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Investigation of Diving Fatalities for Medical Examiners and Diving Physicians

Symposium Proceedings

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OPENING REMARKS

"The main purpose of diving fatalities monitoring is to acquire knowledge necessary for preventive intervention that will reduce the risk of deaths in diving. The first step toward success is to acknowledge that mishaps are not accidental but rather a consequence of root causes that are preventable. Medical examination should aim to establish possible root causes in individual cases as well as possible risk factors identified at a group level. Preventable causes are those that precede drowning and thus the ME should look beyond drowning."
- Petar Denoble, MD, DSc, Chairman

"A meticulous, thorough postmortem examination with toxicology is essential to investigating any diving related death. A few modifications in protocol will maximize the information gleaned from the autopsy and the autopsy findings must be put into the context of the dive profile and the circumstances surrounding the death."
- James L. Caruso, MD

"Of all the divers a Medical Examiner might encounter, rebreather divers may be the most enigmatic. I know at least one diver who asks his rebreather before every dive 'How will you try to kill me today?' His rebreather might just answer through a perversion of Elizabeth Barrett Browning's poem by saying, 'How do I kill thee? Let me count the ways...' We will enumerate all the ways a rebreather can send even a cautious diver to the Medical Examiner's table. Seldom is the root cause of death obvious on autopsy."
- John Clarke, PhD

"Diving conditions and diving equipment may cause or contribute to a diver's death, which makes field investigation and equipment preservation critical. Simple evidence preservation techniques and documentation can make the difference in understanding a diver fatality, but those in the field don't have to be divers or equipment experts in order to preserve information and items for later examination.

Check your ego at the door, because we're all smart here: pathologists, diving medicine doctors, equipment experts, scuba instructors, lifeguards, police, Coast Guard personnel, recovery divers, and death investigators each have their own skill sets that can either conflict and muddle a diver death investigation if they don't communicate, or complement each other well to form an expert review panel. A model committee formed in San Diego brings together representatives from all of these groups for review of diver deaths to maximize the knowledge and experience of everyone for the benefit of the investigation and to promote safety for the local diving community."
- Craig L. Nelson, MD

"The correct identification of the actual cause of death in divers allows us, as diving physicians, to try to deal with the root causes of these events and therefore help prevent such deaths in the future. There are also important legal ramifications to correctly identifying the cause of death. Emergency Department physicians probably see more sudden cardiac deaths than any other specialty thus, this presentation will include a discussion of sudden cardiac deaths in divers from the point of view of an experienced diving medicine and emergency medicine physician. We joke that all too often the average Medical Examiner's response to a diving death is - He's wet, he's dead; he drowned - , but there is much more to it than that."
- Tom S. Neuman, MD, FACP, FACPM, FUHM

"As we evaluate for alternative causes of death in divers, perhaps the most likely culprit is sudden cardiac death, either from arrhythmia or ischemia. The post mortem evaluation of these patients necessitates an interpretation of their heart weight and coronary arteries. The purpose of this talk will be to highlight the problems of so-called "normal" heart weights, as well as the pathophysiology of sudden cardiac deaths from arrhythmias and ischemia."
- Charlotte Sadler, MD

"I have been requested as an expert witness in diving fatalities for the Coroner's court in the past decades in Western Australia, I have to admit that I have seen mostly ''less than helpful" autopsies for the diagnosis of the cause of death. Sadly, the pathologists do not seek out diving physicians for assistance or advice, and most of them are not divers and are usually not familiar with compressed air diving injuries or deaths. They tend to perform standard autopsies for any fatality rather than to seek out information specific for diving. No consideration appeared to be given to dive profiles, circumstances surrounding the deaths, and little liaison with the Water Police who do the dive equipment testing. And because the victims are found in water, the most common cause of death was labelled as' 'immersion" or ''drowning".
- Robert Wong, MD

SYMPOSIUM SPEAKERS

Petar Denoble, MD, DSc

Dr. Petar Denoble is Vice President of Research at Divers Alert Network (DAN) and leads the DAN Injury and Fatality Survey. He joined DAN twenty-two years ago after a career in Naval and Diving Medicine in the former Yugoslavia.

At DAN, Dr. Denoble prospectively studied the exposure and outcomes in recreational scuba diving and introduced a root cause analysis in scuba fatality monitoring. His current research focus is on cardiac risk factors for scuba fatalities and efficacy of preventive interventions for human errors.

James L. Caruso, MD

Dr. James Caruso was recently appointed the Chief Medical Examiner and Coroner for Denver, Colorado after completing over 29 years in the United States Navy. Dr. Caruso's Navy career included certification and tours as a Navy Diving Medical Officer, Navy Flight Surgeon, and Armed Forces Medical Examiner. He was the Deputy Chief Medical Examiner for the Department of Defense and also the Regional Armed Forces Medical Examiner for the Pacific.

Dr. Caruso is board certified in Anatomic, Clinical, and Forensic Pathology. In addition to his training in Pathology, Dr. Caruso completed a fellowship in Diving and Hyperbaric Medicine at Duke University Medical Center and achieved board certification through the American Board of Preventive Medicine.

Dr. Caruso has numerous journal articles, book chapters, and formal presentations to his credit. He is a member of the DAN medical staff as a consultant in Forensic Pathology and Clinical Diving Medicine. In addition to Dr. Caruso's contributions to the technical diving medicine literature, he is the current medical editor for Sport Diver magazine and formerly had the same role for Asian Diver magazine.

John R. Clarke, PhD

For 23 of his 35 years working for the U.S. Navy as a respiratory physiologist and physical scientist, Dr. John Clarke has directed numerous forensic diving equipment investigations, authored investigative reports on the most complicated diving accidents, and served as a subject matter expert in Coast Guard investigations and at trials.

He lectured on Rebreather Accident Investigations at DAN's 2008 Technical Diving Conference, and is Chair of the Diving Control Board for the Florida State University Advanced Scientific Diving Program and Crime Scene Investigation Program. He's been the Scientific Director of the Navy Experimental Diving Unit (NEDU), since 1991. He is a prolific blogger writing on diving, aviation, nature, science and technology at http://johnclarkeonline.com. He is also a published novelist writing on diving, diving accidents and deep sea physiology.

Craig L. Nelson, MD

Having recently taken a position as an Associate Chief Medical Examiner for the State of North Carolina, Dr. Craig Nelson was previously a Deputy Medical Examiner for the County of San Diego. With a background including the 1996 Rolex Our World Underwater Scholarship and experience in underwater body recovery, he joined the San Diego Lifeguards and helped form the San Diego Diver Death Review Committee to further diver death investigation and promote local diver safety. Now closer to DAN, he hopes to continue work in diver death investigation.

Tom S. Neuman, MD, FACP, FACPM, FUHM

Dr. Tom Neuman is a co-editor of the 5th Edition of Bennett and Elliott's Physiology and Medicine of Diving that is one of the most widely used textbooks in the world in the field of undersea medicine and he was the Editor-in-Chief of the Journal of Undersea and Hyperbaric Medicine. Dr. Neuman is on the American Board of Preventive Medicine committee responsible for formulating the Board Examination in Undersea and Hyperbaric Medicine. The UHMS has awarded Dr. Neuman the Craig Hoffman Memorial Award for contributions to diving safety, the Merrill Spencer award for Lifetime Achievement, and the Albert Behnke Award for outstanding scientific contributions to advances in the undersea biomedical field. In 2011, Dr. Neuman was selected to be the DAN/Rolex Diver of the Year. Dr. Neuman continues to do research in gas exchange and exercise physiology and the causes of diving fatalities.

Charlotte Sadler, MD

Dr. Charlotte Sadler is currently in fellowship in Undersea and Hyperbaric Medicine at the University of California, San Diego. She is also on faculty in the Department of Emergency Medicine at the University of California, San Diego and is a member of the San Diego Diver Death Review Committee.

Robert Wong, MD

Dr. Robert Wong is at present Emeritus Consultant at the Department of Anesthesia & Pain Management, Royal Perth Hospital; and Emeritus Consultant in Hyperbaric Medicine, Fremantle Hospital. In his professional career he served in various leadership roles in military, commercial and recreational diving, including: Consultant in Underwater Medicine to the Director General, Naval Health Services and to the Royal Australian Navy. Diving Medical Consultant for Australian Pearling Industry, Examiner in Physiology for Australian & New Zealand College of Anesthetists, Examiner and Chairman in Diving & Hyperbaric Medicine, Australian & New Zealand College of Anesthetists.

Among numerous awards for his work, Dr. Wong received the UHMS Craig Hoffman Memorial Award for major contributions to safety and health of divers, UHMS Oceaneering International Award for contribution of increased productivity and performance of divers, Western Australian Fishing Industry Council Safety Award for contribution to safety in pearl diving, Australian & New Zealand College Council Citation for contribution to education in Western Australia, and the Australian and New Zealand College Medal for outstanding contribution to diving and hyperbaric medicine. As editor, reviewer and writer he contributed to several workshops, scientific journals and books about diving medicine.

Why It May Not Be Drowning

Tom Neuman, MD, FACP, FACPM, FUHM

Introduction

To begin, I would like to thank Dr. Denoble for inviting me to speak on a subject that has interested me for many years. The issue of why divers die is not at all clear cut and it has always confused me why good swimmers with air on their backs, swimming back to a boat can suddenly "turn turtle" and have "drowned." Yet we have all been taught, and probably even have taught others, that the most common cause of death in divers is drowning. Certainly, if one runs out of air and is in an overhead environment or is trapped under water by being entangled, drowning makes perfect sense. But how do we explain the death of an experienced diver who still has air in his or her tank, and prior to being found dead gave no evidence of any difficulty to his or her buddy. It seems that we may be getting the diagnosis wrong and perhaps there is another cause of death that we have been overlooking or ignoring in these cases.

But before one ventures out trying to find the "correct" diagnosis, we should address the question of why is it important to get the diagnosis right in the first place. There are a variety of reasons that we, as divers and professionals interested in the field of undersea medicine, should be interested in establishing the correct diagnoses in fatal diving incidents. First of all, if divers are truly "drowning" then should we be spending time and resources trying to teach divers to swim? But if there are other diagnoses (such as cardiac disease) then perhaps we should be allocating our resources to finding measures that can be taken to try to prevent these deaths. There are also other societal issues that arise when a diver dies. Most individuals carry life insurance and frequently there is a different payment if a death is accidental rather than natural. There are also potential costs to the diving industry and, therefore, ultimately to us as divers if a death is erroneously ascribed to drowning. If an individual "drowns" while on a dive trip, the very word carries the implication that with better supervision, training, or a speedier rescue, that the individual could have/would have survived. In our society it is no surprise that most diving fatalities result in litigation and when "drowning" is the cause of death, that alone creates the impression of fault. Finally, when a death is improperly ascribed to drowning, limited resources are potentially squandered investigating such an event. The coast guard, police, sheriff's, etc. all might have a very different view of a death investigation if the medical examiner or coroner ascribe a death to natural causes as opposed to an accidental drowning.

So if we entertain the possibility that many of these "drowning" deaths are really something else, we must ask the question, why do pathologists so often ascribe in-water deaths to drowning? The answer to that question requires the appreciation of the culture and philosophy of forensic pathologists as well as an understanding of the limitations of anatomic findings at autopsy.

Culture and Philosophy

Perhaps the most important reason we are told that most diving related deaths are "drownings" can be found in a quote from the 2002 National Association of Medical Examiners (NAME) Guide for the Manner of Death Classification. Quoting directly, "When a natural death occurs in a hostile environment, as when someone has a myocardial infarction while swimming, there is a likelihood that the person was alive when the face became immersed (i.e. the person was still alive while in the hostile environment), preference is usually given to the non-natural manner unless it is clear that the death occurred before entry into the hostile environment." many of us have previously joked that the pathologist's mantra is "He's wet; he's dead; he drowned." Or to put it in a more easily understood statement; "He went into the water alive; he came out dead; therefore he drowned." From my own experience in Southern California, I have seen that the Los Angeles County Medical Examiner's philosophy is just that of the NAME. Unless there is a "smoking gun," diving deaths are routinely ascribed to drowning, regardless of statements of eyewitness observers or other circumstances that for all practical purposes make the diagnosis of drowning impossible. I have seen one chief medical examiner of a southern California county, when asked how he established the diagnosis of drowning in a commercial hard hat diver, state, "He was in the water, wasn't he?" When coupled with a general unfamiliarity with diving, it is not surprising that the recommendation of the NAME is followed and the default diagnosis of drowning is given.

Additionally, there is the issue that water is a "hostile environment." Philosophically, this appears to mean that had the event occurred on land, the individual would have likely survived. "But for" the hostile environment, the death would have been considerably less likely to occur or may not have occurred at all. Clearly, in some circumstances this is true. If one has a generalized seizure that causes unconsciousness on land, the likelihood of death is quite small. However, if one has a ventricular arrhythmia that causes a loss of circulation in water, the likelihood of survival would be quite an unlikely event. Indeed looking at "all comers," neurologically intact survival from an out of hospital cardiac arrest (OHCA) is somewhere around 4-6%. [1] Outcomes of chest compression only CPR versus conventional CPR conducted by lay people in patients with out of hospital cardiopulmonary arrest witnessed by bystanders; nationwide population based observational study.[1] If one looks at the likelihood of neurologically intact survival from the most common cause of OHCA (i.e. a bradyasystolic arrest) it is essentially zero. It is true that a "hostile environment" prevents or reduces the likelihood a resuscitative effort can be undertaken; however, "if a cardiac arrest occurs in backcountry wilderness far from medical care and there is no resuscitation, it is still considered a natural death. Thus proximity to medical care cannot and should not be a consideration in determining the cause and manner of death; it is not the event that led to the death."[2]

Finally, there is simply the matter of being overworked and then commencing an autopsy with an already established bias in your mind concerning the final diagnosis in the case. I will quote a well-known forensic pathologist and a medical examiner who is familiar with diving fatalities, "… but there is also laziness in my profession whereupon they just don't want to take the extra time to figure out WHY this person drowned, IF in fact they really did. Far easier to put down 'Drowned while scuba diving' and move onto the next case."

Limitations of Anatomic Findings

Unfortunately, there are very few (if any) findings that definitively establish the diagnosis of drowning; however, there is one finding that essentially rules out the diagnosis of drowning. Aspiration (of the fluid in which the drowning takes place) is the hallmark of drowning. Thus, completely normal lungs at autopsy without any evidence of aspiration greatly reduces (if not eliminating entirely) the likelihood of drowning. Evidence of aspiration is necessary to make the diagnosis of drowning, but it is not sufficient to make that diagnosis.

Elevated lung weights or evidence of aspiration do not establish the diagnosis of drowning. Elevated lung weights are routinely found as artifacts of resuscitation. These elevated lung weights can be simply due to CPR as well as gastric aspiration that frequently occurs in individuals undergoing resuscitation.

Furthermore, "agonal breathing" occurring while underwater cannot be distinguished from the aspiration of seawater that results in a drowning. Although you may have been taught that "agonal breathing" only results in small breaths incapable of producing any significant aspiration, a video clip from "Good Morning America" of John McSherry collapsing on opening day clearly demonstrates just how large the tidal volumes during agonal breathing associated with cardiac arrest can be.

Thus, because of artifact and terminal aspiration, examination of the lungs in the vast majority of cases does not provide definitive evidence of a drowning. To further emphasize that point, the following graph adapted from the work of Zhu[3], clearly demonstrates how much overlap exists in lung weights at autopsy between victims of drowning and victims who suffered a sudden cardiac arrest on land (see Figure 1).

