them of the last 1 to 2 minutes before the incident. For some reason they have a blank memory of what happened. I don't know what the others found.

Dr. Edmonds: It's very likely hypoxia, isn't it? The other thing you were mentioning, about the two divers going back together. In Australia, we have managed to even make that an unsafe practice, and we do it by a thing we call victimization of the buddy, which means that one dive leader controls a number of divers. Of course, he doesn't control them, he just collects money from them. But what happens is that he tells them right at the start that they must let him know if they're low on air.

So they all go out and quite obviously the most inexperienced diver runs low on air first and the inexperienced diver is usually at the end of the queue. They've got to paddle right up in front to catch up with everyone, using up what little air is left to show the dive instructor their gauge. He looks at it. Yes, you see his mind, yes, yes, you're low on air. So you stay there, stay right where you are, and he goes on to everyone else in the group to find out what their air supply is like, until he finds some one else who's also low on air. He says, you two go back together, which is a guaranteed way of producing death. And this is the diving practice in one of our states called Queensland.

Mr. Huggins: We see just about exactly what you've shown, and one of my concerns has been the education and the training that the people have gotten moving into open water. Over the past 2 years we've seen at least three people who have ended up with embolism and near drowning because they had a mask flood. And I think focusing on something like this and recommending things to training agencies about getting people comfortable in the water, able to be in the water without the equipment, as well as working with their equipment, would be a major step to helping prevent this. And I don't know if the agencies would pick up any of that. I think comments along that line would help.

Dr. Edmonds: I personally have never been influential in affecting the agencies in any way.

Dr. Desola-Ala:. I would like to know to which time period corresponded to the data you have shown.

Dr. Edmonds: 1995 backward to about 1985. I can't get diving deaths for 1997 but expect to have all the information.

Dr. Desola-Ala: Have you the feeling that in the last years, taking into account the new operators, that buoyancy control may be easier. Suggestions like those of Bill Hamilton of making buoyancy controller stops at a few meters. Have these changed in the last years?

Dr. Edmonds: To my knowledge, most people have been using this for the last 5 to 10 years. I would like to agree with you and Bill Hamilton that the diving attitude and practice will change. I see no evidence of any change in the figures compared to the figures I presented 10 years ago as regards buoyancy control. No evidence at all. On the contrary, I find exactly the same problem, almost the same frequency. But remember, the old figures were done on recreational scuba deaths, not just drownings.

But the answer to your question is no, I haven't seen any change and I don't know that I'm going to see any changes, because I don't really believe that people can be taught buoyancy control in four dives before they get their C card. Two-thirds of the deaths occurred in the first 20 dives. So I don't really believe we're going to influence the overall numbers a great deal with that, but I'm fully in favor of this technique.

Dr. Hattori: Just a couple of additional things. I've had no survivors who came in on complete CPR to the ER. And also, I have no survivors who came in with blood pH of less that 6.9. And I think Modell said that if you don't regain consciousness within 72 hours, that you probably never will, and that has been my case also.

Dr. Dueker: Tak, we're going to discuss that a little bit later. Now, I would like the people from this morning who were presenters to sit at the table and we'll talk a little bit among ourselves and then questions from the floor will certainly be welcomed on what we talk about.

MORNING DISCUSSION

Dr. Dueker: I'm going to take the prerogative that I've got because I'm standing here, and ask Dr. Lundgren, about the UHMS workshop that he chaired on breath holding. Maybe it's temporal, because it was several years ago, but there was not a conclusion that the diving response was oxygen conserving in humans. And it seemed to me that what you were presenting today implied, at least, that the diving response indeed was protective. What's changed?

Dr. Lundgren: Well, what has changed is that the observations in these expert elite breath-hold divers certainly point to a conservation of oxygen, and it does so because, as you saw, there is a shift—there is a substantial lactate formation and there is also the preservation of remarkably high Po_2 for a very long period of time. I have knowledge as a reviewer of some recent manuscripts, that there is shortly to be published measurements of breath-holding times showing indeed that when the diving response is elicited there is a gain in breath-holding time.

Now, the gain in voluntary breath-holding time certainly was something we were looking for in those experiments where we had subjects submersed in the cool water. We didn't see it, and as I mentioned that might have been because we had them sitting for some period of time in the cool water before we could do the measurements. Then they had this increased metabolism that we talked about. It could be that when the exposure to the cold water is briefer the diving response dominates over the boost in metabolism, or rather the boost in metabolism hasn't had time to be fully developed.

So to answer your question, I think there is accumulating evidence that the dive response does what we all expect it to do. Whether it does so in a quantitatively significant way when it comes to the near drownings is a different question all together, but my bias would be toward thinking that it probably does so.

Dr. Dueker: Well, we're going to discuss this a little bit later this afternoon, but I think it's important because up until this time, although it's been postulated that the diving response is protective, the evidence was really not there. And when you looked at the good work that had been done, it didn't support protection. Even your breath holding in cold water experiments tended not to support it. And I think it's very interesting today that we're getting from somebody who knows what's going on, evidence that maybe there is protection.

Now, again, the significance of it, I think, is a different issue. I'd like to save it until this afternoon and incorporate it into what I'm going to talk about on expectations in near drowning and prolonged immersion. I'm not sure that we'll get an explanation for those rare cases. For those people who aren't going to be here this afternoon, in those rare cases of prolonged survival, you can pretty much—even if

you take the anecdotes from your Aunt May—count them on three hands, anyway, if not two. And the number of people who are submerged and don't do well is certainly predominant. But I really thank you for presenting this, and I hope everybody appreciates what they have heard, because it's a very significant contribution.

Dr. Lundgren: If I may add only one other comment, there is some information in the literature indicating that there is quite a wide spread in the population, in terms of ability to develop the diving response. I was showing you results in these elite divers who certainly have a very well-developed response, whether due to training or genetics is not clear, but in a larger population, if I recall correctly, it is claimed that a real good diving response is seen in 10% of the population.

Now you take that distribution and look for a diving response that would be saving in a drowning incident, then you end up with very few persons who are candidates to be long survivors.

Dr. Dueker: Yes, that might be a factor in it. That's a very good point.

Dr. Edmonds: Just before the audience comes in and to show that we haven't connived, I have difficulty in accepting your rejection of the hypothermic result. Now, I'm not saying that hypothermia does protect, but what I am saying is because I don't know, but I do know that of the near drowning cases that we've had, many of them have quite low temperatures. These have been diving not in very cold water, about 15°C. They have been diving for quite a long time and they're reasonably protected, but they come in with bdy temperatures somewhere between 32° and 35°C.

I think what you've said infers that they're not going to drop their temperature, and I think they are going to drop their temperature because I know they do. But I don't know whether it's of value or not.

Dr. Dueker: Well, let me just share that 32 to 35, I would maintain, is not clinically very protective. The surgical consideration for hypothermia—for doing, for example, cardiac surgery—is quite a bit lower than that, and this is not arrest surgery. Almost all the open heart surgery that's done now is done with cardiopulmonary bypass—even on little kids. The old way of getting a small child really cold, stopping their heart—usually it would stop in the cold anyway—and then operating on them with a limited period of time is not used, at least in the United States, very much any more, largely because it didn't work very well.

But in the range, certainly 35 is not going to be a protective one, and 32, I'd be pretty doubtful.

Dr. Edmonds: I'm not questioning that because I don't know the answer to that, but when they do come in they have these low temperatures. The inference was that they're still shivering, but by the time we see them, they're not shivering. They often start shivering during the treatment as the temperature comes back, but at the time we see them, they really aren't shivering any more. They're well beyond that.

Dr. Dueker: Well, that's right about the shivering, it will certainly come back. I think that the amount of time that it takes to get—in a diver—even down to that temperature, which I question the protective value of, would be a little bit long.

Dr. Lundgren: I didn't bring up the question that I wanted to ask, and maybe Carl or somebody else has an answer to that. Do you know of any cases of near drowning in divers where there is definitive information about the time that the diver has been lying on the bottom, say, with the mouthpiece out? I'm willing to bet that it doesn't come anywhere near these long survivors that we have in the more classical drowning cases.

Dr. Edmonds: I'm absolutely sure you're right and I'm also sure that if we could get any reports of those divers, they would be like a series of one to maybe two. That's one of the big defects in our knowledge in the diving area.

Dr. Dueker: Just for those who won't be here this afternoon or who are going to be asleep when I'm talking—if you take a series of cases that are brought to emergency rooms, in most cases temperature is an inverse relation factor with survival. In other words, the coldest victims generally have the least good survival. That usually just means that they were in the water a lot longer.

But if you were to try to correlate being cold with that being good, you certainly could not do it on the series of cases that are reported..

Dr. Hattori: I agree with Dr. Edmonds' finding that the majority of the near drowning cases that I've got, the rectal temperature always was around 90° to 92°F when they came in.

Mr. Graver: In response to Dr. Lundgren who was asking about survival times of people who had been under water for quite a while. I've been tracking that to the best of my ability for many years. The longest time of submersion that I'm aware of that's documented for a scuba diver who has been unconscious and under water and has lived, is 10 minutes. That case occurred at Catalina. Ron Ryan was in charge of supervising the recovery.

Dr. Lundgren: Can I ask you what was the depth that the diver reached?

Mr. Graver: I'm guessing now, but I think it was in the

order of 70 to 75 feet. And the person lost consciousness at the bottom, not at the surface.

Dr. Lundgren: Which is of course very significant because that means that he had roughly three times as large a lung oxygen store as if he'd been at the surface.

Mr. Graver: Correct.

Dr. Dueker: Carl mentioned that earlier as a possible explanation in diving, and I'm glad you mentioned it again. The water in Catalina is not very cold.

Dr. Harvey: Do you feel there is a role for cardiac dysrhythmias as a result of sudden cold exposure, as opposed to a core temperature drop and its effects?

Dr. Dueker: I'll answer it and maybe we'll have some other answers too. There is some nice work on sudden chilling that includes people who died in cold showers after they had gotten out of swimming pools. That was a part of the work that was done by Rawlins. And I think that those would almost have to be that kind of thing. He did not feel that that was the usual explanation for sudden death in cold water immersion. There is a school of thought that still believes that's the case, and it was a more popular thought until it was looked into more thoroughly.

I think most people now feel that at least in immersion, that it's gasping, respiratory distress water-aspiration type of death, rather than a dysrhythmia, but I'm sure they occur.

Dr. **Lundgren:** I can offer a bit of a comment to that. One is that in immersion, say, at the surface, head out, the redistribution of blood, as I referred to earlier is quite marked. We've measured with dye dilution technique up toward a liter of blood being forced into the chest. You have a profound distention of the heart and increase in preload—and distention of the heart is arrhythmogenic by itself. And the other thing is that with the diving response we have seen amazing levels of arrhythmia in these healthy people. One of our subjects had a period of 45 seconds without a single sinus peak.

Dr. Dueker: In your breath-holding workshop report, though, there are some other EEGs that were actually measured during the dive, and there were some very upsetting dysrhythmias. One of my senior middle-aged partners took an old- fashioned scuba course a few years ago where you had to do skin diving instead of going right to 300 feet on a scuba. He called me one morning and said that he had done the skin diving in the pool and had gone into atrial fibrillation. He was a cardiologist, he knew what it was. He digitalized himself that night and called me in the morning; asked me what I thought he should do. I suggested he could keep up with his skiing and that's what he elected to do.

Dr. Wong: I have a question about dysrhythmias. Have you any recorded cases with postmortem findings which show patients with coronary artery disease? What percentage showed coronary artery disease?

Dr. Edmonds: Bob, what group are you asking about?

Dr. Wong: Patients suspected to having died of dysrhythmias, and whether the deceased had postmortem examinations performed on them and, if so, what were the findings of their coronary arteries?

Dr. Dueker: There are a couple of small papers in the literature that have looked at people with known coronary disease to see how they react to breath holding, and they had more dysrhythmias. That's not the answer to your question, but I don't think there is one. I talked to Dr. Caruso, the pathologist who is working with DAN now, and he couldn't offer that kind of correlation. He admitted that if they didn't have coronary disease and had a fatal dysrhythmia, you may well not find anything on autopsy.

Dr. Edmonds: Now, hold on. About half the cases that have been presumed to have died of cardiac disease in the water have very obvious evidence of almost expected myocardial infarction. In other words, you're going to have a coronary artery of greater than 90% obstructed or previous evidence of an infarct. The other half have obvious reasons for arrhythmias. A couple of years ago I looked into the cardiac deaths with exercise and there is a wonderful monograph on it. The author did a wonderful job of assessing each one of the cases. There wasn't one case that wasn't sort of a fairly obvious cardiac reason for the death. Now, that was in exercise in general.

But in the diving cases, you might remember about 10 years ago, about 13% of the deaths were cardiac deaths. However, there have been quite a few deaths since then. DAN has got quite a few and I'm surprised that DAN has got no answers here about autopsies.

Dr. Dueker: Well, no, DAN itself does not do autopsies, but their autopsy rate for their reports is going up steadily. The autopsy rate right now for the ones that DAN reports is pretty good. And the cardiac death rate for divers— recreational divers—is very high. If somebody has a myocardial infarction and it's a fatal one, then I don't want to call that a drowning, and Dr. Caruso doesn't either.

Dr. Lundgren: Nor should we.

Unidentified: I've noticed too in the last 10 years that more of our fatalities are showing up because of, I think, cardiac disease, and I think a lot of these—when we do a post and we see at least one of the coronaries narrowed down to 80 to 90% and these people probably on land —you know, I've had two by-passes, so I'm talking from experience—if you're 80 to 90%, you can go up a flight of stairs running up and not have any symptoms. But I think

if you get stressed long enough, as in the water, that you may have arrhythmia that causes the drowning.

Because in the cases that we've done the autopsies on, there have been 80 to 90% narrowing and we have no evidence of an infarct or clots or anything, so we kind of arbitrarily assigned it to probable arrhythmia, followed by drowning.

Dr. Neuman: Clearly when somebody has myocardial infarction I don't think anybody's going to argue that they're not a drowning. The problem is that there is no pathologic evidence for a fresh myocardial infarction, microscopically. If you remember back to the mid-1950s, the pathologists kept on telling us that myocardial infarctions were not caused by clots in the coronary arteries because by the time they looked at dead people there was no clot there.