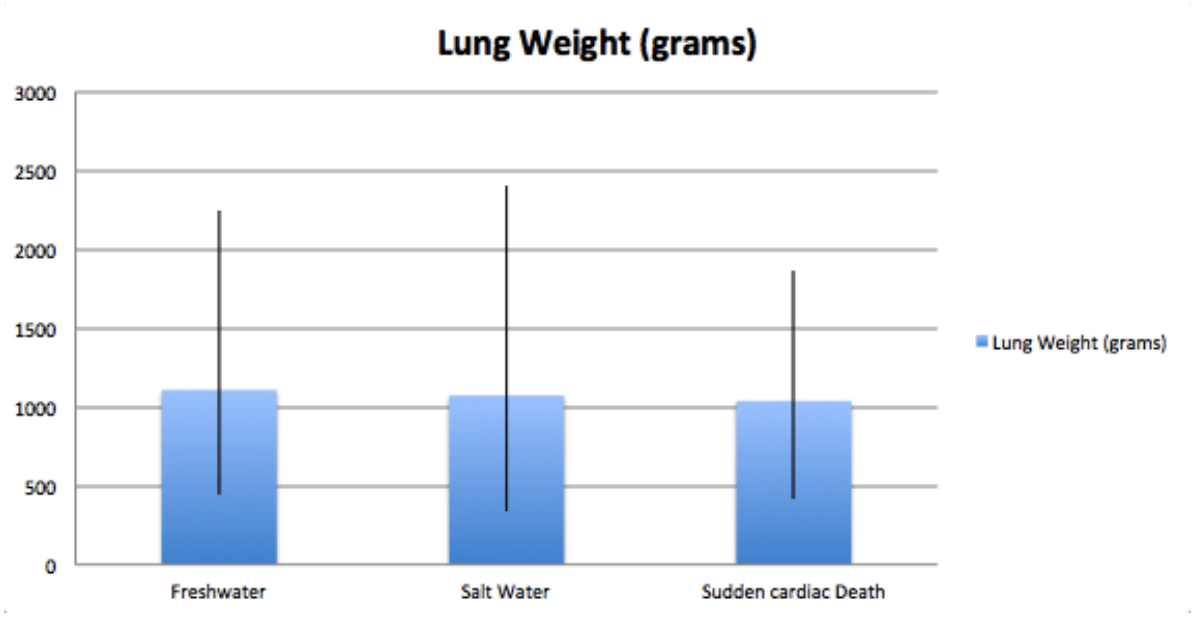

FIGURE 1. The overlap in lung weights from drowning (whether in fresh water or sea water) and sudden cardiac death is so large that one cannot make a useful determination as to their cause in a diving fatality.

Similarly, fluid in the sinuses is not definitive evidence of drowning either. It appears to be more of a consequence of immersion than of actual drowning. Although most individuals who drown do have fluid in their sinuses, slightly more than 50% of those who do not drown also have fluid in their sinuses.[4]

Thus, there is no anatomic finding that definitively identifies the cause of death as drowning or, for that matter, which completely rules out the diagnosis of drowning. There are of course exceptions to every rule and there are exceptions to this as well. As mentioned previously, completely normal weight lungs without any evidence of aspiration greatly reduces the likelihood of drowning and should cause a careful evaluation for another cause of death. There is no such thing as a "dry drowning."[5] On the other hand, a foam column from the victim's nose and mouth associated with large amounts of foam in the upper airway, trachea, and large bronchi, associated with lungs that are very heavy, strongly suggest that the victim did die of drowning.

Thus, without definitive anatomic findings, one is left with critically evaluating the circumstances surrounding the victim's death. This includes interviews with first responders, the buddy, other divers and eyewitnesses. Dr. Nelson will go into the importance of these interviews in much greater detail, however one must realize one of the most important aspects of establishing the diagnosis of drowning are the details of the event. Obviously, the victim has to have been in the water. In this regard I am not joking. I once saw an autopsy of an individual who died approximately half an hour after he had gotten out of the water. He was still in his wet suit when he suddenly collapsed and could not be resuscitated. The diagnosis was delayed death subsequent to near-drowning. But aside from absurdities such as this, a history compatible with drowning should be obtained if possible. Drowning is an event that takes minutes (not seconds) whereas a sudden cardiac death (SCD) is nearly instantaneous. The same video from GMA mentioned previously demonstrates just how quickly an individual goes from functioning normally to pulseless, apneic collapse in the setting of a SCD.

The Differential Diagnosis

It is not my intent to cover the entire differential diagnosis of death in the water. Instead, I will just focus on two of the "usual suspects." The first is arterial gas embolism (AGE). Although a legitimate concern, in my experience, a spontaneous (i.e. sometimes called "undeserved") gas embolism is a very unusual event. I certainly have seen such cases; however, they are extremely rare and I have never seen a fatal spontaneous gas embolism. Most frequently AGE is associated with a panicked and breath hold ascent. Frequently, this is caused by an out of air situation but occasionally some other event results in this behavior. In these cases the victim suddenly surfaces and may be conscious for a period of time sufficient to cry for help. They then become pulseless and usually apneic. This scenario occurs in approximately 4% of all cases of AGE. In this situation however, the autopsy findings can be definitive.

But before going into the actual autopsy findings, it is instructive to review the mechanism of death associated with AGE. It had been hypothesized for many years that the mechanism of death in AGE was either a reflex arrhythmia from brainstem air embolism or an arrhythmia induced by coronary artery air embolism. Air filling of the central vascular bed (i.e. "vapor lock") was rejected as a mechanism based upon dog studies done decades ago wherein air was injected directly into the left ventricle and it was observed that cardiac arrest did not occur. This was further reinforced by placing a very compliant balloon into the left ventricle and filling it with air and then also being unable to induce cardiac arrest (see Figure 2).

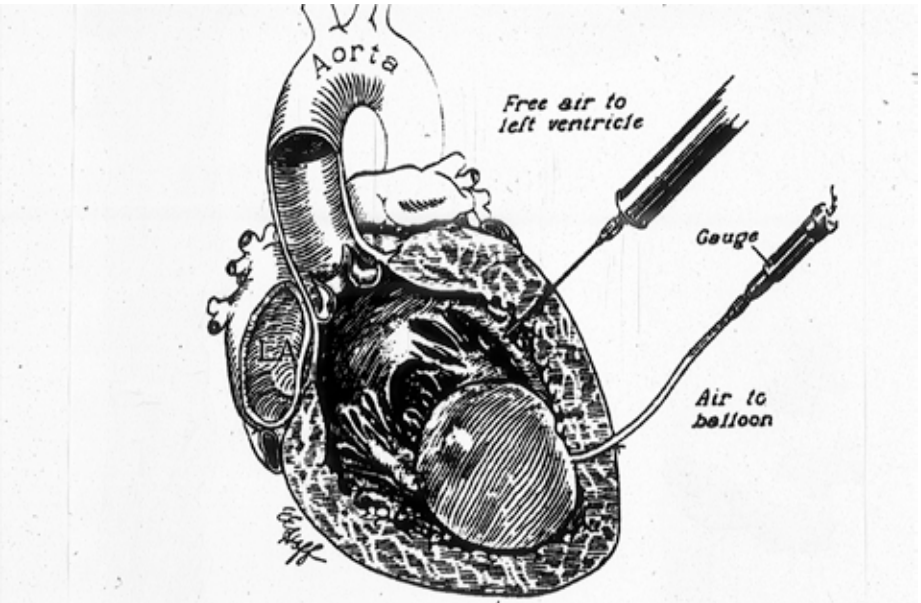

Fig. 2. Diagram of experiment contrasting the effects of free air and contained air in the left ventricle.

Figure 2. Diagram of experiment contrasting the effects of freee air and contained air in the left ventricle.

There are, however, very strong reasons to revisit whether the former two mechanisms make physiologic sense. First of all, the course of human stroke victims is not that of nearly instantaneous death. Secondly, in all animal experiments, in order to produce death, multiple embolizations are required and then death does not ensue until minutes after ventilation stops. Thus, the clinical course of death from AGE is not the clinical course observed in strokes in humans nor is it the course of death as seen in animal models of AGE. If one prefers to invoke coronary artery air as the mechanism of death in AGE, similar problems exist. Unfortunately we now have a considerable experience of the effect of accidental air injection into the coronary arteries due to misadventures in the cardiac catheterization laboratory. When such an event occurs, sudden death has not been described. Rather, chest pain and a current of injury are noted on the EKG.

It is only relatively recently that radiographs were able to be obtained on victims of fatal AGE during their attempted resuscitation (The majority of published series of AGE's had occurred at the top of submarine escape training towers where radiography was not available). With the availability of such radiographs, it has become clear the mechanism of death in many (if not all) cases of fatal AGE is complete filling of the central vascular bed with air, which then may be turned to foam by the ineffective (pulseless) continued beating of the heart.

Figure 3 is just one of many radiographs that we have obtained in cases of fatal gas embolism.[6] Thus, given appropriate circumstances and the finding of large amounts of air and foam within the heart and great vessels (arterial and venous), the diagnosis of arterial gas embolism is reasonably established. It is worth stating that the amount of gas seen in Figure 3 and in similar cases is not compatible with simple post-mortem putrification; nor is it compatible with the amount or distribution of gas that is seen as post mortem effervescence that is from absorbed gas secondary to usual diving activities.

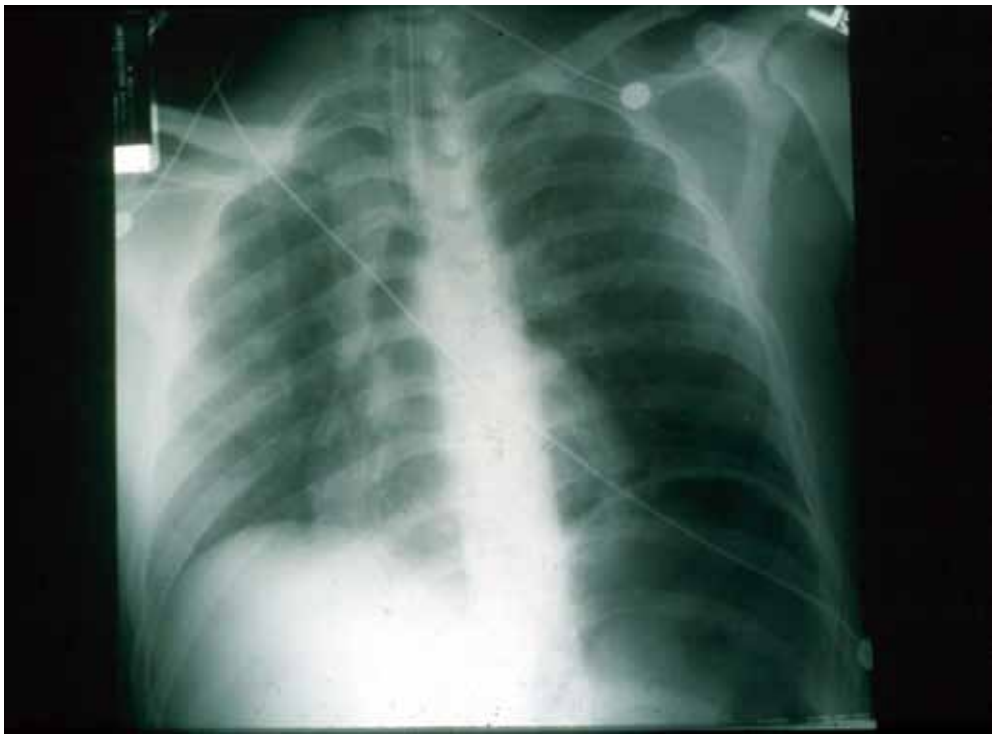

Figure 3. A case of fatal AGE. Air is filling the heart, aorta, aortic arch, axillary vessels, and the carotid vessels is clearly visible. Pneumomediastinum can also be appreciated along the border of the LV and below the left pulmonary artery.

The other "usual suspect" to be discussed in depth today is that of SCD. This is to be covered in detail in the following presentation by Dr. Sadler; however, I will give you a few introductory remarks where I put on my hat as one of the previous Editor in Chiefs of the Journal of Undersea and Hyperbaric Medicine. One of my most important duties in that position was reviewing manuscripts for sound methodology so that serious errors of synthesis did not get published. It is in this area that I would like to make a few remarks.

Although SCD can occur in individuals with completely normal hearts, it is far more likely to occur in individuals with abnormal hearts. It then follows that in order to assess whether a heart is abnormal at autopsy one has to know what a normal heart is. Unfortunately, almost all observational studies of heart weights in autopsy studies suffer from a variety of severe selection biases giving a false impression of what a normal heart can weigh. There are many reasons for such biases. In accident victims, alcoholics are over represented as are drug addicts. Both of these conditions can lead to enlarged or hypertrophic hearts. Similarly in a group of accident victims, individuals with chronic diseases are generally not excluded. Thus the 30% of Americans who have hypertension and potential left ventricular hypertrophy (LVH) will be included in the sample group. Even if one attempts to exclude victims with any known disease, the 7-10% of Americans with undiagnosed hypertension will still be included in the sample group. Such a selection bias leads to a skewed distribution of the normal weight of a heart. Such a bias is not insignificant. LVH produces an increased hazard ratio for sudden death and as little as 50 g/m increase in left ventricular mass also increases the risk of sudden death.[7]

Summary

In conclusion, it is my opinion from my experience investigating diving fatalities for now approximately 40 years, that a large number of deaths in scuba divers that are ascribed to drowning are in fact due to other causes; specifically SCD and to a lesser extent AGE. In the latter case, I believe there is room for improving training and there is room for better hardware. It is unclear to me however how the incidence of SCD occurring in the water can be easily reduced without absurd demographic limitations. In reality, there are actually very few deaths associated with the sport of diving in relation to the number of active participants and until we are in a position to predict who is at risk for SCD at all, whether or not they are diving, I do not see how we are in a position to determine who is at risk while they are diving.

Thank you very much for your attention and I would be pleased to now try to answer any questions.

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Looking for Preventable Causes of Death

Petar Denoble, MD, DSc

Divers Alert Network's fatalities monitoring program is the major source of information about recreational scuba diving injuries in the USA and in the world.[1, 2, 3] While the statistics of scuba fatalities are always sought after, there is less awareness of injury prevention which is a more important scope of DAN's program. Scuba fatalities that occur in an underwater environment may be perceived differently than fatal injuries occurring in land-based recreational activities or falls that occur at home; they are, nevertheless, part of the same global health problems high on the list of strategic public health prevention goals.[4, 5] Diving injuries do not make the list of the most common fatal injuries in the general population; but for the exposed subpopulation, their prevention is of great interest.

Injury research

In general, for prevention to be successful, it must be evidence based and thus depends on injury research which includes data collection, identification of causes and risk factors, development of preventive interventions, implementation, and follow-up evaluation as listed in Table 1.

Table 1: Injury Research

Surveillance (data collection): What is the problem?
What are the most common and most severe injuries?
Who, where, when, what and how?
Risk factors identification (analysis): Why it happened?
Develop causal models for homogenous subsets of injuries
Find changeable factors that will reduce injuries (control for confounding factors)
Develop preventive interventions: What works?
Change causal factors: remove hazards, protect body, improve processes, improve equipment
Implementation: How do you do it?
Follow up: Evaluate the effectiveness of an intervention
In a realistic settings, controlling for "other factors

While this workshop is addressing only medical investigation and some risk identification aspects of injury research, it has to be guided from the outset with the ultimate purpose in mind and that is the prevention of injury and death. Thus, when we are looking for causes, we mean the preventable causes.

Science of injury prevention

The science of injury prevention shares some basic principles with the epidemiology of diseases. The classic triad of epidemiology considers the environment, the host and the agent. How it is applied to injuries, is shown in Figure 1.

The injury is defined as a damage to the body produced by abnormal energy transfer or interference that have relatively sudden discernible effects.[6, 7]

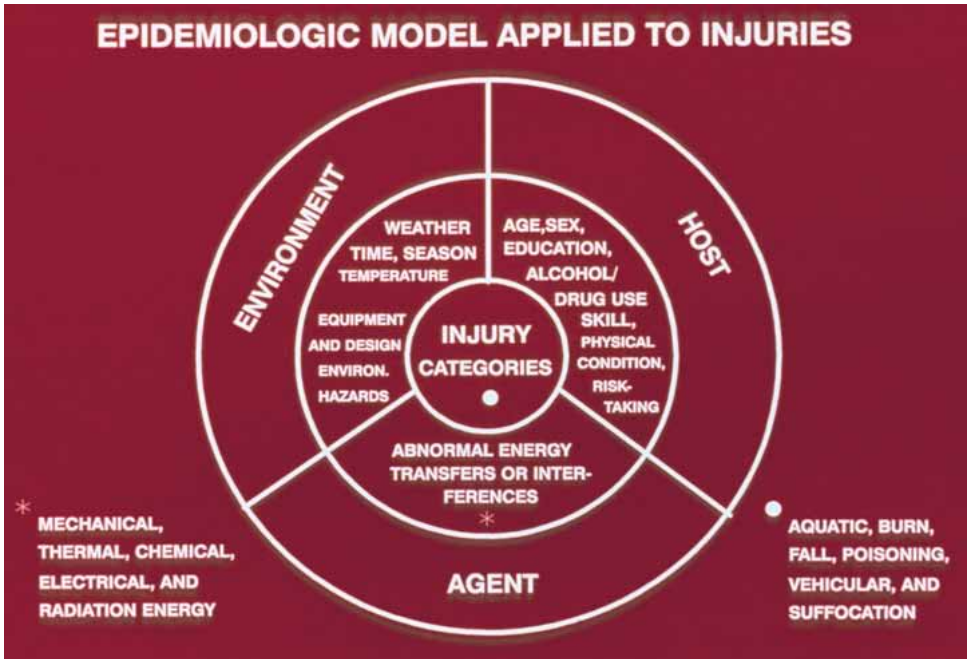

Figure 1. Epidemiologic Triad

This is reflected in the Epidemiologic Triad (Figure 1) as it has been applied to the study of injury by scientists at the Centers for Disease Control and Prevention (CDC). For example, the agents for diseases are bacteria and viruses; for injury, they are kinetic or mechanical energy, chemical energy, thermal energy, electricity, and radiation. There are many possible vehicles and mechanisms to determine how energy acts on the body.