So as Dr. Caruso says, the problem is that in these people who die sudden arrhythmic deaths, there is no way of telling what has happened to them, and you can only look for sort of corollary evidence that they may have a risk factor for a sudden arrhythmic death. The trouble is the vast majority of pathologists, if they've got somebody with heavy lungs and they've got somebody with an enlarged heart, if he dies on land —heavy lungs notwithstanding, because they're going to have the same heavy lungs on land as in the water because of CPR and a variety of other things—so if they die on the land they call them a sudden arrhythmic death, and if they die in the water, they call them a drowning.

And so this is one of the epidemiologic problems that you have to face, that we simply are going to have to accept the fact that it may not be possible to tell.

Dr. Dueker: I think that's absolutely beyond doubt, and if some one has an arrhythmic death, which certainly can occur, without coronary calcifications, but even with coronary problems, the pathologist isn't going to be able to help.

Attendee: And call it a drowning.

Dr. Dueker: No, if it's in the water, it's going to be a drowning, and it's not just that it's sort of idle curiosity or a desire to get things right; some of it can be therapeutically important. Certainly diving physicians know that if you have an unconscious person after scuba diving, there are several things that could be going on, and treating one of those and not looking at the others may give you less than a great result. You have to decide what you have to do. If the heart stops, you obviously start there.

If you had a massive air embolism and a drowning or a near drowning, both of those would have to be addressed, and the same thing could easily happen with a heart attack. If a person has a non-fatal heart attack in the water and nearly drowns, you've got both of those problems to treat. I'm hoping that Steve Brown will get into that a little bit when he talks about hospital care, because once you get the emergency taken care of, you need to consider what else is going on with the person. They may need some help other than just for near drowning.

Dr. Edmonds: Yes, again, I can come back to what I said before, there's no question that there is a sudden death syndrome in divers. But why should we confuse this workshop with that?

Dr. Dueker: You're absolutely right, and the reality is that probably most of the scuba deaths that occur are drownings and so it's a little bit hard to just say, well, this clearly isn't. We've got to work at it. I couldn't be more enthusiastic about your point of view.

Dr. Edmonds: Yes, let's not make the same mistake as Swan.

AFTERNOON SESSION

Dr. Brown: Welcome to the second half of this workshop on near drowning. The morning has been devoted to the theoretical or pathophysiological mechanisms of near drowning, and some of the epidemiology of circumstances and victims.

The second half of the day is devoted more toward the management, both on site—which is the appropriate place to start—and different circumstances or problems you may encounter in the course of near drowning. After that we'll discuss the top of that pyramid, the intensive care unit, to which, fortunately, relatively few near drowning victims come, but where a few finally get their care. Finally, Chris will sum up the session with the expectations of recovery.

So I'd like to introduce Dennis Graver to discuss open water rescues and field resuscitation.

NEAR DROWNING: OPEN WATER RESCUES AND FIELD RESUSCITATION

Dennis K. Graver

Scuba divers may become incapacitated and lose consciousness for many reasons. Loss of consciousness in water frequently causes drowning or near drowning. Many factors affect the survival chances of a near-drown scuba diver. The purposes of this paper are to trace the history of rescue and resuscitation techniques, identify the complicating factors of scuba diving near drowning, and present the latest information concerning the rescue and resuscitation techniques for nearly drown scuba divers.

HISTORY

Rescue procedures change—as well they should—with time. It was suggested in 1970 (1) that surfacing unconscious divers in an inverted position would prevent embolism and maintain a clear airway. In 1974, Dr. George Harper (2) explained why unconscious divers do not embolize when brought to the surface. Regrettably, until the 1980s, publications and instructors taught that rescuers must tilt the head of an unconscious back to allow expanding air to escape during a rescue ascent. What is even more unfortunate is that many contemporary divers and dive leaders continue to accept this theory and use the technique (personal knowledge from interviews and accident report reviews). The PADI Rescue Diver Manual (3) instructed rescuers that "the victim should be held near the head and maintained in an upright, normal anatomical position throughout the ascent." As we shall see later in this paper, there is a better position for unconscious divers.

Resuscitation procedures also change with time. As the American Heart Association (AHA) Standards and Guidelines for CPR and Emergency Cardiac Care change, so have rescue procedures for scuba diving. The dive community faithfully adhered to the AHA Standards for years until it became apparent that the rescue requirements for scuba diving accidents differed significantly from those for a swimming accident. The first documented approval (4) for deviation from AHA Guidelines occurred as a result of a 1991 Near Drowning Workshop held in San Diego, CA. The workshop was sponsored by the Council for National Cooperation in Aquatics (CNCA), the National Association of Underwater Instructors (NAUI), and Divers Alert Network (DAN) and supported by the Undersea and Hyperbaric Medical Society (UHMS). One example of a different resuscitation technique than that suggested by AHA Guidelines is the resuscitation rate. At the near-drowning workshop Butch Hendricks, owner of Lifeguard Systems, proposed two breaths every 10 seconds for in-water rescue breathing. His proposal was unanimously supported by all attendees of the workshop and has since been published in the NAUI Scuba Rescue Diver Manual (5).

Another example of changing resuscitation philosophy is use of the Heimlich maneuver. Dr. Heimlich has long recommended the use of his procedure for near-drown victims, but Rinke (6) and Shaw (7) state that the procedure is a waste of valuable time, may cause regurgitation, emesis, and aspiration, and should be employed only if the rescuer is unable to ventilate the victim and a foreign body is suspected. The physicians attending the 1991 near-drowning conference in San Diego agreed unanimously with the recommendations of Rinke and Shaw. The issue seemed clearly resolved, but support for the Heimlich procedure for near-drownings was recently resurrected by Sugden (8).

In-water resuscitation techniques have improved during the past 30 years. Warren Glaser developed the "do-si-do" rescue breathing technique in 1969. The procedure became and remained widely accepted. In April 1981, I discovered that the Laerdal Pocket Mask was an extremely effective for aquatic rescue breathing. My recommendations concerning the Pocket Mask were published in 1984 (9). A 1991 letter to Jim Brown of the NAUI USA Training Department from Hans Telford of the NAUI Australia Training Department stated that with the do-si-do technique, "the jaw does fall back, which unfortunately has a tendency to allow ventilated air to go to the stomach rather than to the lungs." His letter prompted me to research the technique. I confirmed his concern and wrote an article comparing rescue breathing techniques. That article (10) recommends mouth-to-mask rescue breathing (centerline position) as excellent for maintaining an optimum airway. In my opinion, ventilation via a pocket mask remains today's preferred method of aquatic rescue breathing (11).

The 1992 American Heart Association/JAMA Guidelines for CPR (12) state that "Chest compressions should not be attempted in the water unless the rescuer has had special training in techniques of in-water CPR . . .." To the

best of my knowledge, there is not effective means of administering CPR in deep water. Participants in the 1991 CNCA Near Drowning Workshop in San Diego, California, were in agreement that "in-water CPR is ineffective at best." A rescuer cannot detect a pulse while in the water. The PADI Rescue Diver Manual (13) states that "A person apparently requiring external cardiac massage must be removed from the water because highly specialized training and equipment are required to administer CPR effectively in the water. Because of the extreme difficulty in detecting a pulse while in the water, however, the person should be ventilated during transport in case the heart has not stopped." The following statement concerning in-water CPR is included in my book (14), which was reviewed by experts: "You probably will not be able to detect a pulse in the water, so don't bother trying. You cannot administer CPR in the water; you must remove the victim from the water, and position him or her on a firm surface."

Vertical lifts and carries have been common rescue extrication techniques for many years. Surgeon Rear-Admiral Golden, of the Royal Naval Hospital in England, published an excellent paper (15) explaining that it is essential to maintain a horizontal position throughout rescue procedures. Golden's recommendations influenced the rescue exit techniques contained in my book (16).

For years the Trendelenberg position was an automatic first aid component for an injured scuba diver. The value of the technique is questionable. The Divers Alert Network (17) and NAUI (18,19) modified use of the procedure. DAN says that "a brief (less than 10 minutes) head-down position might conceivably facilitate the clearance of bubbles from the cerebral circulation after AGE, it seem prudent thereafter to keep the patient in the supine position rather than in prolonged head-down position." NAUI's publication says that "Supine is fine."

The survival chances of nearly-drown, submerged scuba divers are poor. The majority of divers believe that nearly-drown scuba victims have a good chance of living because of Dr. Martin Nemiroff's 1978 Cold Water Near Drowning information published by the U. S. Coast Guard (20). As Dr. Christopher Deuker has pointed out during several presentations, the cold water response applies to young people who are cooled rapidly, not to adult scuba divers who wear insulating exposure suits.

COMPLICATING FACTORS

Many factors compound the rescue of an injured scuba diver, who may suffer trauma, barotrauma, hypothermia, and near drowning simultaneously. Deep water and long distances to shore compound rescue procedures. Surface chop makes it difficult to maintain an open and dry airway during rescue breathing. Remote locations may make rapid EMS response impossible. Few rescuers are equipped with suction devices to protect a victim's airway—an important first aid component for the nearly drown.

The aftereffects of alcohol use, which average 38% amongst diving accident victims (21:32–33), alter physiology and hamper rescue efforts. Heart dysrhythmias and acute pulmonary edema (more later) are complicating factors that are not well understood and which hamper rescue efforts substantially. Inappropriate and incomplete autopsies of deceased scuba divers fail to provide valuable information that could help provide greater insight into the causes of scuba accidents.

A scuba diver who loses consciousness at depth, nearly drowns, and is rescued promptly stands a better chance of surviving than a diver who loses consciousness during ascent or at the surface, sinks, and is recovered promptly. Loss of consciousness usually stems from hypoxia. Sinking increases partial pressure, which consumes remaining oxygen. Bringing the victim back to the surface lowers oxygen partial pressure and makes the victim extremely hypoxic and far less revivable than if the diver had not sunk. Unfortunately, more than 42% of fatal scuba accidents occur while ascending or at the surface after a dive (21:73). An additional 16.5% of fatal scuba accidents are unobserved and undoubtedly increase the percentage of accidents that occur at or near the surface.

CURRENT RESCUE AND RESUSCITATION RECOMMENDATIONS

If a distressed diver is at the surface, do everything possible to support the diver before loss of consciousness and sinking occur. Distressed divers are seldom buoyant and sink quickly after loss of consciousness. The first rescue priority for a distressed diver at the surface is to provide buoyancy. Rescue diver courses teach various buoyancy-establishing techniques. All divers should complete a Rescue Diver course.

Divers also need to become aware of the Instinctive Drowning Response, which was discovered by Frank Pia, a lifeguard for 20 years at Orchard Beach, NY. As he points out in his film (22), everyone resorts to instinctive actions when about to drown. Victims are incapable of communication, grasping objects thrown or extended to them, or assisting themselves in any way. The only way to establish buoyancy for panicked people exhibiting the instinctive drowning response is to place support beneath their outstretched arms or to make contact with them and physically support them or make them buoyant.

When a rescuer encounters or locates an unconscious scuba diver underwater and confirms that the victim is

unresponsive, establishing buoyancy for the victim is again a priority. The rescuer should pause briefly before establishing buoyancy and look at the victim's face to determine if the regulator is in the mouth and if there is water inside the victim's mask. If there is water in the mask, it should be removed because air expanding inside the mask during ascent will force the water into the victim. If the regulator is in the victim's mouth, it should be held in place. If the regulator is not in the victim's mouth, it should not be replaced. If the victim is in a face down position, the rescuer should maintain the victim in that position while establishing buoyancy to help keep water from entering the victim's mouth and airway. Rescue diver courses teach various buoyancy-establishing techniques. It is important to emphasize that when a rescuer removes the victim's weight belt, the rescuer must pull the belt completely clear of the victim before discarding it. Exercises during numerous rescue courses and workshops have shown that if the weight belt can catch on anything and remain in contact with the victim, it more than likely will. I reiterate that all divers should complete a Rescue Diver course.

Because of airway positioning and possible circulatory bubbles from decompression, the next priority should be to maintain the victim in a face down, horizontal position. Testing at rescue workshops, rescue courses, and Instructor Training Courses has shown that a rescuer can find a balance point on a victim's scuba tank and control the victim's position with one hand while using the other hand for buoyancy control during ascent. An additional advantage of the horizontal rescue ascent is that it increases the victim's cross-sectional area and allows the rescuer better control of the ascent rate.

The next priority for the rescue of an unconscious, submerged diver is the rate of ascent. It is important to get the victim to the surface as fast as is safely possible. When the depth is shallow and the risk of decompression sickness is negligible, the only concern for the rescuer is avoiding arterial gas embolism. But when decompression illness is a concern, the rescuer must limit the rate of ascent to avoid injury. When a rapid rescue ascent is inappropriate, the rescuer may choose to send the victim to the surface unaccompanied and then follow at a reasonable ascent rate. Other divers at the surface may see the victim surface and provide aid until the rescuer arrives. Rescuers are not required to jeopardize their safety to save someone and should not risk injury unduly while attempting to rescue a submerged diver.

Some dive accidents occur between the surface and the bottom. A victim loses consciousness and sinks. A buddy attempts to support the unconscious victim by swimming efforts, establishing personal buoyancy, or trying to inflate the victim's buoyancy compensator. These efforts are usually unsuccessful and the rescuer winds up releasing the victim, surfacing in a panic, and feeling tremendous guilt for the inability to rescue the unconscious victim. Rescue training needs to include the appropriate response. Immediately upon loss of consciousness and the initiation of sinking, a rescuer should establish contact and pull himself or herself down toward the victim far enough to be able to grasp, pull clear, and release the victim's weights. The instant that this task is accomplished, sinking will cease. The rescuer should then rotate the victim into the face down position, hold the regulator if it is in the victim's mouth, and take or send the diver to the surface.

Panicked divers struggling at the surface must be assisted quickly. According to Smith (23), "The terminal, silent surface struggle normally takes no more than a minute." It is essential to provide physical support for the victim as quickly as possible. A rescue-trained buddy who can recognize the difficulty and establish buoyancy for the distressed diver is the best solution to the dilemma. Unfortunately, a buddy may not be present. Solo diving and buddy separation leave divers without immediate assistance when panic occurs. A similar problem occurs when people dive in groups and do not have specific buddy assignments. Sixty-two percent of the 1994 diving fatalities occurred in dive parties with three or more divers (24).

A rescuer should avoid a face-to-face encounter with a panicked diver. A trained rescuer should, if possible, establish buoyancy by releasing weights after approaching the victim beneath the surface at a depth eye level with the victim's waist. An alternate surface technique for assisting a panicked diver involves grasping the top of the victim's wrist and pulling the victim forward while rotating the victim so that he or she faces away from the rescuer. This maneuver positions the rescuer behind the victim—a safer position than a frontal one—and allows the rescuer to control the victim by holding the victim's scuba tank valve. While one hand controls the victim, the rescuer may use the other hand to establish buoyancy.

The instant that a rescuer has an unconscious victim at the surface and buoyant, the next priorities are to summon assistance and EMS, have others account for the victim's buddy immediately, and maintain the victim's airway and breathing. Trained rescuers are aware that an additional diver may need to be rescued immediately, "fix" the position where the victim was recovered, and deploy a team of rescue-trained divers to locate the victim's buddy if that person cannot be located immediately. Other trained rescue divers on scene will summon EMS, assist the rescuer, prepare first aid equipment, and prepare for the rescue exit. The need for advance rescue coordination during dive

operations is obvious. A written emergency action plan, advance coordination, trained responders, emergency equipment, emergency communications, early problem detection, effective rescues, proper first aid, evacuation, and accident management are essential for the survival of seriously injured scuba divers. Regrettably, one or more of these elements are usually missing when a fatality occurs. Divers need to be taught that discussion of an emergency action plan is an essential part of every dive briefing and must involve more than telling the divers to let the divemaster know of any problems. People with special training, such as doctors, nurses, paramedics, emergency medical technicians, first responders, scuba instructors and divemasters, and rescue divers should be identified and their role in an emergency situation should be coordinated before an emergency occurs (16:36).

To determine if a victim is breathing, the rescuer must first remove the mask from the victim if the mask is still in place. In calm water, the rescuer should also remove his or her mask because rescue breathing and swimming are easier when the rescuer can breathe through both the nose and the mouth.

The next rescue step is to open and maintain the airway. Maintaining an airway for an unconscious scuba diver in the water is not an easy task. The skill requires training and practice. Even Instructors, who are critically evaluated for rescue ability, often forget to maintain the correct head position to ensure a proper airway (personal experience evaluating instructors since 1970). Unless special circumstances—such as a fall or being struck by a passing boat—suggest otherwise, cervical-spinal immobilization will not be required for unconscious scuba divers. While Director of Training at PADI, I conducted a survey to determine if any PADI members were aware of any dive accident in which a diver had suffered a neck or back injury while in the water. Not a single incident was reported, nor have I been able to substantiate any since.

While C-spine stabilization usually is not required for scuba rescues, use of the jaw-thrust or a chin-lift maneuver to open and maintain the airway is desirable because the maneuvers provide benefits. Chin-lift from a position alongside a victim or use of a pocket mask from a position in front of the victim require buoyancy to place the rescuer above the victim (10). While maintaining the airway the rescuer should look, listen, and feel for breathing. Removal of the rescuer's weights after the victim is buoyant helps provide lift to get the rescuer above the victim to check breathing and provide ventilation, if needed.

If the victim is not breathing, the rescuer should attempt two rescue breaths initially. If the breaths do not go in, the rescuer should retilt the victim's head and attempt additional breaths. If the breaths still do not go in, the rescuer should position himself beneath the victim, give five abdominal thrusts, sweep the victim's mouth, and reattempt rescue breathing. The procedure should be repeated until the breaths go in, the rescuer is exhausted, or qualified personnel relieve the rescuer.

When rescue breathing is required, breaths should be given twice every 10 seconds or once every 5 seconds. Two breaths every 10 seconds is preferred because it keeps the victim's airway drier, conserves rescuer energy, expedites equipment removal, and allows a more rapid transport and extrication. Use of the Pocket Mask is the most desirable aquatic rescue breathing technique (10). Every trained Rescue Diver should be encouraged to carry this device in the pocket of his or her buoyancy compensator. Rescuers should be aware of the priority order for aquatic rescue breathing: Pocket Mask, mouth-to-mouth buoyant chin lift, mouth to nose, and mouth-to-snorkel. Rescuers should be trained in more than one method of aquatic rescue breathing. The mouth-to-snorkel technique has diminished in popularity because it does not work with many modern snorkels. The procedure also requires extensive training and practice for effective application.

Any time the rescuer detects fluids or vomit in the victim's airway, the rescuer should roll the victim to the side and clear the airway before proceeding with rescue breathing. Blowing water and vomitus into the victim's lungs could be fatal. The do-si-do position allows the rescuer to roll the victim using one arm while the free hand can be used to sweep the mouth clear of debris. The do-si-do position is also an effective control method for rough water conditions.

After two successful initial breaths, the rescuer should continue rescue breathing with two breaths every 10 seconds while preparing to transport the victim. The rescuer should not waste time attempting to palpate a pulse. Transport preparation includes removing equipment from the rescuer and the victim. Equipment removal expedites rescue swims and is necessary for rescue exits. The priority for equipment removal is affected by several factors, but the general recommended order is: Victim's weights, rescuer's weights, victim's mask, rescuer's mask, rescuer's scuba unit, victim's scuba unit, and—in water shallow enough to stand—the rescuer's fins (just before exiting the water).

Rescue breathing interruptions should not exceed 30 seconds during rescue exits. If possible, rescuers should maintain the victim in a horizontal position continuously (15). Use of the head-down, Trendelenberg position should be avoided (16–19). A supine position is suitable for non-breathing victims and the recovery (left side) position

is best for victims who are breathing. Victims should be handled as gently as possible because severely cooled victims are extremely susceptible to ventricular fibrillation (26).

POST-EXIT RESUSCITATION

Rescuers should check the victim's pulse as soon as possible after the victim has been removed from the water. Ideally a trained rescuer who is warm and dry should check the pulse because the rescuer who removed the victim from the water will find it difficult or impossible to detect a pulse. The rescuer's strong pulse from the physical exertion required for the rescue exit combined with the effect of cold water on the sense of touch severely hinder the senses. Pulse check duration should be for 45 to 60 seconds for a hypothermic victim. Any pulse detected, however slight, should contraindicate chest compressions (27).

Scuba diving near drowning frequently occurs at remote locations where the response of EMS personnel may be delayed or impossible. The scuba industry encourages scuba divers to have first aid, CPR, dive rescue, and oxygen first aid training and equipment. Because many scuba divers dive as single buddy teams at remote sites and because one buddy rescuing another can do little more than rescue breathing and CPR, I encourage the industry to promote the following recommendation: Scuba diving excursions should consist of two or more buddy teams. Each diver should have current first aid, CPR, dive rescue, and oxygen first aid training and equipment. At least one buddy team should remain above water during dive operations to provide assistance and summon aid in the event of an emergency.

Current American Heart Association Guidelines recommend that "both trained and untrained bystanders should be instructed to call 911 or local emergency telephone numbers as soon as they have determined that an adult is unresponsive (28). Because it is obvious that leaving a non-breathing or pulseless scuba diver to summon emergency help while at a remote location is senseless, both DAN (27) and the National Safety Council (16:5) recommend that rescuers "not leave a seriously injured scuba diver unattended".

Regurgitation of stomach contents is a common near-drowning complication. Water from the stomach of near-drown victims hampers resuscitative efforts frequently. It would be a good idea to recommend that the use of manual suction devices become part of the curriculum for scuba rescue training and that suction devices become part of the recommended equipment for scuba diving emergencies. Dr. Edmond Kay of Seattle, Washington, has developed a simple suction device with a quick disconnect that attaches to a scuba tank low pressure inflator hose.

Rescuers should provide high flow oxygen as soon as possible (27). Medical grade oxygen has a low specific heat content, so it does not carry or provide warmth. Expansion of oxygen as it exits a compressed gas cylinder cools the gas to a low temperature, which may further cool the victim. Attempts should be made to warm emergency oxygen when it is provided to near-drown victims. Simple field procedures include wrapping the oxygen cylinder and tubing with heat packs or coiling the oxygen tubing in a container filled with hot water (27).

If oxygen, but no appropriate breathing mask is available for mouth-to-mask ventilation with supplemental oxygen, the rescuer can take a breath of 100% oxygen occasionally and exhale the oxygen into the victim. The rescuer should not breathe pure oxygen continuously because it will affect respiration (4). A rescuer who has oxygen but lacks a breathing mask could also hold a constant-flow oxygen tube in the corner of his or her mouth to provide a mouthful of pure oxygen with every rescue breath. This technique should not affect a rescuer's respiration (4).

Ventilation techniques for the near drown vary from standard procedures. Water in the lungs affects compliance and requires stronger breaths than usual. Firm inflations are recommended (4). Inflations should be firm, but not quick. If a rescuer blows hard and quick, the air is likely to travel down the esophagus, distend the stomach, and increase the likelihood of regurgitation of stomach contents.

EMS near-drowning protocols recommend positive-end expiratory pressure (PEEP) to help keep the air sacs in the lung from collapsing. Various manual techniques, such as reducing the size of the exhaust tube on a breathing mask, have been suggested as a means to mimic PEEP devices. Field resuscitation efforts without appropriate equipment should not include attempts to obtain positive-end expiratory pressure by manual means.

Near-drown scuba divers should be treated for shock. Rescuers should remove victims' exposure suits unless the exposure suits are the only means of thermal protection. Ideally victims in cold environments should be gently stripped, dried, and bundled to prevent heat loss.

Rescuers should not allow injured divers who recover or whose condition improves to sit or stand upright. Victims should remain horizontal until examined by medical personnel. At least two deaths have occurred when divers—who seemed to have recovered—were allowed to sit up.

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RESCUES IN SPECIAL CIRCUMSTANCES

R.W. Bill Hamilton

This analysis covers two specific rescue situations relevant to the topic of near drowning, with case reports. First is the situation in which a diver surfaces with grossly inadequate decompression. The other is the rescue that may follow an oxygen convulsion in which a diver loses the mouthpiece, and will inevitably drown unless rescued. These may result from a new self-contained recreational diving practice known as *technical diving*, which involves diving deeper than the traditional recreational limits using special equipment, gases, decompression tables, and techniques. A double fatality case of missed decompression emphasizes the difficulty of surviving such exposures. Three cases of successful rescues of divers who had convulsions in the water show that such rescues are possible with good in-water and topside support. Such support should provide divers surfacing with missed decompression the opportunity to go back in the water and complete their required decompression, conservatively, using oxygen and oxygen-enriched gases. For divers convulsing, it is impossible to reinsert the mouthpiece, so the best option is to get the diver out of the water as soon as possible to prevent drowning; embolism is unlikely under these circumstance; drowning is inevitable otherwise. Opinions are those of the author, who disclaims responsibility.

INTRODUCTION

Two specific special rescue situations are relevant to the topic of this workshop. First is the situation in which a diver surfaces with grossly inadequate decompression. The other is the rescue that may follow an oxygen convulsion in which a diver loses the mouthpiece, and unless there is a rescue will inevitably drown.

Both of these topics jump to the forefront because of the relatively new practice of *technical diving.* Technical diving involves self-contained diving operations beyond traditional recreational limits and is made possible by the use of special equipment and breathing mixtures, specifically including the necessary decompression procedures, special equipment to enable enough gas to be carried, adequate thermal protection, buoyancy control, and a myriad of other factors that become more prominent as the distance from the surface increases. This report threads rescue philosophy into several specific case reports on these topics.

RESCUING A DIVER WITH A MASSIVE DECOMPRESSION OBLIGATION

In addressing this category, the first consideration is the nature of the events that can create this situation. Normally something has gone wrong operationally. This may be that the diver surfaces inadvertently, a term sometimes called "blowup" (1). This is usually due to something like panic or loss of buoyancy control. Or, there may be a reason to surface prematurely, such as loss of gas, fear of shark attack, or equipment failure. Or, it may simply be "omitted decompression" in the classical sense, the table is not carried out correctly. Such events are handled routinely in commercial and much military diving because a decompression chamber is available and can usually deal with the problem. When there is no chamber, alternative procedures are needed.

The primary requirement in these cases is to get the diver back under pressure to complete decompression. This situation, of course, has wide variations of degree, in terms of the extent of the decompression obligation, the operational capabilities for recompressing the diver, availability of gases such as oxygen, and other such factors. A major decision point is the condition of the diver: Are there symptoms and how severe are they?

Decompression Sickness Case Report: Double Fatality

A case report of the rescue process following the inadvertent surfacing of two divers is useful to illustrate some of the points of concern. The intent here is to concentrate primarily on the possibilities of rescue, not so much on the operational aspects of the case.

Two highly experienced divers, diver 1 whose age was in the 40s, and diver 2 who was in his 20s, were healthy males making a dive with **air** to a sunken vessel at 230 feet of sea water (fsw) [70 meters of sea water (msw)]. They left their "stage bottles", tanks of oxygen and oxygen-enriched air decompression mix at the entry point to the wreck. While diver 2 was inside the wreck a panel collapsed on him, he became disoriented, and he had to be rescued by the other diver. Visibility went to near zero as the area "silted up". He thought he was tangled up in monofilament fishing line, but none was found at that site by divers who checked the area a few days after the accident; this is a typical manifestation of nitrogen narcosis.

The two divers exited the wreck but did not find their stage tanks (they must have come out of the wreck a

different way). By this time they were a few minutes beyond their planned dive time and were running low on air. They ascended directly to the surface at a time estimated to be 41 min since entry; diver 2 lost his regulator during ascent, which may have been a reason for the direct ascent. On arrival at the surface, they were missing about 2 h of required decompression. There was a 6- to 8-foot sea. Oxygen was available hanging at 20 fsw but they did not use it. While at the surface diver 2's manifold was broken due to wave action.

They had trouble getting on board. Diver 1 indicated he was dying, asked rescuers to concentrate on the other diver, and died within a few minutes. Diver 2 asked to go back in for decompression, but as mentioned his breathing equipment was not functioning, and by this time he was paralyzed from the chest down. He was lucid, however, able to talk and breathe O_2.