Examples of diving injuries and the energy that caused them are given in Table 2.

Table 2: Diving injuries and the energy that caused them

Damage to the body	Energy transfer
Pulmonary barotrauma	Expansion of gas
Decompression sickness	Inert gas pressure gradients
Oxygen seizures	A chemical surplus
Unconsciousness due to hypoxia	Lack of oxygen (chemical)
Hypothermia	Excessive loss of heat

Some conditions usually listed among disabling injuries/conditions may not obviously fit the definition of injury based on energy transfer. One such condition is sudden cardiac death (SCD) in diving; is it an injury or disease?

While this may seem irrelevant for other purposes, it is an important question for prevention. A question for injury research here is: does SCD occur at a greater rate in underwater diving than in land-based recreational activities of similar level of exertion? If the answer is positive, there may be a dive specific provocative factor in a form of energy exchange causing relatively "sudden discernible effects" which leads to SCD. For example, the energy transfer may occur in the form of blood shift caused by immersion, work of breathing, or pressure, in combination with a vulnerable host. One of the hypothesized conditions linked to SCD is left ventricular hypertrophy (LVH).[9] LVH may be established at postmortem medical examination but the necessary measurement is not regularly performed. A recent study found that medical examiners sometimes establish a diagnosis of LVH without any measurement; or their methods of measurement do not fit standards.[10] The chances of discovering preventable causes in this and other cases depend on meticulous medical examination.

Data collection

Data collection in diving fatalities has been described previously.[1] The objective is to answer the questions: who, where, when, what and how. We have previously classified our finding as shown in Table 3.

Table 3: Classification of diving fatalities

Category	Question	Functional role	Comment
Cause of Death	What	Coronial category	Final outcome. In aquatic environment it is most often drowning.
Disabling Injury	What	Diagnosis	The original damage to the body caused by specific energy transfer which led to fatality.
Disabling Agent	How	Mechanism	How the energy transfer acted upon the body and caused the damage.
Triggers	Why	Root causes	Why the conditions for the energy transfer occurred.

The cause of death is a coronial answer to what happened. More meaningful from the perspective of prevention is the disabling injury/condition or a diagnosis of the damage caused to the body. The answer to both of these categories is expected from the post-mortem medical examination although there are significant limitations which often preclude it. Identification of the disabling agent or mechanism which transferred energy to the body is sometimes possible based on witness reports or corroborating findings of equipment investigation. The disabling agent is sometimes called a provocative factor and is used to explain autopsy findings that otherwise may be inconclusive. Triggers are root causes that put in motion and propel adverse events leading to injury. This is the least complete and most problematic part of data. As much as we would like to know about it, official investigations rarely drill deep enough to identify root causes. Involved parties that could provide insight in the accident often fear litigation which affects their reliability as a source. In many instances, root causes are guessed retrospectively.

Fatality investigation vs. fatality research

Fatality investigation is conducted by legal authorities focused on a single case. The main purpose is the

attribution of legal responsibility and this determines how the causation is established. In most cases, the inquiry ends with establishing the proximal cause of death. In cases when the death is ruled unintentional or due to natural causes, investigation usually stops short of pursuing root causes. Medical examination is a part of legal investigation which determines its scope and extent and thus is likely to be subjected to the same limitations.

On the other hand, injury research is focused on a class of injuries and affected subpopulation. The main goal is to identify changeable root causes and pursue prevention.

Injury research depends on the quality of data provided by investigation. Legal investigation may provide answers on questions of how it happened but often not concerned with "why". The medical examination may answer what were the cause of death and the mode of death. For injury research, this usually provides enough answers needed for the study of most common causes of injuries but may be lacking information about some specific risk factors and subpopulation at risk, unless the medical examiners are cognizant of diving injury research and prevention goals.

Data analysis

Scuba fatalities usually get a lot of media attention. Most cases are reported in local newspapers, in social media and some make it up all the way to the national media. However, the urge for media to provide an immediate explanation as to why a diver died often results with misconceptions both as a consequence of lack of data as well as due to common biases about scuba diving. Here are some biases that are sometimes shared by the general public and many divers:

- *Need to blame:* there must be a cause and culprit (retrospective determinism)
- *Experience bias:* only inexperienced divers die
- *Health bias:* healthy divers do not die
- *Equipment bias:* it is the user, not the equipment (Guns don't kill people. People kill people)

Medical examiners have their own biases too, for example:

- Underwater diving - drowning
- Free gas in circulation - arterial gas embolism
- Atherosclerotic changes - sudden cardiac death

The purpose of this workshop is to discuss how to conduct a medical examination in order to obtain the most complete information and to avoid bias in establishing causation.

An important sociocultural bias affects how people attribute causes. Some people tend to explain accidents by internal causes and thus they blame human error. Usually, the human error is blamed on the deceased. Others tend to explain accidents by external causes. The choice of internal vs. external may also be affected by conscious self-interest. While the human error plays an important role in many accidents, "The focus of attention on the human actor involved tends to detract from an examination of the full range of factors that contribute to injuries and, particularly, their severity."[7]

A systematic study of causation is an important step in injury research. However, one has to keep in mind that accidents are usually caused by multiple causes that interact in various ways and affect the final outcome in various degrees. There are many popular models for causation analysis that could be used but there is no single one that meets everybody's expectation. For the public, it is important to realize that it is not always

possible to identify a single root cause and culprit for the accident. For injury research, that is not a problem. Rather than asserting blame, injury research looks for apparent root causes as well as for conditions conducive to human errors and organizational failures.

For evaluation of possible risk factors discovered with the analytical process, research uses statistical methods to establish possible associations. The final goal is to explore a wider context of an accident as it can be presented in the Haddon Matrix. Identification of possible intervention points could prevent the occurrence or change the evolution and mitigate the outcome of an adverse event.[8]

Table 4: Haddon Matrix

Factors	Factors			
	Human Factors	**Agent or Vehicle**	**Physical Environment**	**Sociocultural Environment**
Before dive				
During dive/accident				
Post accident				

Constructing a Haddon matrix is beyond the scope of medical examination. However, for the interpretation of autopsy findings, analysis of equipment, environment and procedures, it is often the key and medical examiners are in a position to request it. At present, toxicology analysis is regularly done but the equipment examination and gas tests are not.

Final analysis and completion of the Haddon matrix may be completed with consultation of a panel of experts who can address the technical, legal and medical aspects of diving accidents. The experience of the San Diego Scuba Fatality Panel is a successful model of such an approach which may be adopted by other entities.

Conclusions

Medical examination contributes to injury research and prevention by establishing the cause of death and disabling injury, possible mechanisms of injury, and medical conditions that may have contributed to the outcome. Thorough medical examination is always warranted in the case of diving fatalities even if the cause of death seems obvious.

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Dilemma of Natural Death While Scuba Diving

Charlotte Sadler, MD

What is the dilemma?
The majority of scuba divers who die in the water have their deaths attributed to drowning. In fact, 70% of diving deaths since 1992 have been attributed to drowning.[1] You just heard why it may not be drowning; but, this brings us to the dilemma: If it's not drowning, what is it?

What I will present to you today is that the investigation of the circumstances surrounding the death, equipment analysis, and autopsy findings may lead to the true cause of death, which may be from cardiac death, a natural death.

Let us start with a case that we reviewed in San Diego, and then we will discuss the most common causes of sudden cardiac death in adults which are coronary artery atherosclerotic disease and left ventricular hypertrophy (LVH). We will also discuss the difficulties in defining a normal heart weight and diagnosing LVH. And this is a really important point because if we can't define normal, then how can we define abnormal? Finally, we will discuss why these cases may be the result of cardiac disease and not drowning and the potential implications this may have in dive medicine.

Case:
This was a 65-year-old-male who was on a liveaboard dive trip. On the third day of the trip, he made his first dive to 75 fsw (23 msw) for 55 minutes and had a normal ascent and safety stop. He had a 90-minute surface interval and put his wetsuit back on - witnesses later recalled that he seemed flushed and sweaty. He then entered the water and descended. His dive buddy followed him down to the bottom at 44 fsw (13 msw) and found him prone and appeared to be struggling. By the time she got to him, he was unresponsive with his mask and regulator still in place. She ascended with him to the surface and yelled for help. The boat crew pulled him aboard and started CPR, but he was unable to be resuscitated.

So what happened? Did he drown?

Additional information that we need to answer this question includes the dive profile, the analysis of the dive gear, and the autopsy results. I'm going to defer the topic of diver death investigation to other speakers a little later in the day, but I'd like to spend a little bit of time talking about the autopsy results in this case.

The results of the investigation showed that his dive profile was not indicative of DCS or AGE. His dive gear functioned normally, and his toxicology screen was unremarkable. His right and left lung weights were 640 g and 510 g, respectively. As you have just heard, these are slightly heavy, but this could be the result of multiple factors, including his prolonged resuscitation and chest compressions. Finally, his heart was 420 g with 90%

stenosis of the mid right coronary artery. I'm going to go into great detail about what these findings mean, but they are very, very abnormal.

After the results of this investigation, it brings us to the question, what was the cause of death? Was it drowning, which is an accidental death, or something else? Most medical examiners in this country would call it a drowning, simply because he was in the water. Or was it sudden cardiac death from atherosclerotic disease, which is a natural death?

Sudden cardiac death

In the United States, the two most common causes of sudden cardiac death (SCD) in adults are coronary artery disease (CAD) and left ventricular hypertrophy (LVH). These are separate entities, but are often co-existing. Here are other, less common, causes of sudden cardiac death that I will be not be discussing today, including hypertrophic cardiomyopathy, coronary artery of anomalous origin, arrhythmogenic right ventricular dysplasia, Long QT, and Brugada Syndrome.

Atherosclerotic heart disease

In atherosclerotic heart disease, the single most likely cause of sudden death is the spontaneous development of a lethal tachydysrhythmia in someone with underlying ischemic heart disease.[2] This is a very important point. It is not the heart attack that kills people instantly. Heart attacks and the subsequent damaged myocardium kill people over a time course of hours to days. It is the dysrhythmia that kills people instantly. And there are two mechanisms by which this occurs. The first is coronary artery plaque rupture or coronary artery thrombosis. The second is critical stenosis (defined as >75% narrowing of lumen) resulting in ischemia and leading to spontaneous lethal arrhythmia.[3]

The first mechanism, the idea of a ruptured plaque or coronary artery thrombosis is, I think, conceptually easier to understand. There is a mechanical blockage, which leads to ischemia. The ischemia causes a dysrhythmia, which then leads to sudden death. If there is no dysrhythmia, it will lead to necrosis of the myocardium over the following hours and days, which should be evident histologically.[4,5] One would hope that you find the ruptured plaque or thrombosis on autopsy, but you could conceivably miss it when cross-sectioning the coronary arteries.

The second mechanism, critical stenosis, is essentially an electrical event in an unstable and excitable myocardium, induced by hypoxia and ischemia from vessel narrowing.[2,5–7] The most common lethal arrhythmia is bradyasystole, but any dysrhythmia poses a bit of a problem post-mortem because you can't see a dysrhythmia on autopsy.

Left ventricular hypertrophy

Atherosclerotic disease often co-exists with another risk factor for sudden cardiac death and that is LVH. This was first identified as a risk factor for sudden death in the Framingham study.[8] The Framingham study was a revolutionary study for many disease processes, including heart disease, but also subjects such as sleep medicine, brain and bone research. The Framingham study was based out of Framingham, Massachusetts and it was a cohort study that followed 5,000 patients. It is still going on and is currently in its third generation. This study is where we got a lot of our information about risk factors for heart disease and sudden death, including LVH. I think that LVH is pretty easy to conceptualize, the name is self-evident, but it's a lot harder to define. And this is really important, because if you don't recognize it, you're missing a huge risk factor for sudden death. So before we talk about how to recognize the abnormal, we have to define the normal.

What is a normal heart weight?

The short answer is, we don't know. There have been many studies done, but there were pretty major flaws in these studies. You've already heard about the inherent selection bias of autopsy studies and we're going to review a few of the major studies and some of their flaws. It is critical to realize that there is no universally accepted definition of a normal heart weight, and this obviously causes a problem if you're basing autopsy conclusions on "abnormal."

The first study that I'd like to review was done by Zeek.[9] This was an autopsy study done in 1942 in Cincinnati. This study was done on hospitalized patients who died during their inpatient hospitalization. They attempted to exclude any patients who would have obvious causes of an abnormal heart weight, as well as the obese and malnourished. She concluded that normal heart weights should be based on a person's height, as this is unlikely to vary with disease state. This was an autopsy study, so it obviously had the same problems with selection bias that all autopsy studies have, but additionally, these were all people who died in the hospital. By definition, this means that they were not "normal, healthy" people, so how can we define a normal heart weight based on them?

The next study that I would like to discuss was done by Kitzman in 1988 from the Mayo clinic.[10] Again, this study was done on hospitalized inpatients, and the only exclusion criteria that they used was a clinical history of cardiac disease, systemic or pulmonary hypertension, or coronary artery atherosclerosis >50%. This is a very limited list. There are many other conditions that can cause a cardiomyopathy. Kitzman then concluded that a normal heart weight should be based on body weight and developed his own nomograms from his results. These are some of the most widely used nomograms used by pathologists today.

This study had a lot of the same problems as Zeek's study. It was an autopsy study, it was done on hospitalized inpatients (again, definitely not normal, healthy specimens) and it concluded that normal heart weights should be based on body weights. This is hugely problematic; how can you conclude that a normal heart weight is based on body weight when the weights that you used to make this conclusion were from hospitalized inpatients? Disregarding their heart weights for a moment, they most certainly did not have normal body weights. Chronic illness has a huge effect on body weight, whether it be ascites, cachexia, tumor burden, etc. And yet, his nomograms are some of the most widely used by pathologists today.

Molina and Dimaio published a study in 2011 that tried to eliminate many of the selection bias present in autopsy studies.[11] They took 18-35 year old male trauma fatalities (thus eliminating the bias of using hospitalized inpatients) and then they went back through their medical records and eliminated anyone with a medical history that would cause an increased heart weight (such as hypertension). And this did eliminate at least some of the bias, but there is still a significant amount of the United States population with undiagnosed and uncontrolled hypertension that is unable to be detected post-mortem. However, even with some of this selection bias still present, they came up with lower values than Zeek or Kitzman, which was, a normal heart weight in males was in the range of 233-383 g, with no correlations to height or weight.

There are other definitions in the literature, ranging from absolute values of 380-400 g for men and 350 g for women, or percentages of body weight (0.45% of body weight in men and 0.40% in women).[12,13] Again, all of these come from autopsy studies and suffer from the same biases as the previous studies.

Take your average 5 ft 11 in (180 cm) and 176 lbs (80 kg) man. What would each of these studies say is his normal heart weight? According to Zeek, it would be 340 g, with a range of 300-380 g. According to Kitzman, it would be 349 g, with a range of 265-461 g, and according to Molina and Dimiao, it would be in the range of

233-383 g. Take a second to really look at these numbers. These are very different numbers, and perhaps even more importantly, the ranges (especially in Kitzman) are huge. So how do we really know what normal is?

We must return to the Framingham Study. They were the first to use echocardiogram on healthy adults to develop a new definition of normal heart weight, as well as LVH.[14] They defined LVH as greater than two standard deviations away from normal or >143 g in men and >102 g in women. This was the first cohort study done on healthy, living subjects. They could see and evaluate these patients, take their blood pressure and effectively eliminate the selection bias that was present in the previous autopsy studies. Unfortunately, all of these results were published in the cardiology literature. I don't know about you, but I do not expect physicians to be well versed in literature way outside of their specialty; and so these values are not widely used by pathologists.

Returning to our average man, according to the Framingham values, his normal heart weight would be 257 g. This is significantly less than the other values. So you can see how it may be very easy to miss some pathologically enlarged hearts simply based on what value of "normal" you use.

Dr. Denoble and his colleagues also thought that LVH may play a significant role in sudden cardiac death in divers and that perhaps the stress on the body from diving may precipitate arrhythmias and death.[15] They hypothesized that the incidence of LVH in diver autopsies will be higher than that of the control group, which were traffic fatalities matched for age and sex. Additionally, they excluded from the control group all conditions that would disqualify someone from diving, as well as evidence of substance abuse. And their hypothesis turned out to be correct. They found that the divers had significantly larger hearts than their matched controls in traffic fatalities. This was a really important study. It does not prove causation, but it certainly adds to the body of evidence that LVH may be associated with these deaths.