The helicopter arrived 1 h 20 min after the divers surfaced and took them to a nearby chamber. The diver breathed O_2 on the way to the chamber. Diver 2 was recompressed 2 h 43 min after surfacing. Diver 1 was declared dead. Diver 2 had normal temperature, BP 120/80, HR 120, RR 40 with dyspnea, breath sounds clear and equal. A Foley catheter yielded a small amount of dark amber urine.

Recompression to 2.8 atm was ineffective, so they continued compression on to 6 atm for United States Navy (USN) table 6A, which was carried out with 50% oxygen/nitrogen. There was some relief, with the diver still conscious and in extreme pain. Patient's lower extremities were paralyzed, but lower body sensation returned after compression. They carried out five O_2 cycles at 2.8 atm (2 extensions), and the patient received Valium and Percocet for pain and anxiety and was still able to converse. During the air break after the first 60-min cycle at 1.9 atm the diver had heart failure after showing some confusion and other warning signs. The chamber was reduced to room pressure for resuscitation, which was unsuccessful. The heart was full of gas and foam.

20:20 Hindsight Analysis of a Difficult Treatment

These divers, once this much decompression was missed, had a very slim chance of survival, especially because recompression was delayed. Some experienced diving medical officers consider this type of situation to be extremely difficult to resolve even when a chamber is immediately available. This may be due in part to lack of established and reliable treatment protocols for this type of exposure. Saturation is an option (2).

Operations.

From the perspective of decompression, a case can be made that the divers should have acquired decompression gas at the surface and gone back into the water to complete their required decompression. This decompression profile would best be more conservative than their original one to compensate for the time at the surface during which bubbles are likely to form and grow. The divers were not able to do this. Alternatively, they could have gone to 20 or 30 fsw and breathed O_2. In this case they could have taken O_2 at 30 fsw, in cycles. They did not have enough organized operational support to make either of these options possible.

Some would argue that a diver should not go back in the water for decompression if there are symptoms. This seriously cuts the options, and would depend on the symptoms, of course. If the diver is otherwise capable of doing the reentry for decompression then symptoms only emphasize the need. It now becomes a treatment and not the completion of a scheduled decompression, and falls under the rules of a different workshop (3). One could do this while waiting for the helicopter.

In today's practice of recreational (i.e., not commercial) diving, a dive to this depth (230 fsw; 70 msw) would normally be made with a trimix containing helium to reduce the narcosis. The divers elected to use air on this occasion. This was, however, supposed to be a "technical" dive since an intermediate mix of 60% O_2-enriched air and O_2 were to be used for the decompression (one definition of a "technical" dive is one in which the diver uses more than one breathing mixture).

As an editorial comment unrelated to near drowning as such, this incident is further evidence that this is too deep to do a wreck penetration dive with air as the breathing gas.

Chamber

The diver began to deteriorate after pressure was reduced to 1.9 atm, the third stage of treatment table 6A.

One might ask whether the diver should have been held at 2.8 atm for a longer time, possibly even put into saturation, since he was still paralyzed. In hindsight, yes of course. But were the signs strong enough to justify that position at the time? In a commercial diving operation offshore this would have been a natural choice, but most hospital chambers are not equipped for saturation, and this factor may have dictated the choice that was taken. Current treatment practices are to complete the original treatment, perhaps with extensions, and to treat on subsequent days until symptoms are resolved or no further progress is being made (2).

One might also wonder if recompression back to 6 atm after heart failure would be of any help? Even in hindsight the answer to this one is not so clear. The cause of heart failure might well have been "vapor lock", too much gas in ventricle for effective pumping. Whether a recompression from 2 atm abs to 6 atm abs would have resolved this is a

good question. This might not have been enough Boyle's Law recompression to be effective.

Conclusions on Difficult Treatments

When decompression is missed and a chamber is not available, the best thing to do if it is operationally feasible is to go back into the water and do the decompression. Use a longer table if possible. This assumes decompression is with one or more intermediate, high-O_2 breathing mixtures and O_2, not air. Also, it assumes that substantial operational support is available.

With air the only gas available, options are few and chances are slim. One lesson here is that it is foolish to do extreme diving without O_2 available!

RESCUE OF DIVERS DURING AN OXYGEN CONVULSION

Considerations

Divers doing extreme ("technical") dives accumulate big decompression obligations. Intermediate mixes and O_2 are almost essential. Divers try to limit their O_2 exposure, but even if this is done, CNS toxicity is possible. The consequence of concern here is a convulsion. In a convulsion, a diver is sure to spit out the mouthpiece. A full-face mask or helmet helps to prevent this, and is a sensible option when diving with high O_2 exposures.

It is virtually impossible to reinsert a mouthpiece during and just after a convulsion, a matter of some concern to a would-be rescuer. If a scuba diver convulses in the water there are two basic options, *drowning* or *ascending and risking embolism*. The latter is normally the best choice.

It is possible that the glottis is closed during the initial tonic phase, but it will open after it. It is unlikely that the airway will be so restricted that gas cannot escape if the diver ascends. However, the choice is obvious in any case. While embolism is a possibility, drowning is inevitable if the diver stays in the water. The rescue plan, then, is to get the diver out of the water. The only alternative to this would be the case in which a full-face or oro-nasal mask is available to provide breathing gas to the diver; this would be a preferred method when possible.

The convulsion will cause a period of apnea, and when this is over the diver will attempt to take a big breath, and may not be conscious enough to make a voluntary choice of not breathing water.

Once at the surface CPR may or may not be needed, depending on whether the diver is breathing. Most cases where rescue has been prompt have not needed CPR, or have required it for only a brief period.

The rescue will be complicated if the diver has a big decompression obligation and there is no chamber. Even so, the diver has to breathe first, then worry about decompression.

One additional point. For these rescue techniques to be of value, someone has to do the rescue. It is generally not sufficient just to have a buddy in the water, stressful dives need some "topside" support as well, so operations should be planned accordingly.

Central Nervous System Incident, Case 1

A diver in training was decompressing from a technical dive to 210 fsw (65 msw)/25 min. He went on O_2 at the first stop, about 90 fsw, instead of the intended intermediate mix of O_2-enriched air. At 60 fsw the diver began to have a convulsion. Instructors took the diver directly to the surface. He regained consciousness immediately. He was disoriented, but recovered rapidly. At no time did he have symptoms of DCS. Because he had been breathing O_2 deep on the dive, he did not miss an appreciable amount of planned decompression. Nevertheless, he was taken to a chamber and given a precautionary table 6 treatment, with no sequelae.

Analysis

The O_2 breathing may have helped protect this diver from both DCS and embolism. The instructors were alert, well trained, and well organized. Effective operational support pays off.

Central Nervous System Incident, Case 2

A diver on a prototype dual-system rebreather at 90 fsw (28 msw) was 500 feet into a cave. He had no decompression obligation. The O_2 level display showed his O_2 was low, and he opened the valve to add O_2 manually to the breathing bag. The oxygen reading did not increase, and he added more O_2. He repeated this several times.

The diver had a convulsion, spit out the mouthpiece, and aspirated water. His buddy diver pulled him to the surface and began mouth-to-mouth resuscitation within about 6–7 min after the convulsion. After a few breaths the diver began to breathe spontaneously, was taken to the hospital for observation, but had no further problems. There was an experienced doctor, in fact an anesthesiologist, on the surface support team, but he did nothing special. Standard expired air resuscitation worked fine.

The investigation showed that the diver was reading the O_2 level on one of the breathing bags and adding O_2 to the other one (from which he was breathing), which is why he was able to build up a high dose. The actual level of O_2 reached is not known.

Analysis: (About the Same as Case 1)

The O_2 breathing probably helped protect the diver from embolism and the effects of aspirating water. This diver

was fortunate to have a buddy competent to make the rescue and a good surface support team.

Central Nervous System Incident, Case 3

A complex, well organized technical diving operation was diving on the *Lusitania,* which is located at a depth of about 90 msw (300 fsw), using trimix and technical diving techniques. The team's organization had a standby diver on the boat and a support diver in the water; duty for these functions was rotated among the dive team.

The diver was decompressing from a dive to 87 msw for 16 min, hanging with other divers in the water on a fixed underwater station (a suspended framework) at a depth of 6 msw. He had been there, breathing O_2, for only 14 min. He felt a convulsion coming on, then convulsed and spit out his mouthpiece. The other divers could not reinsert it. His head was tilted back, eyes closed, and a wisp of blood came from his mouth. Before ascent they tried to force gas out of his chest but could see none coming out. He was given a few breaths of resuscitation at the surface but while still in the water.

They hauled him on board in about 2 min. He was ashen, cyanotic, and looked dead. Mouth-to-mouth resuscitation for a few moments started him breathing again. He was put on O_2. He regained consciousness, was lucid, took something to drink, but appeared to be "a little drunk" for a while.

The helicopter came in 50 min to take him to the chamber. There the doctor kept him under observation for an hour, but he had no strong DCS symptoms. He was nevertheless given a precautionary table 6 and hospitalized for 2 days. He was completely recovered long before they would let him go home from the hospital.

Analysis

It is impossible to reinsert the mouthpiece in a convulsing diver. The convulsion comes with little or no warning, and if there are some symptoms, it is usually too late. This victim had amnesia from the moment before the convulsion until fully conscious afterward. The appearance of this diver on deck after being taken from the water was worse than one would expect.

His O_2 exposure was a little over half of what he should have been able to tolerate without symptoms. Many possible causes were considered, but we found no explanation; no conditions that would predispose to a convulsion were noted. This diver had done about 100 such dives. One never knows.

He got away with missed decompression, but he did get a chamber treatment. A conservative calculation using a method well established for technical trimix diving, DCAP with matrix 11F6 (4), showed the diver should have taken 29 min more oxygen breathing at 6 and 3 msw out of a total of 94 min total decompression time. Thus the missed decompression was enough to increase the risk of DCS, but this exposure would be regarded as being quite easily manageable in a treatment, unlike the double fatality case, above.

Convulsing people may bite their tongues, so a little blood does not necessarily mean embolism.

LESSONS LEARNED

The cases presented here, and even if many others are included, are far too few on which to base firm conclusions about rescuing divers with extreme decompression obligations or after a convulsion. However, this experience coupled with that from elsewhere offer a basis for some speculative advice.

One principle of successful rescue that dominates all others is the simple (and familiar) motto, be prepared. Extreme diving of the sort that creates the problems covered here is a dangerous sport at best, but the risks can be sharply reduced if adequate rescue plans are in place from the beginning. The first time one thinks about a problem like these should not be when it is happening. There should be an operational structure that provides a competent person at the surface to manage a rescue and the personnel and other resources to carry it out.

What if a rescue diver has a big decompression obligation? Short excursions to the surface or a lower pressure are tolerable, and are the best option for a diver threatened with drowning. Unless there is topside support to take over, this can be a dilemma. How much risk to take is a matter of condition and support (Can the operation afford two casualties?).

RESCUE OF A DIVER AT THE SURFACE WITH AN EXTREME DECOMPRESSION OBLIGATION

The classical preparedness here is to have a recompression chamber at the dive site. Portable chambers are becoming more common and are the next best thing, but definitive operational plans for using them are not yet common. The next line of defense is a good evacuation plan. If there is a choice, go to a chamber with saturation capability.

Treatment needs to be aggressive. Traditional practice, especially as is the routine for hospital-based hyperbaric medical facilities not equipped for long treatments, is to complete a standard treatment such as table 6, perhaps with extensions, even though all symptoms may not be relieved. Then subsequent daily or twice daily continuation treatments are used; these have been found to be effective in most cases in providing continuing relief of symptoms,

sometimes over many days. In life-threatening situations, this may not always be the best course; it may be best to hold the patient at pressure.

The benefits of going to pressure higher than the 2.8 atm of table 6 are hotly debated. If a few hours have passed since the diver surfaced, the chances of getting major benefits from compression to 6 atm are relatively slim. But this rule should be weighed against the severity of the symptoms. One thing to remember is that there are protocols for significant extensions of table 6, and these are often effective (5).

A good rule when a case like the double fatality described above comes in is to get help from colleagues. It is helpful for even the most seasoned diving doctors to be able to "bounce ideas off" a colleague.

RESCUE OF A DIVER AFTER A CONVULSION IN THE WATER

The small risk of embolism of the victim is a much better choice than drowning, so make every effort to get the diver out of the water as quickly as possible.

Some CPR in the water at the surface may be helpful, but it is best to spend that effort getting the diver out of the water. Despite the fact that a diver may *look* dead, the rescue attempt should proceed. Note, however, that all the cases discussed here are for relatively short exposures in the water after the convulsion.

If the rescued diver has a big decompression obligation, the diver should be taken to a chamber, on O_2 at the surface, and given a treatment even if it is "precautionary". Risk of ascent is reduced after O_2 breathing.

If a lot of decompression is missed and there is no chamber available, as soon as the diver is capable, go back into the water and finish it, using O_2-enriched mixtures and O_2. For the time spent at the surface add extra O_2 time, say 3 or 4 times the time spent at the surface (as a guess!).

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NEAR DROWNING: HOSPITAL MANAGEMENT

Steven D. Brown and Claude A. Piantadosi

The pathophysiology of near drowning (ND) is caused by asphyxia. The major target organs are the lung, brain, heart, and kidneys. In adults, ND is associated often with drug or alcohol ingestion and may be associated with other injuries. Adult respiratory distress syndrome complicates 40% of NDs, may be delayed, and can be aggravated by aspiration of stomach contents and other foreign debris. Pneumonia or sepsis induced by unusual organisms from contaminated water may complicate the ND victim's course. Prophylactic antibiotics, however, are not indicated.

INTRODUCTION

Millions of swimmers, boaters, and divers of all ages, with various degrees of physical skill and judgment, participate regularly in recreational activities involving water. The physical environment of the recreational swimmer and underwater diver, however, is deceptively hazardous. Too often, swimmers or divers impair their faculties with alcohol or ignore their physical limitations and venture into unsafe conditions, with tragic results. In some situations, such as with young children, the encounter with the aquatic environment is inadvertent or unsupervised. The number of drownings, diving accidents, and other aquatic misadventures each year is difficult to estimate. There are at least two million underwater divers and many more recreational swimmers in the United States. This activity translates to thousands of drownings and near drownings (ND) each year. Many victims survive the actual event only to die hours or days later in the hospital. Thus, it is incumbent on hospital physicians to be knowledgeable about the potentially life-threatening clinical consequences of diving accidents and ND.

SHALLOW WATER BLACKOUT AS A RISK FACTOR FOR ND

During immersion, the physiologic responses to breath holding have special significance (1). With apnea, the lungs act as a reservoir for exchange of O_2 for CO_2 in pulmonary capillary blood. During breath hold in air, the mean alveolar P_{O_2} decreases linearly with time and its rate of change is a function of the decline in the mixed venous P_{O_2}. As alveolar P_{O_2} declines, whole body O_2 uptake eventually falls and anaerobic metabolism increases. Carbon dioxide enters the lungs during apnea in proportion to both the pulmonary blood flow and the diffusion gradient for CO_2 between mixed venous P_{CO_2} and alveolar P_{CO_2}. The rate of CO_2 transfer is initially high, but decreases rapidly because its diffusion gradient decreases as alveolar P_{CO_2} approaches the mixed venous value. Further CO_2 production increases mixed venous P_{CO_2} which again causes alveolar P_{CO_2} to rise. The point at which high P_{CO_2} causes breathing to resume is the break point. The time to the break point can be extended by maneuvers that lower P_{CO_2} or raise P_{O_2} such as hyperventilation or oxygen breathing. Notably, the body's O_2 store is not increased appreciably by hyperventilation because the increase in alveolar P_{O_2} resulting from a fall in alveolar P_{CO_2} increases blood O_2 content only slightly. Therefore, hyperventilation greatly extends breath-hold time though it can produce profound hypoxia before the CO_2 rises to the break point. Alveolar gas exchange during breath holding is altered by underwater descent when lung volume decreases from thoracic compression, and the partial pressures of O_2, CO_2, and N_2 increase in the lungs. On descent, alveolar O_2 and CO_2 concentrations decrease due to a greater transfer of those gases to pulmonary capillary blood compared to inert gas (N_2). Alveolar P_{O_2} also is higher during breath-hold diving than during simple breath holding, and the transfer of CO_2 during early descent is opposite normal, i.e., CO_2 moves from alveoli to pulmonary capillary blood. During ascent, the lung reexpands and alveolar P_{O_2} and P_{CO_2} decline. Near the surface, the alveolar P_{O_2} may be almost as low as the mixed venous P_{O_2}. In strenuous dives, expansion of hypoxic alveoli during ascent may result in reverse O_2 transfer from mixed venous blood to alveoli. Carbon dioxide retained in the blood during the dive also leaves the blood on ascent as the alveolar P_{CO_2} decreases. Carbon dioxide elimination usually continues postdive as does exchange of the small amount of N_2 that entered the blood during the dive.

Hyperventilation before breath-hold diving is a dangerous way to extend the dive. During the dive, compression of the lungs causes the alveolar P_{O_2} to increase. Thus, the primary signal to return to the surface is the P_{CO_2}. When the diver hyperventilates before the dive, the arterial P_{CO_2} begins at a lower level, and the time to the breath-hold break point is extended. Alveolar O_2 concentration, how-

ever, is depleted more severely during longer dives, and life-threatening hypoxia may occur when the diver approaches the surface. Loss of consciousness by this mechanism, known as *shallow water blackout,* causes a large number of drownings.

The body's response to breath-hold diving can be modified by a diving response induced by apnea and facial immersion, particularly in cold water. This diving response, manifested primarily as bradycardia, is most pronounced in young children. The diving response has been interpreted as an O_2-conserving response that redistributes blood flow from organs resistant to hypoxia to organs with continuous requirements of oxygen such as heart and brain. This interpretation in man is controversial, although a diving response may contribute to survival of children resuscitated up to an hour after submersion in cold water.

NEAR DROWNING PATHOPHYSIOLOGY

The immediate consequences of ND are attributable to asphyxia. Effective pulmonary gas exchange ceases, and the victim suffers the physiologic consequences of hypoxia and hypercapnia. Excellent reviews of the on-site management of ND and its demographics have been published and will not be repeated here (2–4). This section will summarize the pathophysiology of the systems most frequently injured during ND and supported in the ICU: the lungs, brain, heart, and kidneys.

Lung

The pathophysiology of lung injury in ND is complex. Airway obstruction is initiated when either fresh water (FW) or sea water (SW) contacts the mucosa of the lower respiratory tract and stimulates intense laryngospasm. Laryngospasm may be protective in some cases if the duration of hypoxemia is limited by a brief immersion time. Approximately 10–15% of ND victims aspirate trivial volumes of water, yet some of these individuals develop O_2 deprivation of sufficient magnitude and duration to produce hypoxic encephalopathy or ventricular arrhythmia as a direct result of laryngospasm (5). Aspiration of both SW and FW also induces mechanical airway obstruction with a small airway component. Small airway obstruction is aggravated by bronchoconstriction, mucosal edema, and plugging by water and suspended debris such as algae, diatoms, sand, mud, teeth, and stomach contents (6).

Aspiration of even small quantities of either SW or FW produces a prompt and profound decrease in lung compliance and results in persistent areas of low ventilation:perfusion ratio and shunt (6,7). Therefore, aspiration of water may produce a longer-lasting hypoxemia than laryngospasm alone. Some of the mechanisms of the early changes in pulmonary gas exchange have been elucidated in animal models and are attributable, in general, to loss of surfactant or its activity, damage to the alveolar epithelium and capillary endothelium, and alveolar flooding. In human beings, vomiting and aspiration of stomach contents during ND is common and aggravates mucosal and alveolar epithelial injury.

The combination of alveolar flooding and loss of surfactant with atelectasis and alveolar damage may give rise to progressive hypoxemia due to intrapulmonary shunting, which may reach 70% of the cardiac output in severe cases (6,8). The pathophysiologic injury culminates in adult respiratory distress syndrome (ARDS) several hours to days after the ND event in about 40% of ND victims (9,10). Hypoxemia usually necessitates treatment with supplemental oxygen at high inspired oxygen fraction (Fio_2) which may superimpose pulmonary oxygen toxicity on ARDS. Fortunately, ARDS that develops after ND is more likely to be reversible relative to ARDS of other etiologies.(10)

Brain

Near drowning is the second leading cause of brain death, after trauma, in children admitted to a pediatric intensive care unit (11). The pathophysiology of brain injury in ND is that of global anoxia or severe hypoxia and differs little from other etiologies. Prolonged anoxia or hypoxia produces diffuse neuronal damage which, if severe, compromises the function of the blood–brain barrier with resultant cerebral edema. Intracranial pressure (ICP) may rise as edema develops, further decreasing brain perfusion, exacerbating intracellular hypoxia, and in severe cases leading to herniation. Profound increases in ICP are infrequent after ND, but tend to appear more than 24 h after initial resuscitation in patients who already have some evidence of neurologic dysfunction (10). Some clinicians consider a spiraling increase in ICP an indicator of the severity of initial and ongoing neuronal injury rather than a major source of the insult (10,12).

The major potential differences between ND and anoxic brain insults of other etiologies are the diving reflex and body cooling (hypothermia) (13). In human beings, the diving reflex is vestigial and most demonstrable in young children during exposure to cold water (13,14). ND in cold water with prompt hypothermia slows cerebral metabolism and thereby postpones the deleterious effects of anoxia (15). These factors may be important as they are associated with a better prognosis after apparently severe ND in some patients (14–16).

Heart

Atrial and ventricular dysrhythmias, particularly ventricular fibrillation, occur in severe ND although some authors

only report "cardiac arrest." (17). Older studies of drowning in animals demonstrated hemolysis and large rapid shifts in the concentrations of blood electrolytes after instillation of FW and SW into the lungs. These responses were correlated with the occurrence of ventricular dysrhythmias. Human studies have failed to confirm significant electrolyte changes even in patients with ventricular fibrillation (18). A notable exception is drowning in the waters of the Dead Sea which has a much higher mineral content than other SW. Dead Sea drowning victims developed hypernatremia, hyperchloremia, hypermagnesemia, and hypercalcemia slowly over 24 h following exposure because of absorption of electrolytes from the gastrointestinal tract after swallowing large volumes of water during the episode. These electrolyte changes were associated with fatal ventricular fibrillation in one of eight patients reported (16).

Near drowning in human beings differs in a major way from animal studies in the volume of fluid aspirated; human victims rarely aspirate enough water to produce significant electrolyte changes (18). In addition, pathologic studies in humans after SW or FW drowning demonstrate myocyte hypercontraction and hypereosinophilic sarcomeres in the heart. These pathologic changes are characteristic of catecholamine excess and suggest that intense adrenergic stimulation contributes to the dysrhythmias after ND (19). Thus, the etiology of ventricular fibrillation in human beings is most likely related to hypoxia, respiratory and metabolic acidosis, and catecholamine excess. A retrospective review of cases of children with brain death demonstrate myocardial infarction to be commonly associated with ND (20).

Kidney

The incidence of renal insufficiency following ND is unknown but far less frequently reported than lung, brain, or cardiac injury. The renal complication cited most often is oliguria, attributable to acute tubular necrosis (ATN) (21). The etiology of ATN in this setting is likely hypoxemia and hypotension. Infrequently, ND may be complicated by rhabdomyolysis and hemolysis with disseminated intravascular coagulation (DIC) (22,23). Hemolysis and DIC also may contribute to ATN. Although patients with acute renal failure after ND may require transient dialysis, recovery of adequate renal function can be expected in the majority of patients.

MANAGEMENT

General Measures

Near drowning in adults and adolescents is associated with a number of predisposing factors and complicating injuries. These factors are easily overlooked in the unconscious, critically ill ND patient. They must be suspected in every case because they may affect the treatment and prognosis of the patient.

Alcohol and other CNS-altering drugs are commonly implicated in adult ND (24). Sedatives and alcohol in particular may complicate the patient's initial ICU course by exacerbating hypothermia and hypotension, impairing mental status, and depressing respiratory drive. Levels of toxic drugs and blood alcohol should be measured in complicated patients admitted to the ICU.

Other predispositions to ND include untoward pathophysiologic events in the otherwise unimpaired adult swimmer. Myocardial infarction, cardiac dysrhythmias, seizures, subarachnoid hemorrhage, and AGE in the scuba diver have been implicated in many episodes of ND (24). These events may require extraordinary diagnostic efforts in the immediate postresuscitation environment of the ICU. Electrocardiography (ECG) should be obtained routinely because the heart is a target organ for hypoxemia in ND victims. Serial measurements of cardiac enzymes in ND are useful in confirming the diagnosis of myocardial infarction when ECG changes are nondiagnostic. Acute intracranial hemorrhage or status epilepticus may need to be excluded in patients whose course in the ICU is complicated by neurologic dysfunction. Recompression and HBO_2 therapy should be considered in the near drowned scuba diver with obtundation, coma, or other neurologic deficit.

Injuries to the spine and skull are common in ND victims. These most often occur when a swimmer dives into shallow water and strikes the head on the bottom or on a submerged object (24). Another common scenario involves a motor vehicle accident which leaves the passenger submerged in a body of water. Burst fractures of the cervical vertebrae resulting in tetraplegia have been reported in these settings. Additionally, skin, middle ear, or sinus trauma sustained during ND may serve as portals of entry for infection (25). Such injuries must be considered in the unconscious ND victim in the ICU.

Management in the ICU

The major indications for admitting the ND patient to the ICU are respiratory failure, cardiac arrest or dysrhythmia, and obtundation or coma. The patient is often intubated before arrival in the ICU. Clinically, the patient may exhibit cyanosis, tachycardia, hypo- or hypertension, hypothermia (which may require a low temperature thermometer to measure), respiratory distress with frothy, blood-tinged sputum, diffuse crackles, and wheezing on examination. Initial laboratory evaluation often reveals a metabolic acidosis (due to lactic acid) and hypoxemia on arterial blood–gas analysis. Serum electrolytes, with the

exception of a decreased bicarbonate concentration, rarely are perturbed significantly (26). However, ND in industrial fluids or the Dead Sea can perturb serum electrolytes. As an example, a 28-year-old male developed severe hypercalcemia after a ND incident in an industrial vessel with oil-well drilling fluid that contained calcium salts (27). Hypoglycemia is common (28). Hemolysis and rhabdomyolysis, if seen, tend to occur early in the clinical course unless they are the result of late sepsis. ECG abnormalities include evidence of ischemia or injury and ventricular and atrial dysrhythmias. Initial chest radiographic findings range from patchy infiltrates to diffuse air space disease. Progressive increase in parenchymal infiltrates over hours to days is not unusual.

Respiratory Care

Mechanical ventilation can be a major challenge in the severely injured ND victim. As discussed previously, atelectasis and pulmonary edema from the lack of functional surfactant and epithelial damage with intrapulmonary shunting are encountered in all types of ND. ARDS can develop with accompanying elevated airway pressures which predispose to pulmonary barotrauma and air embolism. These conditions can be further complicated by the presence of foreign bodies in the airways. Sedation or paralysis with drugs such as pancuronium are best avoided in ND patients because they impair the clinician's ability to follow the neurologic examination. Careful use of these agents, however, may improve mechanical ventilation by synchronizing the patient with the ventilator and decreasing airway pressures and the risk of barotrauma. The advantages of positive end-expiratory pressure (PEEP) in decreasing intrapulmonary shunt can be dramatic in severe hypoxemia after ND. Treatment of children with ARDS following ND with artificial surfactant did not improve outcome but improved pulmonary function modestly (29).

Respiratory insufficiency in ND may be complicated by factors other than atelectasis and intrapulmonary shunt. Airway obstruction may occur either as bronchospasm or from a foreign body. Bronchodilator therapy with inhaled ß-agonist agents and theophylline may benefit the patient with diffuse wheezing. Patients with localized atelectasis that fails to improve with effective ventilation or those who exhibit localized wheezing should undergo fiberoptic bronchoscopy to exclude or remove a foreign body.