We have certainly seen that LVH is hard to define, but why is this important? Because it is a risk factor for sudden death. The Framingham study was the first to show that LVH was an independent risk factor for sudden death even in the absence of coronary artery disease.[8] They followed subjects with LVH for up to 14 years after their initial exam and they found them to have an increased risk of sudden death with a hazard ratio of 2.16 or over twice as likely to die as their counterparts. Perhaps even more interesting, they found that this risk increased as the heart weight increased. For every 50 g increase in mass, the risk of sudden death increased with a hazard ratio of 1.45. This is a very significant finding and emphasizes the point that if you don't know what normal is and you're not looking for LVH, you could be missing a huge risk factor for sudden death.

So how does LVH cause sudden death? In animal models, where both myocardial fibrosis and hypertrophy have been independently linked to arrhythmias in the absence of CAD, it is thought to be due to the remodeling of cardiac myocytes and increasing collagen deposition.[16] These changes make the myocardium more susceptible to arrhythmias.

Arrhythmias can be stress induced from enhanced sympathetic or decreased parasympathetic tone, hypoxia, acidosis, or hypercapnea.[2] They can occur in injured myocytes in localized substrates, such as healed infarcts aneurysms, or interstitial fibrosis. But they can also occur in myocytes that appear normal histologically and in these cases, we may need to look for surrogate markers of damage, such as LVH or increased heart weight.

When we talk about the stressors that can induce arrhythmias, it is many of these same stressors that occur while diving, including increased peripheral and systemic vascular resistance, increased blood pressure, and

increased oxygen consumption.[17] Exertional stress may lead to systemic lactic acidosis. Not that all of these stresses are unique to diving. Many of them occur with exercise in general, which is also a risk factor for sudden death.

Both coronary artery disease and LVH are risk factors for sudden death, but may not have an obvious cause of sudden death on autopsy. And these are the cases that we may need to use surrogate markers that identify patients at risk for sudden death, such as atherosclerotic disease, LVH or evidence of previous damage, such as myocardial scarring, aneurysms, or myocardial fibrosis.

To return to our case, let's review those autopsy results again. Our gentleman had a 420 g heart with 90% stenosis of the right coronary artery. That's certainly significant coronary artery stenosis and a large heart no matter what definition you use. Using the Framingham values, he would have had to have been 9 feet (3 meters) tall for that to be normal. According to Zeek, even if he were 6 ft 6 in (200 cm), he still would only have a 378 g heart. The pathologist who reviewed this case recognized these abnormalities and their significance and listed the cause of death as a sudden cardiac death while scuba diving due to coronary artery atherosclerosis and it was ultimately determined to be a natural death.

So to return to our initial question, if it's not drowning, what is it? You've already heard why it may not be drowning and later today you're going to be hearing about the components of a diver death investigation and how pathologists classify deaths, natural vs. accidental. I think there are really two main reasons why the true diagnosis may be missed. First is the failure to recognize LVH and the second is the concept of the hostile environment.

I know you've already heard this quote once today, but I'm revisiting it again because it's such an important concept. "When a natural death occurs in a hostile environment, as when someone has a myocardial infarct while swimming, there is a likelihood that the person was alive when the face became immersed (i.e., the person was still alive while in the hostile environment), preference is usually given to the non-natural manner unless it is clear that the death occurred before entry into the hostile environment".[18] This is straight from the guidebook of the National Association of Medical Examiners. However, this is referring to medical conditions or events that when they occur on land they would most likely not be fatal, but when they occur in the water, result in death. For instance, a seizure. The vast majority of seizures that occur on land are not fatal but if they happen in the water, they often lead to aspiration and death. But does this same standard hold true in cardiac arrests?

The outcomes of out of hospital cardiac arrests are quite poor. In fact, most studies report rates of survival to discharge with a favorable neurologic outcome as somewhere in the range of 3-5%.[19–23] If the presenting rhythm is not ventricular fibrillation or tachycardia, the survival drops to less than 1%. In the U.S., the incidence of ventricular fibrillation is decreasing and the most common presenting rhythm is now bradyasystole. Unfortunately, sudden cardiac arrest is largely not a survivable event, whether it occurs at home, on the street, in the airport, or in the water. People die on land from cardiac arrest just like they die in the water-and it has nothing to do with a hostile environment.

So what does all this mean? Is there anything we can do to prevent some of these deaths? More than likely, some would die anyway, despite appropriate screening. But, should we be doing more?

To try and make an abstract idea a little more concrete, I'd like to make some hypothetical and very rough calculations to just give you an idea of the exact numbers I'm talking about. Assume that the overall risk of

sudden death is 10% per year in patients with presumed cardiovascular disease (though this is probably a bit high, it is more likely in the range of 3-5%).[24] We know that there is an increased risk of sudden death with exercise in patients with coronary artery disease, though the studies quote a wide range of numbers, anywhere from five to 56 times increased risk.

And we know there is a risk of death in diving. Again, this is hard to calculate, because no one knows exactly how many divers are out there. In an article based on DAN divers, the estimated risk of diving ranged from 3.2-34/100,000 divers and 0.37-4 per 100,00 divers.[25] In a period of seven years and 187 deaths, they found that the overall rate of death was 16.4 per 100,000 persons. And you can see from this table that about 26% of these deaths were attributed to cardiac causes and this was significantly higher in divers greater than 50 years old.

So, assuming a rate of death of 16.4 deaths/100,000 divers and an estimated 1.2 million active divers, this comes out to be about 197 deaths/years.[26] And assume now that 26% of these deaths were from cardiac causes, this comes out to be about 51 deaths per year. Given a baseline risk of sudden death from any cause of 10% in patients with cardiovascular death, this accounts for about five deaths. And if exercise increases the risk of sudden death even five to tenfold, this brings us to about 50 deaths, which are in the range of what we are actually seeing.

In conclusion, if it's not drowning, what is it? It may be heart disease-specifically coronary artery atherosclerosis and LVH. You can't see an arrhythmia on autopsy, which is why we need to identify surrogate markers and know how to look for them. If we know what risk factors to look for, we may be able to improve our fitness to dive screenings and, potentially, prevent some of these deaths.

Thank you very much.

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Field Investigation; Preserving the Evidence

Craig L. Nelson, MD

Abstract

Diving conditions and diving equipment may cause or contribute to a diver's death, which makes field investigation and equipment preservation critical. Simple evidence preservation techniques and documentation can make the difference in understanding a diver fatality, but with proper training, those in the field do not need to be divers or equipment experts in order to preserve information and items for later examination.

Introduction

Although investigation of a diver death generally begins with law enforcement or other public safety personnel, such as lifeguards, anyone who may be involved with such an investigation should have an understanding of the requirements for the field investigation of a diver death. The initial responders may not be aware of what is needed in terms of securing equipment properly and identifying proper sources of information, so guidance from those who may later be involved in a future investigation or who are involved in an ongoing investigation will be necessary. Moreover, those who may be involved in later litigation, whether criminal or civil, should have a thorough understanding of what can be done to ensure the most accurate collection of facts and evidence.

Approach to diver deaths

My general approach to the death of a diver is a gross oversimplification of asking, "What could possibly go wrong?" That overly broad approach then allows one to start breaking down the possibilities into categories: natural disease; human error; equipment problems; underwater hazards; and diving-related injuries (barotrauma/gas embolism, immersion pulmonary edema, and, less likely, decompression sickness); or a combination thereof may all be factors. Each, in turn, should then be a component of an investigation.

This approach of "what can possibly go wrong?" is in contrast to the forensic pathology concept of the "Hostile Environment." The mentality of most forensic pathologists is that a diver is at risk for drowning because he or she is in a "hostile environment." In other words, the tendency is to assume that if a diver has died in the water, it simply must have been due to drowning, because of that environment. However, it is worth noting that if we have a healthy diver with functional gear and a sufficient gas supply, that hostile environment has been negated and should not, therefore, factor into the cause of death unless it is somehow interrupted. Therefore, a diver should never be presumed to have drowned unless one can offer an explanation for why the diver drowned and the hostile environment came into play.

The three general patterns of diver deaths

For purposes of discussion, it helps to note that diver deaths follow three general patterns, each of which offers advantages and disadvantages for the investigator.

The first is a diver death that occurs underwater, with no rescue or resuscitation attempted. In this situation, there is usually ample time for documentation of findings in situ, such as the position or loss of equipment, whether straps are fastened, any entanglements on the diver, or a variety of other important factors. However, disadvantages may be introduced by possible delay in between when the diver dies and is then recovered. Autopsy information can be altered or affected by postmortem changes of decomposition or animal scavenging. Dive computer data may be lost, particularly in older models with limited memory. Gas may be lost from the diver's cylinder(s), bearing in mind that a second stage may free flow, equipment may leak, or gas may bubble as a normal part of regulator function, as in some Sherwood regulators.

The second pattern is when a diver has a triggering event in the water, but is then brought to shore or on board a boat for attempted rescue, but dies prior to transportation to a medical facility. These situations typically provide the investigator with witnesses who can report on what happened and the diver's initial response, if any. However, responders who are operating in a rescue mode, of course, may not take the time to document certain things, such as the positions of equipment or what the diver's gauge readings were. Also, they will remove, and possibly even lose, dive gear in their intense efforts to rescue the diver.

The third pattern is when a diver is transported to a medical facility and survives for a few hours or days or is resuscitated and then survives for a few hours or days. Advantages for the investigator are that imaging and laboratory tests, which may guide determination of the cause of death, will be documented. However, autopsy findings may be altered by the survival interval and medical intervention. Also, information may be lost as witnesses leave or forget, and, as in the second pattern, when equipment is broken down or, worse, returned to family or a dive shop.

The Field Investigation

Bearing these above patterns in mind, as well as their advantages and challenges, one can break a field investigation into six major parts:

- History
- Antemortem events
- The environment (scene)
- Body recovery
- Medical care administered before death
- Body and equipment recovery and documentation and preservation of evidence

History: The history, or background information, sets the stage about the decedent. Important areas for the investigator to explore will include medical history and family history (particularly paying attention to any family history of sudden death), drug or medication use, diving history and experience, and the diver's last dives before this event, including where, when, how deep, and how long. Family members can be helpful for providing medical information and guidance towards the diver's primary care physician, who can provide records and further history. In many cases, though, family members have limited knowledge about a diver's certification and diving history. So, discussion with dive buddies, the dive shop, and boat operators may be of use.

Information uncovered in the history may indicate that a diver has health issues that may have played a role in the death. Or, an investigator might find that a diver lacks experience for the particular environment, or has a habit of showing poor judgment undertaking risky dives in unfamiliar areas or in unsafe conditions. We may also uncover trends of failure to follow regulations and protocols (buddy system, equipment maintenance, gauge monitoring, or depth and time restrictions).

Antemortem events: Investigation of the events immediately surrounding the death is crucial, and may indeed be the information that allows proper determination of the cause of death. Basically, this portion of the investigation tells the story of what happened to the diver. The investigator should learn the purpose of the dive, the location, conditions (both above and below the water), who was involved, and a timeline of events, paying particular attention and seeking out additional detail on any problems that occurred. To many, these points may seem obvious. However, it should be noted that an untrained investigator, or one not familiar with diving, may not know to ask about these points, and my experience has shown that if a question is never specifically asked, the answer may never be brought forth.

The information regarding antemortem events can come from a variety of sources. A dive buddy or instructor is usually the first choice, but bystanders (in the water, on a boat, or on the shore) can be of help, too. Recovery/rescue personnel and medical providers can also be of tremendous value. For example, statements from rescuers such as, "The foam would just not stop coming up out of her airway!" can be a strong indication that immersion pulmonary edema may have played a role in a diver death.

What is learned from the antemortem events will at least guide the entire investigation, and may itself explain a death. For example, two divers at autopsy may both show marked coronary artery disease and edematous lungs, but the history about one may be that he suddenly became unresponsive while cruising slowly over a coral reef in pristine conditions, and the history about the other may be that he ran out of air at 140 fsw (43 msw) while attempting a wreck dive beyond his limitations. Indeed, the history will very likely trump the importance of the autopsy findings!

The environment/scene: another seemingly obvious aspect of a field investigation is the environment and scene. Weather reports, sea surface conditions, dive reports, and witness statements will contribute to this portion. Discussion with a diver familiar with the area, such as a wreck and its hazards, may also be of help. The investigator may learn that an entrapment or entanglement occurred, or discover that the diver likely was subject to stressors such as cold or hard work fighting surge or current. The potential for physical injury may also be uncovered, such as a situation in which a diver was tumbled in surge around a wreck, struck his head, lost consciousness, and died.

Medical care before death: as mentioned previously, medical care providers and laboratory or imaging studies performed prior to pronouncement of death can be excellent information sources. Information about initial levels of consciousness, initial rhythms, and responses to treatment can guide the investigation. Moreover, if there is sufficient delay between the triggering incident and death, any information about the cause may end up being solely from the medical record, with very little useful information from autopsy.

Body and equipment recovery: The final aspect of the field investigation is recovery of the body and diver equipment and the importance of documentation and preservation of evidence. Any discussion of evidence preservation must include the term "chain of custody," which is defined as the documentation of seizure, custody, control, and transfer of evidence. In other words, a person collecting evidence will document what was collected, when, and where, and record will be kept with the evidence to whom it was transferred and when,

with notes made of any alterations. This process allows investigators, and later, court proceedings, assurance that any findings from the evidence are not an artifact of handling or manipulation after recovery. Certainly, this procedure must be applied in dealing with diving deaths.

In a diving death, the goal for equipment is to preserve information for expert examination at a later time. Thus, the field investigator should document that which may be lost, but, unless fully qualified, not attempt a detailed field examination. Too often, a responder who is a diver undertakes it upon himself to "examine" the equipment, and ends up making alterations that can obscure a true equipment problem related to the death. The discussion of equipment examination is beyond the scope of this manuscript, and indeed beyond the scope of my expertise. The thorough equipment "necropsy" is best left to qualified professionals!

Ideally, the documentation of equipment should begin before the diver is removed from the water, though, of course, this will not happen in situations where rescue is attempted. If the diver is to be recovered by a dive team, the documentation should be discussed beforehand, making requests for the divers to note the position of the body as well as the position of all equipment, paying special attention to weights, buckles, fins, locations and orientations of second stages, and the mask. Documentation underwater is important because dive equipment usually must be removed for body recovery. Photography and videography are ideal and need not necessarily require expensive equipment; even a small camera such as a "GoPro" slowly panned over the body can record a great deal of information in a short period of time. Of course, recovery divers may be hindered by low visibility or safety and time constraints. If so, interviews with dive team members immediately following recovery may substitute for other forms of documentation.

Gauges and computers are particularly important to document; as noted, information about gas supply or dive times and depths may be lost with time, so photographs (or video) of the gauges as soon as possible will be of help. For example, a photograph of a submersible pressure gauge reading "0" at recovery is more meaningful than the same photograph taken two days later when the equipment arrives for professional inspection.

Another area of special concern is the diver's cylinder. First, it should be noted whether the cylinder is properly secured or has slipped from any straps. Second, after documenting the pressure displayed on any gauges, cylinders valves should be closed, which then prevents any further loss of gas. Doing so will allow for any later gas testing. When the valve is closed, the investigator should note the number of complete turns required to close the valve, as there have been diver death that occurred while trying to breathe from a minimally opened valve.

For standard scuba gear, with the cylinder valve closed, the regulator setup should remain pressurized. The investigator should look and listen for any leaking gas. Also, observation should be made for any blockages within the second stage mouthpieces.

Wetsuits generally require little more than a brief inspection and documentation of type and thickness, but the investigator is cautioned that they may be cut off by morgue staff preparing a body for examination. Drysuits, on the other hand, may present a bit of a challenge. The only opportunity for documentation of a leaking drysuit may come in the field, as it will later be removed from the body.

The investigator may then opt to attempt suit inflation, of course documenting any procedures for the chain of custody.

Weights are another area of concern for the field investigator. Despite training that instructs a diver to ditch weights when in distress, weights are often still found in place. If they are not in place, the investigator should specifically ask their location; there is always the chance that the weights were on the diver, but removed to allow easier movement of the body.

Beyond the equipment mentioned, the investigator should consider other possible sources of information that may not be immediately obvious. Logbooks found among the diver's possessions may be of help, as well as text messages on wireless devices. The location of items on shore such as game bags, clothing, or a vehicle can suggest entry points. Cameras should not be overlooked, either; while the photographs or video may be of little help, the metadata can provide important information about the last time at which the diver was still alive and conscious.