Many ND accidents occur in water which is contaminated with human or animal waste or naturally contains pathogenic bacteria or fungi. The lung is the most common portal of entry for a wide variety of these organisms. Infection is heralded by fever 2–7 days after the ND event and should prompt sputum and blood cultures before initiation of antibiotic therapy.(30) Prophylactic antibiotic coverage has not improved outcome after ND, and routine use of antibiotics is not indicated. Reported infections have been summarized in Table 1. Awareness of infection by more unusual organisms is crucial, because they may have specific culture requirements not offered routinely in many hospital microbiology laboratories (25).

Brain Resuscitation

Therapeutic measures for brain resuscitation after ND are controversial. The debate is complicated by the diverse parameters which influence brain injury and recovery, including the age of the victim, the water temperature and submersion time (or period of asphyxia), coexisting injuries, and preexisting disease (35). The matter is complicated further by anecdotal reports of complete or near complete neurologic recovery in association with specific therapeutic measures after prolonged immersion. The initial prognostic uncertainties about brain recovery after ND mandate a full effort at cardiopulmonary resuscitation including correction of hypothermia.(36)

Classification of the victims' neurologic status 1–2 h after resuscitation allows systematic assessment of prognosis. A system with reasonable discrimination for outcome in human beings subclassifies patients after resuscitation into three categories as shown in Table 2 (14,15).

The best discrimination for outcome using this system has been found in children where all category A and B victims (n = 57) recovered completely. Level C victims (n = 39) suffered 33.3 and 23.9% cerebral morbidity (mortality+morbidity = 56.2%) with the lowest survival in the C.3 group (15). In another series of patients which included 52 adults, the category A patients recovered completely while 2 adults and 1 child in category B eventually succumbed to barotrauma or other complications (14). Eight of 11 (73%) adult and 8 of 18 (44%) child category C patients recovered completely in that series.

Use of a brain resuscitation regimen known as HYPER (hyperhydration, hyperpyrexia, hyperexcitability, and hyperrigidity) suggested that the fraction of category C child ND victims with subsequent complete recovery could be increased (15). The acronym addressed the overhydration, fever, excitability, and muscular rigidity noted in some patients. These findings were thought to affect outcome adversely, and aggressive therapy was recommended to minimize them.

HYPER therapy consists of systemic corticosteroids, osmotic diuretics, hyperventilation, barbiturate coma, and muscle relaxants administered to minimize cerebral edema and reduce ICP. Controlled hypothermia (32°C, 89.6°F) to decrease neuronal metabolism also was advocated (16).

Table 1: *Microbial Isolates Associated Wit Infection in ND Victims*[a]

	Type of Water	Reference
Bacteria		
Klebsiella oxytoca	FW	25, 31
Herellea spp	FW	22
Neiserria meningitidis	FW	22
Pseudomonas aeruginosa	FW	31
Listeria monocytogenes	FW	31
Plesiomonas shigelloides	FW	31
Edwardsiella tarda	FW	31
Chromobacterium violaceum	FW	41
Aeromonas hydrophila	SW, FW	25, 31
Escherichia coli	SW, FW	25, 31
Proteus mirabilis	SW, FW	25, 31
Staphylococcus aureus	SW, FW	31, 32
Neiserria mucosus	brackish	33
Pseudomonas putrefaciens	SW	25, 32
Francisella philomiragia	SW	30
Vibrio parahemolyticus	SW	25
Fungus		
Pseudoallescheria boydii	FW	31
Aspergillus fumigatus	FW	31, 34

[a] Organisms isolated from sputum or blood of clinically infected ND victims soon after admission. This list does not include organisms recovered from infected wounds.

Table 2: *Classification of ND Victims After Initial Resuscitation*

Category A: awake, fully conscious
Category B: blunted consciousness, stuporous but arousable
Category C: comatose
Category C is further classified into:
C.1: decorticate posturing
C.2: decerebrate posturing
C.3: flaccid

Monitoring of ICP is necessary to guide such aggressive therapy. This therapy was based on the rationale that control of ICP would minimize neuronal damage after diffuse anoxia. The pathophysiology of brain injury following ND suggests, however, that the increase in ICP which develops 24–48 h after ND may be a result of severe neuronal injury rather than its cause.

Critical reviews and subsequent experience with HYPER therapy have failed to confirm its efficacy and have highlighted its potentially detrimental effects (5,10,36). A retrospective review of 40 ND patients from the same institution that reported the original experience with HYPER found an increased incidence of sepsis and multiple organ failure in patients treated with hypothermia (35). This may be due to cold-induced immune suppression (including neutropenia) complicated by cold-induced bronchorrhea and decreased mucociliary clearance. (35).

Corticosteroids are efficacious in reducing edema surrounding brain tumors but are of no proven benefit in reducing brain edema associated with trauma, intracerebral hemorrhage, or stroke. In the absence of convincing evidence that corticosteroids reduce edema and hence ICP, and that reduction of ICP improves neurologic outcome after ND, these agents should be avoided as they are immunosuppressive and predispose to infection and gastric ulceration (37).

Although hypothermia and barbiturates can reduce ICP in some circumstances, their use has not been demonstrated to improve neurologic outcome. Attempts to decrease ICP by osmotic diuresis also have not been shown to improve neurologic outcome after ND and may induce hyperosmolarity and renal insufficiency. Thus, routine use of corticosteroids, hypothermia, and osmotic diuresis cannot be advocated in ND patients. Mild hyperventilation is a comparably benign intervention to reduce ICP temporarily, and ICP monitoring may help direct therapy in the subset of ND patients with elevated ICP and poor prognosis. More recent experience with aggressive cerebral monitoring and resuscitation of the ND patient has shown no benefit (38).

Prognosis

Overall, about 80% of child and adult ND victims recover without sequelae while 2–9% survive with brain

damage. Approximately 12% of all ND victims die. About 90% of category A and B and approximately 50% of category C patients survive with full recovery while 10–23% of the later group survive but have permanent neurologic sequelae (9,10,14,18). Thus, respiratory insufficiency in the absence of sepsis or infection is seldom the cause of death in ND victims in hospitals with modern intensive care capabilities.

A number of indicators such as serum electrolytes, arterial blood–gas and pH values, electroencephalographic findings or clinical features (body temperature, absence of pupillary responses, cardiac arrest, duration of submersion, initial resuscitative efforts), and cross-brain oxygen content difference (39) have been examined for use as early markers of prognosis. None is sufficiently discriminating to guide early therapy. The absence of spontaneous respirations after resuscitation, however, is a particularly ominous sign and uniformly associated with severe neurologic impairment or death (36). In a retrospective review of 44 children, all survivors, with satisfactory neurologic function were awake with purposeful motion 24 h after the ND incident (40).

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EXPECTATIONS FOR RECOVERY

Christopher W. Dueker

After rescue, resuscitation, and the institution of advanced treatment comes the time for reflection on expectations for recovery. Difficult decisions may need to be made earlier (i.e., should resuscitation efforts continue?), but the ideal time for prognostic evaluation comes after the excitement.

The recognition of the basic injury-asphyxia causing hypoxia dictates the goals in rescue, resuscitation, and treatment. Similarly, the extent of the hypoxic injury will determine prognosis for recovery. Not surprisingly, those with mild injuries do the best. It is, however, important to recognize that the significance of clinical events varies among accident types.

Safar (1), the noted resusitation scientist, postulates that cardiac arrest from asphyxial hypoxia has a worse prognosis than other forms of arrest, for example, electric shock. This theory is supported by the very poor outlook for immersion victims who have cardiac arrest.

Navigating through the literature on outcomes in near drowning is difficult. The reports are obtuse or impossible to compare. Several structural issues explain much of the difficulty. First, the timing of evaluation affects results. The widest range of victims occurs at the accident site. Mildly injured persons will go home and the obviously dead will never receive resuscitation. A report written about beach injuries would have to include both groups though neither would help evaluate the value of on-site resuscitation. But ignoring those who do not need resuscitation would misstate the total recovery and death incidence in water accidents.

Many reports are based on results from emergency departments. Usually conditions are better controlled than in the field, and advanced treatment can be provided. However, this site of evaluation misses the minor accidents, those who were declared dead without resuscitation, and those for whom resuscitation was abandoned.

Hospital ward and intensive care evaluation permit the most sophisticated analysis. However, the majority of immersion victims never reach these sites. Thus their experience does not reflect outcome for the larger population. Survival incidence, therefore, depends on the group selected for evaluation.

Second, outcome requires definition. The simplest distinction is life or death. Death is easy to define except when it is delayed. Should these delayed deaths be called drownings? Opinion varies. Quite a few reports divide results only into death and survival. But survival is quite complicated. Recovery may be complete or the victim may remain in a vegetative state. Complete recovery may contain many gradations of functional capacity. Obviously, the extent of recovery is important in the evaluation of outcome. And a thorough analysis of recovery requires time. Reports from emergency departments or intensive care units cannot predict the eventual condition of those who survive.

Third, data collection, especially in the field, is very poor. Frequently, immersion duration is not known or is not recorded. Water temperature is rarely reported. Postulating the significance of water temperature loses its potency when temperatures remain unknown. The absence of adequate data precludes evaluation of selected groups or treatment methods. For example, it would not be possible to evaluate the outcome of near drowning in scuba divers. They are rarely defined as a specific group even when activity at the time of an immersion accident is noted. Would their water skills be helpful? Does the buddy system aid in rescue? Does physical fitness affect survival? Do exposure suits help or hinder? These, and other questions, remain unanswerable.

Three physical findings usually noted have been valuable in predicting outcome. Loss of consciousness signifies that the immersion has been serious. Conversely, a conscious victim has been less severely affected. However, being conscious does not mean that the accident was minor. Deterioration, and even death, could result from inadequate evaluation and treatment.

Many years ago the Australian physician Pearn (2) postulated that the time to "first gasp" was the most reliable prognostic sign. The presence of spontaneous breathing or a quick return to spontaneous breathing bodes well. Again, proper treatment is necessary even for those who have never been apneic.

Presence of a pulse is probably the best sign that recovery should be possible. The goal of rescue and resuscitation is interruption of the hypoxic spiral before cardiac arrest. Prognosis worsens considerably when cardiac arrest develops.

Even in the hospital it can be very difficult to feel a pulse. Graver has discussed the difficulties presented by cold, gloves, exposure suits, etc., involved in water rescues.

Time should not be wasted in the search for a pulse when this search would delay rescue breathing.

Absence of a spontaneous pulse on arrival in the emergency department is probably the single worst prognostic sign. In 1977, Peterson (3) found that no nearly drowned child who required cardiac resuscitation at the time of arrival in the emergency department made a good recovery. Subsequent reports have been only slightly more optimistic. Recoveries in pulseless patients occur, but only rarely.

Coma in the patient with a pulse and spontaneous breathing has a more variable prognosis. Deranged consciousness is associated with poorer outcome but is not as predictive as pulselessness.

Cardiac arrest and coma signify a severe hypoxic injury. This injury begins with the immersion and will continue until the relief of hypoxia. Poor response implies that the insult was already pronounced when treatment began. Unfortunately, poor response may also reflect inadequate early treatment. First aid, though logically attractive, may be ineffective.

No prospective studies prove that field resuscitation improves recovery from near drowning. The Institute of Medicine reports that there are no randomized trials of mouth-to-mouth resuscitation.(4) The empirically accepted role of mouth- to-mouth resuscitation and the unorganized pattern of emergency care preclude such a study.

But it would be good to look carefully at the results of 1) bystander resuscitation and 2) emergency medical services. Many such studies exist for terrestrial cardiac arrests, but they ignore or exclude near drowning.

An implication that field resuscitation (bystander or emergency medical services) is not ideal comes from emergency departments who receive pulseless near- drowning victims. Some of these patients have received field resuscitation which did not restore spontaneous pulse. Hospital resuscitation in some patients quickly resulted in a spontaneous pulse. Unfortunately, these patients do not recover any more frequently than those who remained pulseless.

Efforts to streamline medical care by increasing the proportion of near-drowning patients treated as outpatients could be hazardous. Injuries which appear minor, i.e., the patient who is fully awake and in no respiratory distress, may progress to serious derangement and even death. This is not "secondary drowning", it is the natural course of asphyxial, pulmonary injury.

Despite methodologic difficulties, the goal of obtaining prognostic data remains worthwhile. Comparison of treatment methods could be used to improve survival, and a realistic prediction of outcome would help the victim's family, the rescuers, and those involved in treatment.

Survival is very good for properly treated patients who awaken quickly. Those who actually drown, of course do not survive. Expectations for the large group in the middle remain undefined. Habib and colleagues (5), in a very recent series of patients from their hospital, estimated that 20–40% of those who survived near drowning "...never reached normal neurological function."

QUESTIONS

Dr. Brown: Is it all comers?

Dr. Dueker: Well, it's not all comers. It's not from the beach on; it's from admission on. It's not a perfect fit with your report (pg) but it gives you a range, and that's a fairly high figure for non-recovery, I think.

A very recent report (6) introduced a new recovery term: Unpredicted Good Outcome (UGO). After careful analysis of their near drowning cases these investigators predicted outcomes. Those patients who did well, despite dire outlooks, became UGOs. They could not improve predictive power to reduce the UGO of below 5% of all patients. The elimination of aggressive treatment would have resulted in the deaths of patients who unexpectedly recovered. Can a predictive power of 95% be improved?

Dr. Brown: I remember reading that study briefly, but how long did they observe these people after they came in and were initially resuscitated?

Dr, Dueker: They treated them all.

Dr. Brown: For what duration?

Dr. Dueker: They gave them the proverbial full court press. And one of the children was in an essentially vegetative state for a month, went home after a month in very bad condition, and 7 years later is virtually perfect. Now, that's a rare thing. I'm not going to argue with you that this has any predictive value, but it does give pause for thought.

PROLONGED IMMERSION

We cannot predict at this time, with complete assurance, that a given patient will or will not recover. Some patients come into the emergency department without a pulse and then fully recover. Unfortunately, a few who never needed cardiopulmonary resuscitation subsequently die.

The occasional survival after prolonged immersions attracts attention. These cases range widely in veracity. Often, no accurate determination was made of immersion time. Survivals beyond 5 minutes of submersion pales in comparison with those persons who drown with less than 5 minutes of submersion.

Almost all of the accurately reported prolonged immersion survivors have been children. And almost all of them have nearly drowned in extremely cold water. Reports of

adult survivors are more rare and the reports are of poorer quality. Failure to measure water temperature, patient temperature, and immersion time weakens the theory of prolonged immersion survival.

Is cold water protective? The advantages and disadvantages of cold water immersion are discussed in *Myths in Near Drowning* (pg-). The moderate temperature associated with most immersions are not protective.

Scuba divers would not be expected to have an improved likelihood of survival after prolonged immersion. They typically do not get very cold. The diving reflex, seen during breath holding, has not been proven to improve breath holding in humans. (In an anecdotal survey at this workshop, no one knew of a scuba immersion accident survival after the victim had been under the water more than 10 minutes).

Victims of cold water immersion require special care. They are especially susceptible to cardiac dysrthymias. Aggressive therapy is justified even in apparently hopeless cases. Prognostic evaluation should not be made until the patient has been rewarmed to normal thermia.

There is no predictive protective value in cold water immersion. In matched cases, for example, two or three children fall into very cold water. Despite identical conditions one may survive while the others drown or recover incompletely.

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AFTERNOON DISCUSSION

Dr. Brown: Would those of you who spoke this afternoon like to join us?. As they assemble, I'd say, yes, you're not dead until you're warm and dead; I actually think that no one challenged that, and it remain a really good adage, that I hope doesn't come under insurance scrutiny.

Panelist: Two questions, the first being the presentation on the initial rescue of the diver, and essentially I am concerned because of this same problem came on with resuscitation of a gas embolus—you know, positioning and we got into that mode rather than treatment of the diver. My question would be just a clarification: a diver who's unconscious on the bottom, we're attempting to bring him to the surface as quickly and efficiently as possible, and bring him to the boat and begin a resuscitative effort. These positions, these various things in water, even to the point of using laryngeal masks, are certainly things to be aware of, but the primary is quickly to the surface, on the boat for resuscitation. Correct?

Mr. Graver: In rescue procedures? It would be nice if there was always a simple answer to every situation, but the fact is that the situation varies drastically. If you surface someone right beside the boat, you should do exactly what you said, get them out of the water and do all your resuscitation efforts right there on the boat. If you're a long way from the boat or the shore, dragging them there without resuscitation is a disaster.

Panelist: Absolutely. It does depend on the circumstances.

Mr. Graver: Thank you for the clarification, though.

Panelist: The second question was with that severe a disease, the other extreme is the actual screening of those patients or potential near-drowning victims, and in those that train a large groups of individuals that are in the water—water survival training—we see the very model cases we could say, of near drowning, where coughing or sputtering on the pool side, essentially a rapid screening device that may be used . Or could you clarify what the lifeguards used?

We were looking at the idea of looking at respiratory rate and pulse oximeter. And one of the points that was brought up earlier was the use of arterial blood gas as being ineffective in this early going as far as picking up lung water in an animal model. Would a pulse oximeter in people that have been previously screened for no lung disorder be very effective in that very early going and particularly in anesthesia. When we do see a fairly significant aspiration we do detect that fairly early on but, again, gastric aspiration. Could you comment a bit on that?

Dr. Dueker: Yes, as an anesthesiologist I like pulse oximetry. I don't know if it would have much value in the field. The other problem with the pulse oximeter, as you know, is that when you're cold they don't work. But I think if you look at the figures, like Modell's—at groups of

nearly drowned people, who come to the emergency department with a completely clear x-ray, fully awake and talking and an O_2, a blood gas O_2 of 50, I would say that that's a clear sign that you'd better watch them for a while.

The screening tests that were used by these lifeguards—they looked at, clearly, real simple things—loss of consciousness, they went to the hospital; pulseless, apnea, they went to the hospital. They like color—I'm not great on color—but they like color. Sputtering, coughing, any kind of distress they treated. Now if there was any question they sent them to the hospital.

Panelist: My only comment about pulse oximeters is that I think they are a bit of a security blanket. As an example, if someone ends up with a small aspiration of water, they will probably hyperventilate and that means their $PACO_2$ will drop, they will become alkalotic, and if you get a pulseox value of 95, that looks good. But actually, you've got to remember that you've shifted the oxygen association curve and that saturation of 95 may be at a PAO_2 that you would not find acceptable. So you have to be very careful about that interpretation because those kinds of shifts in the oxyhemoglobin curve.

So, I think that if you're doing this at beach-side or at pool-side, I don't think a pulse oximeter is going to help you. and I'm not sure it's going to change what you do in keeping an eye on them.

Mr. Huggins: One of the things we see out at the chamber is at least once or twice a year we get reports of a pulseless diver being brought to us and initial report saying the diver was pulled out. They're pulseless, citizen CPR was done on them and by the time they get to us they're awake, talking, and so forth. And I'd just like to get information, like you were talking about, even in the hospital, difficulty in finding pulses in patients who have gone unconscious because everybody I've talked to, that I've been able to contact, were absolutely dead certain that this diver had no pulse when they were being pulled out.

Dr. Brown: I want to reiterate that in the field or with a more naive examiner, pulses are easy to miss. If the victim is cold and vascularly constricted, pulses are very easy to miss. Second, many examiners don't adhere to American Heart Association guidelines by feeling at the carotid. That's the correct place to find a pulse, not anywhere else. I think that's exactly the kind of person that probably should be observed for a period of time in the hospital and not sent home. Again, thinking in a financially correct, rather than a politically correct way, you could put them on observation status and that gives you 23 hours.

Dr. Dueker: Yes, I don't know whether they had a pulse or not. There's no way of knowing. Either the people miss it and that's fine, or they were heroic, and in any event that result was good, and that's really what matters, and they assume that they did not have a pulse, that's tremendous, and then they got a good recovery, they ought to be observed.

Mr. Huggins: My concern is people's overconfidence in citizen CPR being able to help a pulseless person, bring them back.

Mr. Graver: Just two comments on that, Carl. I teach CPR recertification for several fire districts. We're talking about EMTs and first responders who get this every year They know what to do, and invariably pulse checks are of extremely short duration. So the first thing is that pulse checks are supposed to be for 5 to 10 seconds, which is forever when you think you're dealing with a dead person. Secondly, if that person is cold, the pulse check is supposed to be even longer. So I would really doubt that they're doing an adequate pulse check in the field. That would be at the top of the list.

Next, I rescued a person once that wasn't breathing and I established breathing and knew that he was breathing, but I couldn't find a pulse on him to save me, and we were in this kind of climate right here. I was recovered physiologically, It wasn't a heart pounding situation any more. Even on accident victims in motor vehicle accidents, their blood pressure is way down, so it is difficult to find a pulse on them. I would go back to saying that it's highly likely that victims may have a pulse and citizens can't find it because the pulse isn't strong enough, the responders are cold themselves, or they probably don't check it long enough.

Dr. Hattori: First I want to thank you all for this conference. It's been a long time coming and I really looked forward to it. I wish we got into a little bit of the clinical side, maybe we could do it some other time. And as far as the carotid pulse goes, I went through a residency in radiology in the 50s and we did direct carotid sticks for the cerebral arteriogram and I did maybe a dozen a week, and I still have a difficult time trying to find a carotid pulse to figure out where to stick the needle.

You mentioned PEEP. I'd like to know what centimeter of water pressure was used in the PEEP? Because that becomes important. One of the papers that came out of Miami involving children varied their pressure from 10 to 25 centimeter water and they had a 25% incidence of pneumothorax in these patients.

Until I got myself out of near drowning treatment, I had a standing order that anybody that came in semi-comatose or comatose, I wanted to see a tube coming out of their lung and one out of their stomach, because—especially in salt water—you put that endotracheal tube in them and I've

seen solid columns of water come shooting out of the endotracheal tube. And I've seen vomitus coming out of the stomach that got inhaled before they got the stomach tube down.

Another question was the use of sodium bicarbonate. Nobody's mentioned anything about that, and of course I use that pretty liberally, but there have been papers in which there have been warning about getting too liberal with sodium bicarb, so I am more careful these days.

Dr. Brown: On the PEEP issue, it's not the level of PEEP that produces trouble, it is actually the total pressure. From John Marini's work, if you're talking about non-cardiogenic edema—acute respiratory distress syndrome—sustained alveolar pressures that in a pressure control mode of ventilation will be the total pressure, about 35 cmH_2O or greater, is when the risk of a pneumothorax increases.

So it would not matter how much PEEP you applied, it's actually the total alveolar pressure. The correct amount of PEEP is the amount that gives you the desired oxygenation. And to get that oxygenation you may need to sedate and paralyze, and control the patient to get them to synchronize with the ventilator and avoid big peaks in alveolar pressure.

Again, I want to emphasize that I'm saying alveolar pressure and not tracheal pressure or peak airway pressure because the pressure that injures the lung is the alveolar pressure, that often has nothing to do with peak airway pressures as reported.

Second, I agree about vomiting. The vast majority of patients who are near drowning, swallow the water, vomit and then aspirate, so that's the reason why all those things happen.

Sodium bicarbonate in the immediate resuscitation period, I think, should probably be avoided. You've got to remember that as soon as you put bicarbonate into the system it reacts immediately with hydrogen ion with subsequent generation of CO_2. And if you cannot keep up with the ventilatory load of getting rid of that CO_2, you may actually worsen the tissue level acidosis.

If you remember, just a few years ago indwelling arterial pH electrodes and blood gas senssors were very popular. I've seen tracings in humans during this monitoring when the patient went into cardiac arrest and became somewhat acidotic. IV bicarbonate was given. There was a precipitous drop in pH until they were able to increase the ventilation to the point where the pH finally rose. So you actually may worsen things with rapid bicarbonate administration.

And if this is a lactic acidosis only, I think, unless you're dealing with something like a pH of 6.9 or less, I'd like to let nature take its own courset and simply increase minute ventilation. Anyone else?

Ms. Land: We have a high number of drownings in Arizona and I have a couple of questions. One of them is, it is widely reported that we have patients who are awake and alert in the first 24 hours, which you talked about, and then go down again, and I was wondering if there was any connection with the reperfusion phenomenon, and if so, why isn't hyperbaric oxygen used? It was tried in a post-anoxic head injury study and it's been talked about quite a bit with anoxic head injury with carbon monoxide in stopping that reperfusion phenomenon. I wanted to know if anyone had done it?

Dr. Brown: I'm not aware of any trial. Are you aware of a trial?

Dr. Lundgren: Not a trial, a single case.

Dr. Brown: I'm not aware of any trial that's looked at HBO_2 to prevent the reperfusion injury, but you may be right. It's a spiral that once the neurons begin to swell, in part the injury is reperfusion, but once neuronal swelling begins intracranial pressure rises and cerebral perfusion decreases and causes further neuronal swelling. And that part of the spiral is not a reperfusion injury. Once it starts, it becomes a positive feedback cycle. But you may be right, because initially you find reperfusion injuries that appear to be worse in the areas of the brain that contain the most iron giving more credence to the idea that you may actually have a reperfusion early on. But it can be such a diffuse hypoxic injury that it won't matter, but I know the cases you're talking about. It's not a bad idea.

Dr. Lundgren: We had the unfortunate experience I have to say of trying hyperbaric oxygen in a case that was brought in. This was several years ago, in Sweden. It was a suicide attempt that was eventually successful of a youngster of around 17 or 18 years old. And there might have been some drugs involved as well. But he was brought to us as a last attempt to do something when he had slid into coma. We put him in the chamber to see if we could somehow tide him over and do anything with hyperbaric oxygen, and in hyperbaric oxygen he woke up and was able to communicate with us. He then gradually developed pulmonary edema, slid into coma again and we inched up the oxygen pressure and he woke up again, and eventually we lost him because of pulmonary edema that couldn't be controlled.

Can I shift gear here and turn very briefly to a point in Dennis Graver's very nice presentation where I think I can add a few words, and that was the notion of oxygen being absorbed more rapidly if the victim is at depth than if he or she is closer to the surface. What I would like to do is very quickly show you a couple of slides that I borrowed from

Hermann Rahn and Albert Olszowka in our department. They show results obtained with a very elaborate circulation and metabolism model that albert Olszowka, who is very adept at these things, developed. They looked at what is admittedly an unusual situation, namely a 100-meter dive, but it doesn't make any difference in principle. What they looked at was the changes in oxygen tension in the alveolar air in such a dive and also as we shall see in the next slide, what happens during ascent near the surface.

Here, first, the oxygen pressure on a dive, on air, with a duration of 220 seconds where the ascent and descent occur at 2 meters per second.

On compression, the oxygen pressure in the lung will go up and that of course promotes oxygen delivery to the blood and then to the tissues.

And I'll get to what happens during decompression here. This is where the diver starts up, and of course oxygen pressure falls, both due to oxygen consumption, and in particular because of the expansion of the lung gas and the falling gas pressure. So that toward the end here, after 180 seconds, that is to say 40 seconds from the surface, you're going below normoxia and developing gradually increasing hypoxia. But compare this with the situation if this subject only held the breath at the surface, starting out with the same number of oxygen molecules in the lungs. And you will see that this person is hypoxic from the 40th second and all the way through—a much longer period of hypoxia. And if we look at the very last section of the graph here, in the next slide showing the last part of the ascent from the deep dive, something very interesting emerges.

Here again is the oxygen pressure in millimeters of mercury, and we have lung volume here as well. We don't have to pay much attention to that except to note that of course the lungs expand as you approach the surface. But look what happens with alveolar P_{O_2}. It falls, here it becomes hypoxic—below normoxia of 100 mmHg, and it falls—and it should fall according to Boyle's Law and the oxygen consumption included in the model—it should fall according to this line and you would end up, on breaking the surface, at extremely low P_{O_2} of 10 mmHg, certainly not compatible with consciousness.

However, the venous oxygen pressure falls more slowly, according to this line, and where you have this cross over, you actually have venous oxygen being delivered to the alveolar air from the venous blood which helps to keep up the arterial P_{O_2}. So much so, as a matter of fact, that upon surfacing from this dive, we have an alveolar oxygen pressure and arterial oxygen pressure, presumably, somewhere close to 30 mmHg, which is a number that, incidentally, coincides very well with the numbers we have measured in actual sea dives performed by elite divers.