Conclusion

Guides and forms for diver death investigation exist and can be found readily. Some are tedious and repetitive, while others lack detail. Both of these issues are understandable, considering that diver deaths by nature are complex, and it is difficult to account for every possibility with a dropdown or a blank. Certainly, the information I have provided here cannot account for all situations, either. Most useful will be an investigator with diving knowledge and experience who prepares for such an event. Prior discussion with potential responders (the dive team, coroner or medical examiner investigators, and others) can foster solid working relationships and develop understanding of everyone's needs and expectations to provide a solid death investigation. Finally, the investigator should remember to ask for help, because the complex nature of these deaths will often span beyond one person's knowledge and experience.

Post Mortem, How To

James L. Caruso, MD

Very few forensic pathologists have significant experience with the investigation of fatalities involving divers who were breathing compressed gas. These fatal mishaps are fortunately rare, with fewer than 100 combined deaths occurring in the United States, Canada, and the Caribbean each year. The vast majority of these fatalities occur in somewhat predictable locations such as Florida, Southern California, Hawaii, and the Caribbean. Divers Alert Network (DAN) tracks all fatal diving related mishaps and disseminates the details of these events in an annual report.

With the paucity of these types of deaths, most forensic pathologists have limited exposure to performing autopsies and interpreting investigative reports related to diving deaths. Most of the standard textbooks of forensic pathology mention scuba related fatalities to a very limited extent and the board certification examination taken by forensic pathologists generally contains a token question on the phenomenon of air embolism. While a few medical examiner/coroner offices (e.g., Miami, Key West, San Diego, Los Angeles, Honolulu) see several diving related deaths in their practices each year, for most jurisdictions this would be a rare case and expertise among the pathologists and death investigators would be limited.

The proper investigation of a diving related fatality requires both a systematic approach and familiarity with recreational, and sometimes technical, diving practices as well as the physiology specific to breathing compressed gas in an underwater environment. It is far too easy, and serves little purpose, to ascribe to the "diver went into the water alive and was pulled from the water both wet and dead, therefore it is a drowning" adage. For numerous reasons, including ensuring that safe diving practices are propagated, providing closure for the family, excluding foul play and criminal activity, settling life insurance issues and other potential litigation, and just plain good forensic practice, these deaths need to be investigated thoroughly and competently.

There are still a handful of diving related deaths that are reported to DAN each year where an autopsy has not been completed as part of the investigation. The deaths have been simply certified as drowning or on rare occasions as a natural death due to cardiovascular disease, and the body is given only a cursory external examination at best. This is absolutely inappropriate as should become apparent during the following discussion. Most forensic pathologists consider the diagnosis of drowning to be one of exclusion; it is easily argued that without an autopsy not much has been excluded. In another small subset of cases an incomplete autopsy is performed (e.g., excluding an examination of the brain or other important organ or organs). The investigation of any diving related death should include a complete medicolegal autopsy.

The Dive and Diver's History

A complete medicolegal death investigation includes both a thorough evaluation of the circumstances as well as a detailed postmortem examination. In forensic medicine, the traditional history and physical is ac-

complished by the investigation and autopsy. While both components are essential and complimentary, a reasonable argument can be made that the investigation is even more important than the autopsy for diving related deaths.

Not only should the pathologist be aware of the circumstances surrounding the fatal dive mishap, but the diver's past medical and surgical history, recent health status, and any medications taken on a regular basis and on the day of the mishap need to be known. There are many active divers who have significant medical problems, including cardiovascular disease, diabetes, and chronic musculoskeletal problems for which they take narcotic pain medications. Cardiovascular disease in particular is a frequent factor in a diving related fatality, especially in older divers.

The deceased diver's dive training and experience level should be ascertained. More specific even is the diver's experience level with the particular diving activity, such as deep diving, spearfishing/game collection, drift diving, and cave or wreck penetration. Another important consideration is how frequently the deceased diver participated in the activity. Many very experienced divers have changed their activity patterns over time so that they only make an annual or biannual dive trip. Inexperienced divers and students enrolled in an initial dive certification class are overrepresented among fatal diving mishaps.

The pathologist and death investigator should also have at least a basic understanding of diving physiology. Deaths as a result of decompression sickness are exquisitely rare and when they do occur they typically involve a significant survival time after the diver surfaces and most often a period of hospitalization. On the other hand, air embolism resulting in death is relatively common. In fact, a frequent scenario is an out-of-air situation that causes the diver to ascend rapidly and suffer an air embolism. As the diver reaches the surface, Boyle's law has resulted in expansion of the gas held within alveoli to the point of over-expansion and pulmonary barotrauma ensues. If gas enters the injured pulmonary vasculature it may be conveyed back to the heart via the pulmonary veins and then out to the systemic circulation with the cardiac output. Gas entering critical areas of circulation such as the cerebral arteries and coronary arteries can be rapidly incapacitating or fatal.

An understanding of diving physiology allows the pathologist to accurately interpret the circumstances surrounding the mishap. If a rapid ascent has not occurred or particularly if there was no ascent at all and the stricken diver was recovered from depth, air embolism is not likely in the differential diagnosis. Postmortem changes that may be present at autopsy are best interpreted with at least a basic grasp of diving physiology.

The pathologist should be briefed on the scene and in some jurisdictions will respond to the scene in person. Important factors that need to be addressed include the exact dive profile, if known, any history of rapid ascent, insufficient breathing gas, panic on the part of the deceased diver, entrapment, and the exact setting and body position of the deceased diver if recovered from depth. Unfortunately, many dive fatalities involve the deceased diver becoming separated from his or her dive buddy prior to the fatal incident, so eyewitness statements may be limited. There is also a subset of divers who die while performing solo dives, again limiting the role of witnesses.

The Postmortem Examination in a Diving Related Fatality

When faced with performing an autopsy on someone where breathing compressed gas at depth may have played a role in the death, the pathologist should ensure that he or she has a solid understanding of the investigative aspects of the fatality. The approach to the postmortem examination is that of putting the autopsy findings into the context of the event. There is no single observation during the autopsy that will be

pathognomonic for the cause of death. The best approach is to systematically examine the body and record all pertinent observations that may be relevant to determining the cause of death. After the postmortem examination is completed, those observations must be placed into the context of the circumstances of the dive, eyewitness accounts, the diver's medical history and dive training/experience, and the scene investigation to include an equipment evaluation.

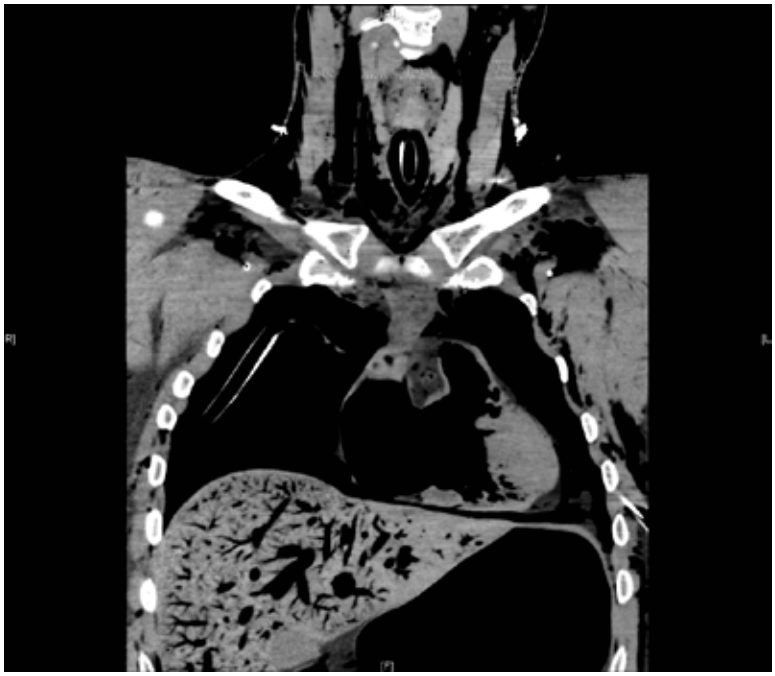

Figure 1. A postmortem imaging study (CT scan) on a diver who died of an air embolism shows extensive gas present in the heart and liver.

A short postmortem interval is ideal as postmortem changes will increase the degree of difficulty in interpreting autopsy findings. Radiographic imaging, either standard x-rays or postmortem CT scans should be obtained prior to the autopsy with a focus on the head and thorax at a minimum (see Figure 1). These are also helpful in screening the body for trauma.

A thorough external examination including a search for signs of trauma, animal bites, or envenomation should be carried out. Palpation of the area between the clavicles and the angles of the jaw should be carried out to determine if there is external evidence of subcutaneous emphysema. The initial opening of the body should include an evaluation for pneumothorax. One of two techniques can be employed, both of which have been proven to be useful. The pathologist may modify the initial incision over the chest to make a "tent" out of the soft tissue (an "I" shaped incision) and fill this area with water. A large bore needle can be inserted into the second intercostal spaces bilaterally and if appropriate laboratory resources are available, any escaping gas can be captured in an inverted, partially water filled container for measurement and analysis. This is impractical for most medical examiner facilities.

An alternative method, and the one used by the author, is to dissect through the intercostal muscles and observe if the parietal and visceral pleura are still somewhat adherent at the time the pleural cavity is breached. If the visceral pleura falls away from the parietal pleura, then any significant antemortem pneumothorax would have been unlikely. As the breastplate is removed any gas escaping from incised vessels or any gas present in the internal mammary vessels should be noted.

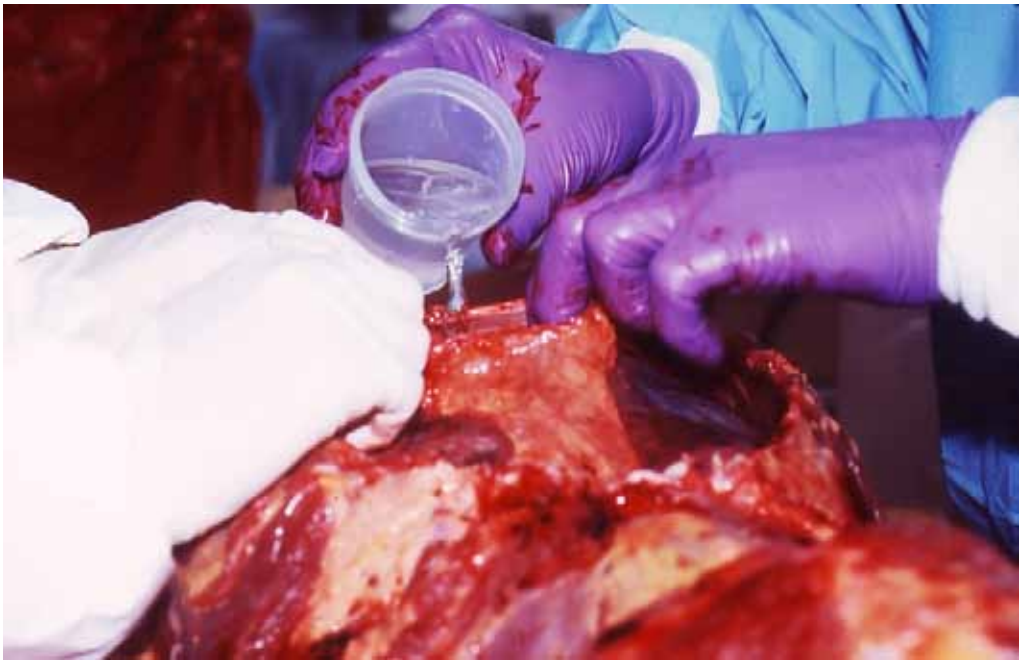

Figure 2. After opening the pericardial sac, water is poured into the sac before incising the left and right ventricles to look for intracardiac gas.

Pleural effusions are common in diving related deaths, particularly since they are fairly common in deaths due to drowning and bodies recovered from water regardless of the cause of death. As soon as the thoracic and abdominal viscera are exposed, an evaluation of fluid present in the body cavities should take place. For the pericardial sac, this can occur in conjunction with an evaluation for the presence or absence of gas within the chambers of the heart. As soon as the pericardial sac is incised and any accumulation of pericardial fluid is documented, the pericardial sac can be filled with water (see Figure 2). At the same time the pathologist can grossly evaluate the pericardium itself for the presence of pneumopericardium. Some forensic pathologists claim that this method of examining the heart for intracardiac gas is difficult and messy, but the author has performed this maneuver several times with minimal difficulty. After the pericardial sac is filled with fluid, a scalpel or needle can be inserted sequentially into the right and left ventricles. As with the chest cavity, the needle procedure can be employed if measurement and analysis of intracardiac gas is planned; otherwise, the use of a scalpel is easier and recommended.

After the mediastinum, heart, and great vessels have been examined for the presence of gas, the water may be evacuated and a standard autopsy may be performed. A careful evaluation of the lungs for bullae, emphysematous blebs, and parenchymal hemorrhage should take place. The dissection of the heart should note the presence of any inter-atrial or inter-ventricular septal defects, with particular attention paid to the area of the foramen ovale or fossa ovalis. As in any forensic autopsy, a detailed documentation of any cardiovascular disease and changes that would compromise cardiac function is important.

A complete medicolegal autopsy requires opening of the head and examination of the brain. This is no less true for the autopsy of an individual whose death was related to breathing compressed gas at depth. The author has reviewed autopsy reports on diving related deaths where the head was not opened and the brain not examined. As in the case of certifying a death as a drowning without an autopsy, this should be considered substandard practice. In addition to limiting the extent of an evaluation for possible air embolism, significant natural disease processes will be missed. Several diving related fatalities have occurred where the death was attributed to a cerebrovascular accident or a malignant neoplasm of the brain (see Figure 3).

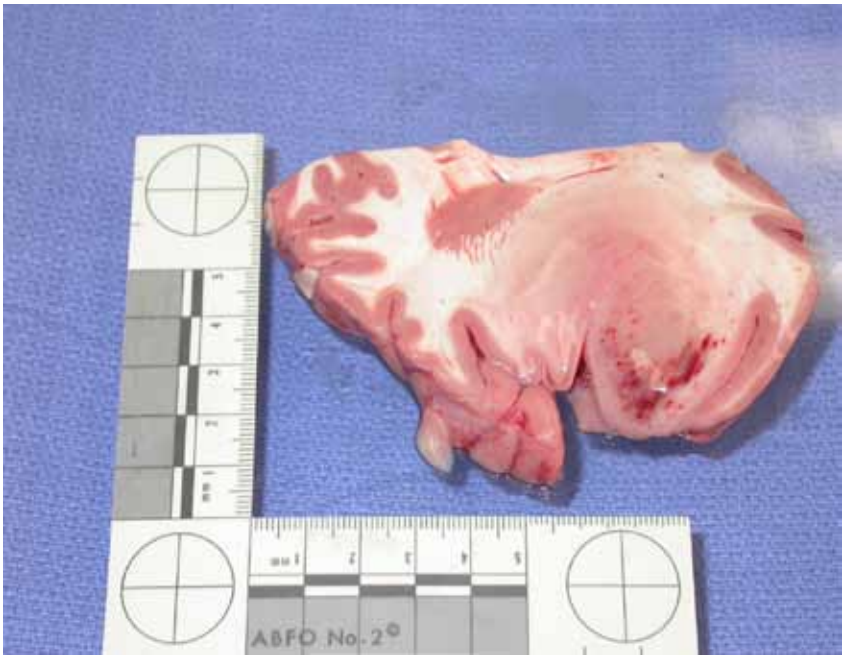

Figure 3. A malignant brain tumor found at autopsy of a diver who lost consciousness while using a pure oxygen rebreather. The tumor and not oxygen likely caused the seizure.

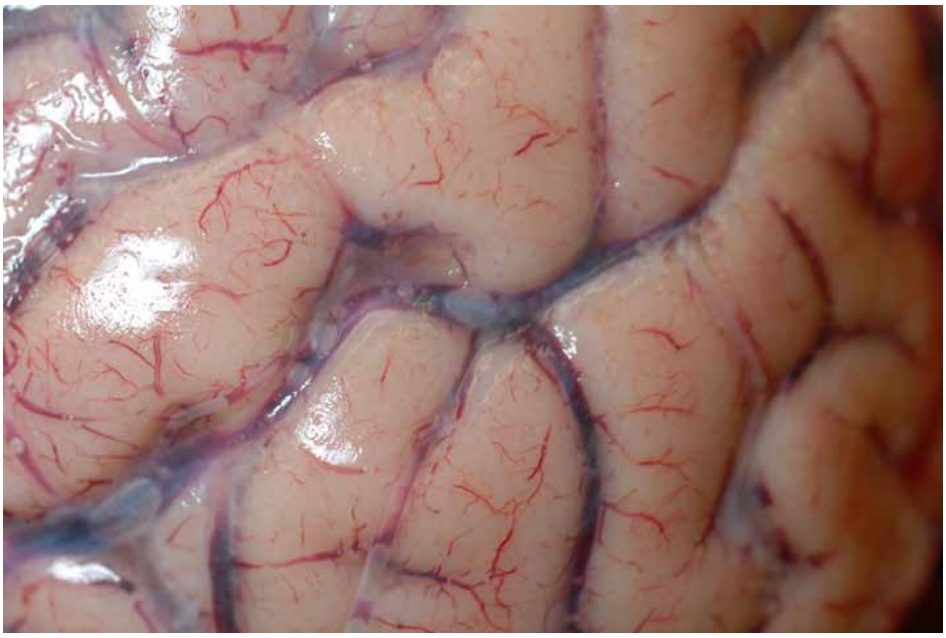

Figure 4. Bubbles within the blood vessels of the crebral cortex.