I'm not saying that you should let your drowning victim drop to the bottom to use more of the oxygen in his lungs, obviously, but I think it's worth considering these finer points when we think about what is going on in terms of the oxygen husbandry of the body.

If we can change the subject for a moment, I would like to bring up—since we had the discussion earlier today about possible causes for the more curious cases of drowning in scuba divers where there is nothing to explain it—there's air in the bottle, decompression sickness is not involved, and the accidents frequently happen as Dennis Graver said, on ascent, right? There is an over-representation of accidents on ascent.

I would like to have us all recall the phenomenon known as alternobaric vertigo. I hold that that is probably a likely cause of drowning in these otherwise very hard to explain cases. And the reason I bring it up again is that you can say something about possibilities for prevention.

Alternobaric vertigo is vertigo caused by changing middle ear pressure. Incidentally, it's erroneously in some literature, written up as an example of barotrauma. Alternobaric vertigo is no more barotrauma than caloric vertigo is frostbite. And the proof incidentally, for that, is that you experience vertigo, a sensation of rotation as you start to ascend. If you stop and stay stable for a while and the world is spinning around you, you can go down 2 or 3 feet and it goes away. That's not trauma. You go up 2 or 3 feet again, and it's there again.

Now, in the more severe cases you can have violent vertigo and vomiting. Those who live to tell about it vomited after surfacing. Aviators whom we have interviewed also describe cases on ascent, on climb out, with violent vertigo, which if they hadn't been high enough, they claim, would absolutely have caused them to crash.

These cases can, both in divers and aviators, are almost always connected with a unilateral problem of pressure equilibration. So when the middle ear tends to build up pressure on ascent, that's what sets the stage for alternobaric vertigo.

Now what often precedes an episode of alternobaric vertigo is a pressure equilibration problem going down, and you force yourself down, you get a squeeze, the eustachian tube swells and now you have the scenario for pressure build-up which can cause this vertigo. And of course, the advice here for prevention is not to dive with a cold, and to be careful with your pressure equilibration.

Dr. Dueker: One thing is very important that Dennis discussed, but I want to give a little bit more information on this issue. The current recommendations for CPR that come from the American Heart Association–Red Cross are based on terrestrial situations in which cardiac arrest is

most commonly caused by a ventricular fibrillation. The recommendation currently is that bystander CPR instead of proceeding through A,B,C, starts with finding a phone and calling and activating the emergency medical service. This has been determined empirically to be valuable because, terrestrially, it looks like people get better survival that way than if people start resuscitation first.

This definitely does not apply in the near-drowning or immersion situation for a couple of reasons. One, even though children do get ventricular fibrillation, more probably than we used to think, they don't get it as much, and they're the ones who most commonly are involved in the near-drowning problems. That's one thing.

The other is the basis, again, of the injury. It's not primarily a cardiac injury, it's primarily a respiratory injury, leading to cardiac problems. And waiting is just not going to be good. You want to treat the near drowning before there's an arrest. I want to make sure that everybody understands that, and I am sure that you would know that in the ocean because in a typical ocean, there's no phone, and so you would know that you would have to go ahead and do things.

But in a pool situation, I can easily imagine that someone would be confused and fish somebody out of the water and then look for a phone. If there are a lot of people around, that's ideal, somebody can call while you begin to work on the patient, but this is an issue that hasn't been resolved. I thought maybe it was going to be in April 1997 by the American Heart Association—in *Circulation*. There's a nice article called "Special Resuscitation Situations".

Under "Near Drowning—Effective resuscitation is seen as paramount in determining long term survival and its quality" and so forth and so forth. "Basic life support modifications during arrest". Well, they don't talk about it other than during arrest, and there's no modification. ALS, also no modification. You wouldn't necessarily modify that.

Dr. Edmonds: A number of things have been said today that I think I would like to make some comments on, and I may be right or wrong. I've got maybe half a dozen very brief points.

First, I think, Chris, you took umbrage with me because I was the one that said that I think that near drowning should be defined as having loss of consciousness. If you don't do this, then where are you going to draw the line between the small aspiration, slightly larger aspiration, medium aspiration, moderately medium or slightly larger aspirations to finally ending up with someone drowned? And so really, that was the only reason why we included the loss of consciousness as a sort of cut-off point for the terminology. Of course there's no way that those three syndromes are separate. They all merge. But you've got to do something.

The other thing is that if you sit in an academic chair, it's all right, you can talk about loss of consciousness, but if you actually look after divers, a lot of them who aspirate have impaired conscious state, and often it depends on whether they're standing up or sitting down, you know, as to whether you'll see the clouding of consciousness. So it's a complex sort of thing. And I think we should have mentioned that syncope on standing is one of the ways of perhaps moving it from an aspiration syndrome to a near-drowning syndrome.

The next thing I was going to mention is that someone, I think it was Bill Hamilton or it may have been Dennis, said something about the difficulty of putting in a demand valve in someone who has convulsed with oxygen toxicity. And there's no doubt that's true, there's no way you can get a demand valve in their mouth. But you know, we're having a workshop on near drowning and that's a far more common problem, that with the massive spasm they have with hypoxia, there's also no way that you can get that demand valve into their mouth. So it's the same concept, we have the same problem with near drownings as you do with convulsions, really.

The next thing was in regard to what Claus said, though of course I disagree with him entirely about terminology, I think that is just another barotrauma, but that doesn't matter. We both agree that it is very important, and that's what matters. But just to add on to what Claus has said, there are lots of other causes of loss of consciousness and of vomiting underwater that we have not mentioned here today, and that was not the purpose, in any case, of the workshop. There are many others that we could name if we wanted to. So, please, I would hate people to get the impression that we have looked at loss of consciousness underwater. We have not looked at that particular problem. That should be the subject of another workshop.

This next I'd like to ask Dr. Brown about, because in the field we had a real problem with the near-drowning cases producing an awful lot of frothy red sputum, and it seemed to me that there are lots of the people who initially had to treat these folk, kept trying to suck it out when I think the people there would have been better just by forcing oxygen down, and we ended up just making them ventilate through the frothy sputum. I'm not quite sure if we were right in telling them to do that and later on I'd like to get your opinion on that whether you should spend too much time in the field, to try and suck out this, because the sputum just keeps coming and coming and coming.

And I am reminded of Maury Campbell at one stage when we were really treating a lot of near-drowning cases,

not in an intensive care unit, but pretty close to where it was happening, and we were lucky to have Maury Campbell come down and I said to him, "I need advice because I can't understand all these treatments we're talking about or the metabolic things we should be trying to tackle if we should", at that stage it was the things like the bicarbonate and whether we should be using respiratory stimulants and all those things.

And Maury Campbell said to me, "Look, mate..." well, he didn't say mate because he's English, he said, "Look, if you can keep their arterial oxygen levels normal by any way you can, everything else will fall into place for you".

So could I just ask you to comment, if you would, on what you should do in the field about trying to suck out all this sputum?

Dr. Brown: You should initially try to suck it out, because first of all you may be dealing with Big Mac and fries down there as well as sputum, and consider the fact that the victim may have aspirated foreign bodies that came out of the stomach or from the bottom of the ocean. But give a good effort to try to suck it out. But number two, go ahead and apply positive pressure ventilation, and that is going to impede the rate of pulmonary edema formation.

About the oxygen, I completely agree. If you get the PaO_2 is increased and the blood pressure is in normal range, the rest of this is going to fall into place. The caveat would be if oliguria occurs later and the patient suffers abrupt acute tubular necrosis, then you have to adjust the electrolytes, but that's an unusual situation.

Mr. Graver: I'd like to comment on field suctioning for near-drowning people. I'm on a crusade to encourage suctioning because it's not there. We need at least manual suctioning, hand powered type of suctioning. We don't need anything that's really complex or mechanical. A turkey baster would be the low end of the totem pole, but they do have other hand-powered types.

If we suction people, the protocol is for no more than 15 seconds followed by hyperventilation, and we should do it more than once to keep them cleaned out. There's also available if you have enough people and if the people know what they're doing, use of cricothyroid pressure, a Sellick maneuver to help keep down the rest of what's in there to keep it out of the airway during resuscitative attempts.

Just one more thing about the inverted ascents, and I hope that no one got the idea that that was an endorsement of that, that was just a historical perspective. There are problems associated with it logistically. People tried it in the past and they think it's a clever idea, but in reality it's not practical. You can get the air trapping, or the same effect for the airway, with the horizontal face down position. I discourage inverted ascents.

Panelist: I have a quick question for Bill Hamilton. We were talking about this at the break. That is a patient, a scuba diver who comes up with loss of consciousness in a suspected near drowning, but you're going to have to make a good guess on which one -- who had the arterial gas embolism in addition to the near drowning. Do you have any suggestions on how to make a decision who gets a Table 6?

Dr. Hamilton: Table 6 is sort of a catch-all for all types of situations, but I would advocate using it where you have a significant amount of missed decompression that you can't make up with surface oxygen.

Panelist: No, no. I'm talking about gas embolism from pressure, barotrauma.

Dr. Hamilton: I would follow standard rules on that. I'm not sure I understand the question. If somebody has an embolism, then you'd treat him. If you can do it right away, use a Table 6A, otherwise later on you'd use Table 6.

Panelist: (not at mike) . . . near drowning . . .

Dr. Hamilton: Do you have a chamber? Are they at the chamber? Your first obligation is to get the patient breathing again, and then he will usually regain consciousness. A lot of times an embolism recovers spontaneously. You still may have a problem, but you often get a prompt recovery, or at least partial recovery. Recovery of consciousness. Really, the only way to detect an embolism is from the history. If you really don't know where they've been, you have a hard time determining that. And so you have to do your best to determine what happened, and then if the patient has been through a profile that could cause an embolism, I think you more or less have to assume that is what they have, if they don't wake up promptly.

Panelist: But a profile doesn't have anything to do with an embolism in 3 feet of water.

Dr. Hamilton: Well, if they have not been through the right kind of profile, then an embolism is unlikely and you might not have to worry about it.

Dr. Edmonds: I think the question's a genuine one. I've seen too many of these cases exactly as you described, and you really don't have a clue as to whether they've got an air embolism or not. What you do know is that they have certainly got aspiration, and they could well have an embolism as well. And wh I at do you do with these? I can tell you it's terrible to treat. What we've done, and sometimes it's been successful, we treated the embolism initially by taking them down to something like 18 meters, but not adhered to the protocol if, after an hour or so, it doesn't look as though we're getting anywhere. It's hard to treat these people from the respiratory point of view in the

chamber. After about a half an hour to an hour, if we're not getting anywhere, I'd prefer that the patient be in the hospital and treated for the near drowning. We will at least know we can do something about that. Their embolism may or may not be there. The near drowning is there, so at least let's take them back to the hospital. That does mean bringing them back up after perhaps an hour in the chamber. If you keep them down in that chamber for 6 hours and just myopically think it's embolism plus near drowning, you end up keeping that person there for days while they gradually die.

Dr. Hamilton: I agree, because if it is embolism and you put them in the chamber and treat it, you're going to get a response. If you don't get a response, then that is not the right treatment.

Dr. James: In that scenario, who is concerned about the additional toxic insult of the oxygen in the chamber to the already traumatized lung?

Dr. Brown: I don't think that's as issue in the first few hours. As a matter of fact, some information suggests that a previously injured lung is actually protected against toxicity from a high FiO_2. The times that I've worried about O_2 toxicity is in people who have received bleomycin previously or during a Table 7.

Mr. Huggins: We see people who end up near drowning, embolism combinations at our chamber more frequently than I'd like to, but what's looked at by our physicians is level of consciousness and whether there's a change upon pressurization, and if there isn't, then we work at an evacuation routine to get them out to the mainland, to the hospital. But most of the cases that I've seen since I have been there have gotten initial improvement, level of consciousness has improved, and then we may be getting lung involvement later on with difficulty breathing, very rapid, shallow breathing as the treatment for embolism progresses and some of those cases have been brought out early to be taken back to the hospital for management of the lung problems.

Dr. Brown: Are you suggesting that there's oxygen toxicity being manifested?

Mr. Huggins: No, not oxygen toxicity, possibly pulmonary edema, even with the higher oxygen pressures, you're still having respiratory distress.

Dr. Brown: Yes, from stiff lungs. It sounds like the consensus is that a lot of people would err toward the treatment of gas embolism if that was a strong suspicion.

Panelist: I want to address what Dennis was talking about the suction by the tube. There was an article out about a year or two ago saying that you've got to get an endotracheal tube in because they showed that sticking the suction tube down you mainly cleared up the laryngeal region but you really didn't get down into the trachea.

Mr. Graver: You insert suction no further than you can see. It is advanced life support (ALS) if you go beyond that.

Dr. Brown: And I think we were talking about intubated patients, right?

Mr. Graver: Right.

Attendee: I'd just like to throw something out. I don't know whether the panel would agree with this, is rather than have like artificial barriers saying near drowning, AGE, arrhythmias, sudden death syndrome, would it be convenient to talk about these in future forms or when you're talking about near drowning? The very good description you gave of the near drowning victim is well described because there's so many near-drowning victims on the surface, as there are many lakes and rivers and what not.

The same may apply to those that are under water, but we don't have that same description, that same panic that occurs under water that you can see above the water. Therefore, we have an individual who is not going to do some of the things you said, like release the weight belts as Dr. Edmonds so clearly showed that we have a panic situation setting up in the divers in better than what? 65% of the time; it's lack of preparation in those divers.

So the diver has several options. One is to make—he's already made one mistake, he's ill prepared for his dive, such as having the gas off or what not, flooding the mask and unable to cope with that—he makes an urgent ascent. He may successfully complete that, which is the end of the drill, he may make that ascent and embolize while holding his breath because we see that they no longer rationally think, or they may not even have the reserve to do that, and go on to drown.

And on top of that we've already increased the after load, preload, he may already have had coffee, Sudafed, some catechols. In addition, we're putting this individual under stress and also increasing both, making that person both hypercarbic and hypoxic at the same time, perfect ingredients for a situation to see some very malignant arrhythmias.

So in the future I think you have to talk about these together. We don't necessarily have to talk about the treatments, but I think it might be a suggestion in the future.

Dr. Brown: Well, thank you all for coming. This concludes the workshop.