In an effort to prevent air from entering the intracranial vessels as a result of negative pressure generated when the head is opened, the vessels in the neck should be tied off prior to breaching the intracranial cavity. In addition to noting any trauma, particular attention should be paid to the basilar artery, the vertebral arteries, and the Circle of Willis. Intravascular gas in those vessels may be significant (see Figure 4). Also note any

gas in the vessels that extend over the cerebral hemispheres. The appearance of bubbles is more likely to be sausage shaped structures within the vessels rather than the classic, round shape. The autopsy should then be completed in the standard fashion with appropriate written record and photodocumentation of pertinent findings. One final trauma check of neck structures should be accomplished prior to releasing the body to the funeral home.

Ancillary studies should include a thorough toxicological evaluation with testing of blood and urine for ethanol, common drugs of abuse, and select therapeutic medications as dictated by the medical history and the pathologist's judgment. A blood carboxy-hemoglobin saturation should be determined, to exclude the possibility of tainted breathing gas. If the deceased diver has a history of diabetes or renal disease, or if a contribution from dehydration or hyperthermia is suspected, vitreous fluid should be analyzed for concentrations of electrolytes, glucose, creatinine and urea nitrogen.

Interpretation of Findings

The presence of gas in any organ or vessel after a scuba diving death is not conclusive evidence of decompression sickness or air embolism. During a long dive, inert gas dissolves in the tissues and the gas will come out of solution when the body returns to atmospheric pressure. This, combined with postmortem gas production, will produce bubbles in tissue and vessels. Many experienced pathologists have erroneously concluded that a death occurred due to decompression sickness or air embolism based on the observation of intracardiac and intravascular gas alone. The autopsy findings must be put into the context of the dive history and the scene investigation. Intravascular bubbles, especially if present predominantly in arteries, found during an autopsy performed soon after the death occurred is highly suspicious for air embolism. Bubbles present in the Circle of Willis and the arteries at the base of the brain are a more significant finding than the presence of gas in the superficial cortical vessels or the venous sinuses. Gas present predominantly in the left ventricle of the heart would also raise the suspicion for air embolism. The importance of the dive profile cannot be overemphasized.

If analysis of collected gas does take place, demonstrating that the gas in the left ventricle has a higher oxygen content than gas present in the right ventricle would lead the pathologist to correctly conclude that an air embolism likely occurred, though this comparative analysis seldom takes place for practical reasons. Intravascular gas from decomposition or off-gassing from the dive would have little oxygen and be made up of mostly nitrogen and carbon dioxide.

It may become apparent in any given case that the cause of death is drowning. While drowning is the most common cause of death in fatal diving mishaps, it is often a final common pathway for which many contributing factors or even a root cause may be identified. The pathologist should question why an individual who apparently knew how to swim ended up as a drowning death. Most diving related deaths are the result of inadequate training or experience, running out of breathing gas at depth, or the diver compromising generally accepted safe diving practices. Natural disease processes, including atherosclerotic cardiovascular disease, also contribute to a significant number of diving deaths each year.

Equipment

Only a brief mention of the dive gear is warranted here, but suffice it to say that all diving gear used by the decedent should be properly secured and thoroughly evaluated by an expert prior to returning it to the next of kin or to the dive shop if rented. Any remaining breathing gas should be analyzed for composition and the regulator and buoyancy compensator need to be examined to determine if properly functioning. The pathologist should be made aware of all equipment testing results (see Figure 5). Nearly all divers now use dive

computers and the vast majority are downloadable to a computer. The information stored is invaluable to the investigation as not only should the last dive be recorded but additional information such as diving habits and diving frequency may be gleaned.

Figure 5. A poorly maintained air compressor belonging to a surface supplied diver. Reenactment proved that the faulty compressor resulted in the diver's death.

Technical diving involves the use of specialized equipment and breathing gas and most often switching of breathing gases occurs during the dive. Types of technical diving include entering a cave system, using a rebreather apparatus, and deep dives that require decompression stops during the ascent. The pathologist and medicolegal death investigator should seek consultation with experts when faced with a technical diving fatality.

Conclusion

While fatal diving related mishaps are rare in most jurisdictions, it is possible for any coroner or medical examiner's office to be tasked with investigating such a death. The investigative information is every bit as important as the findings of a postmortem examination and the latter needs to be placed into the context of the dive profile. A few alterations of standard autopsy procedures may be employed to maximize the amount of information obtained from the autopsy. Postmortem changes, especially those that involve dissolved gas coming out of solution or gas production due to decomposition, can complicate the interpretation of autopsy findings. A thorough investigation with a complete medicolegal autopsy, comprehensive toxicology testing, and an evaluation of the dive equipment is essential in properly certifying the cause and manner of death in diving related fatalities.

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What Medical Examiners Need to Know About Rebreathers

John Clarke, PhD

Figure 1. The U.S. Navy's first computer controlled rebreather.

The insidious nature of rebreather fatalities

Rebreathers are troublesome from the medical examiners' standpoint. They can kill divers without leaving evidence behind.

There are three main root causes of fatal accidents with rebreathers:

1. Diver error – the most common cause
2. Mechanical problems – often caused by diver action or inaction
3. Mechanical-software problem – there is at least one incident of this type to be discussed here

The most common disabling injury in rebreather fatalities is the loss of consciousness due to 1) hypoxia, 2) hyperoxia – resulting in oxygen-induced seizure, and 3) hypercapnia. Unfortunately, an autopsy cannot reveal any of these three most common causes of rebreather fatalities and thus, in most cases of rebreather fatalities, medical examiners cannot detect the root cause.

Taxonomy of rebreathers

Rebreathers are a diverse group of portable life support systems. However, each rebreather may be placed into one of three categories; semiclosed mixed gas, fully closed oxygen, or fully closed mixed gas (otherwise known as "electronic") rebreathers.

Semiclosed rebreathers are among the simplest rebreathers. They are mechanical, which means they are rugged, and there are no electrical systems to fail. The system works by supplying either a constant flow of mixed gas (a mixture of oxygen and usually nitrogen), or by the injection of mixed gas keyed to diver ventilation. There is a plethora of names that are used to describe these units, but the most common is Constant Mass Flow, and Variable Volume Exhaust. Figure 1 is a schematic of the former type rebreather.

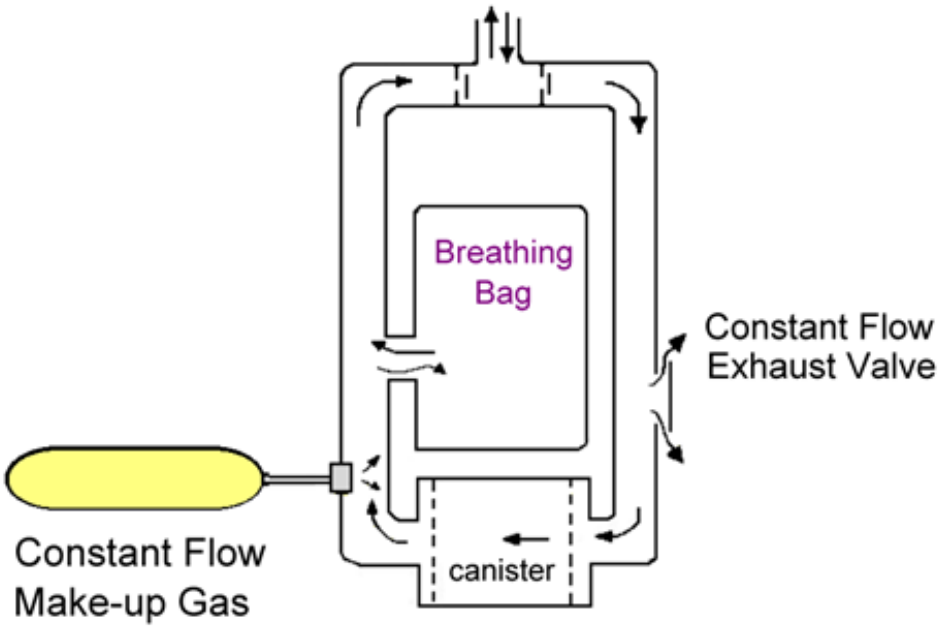

Figure 2. Schematic of a Constant Mass Flow semiclosed rebreather.

For the popular and simple constant mass flow unit, fresh gas is circulated to the breathing circuit, which contains a "counter lung" compliant volume. Exhaled gas then passes through a "scrubber canister" containing a CO_2 absorbent material (sodalime or lithium-based). If the diver's oxygen consumption is less than the fresh gas added, then internal gas volume and pressure will build until the exhaust valve opens, relieving excess pressure. If oxygen consumption exceeds the constant volume added, then collapse of the breathing bag triggers a demand valve (not shown) that adds additional gas.

Medical examiners who have some familiarity with anesthesia may recognize a similar layout in semiclosed anesthesia circuits (Figure 2). The features of lungs, counter lungs, CO_2 absorbent canister, fresh gas injection, and overpressure relief valve are all present.

Anesthesia circuits (semiclosed)

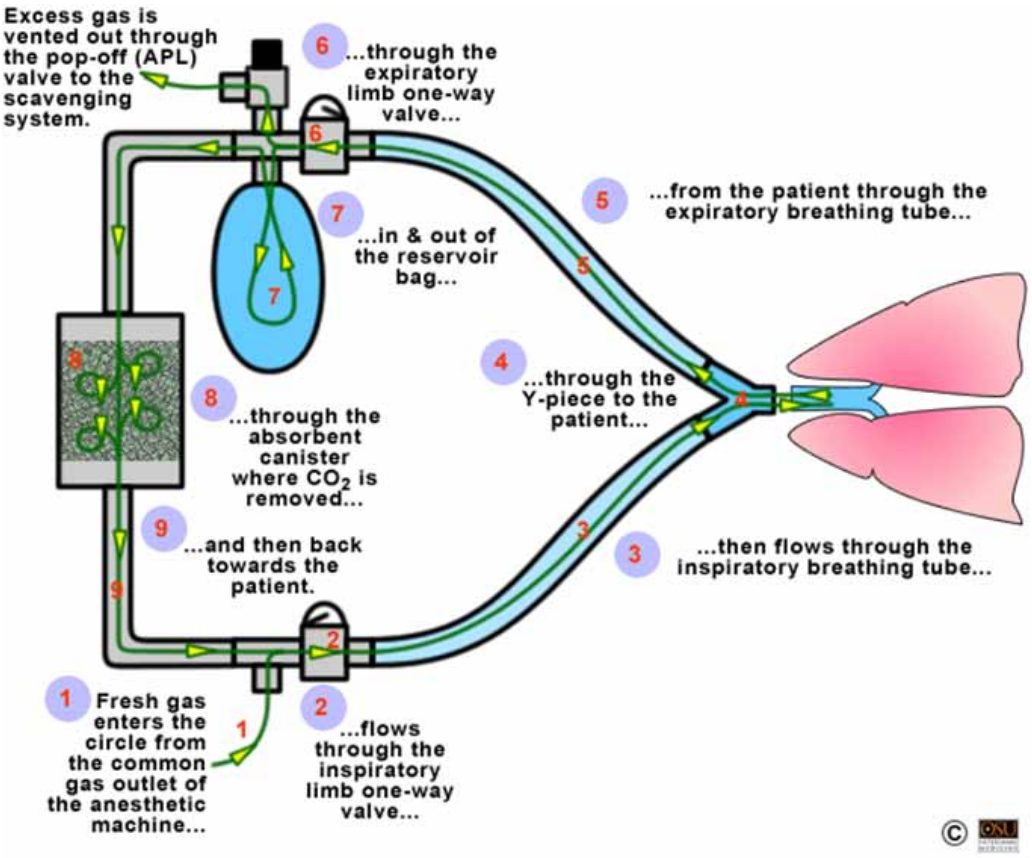

Figure 3. Semiclosed Anesthesia Circuit (Image credit LLee).

From a medical examiner's perspective, divers are active, and anesthetized patients are not. Although patients rarely become hypoxic due to some medical misadventure, divers can, due to their own misadventure, or inattention.

Due to the delicate balancing act between oxygen consumption and oxygen inflow, divers working hard at shallow depth are particularly prone to oxygen starvation, as in the following graph (Figure 4) of data obtained at 15 feet in a Navy Experimental Diving Unit (NEDU) test pool.[1,2] If NEDU had not been monitoring oxygen fraction, and these divers had continued working, they would have lost consciousness. Unattended loss of consciousness under water is usually fatal.

An unwitnessed fatality of this type would have left no clues for the medical examiner.

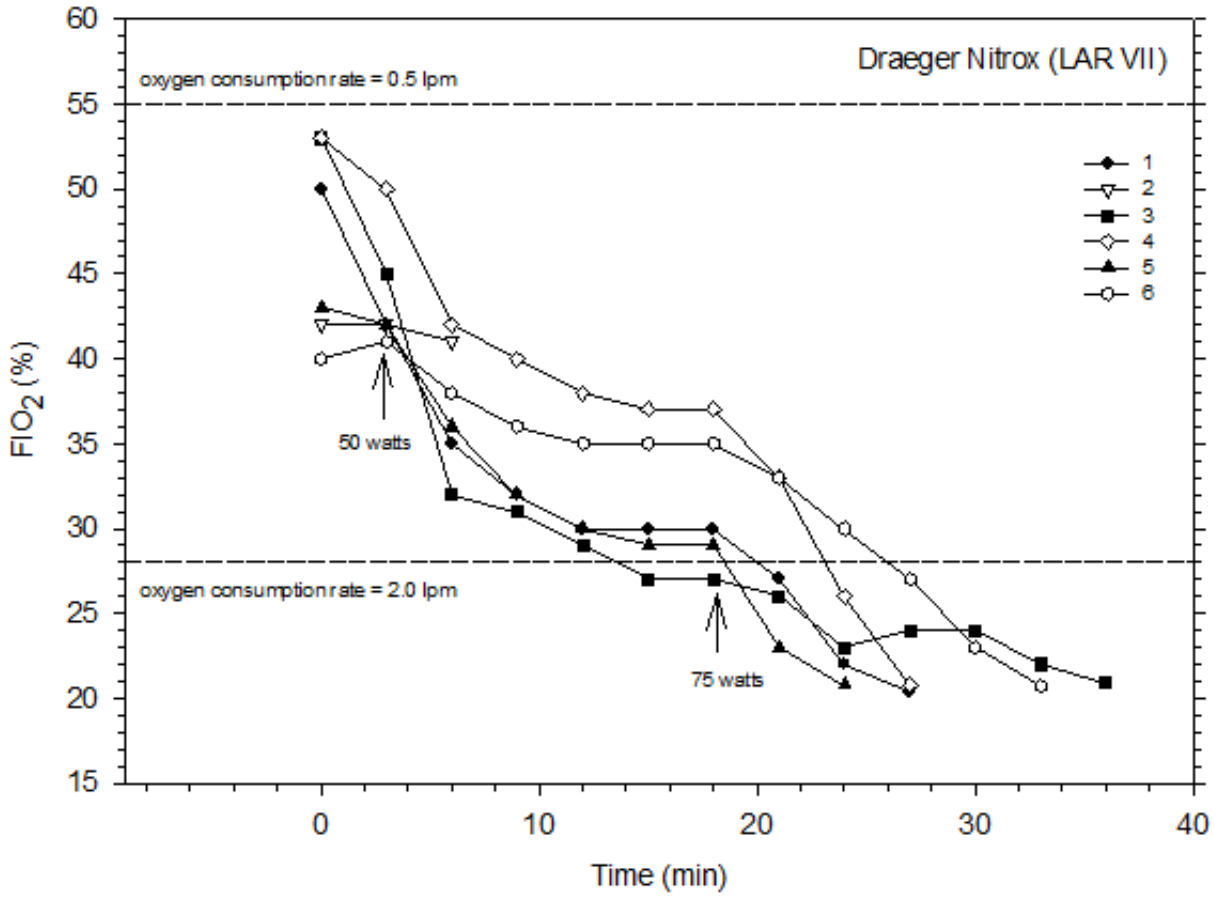

Figure 4. The decline in inspired oxygen fraction in six Navy divers used a constant mass flow semiclosed rebreather.

Also leaving no clues for the medical examiner was a fatality investigated by NEDU. The fatality was caused by both human error and blockage of the mass flow injection orifice in a constant mass flow rebreather, an Azimuth rebreather made by San-O-Sub Italia. On the fatal dive, the rebreather was dived to a maximum depth of 43 fsw (13 msw).

The primary gas supply to the diver was a 37% oxygen-nitrogen gas mixture. The manufacturer recommended constant flow rate for a 40% oxygen mixture was 9.6 L/min. The measured flow rate was 2.2 L/min, providing no more than 0.8 L/min of oxygen per minute for a 37% oxygen mixture.

The cause of the blocked gas flow turned out to be a large accumulation of white powder, presumably powdered carbon dioxide absorbent which would have physically blocked the gas flow orifice (Figure 5).

Figure 5. Powder contaminated flow orifice on an Azimuth rebreather.

According to dive simulation software developed by NEDU, (Figure 6) at a low oxygen consumption rate of 1.0 L/min, and a dive to 43 fsw (13 msw), the diver would have passed out after 16 mins. At a more reasonable rate of 1.5 L/min oxygen consumption, he would have lost consciousness eight minutes into the dive.

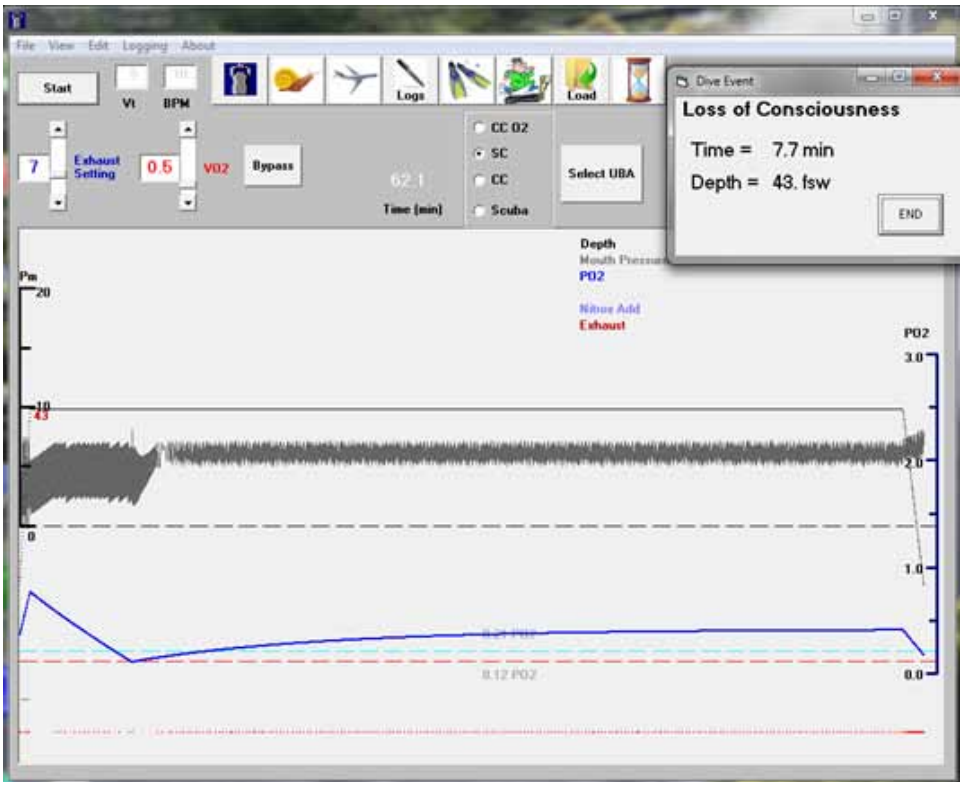

Figure 6. Computer simulation of the effects of light work in the face of low fresh gas flow due to a clogged gas injector.

Sadly, the victim had an oxygen monitor attached to his rebreather, but it was nonfunctional due to a reversed polarity condition of the monitor's battery. Once the battery was installed correctly, the unit alarmed constantly. If the unit had been functional, the diver would have been warned of the low PO2 condition during the dive.

In this case, human error led to poor maintenance of the rebreather, and to malfunction of the oxygen monitor. If the monitor had been malfunctioning with nuisance alarms and the diver had deliberately reversed battery polarity to quiet the alarm, then that decision could arguably have cost the diver his life. Diving with malfunctioning safety equipment is not, in the long run, either expedient or safe.

Fully closed oxygen rebreathers are arguably the simplest rebreather.

Like semiclosed rebreathers, fully closed oxygen rebreathers are mechanical, but instead of containing mixed gas they only hold pure oxygen. There is no constant flow of gas: as the oxygen in a breathing bag is consumed, the bag collapses, triggering the addition of a new bolus of oxygen.

Not much can go wrong with O_2 rebreathers, except that if the diver descends too deeply (deeper than 50 feet or 15 meters), they will be susceptible to oxygen seizures due to high partial pressure of oxygen.

An unobserved seizure under water is typically fatal, and usually gets classed by the medical examiners as an accidental drowning. If an oxygen rebreather is found on a diver at more than shallow depth, consider the possibility of an oxygen seizure.

Figure 7. Two 100% O_2 rebreathers: a Dräger LAR V and Aqualung Code.

CO_2 absorption

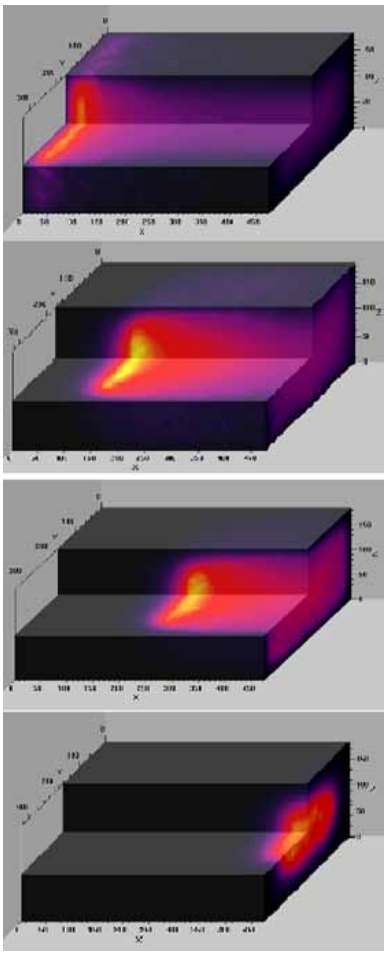

Figure 8. The dynamics of CO2 absorption and heat exchange throught a canister's life

Figure 9. The top of a rebreather canister showing granular absorbent

The CO_2 absorption process is a dynamic exothermic process: CO_2 absorption causes the release of heat. The process is dynamic in that the absorption process depletes the absorption sites in the absorbent, and then moves downstream to the next available absorption sites (Figure 8). The sequential images in Figure 8 were derived from a stochastic computer simulation of a rebreather canister during the absorption process.[3]

From a medical examiner's perspective, many things can alter the efficiency or speed of the thermokinetic reactions involved in CO_2 absorption. Disturbance of the process usually leads to premature canister expenditure, exposing the diver to an exponentially rising concentration of CO_2. Once CO_2 begins to rise, it does not take long for the CO_2 to reach toxic levels, resulting in loss of consciousness.

Flooded canister

Another form of incapacitation in any rebreather is a flooded scrubber canister. It can cause a physical obstruction to breathing. Aspiration of a slurry consisting of highly caustic absorbent and water could lead to tracheal burns, and reflex and potentially lethal bronchoconstriction.

All rebreather divers should be trained in emergencies procedures following canister flooding, which requires switching to open-circuit bailout gas. However, a mismanaged emergency procedure, or panic, can result in drowning.

If there is a good side to an accident of this type, it is that inhalation of alkali usually leaves tell-tale signs at autopsy.

Depleted or poorly packed CO_2 absorbent

Some divers try to stretch or reuse their CO_2 absorbent since it is a costly consumable (Figure 9). However, when that is done, the diver has no way to know how long his scrubber will remain effective. If stretched too far, CO_2 stops being scrubbed, and can quickly build in the rebreather until the diver loses unconsciousness due to CO_2 narcosis.

Such an occasion leaves no sign for the medical examiner or rebreather accident investigator, especially if the unit floods when the diver loses consciousness.

Sometimes, even the best tests of sodalime performance can go awry in the testing laboratory (Figure 10). The same can happen on an actual dive, leaving a diver with a very short

time of effective use of his scrubber. Unfortunately, he would receive no warning of that potentially fatal menace.

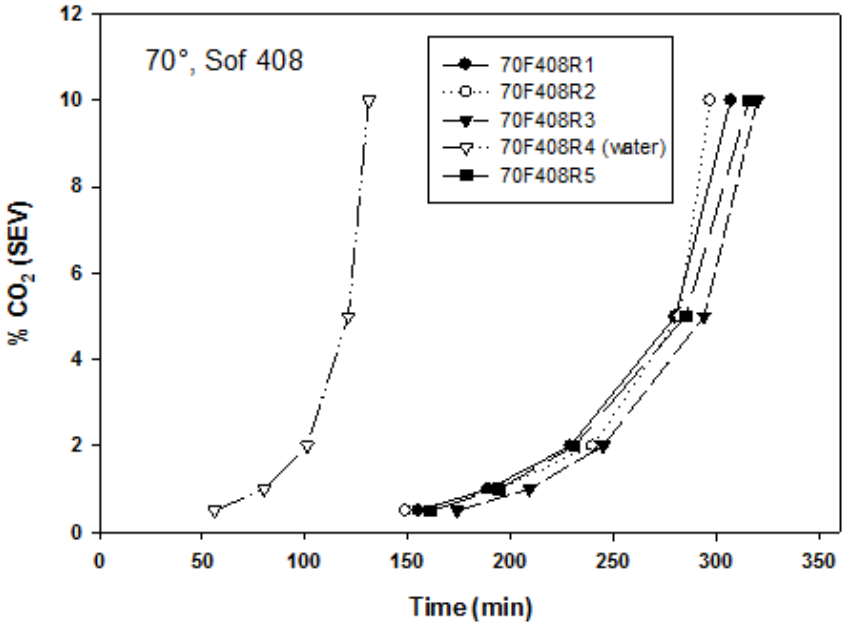

Figure 10. Early scrubber canister break-through believed to be brought about by moisture leaking into one of the five scrubber canisters.

Accident Statistics of NEDU Investigations

As seen in Figure 11, the incidence of open circuit fatal accidents are greater than other forms of diving. That is not surprising due to the large number of open circuit underwater breathing apparatus (UBA), primarily scuba, being dived. The fact that a high percentage of those accidents are fatal suggests that if the accident is an out of air situation, the remedy for that, ascent to the surface, is either not possible, as in overhead (cave or ship wreck diving), or is dangerous, resulting in panic and air embolism. The details of these accidents are not available in this figure.

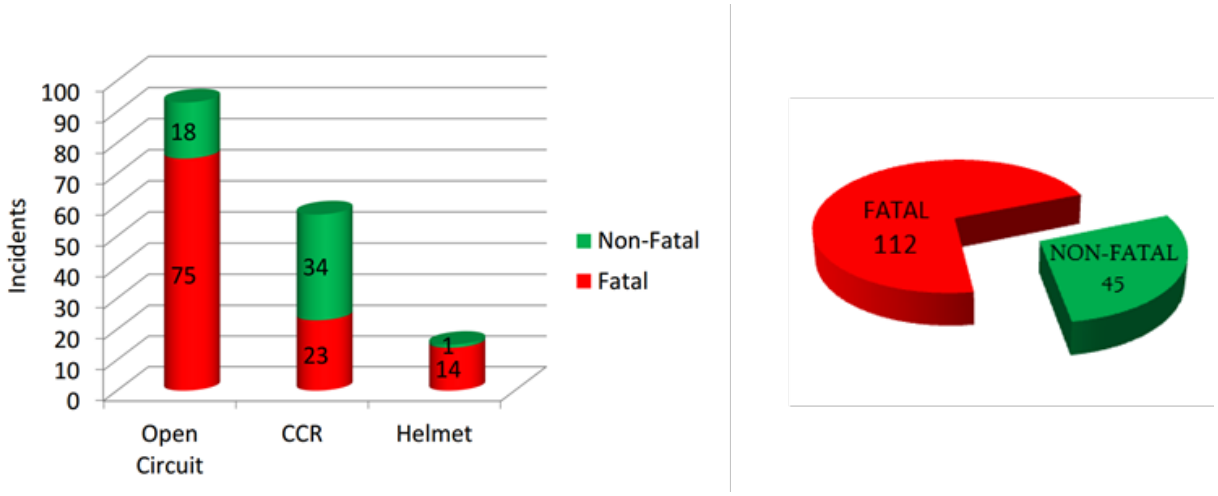

Figure 11. 34-years of accident investigations at NEDU (incident numbers are counts, not percentages).

Figure 11 also shows that although helmet diving is relatively safe, when something does go wrong, it is usually fatal. The data plotted do not distinguish among the various modes of helmet diving failure; loss of surface gas, flooding, or helmet coming off the diver at depth.

Compared to open circuit (scuba) or helmet accidents, accidents involving closed-circuit (CCR) UBA are, in NEDU's experience, not as lethal (From the chi-square test, that difference in proportions is significant).

It is not possible from the existing record for us to know for sure, but it could be that the presence of bailout gas, either open or closed circuit, available in many but not all cases, as well as the presence of UBA alarms and good diver training, may save a greater proportion of the closed-circuit divers involved in accidents compared to other forms of UBA accidents.

The Venn diagram (Figure 12) shows that virtually all accident causes involved human error in one form or another. The human error-based events could be ascribed to a combination of actions involving equipment, or to a lesser extent, maintenance.

As a medical examiner, you should keep in mind that the above statistics represent a relatively small cross-section of all accident cases, namely, those submitted to the U.S. Navy for analysis. Other data samples and analyses, for example the database compiled by Deeplife (www.deeplife.co.uk) will likely yield somewhat different results. However, common to virtually all accidents in diving or aviation, is the ghostly presence of human error.

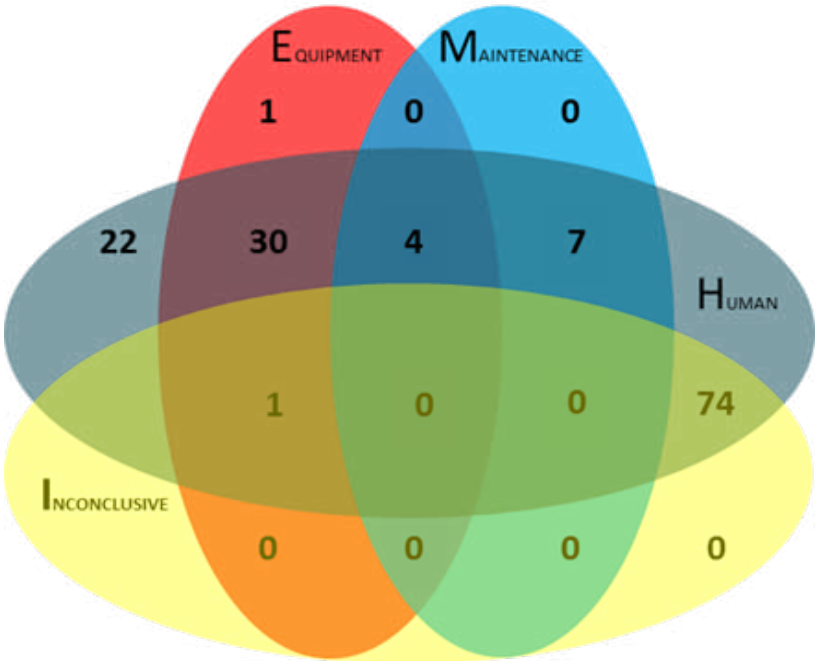

Figure 12. Venn diagram of incident causes.

Oxygen control in electronic closed-circuit rebreathers

One of the ways for a rebreather diver to die without knowing his demise is eminent, is through failure of the oxygen control system.

Figure 13. Triplicate oxygen sensors used in electronically controlled rebreathers.

It is not the objective of this presentation to discuss the safety features and operation of triple-redundant sensor systems in rebreathers. However, we will show that human error can compromise the safest possible oxygen control systems.

When oxygen control systems fail, the failure usually results in either high oxygen levels (hyperoxia) or low levels (hypoxia).

While all physicians are familiar with the results of hypoxia, hyperoxic events are poorly understood by most. When a rebreather starts continuously adding O_2 at depth, very high partial pressures of oxygen can occur, eventually resulting in oxygen-induced seizures. During the tonic-clonic phases, unconscious divers are likely to expel their mouthpiece, and be physically incapable of taking another mouthpiece. The post-clonic phase yields a deep inhalation as the body attempts to reoxygenate after a prolonged breath hold.

Of course, with a mouthpiece out, the diver either inhales water, or suffers laryngospasm due to the attempt to inhale water. Either way, the end result is usually drowning.

The following were incidents where a rebreather's oxygen control system was damaged. The first was a fatality.

MK 16 military rebreather fatality

Damage to a Viton O-ring on an oxygen-add valve (solenoid) occurred due to improper Bendix connector attachments, which allowed water to enter the connection, preventing operation of the solenoid (Figure 14). The diver died because approved emergency procedures were not followed.

Figure 14. Damaged components of a MK 16 rebreather involved in a fatality investigation at NEDU.

Near fatality - MK 16 diver unconscious

Figure 15. Improperly repaired cable connections in a MK 16 rebreather. (Photo credit NEDU)

An improper repair to an electrical connector degraded, causing loss of voltage connection between the electronic controller and the battery (Figure 15). This diver beat the odds.

Malfunctioning oxygen sensors

The performance of triplicate oxygen sensors is monitored by circuitry or software which makes decisions about sensor reliability. The controller votes out a reading which is disparate from the others. Fatalities or near fatalities occur when: 1) voting logic votes out the only correctly reading sensor, or 2) turns off alarms due to changes in sensor reading status (an example of alarm failure is found in Clarke, 2009). A remarkable example of voting logic failure is described below.

Dual sensor failure at Lake Mead: lessons learned

Due to seriously out of date oxygen sensors, the experienced government diver lost consciousness during convulsions at 110 ffw (34 mfw). His loss of consciousness from hyperoxia at 110 ffw (34 mfw) was instantaneous, with none of the often reported premonitory symptoms (VENTID-C[4]). His rebreather mouthpiece was out of his mouth when he was found by his buddy diver.

NEDU suspected there was a problem with the diver's oxygen sensors, a suspicion confirmed by our tests of the triplicate sensors (Figures 13, 16 and 17).

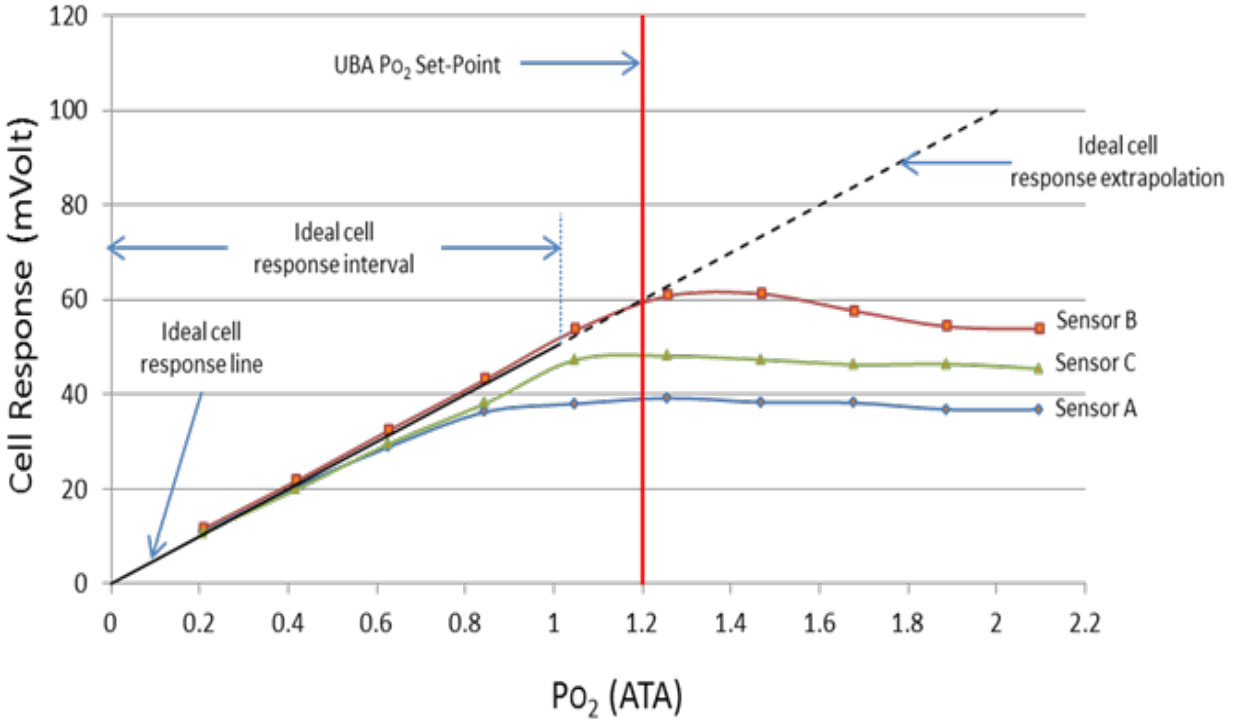

Figure 16. NEDU tests: Oxygen responsiveness determined for each of the three sensors. As PO_2 was increased experimentally, an ideal oxygen sensor would increase its voltage output. All three sensors in this case stopped responding above 1.2 atmospheres. Rebreathers typically attempt to maintain 1.3 ata in the rebreather, and in this case the controller would have commanded continuous O_2 addition in an attempt to reach 1.3 ata.

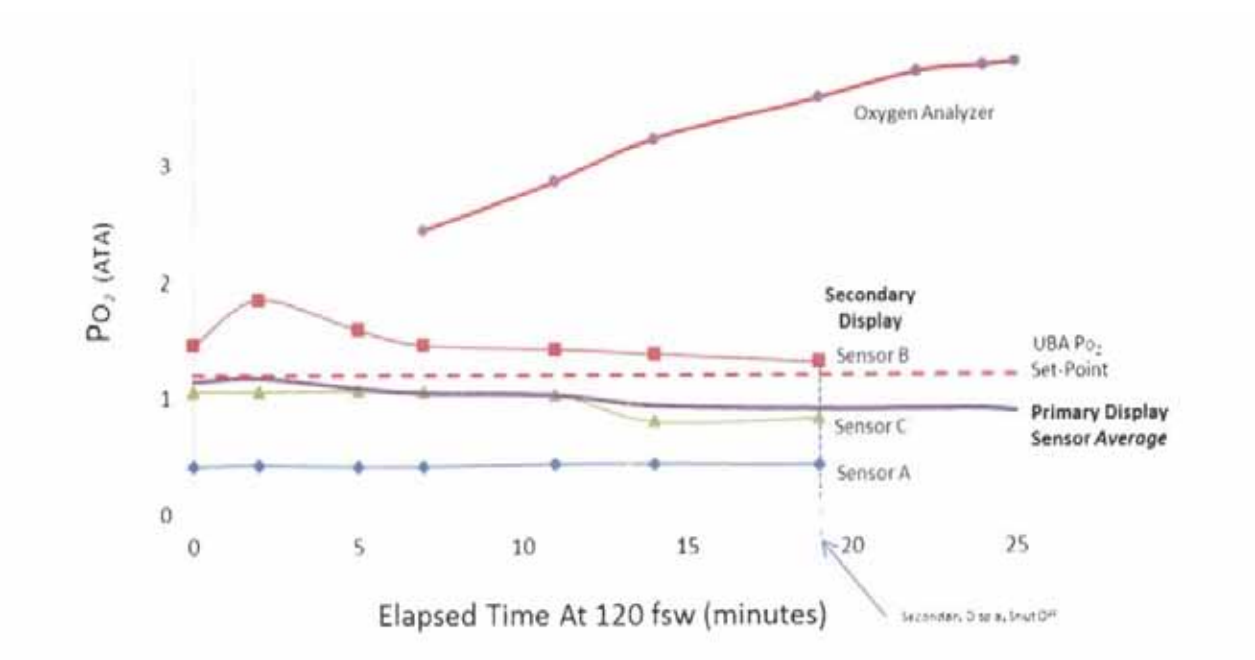

Figure 17. NEDU test: The intact rebreather with the three incident sensors in place. An independent oxygen analyzer revealed that the rebreather continuously added oxygen.

Fortunately for the forensics investigation, the diver's dive computer logged what was happening prior to the rig failure (Figure 18).

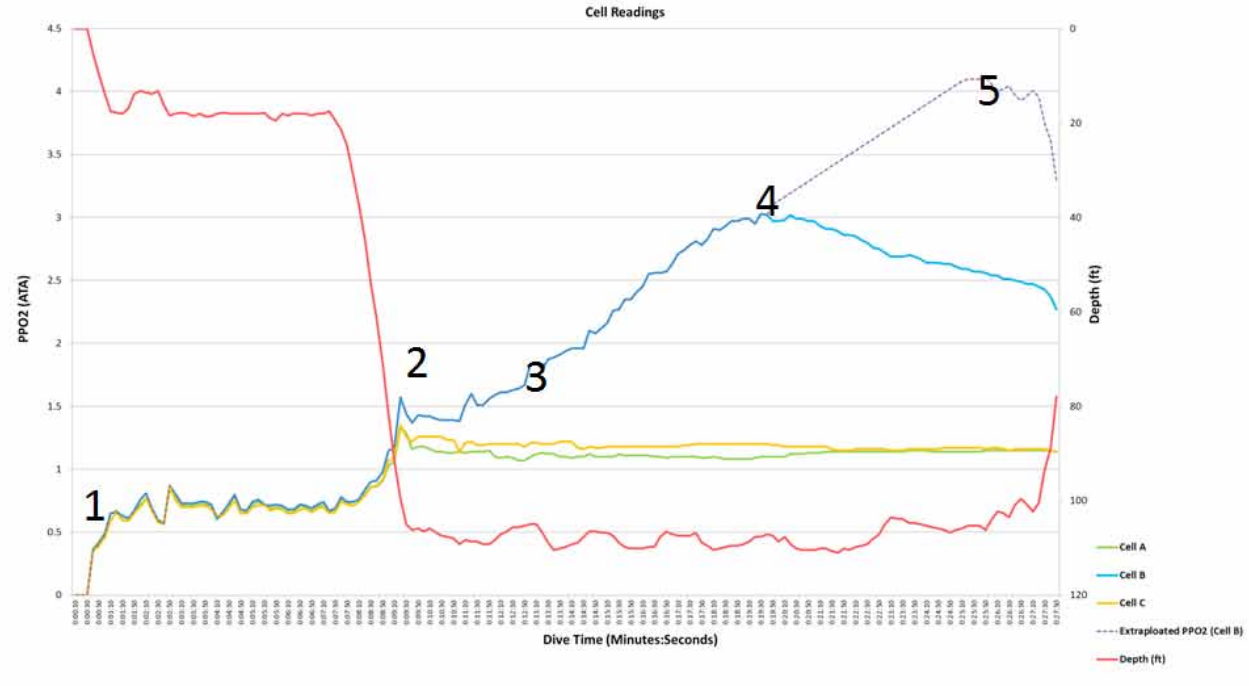

Figure 18. Plot from diver's dive computer/logger showing depth (red line) and three oxygen sensor readings. Numbers accompanying the tracings represent events described below.

The diver's story of near-death (Figure 18) and miraculous survival is best told in his own words.[5]

1. At 1.0 atm O_2 (maximum possible at surface) all three sensors read correctly.
2. 9:20 min into dive (1.3 atm) O_2 sensors start to diverge. Alert indicates one sensor out of range.
3. 2:10 into dive, O_2 in breathing loop exceeds 1.6 atm. Voting logic uses two failed sensors and tells diver that breathing gas is at approximately 1.2 atm (normal).
4. 20:10 into dive, O_2 in breathing loop reads 3.02 atm and third sensor starts to fail.
5. Approximately 26:50 min into dive, diver experiences O_2 toxicity/seizure/loss of consciousness. Diver's partner brings him to approx. 70 fsw (21 msw) and then releases him to the surface in uncontrolled ascent.

While indicated O_2 in the breathing loop was measured at a maximum of 3.02 atm by the functioning sensor, continual addition of O_2, as indicated by the gradual loss of pressure of the O_2 tank, shows that oxygen was still being added to the breathing loop. Inferred PO_2 reached 4.12 atm prior to the seizure.

Summary:

The common theme in these three events, one which was fatal, and two that easily could have been fatal, were induced by equipment failure, caused in turn by human error.

The first accident was caused by a lack of care handling cable connectors, and a failure to implement standard emergency procedures. The second was due to the breakdown of an improper connector repair. Presumably, emergency procedures saved that diver.

The final dramatic failure was avoidable: the sensors were supposed to have been replaced long before the accident occurred. Due to assumptions made about factory repair procedures, the diver had not checked the expiration date of his three sensors. Oddly enough, two sensors failed at the same time, and the controller ignored the one correct sensor. Eventually, after exposure to extremely high values of oxygen, the third sensor began to fail (at "4" on the sensor reading plots).

From a medical examiner's standpoint, if these three events had proven fatal, the pathology would have looked like that of drowning. The fact that drowning would have been preceded by hypoxia (cases 1 and 2) and hyperoxia (case 3), would have been easily missed without witness accounts of a seizure, or the tell-tale record of a dive computer showing either hypoxia or hyperoxia.

Through a combination of improbable events, the third diver survived to talk about his accident. He is the only diving accident victim in 23 years of investigations that I've had a chance to talk to about his accident. Due to the nature of their work, medical examiners will not have that luxury.

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Diving Medicine Expert Witness Perspective: "Consistent with Drowning"

Robert Wong, MD

Investigation of diving fatalities in Australia

In Australia, a coroner is a legal practitioner, usually one who is eligible to be appointed as a magistrate. Each state has a state coroner and is appointed by the governor on the recommendation of the attorney general. Hence, every magistrate is contemporaneously a coroner. He/she is not normally medically qualified. Before the Coroner's Act of 1996, there was no state coroner in Western Australia, a coroner then had the power to commit for trial cases of homicide, murder, manslaughter, infanticide and arson. However, a coroner cannot frame a finding so as to appear that a person is guilty of an offence, or to determine any question of civil liability. An adverse finding (such as negligence, contravening the laws of occupational health and safety etc.) has to be referred to the director of public prosecution to be dealt with. In Australian law, there is no compensation for emotional pain and grief.

When an accidental death occurs, a coronial inquiry takes place. The coroner may direct an inquest to be held, and may direct a medical practitioner to perform an autopsy. This procedure is usually performed by a qualified pathologist; but there have been occasions (in days gone by) when the autopsy was performed by a general practitioner (family medicine practitioner) especially in country towns where no pathologists were available. Even in capital cities, autopsy could be performed by pathologists who were not trained in forensic pathology, particularly involving diving fatalities. Unlike the U.S., a death certificate can only be signed and issued by a legally qualified medical practitioner.

Diving medicine is not normally part of the undergraduate medical curriculum, hence most medical graduates are not familiar with this discipline. Initially, the Royal Australian Navy ABR1991 issued guidelines on the conduct of autopsy of diving fatalities; subsequently, the Royal College of Pathologists of Australasia issued instructions to pathologists on techniques for performing autopsies of diving fatalities.

In Western Australia, diving fatalities have been few, hence most pathologists do not have vast knowledge or experience in the proper conduct of post-mortem of diving deaths. When faced with such a situation, few if any consult a diving medicine colleague or seek advice from an experienced pathologist. Coronial inquests are not automatic following an accidental diving death.

Most diving fatalities are labeled as drowning

I have been involved in providing advice to the coroner's court in Western Australia on diving fatalities for over 20 years. Sadly, most of the autopsy reports I have come across were of little or no use in assisting the determination on the cause of death.

Indeed the cause of death was commonly stated as "drowning" or "immersion", the final common pathway of someone who died in water, but no analysis was made of the "root cause". Some of the cases which have been labelled as "immersion" or "drowning" have subsequently been found to be due to other causes such as inhalation of inert gas (nitrogen), air hose entanglement (entrapment), hypothermia, and cuttlefish attack that caused perforated tympanic membrane, leading to panic, rapid ascent and gas embolism; there were also other causes labeled as drowning.

In reviewing past Australian records, Lippmann et al. reported Australian diving fatalities from 1972 to 2005[1]; of the 351 deaths, 49% were attributed to drowning. In cases reported in 2008, 52% were drowned (10 out of 19), but on review of the data, seven were attributed to cardiac events (in addition to the four cases diagnosed as cardiac caused). In the 2009 cases, 71%, (15 out of 21) were diagnosed as drowned; on analysis, again, four were related to cardiac events, also in addition to the five that were diagnosed as cardiac caused.

A case is presented here to illustrate the common fallacy that someone who died in water is due to drowning because the body was immersed in water. This is a final common pathway for someone who died in water, but what is the cause of "drowning"?

The case in point is one that occurred in January 2012 where the pathologist stated the cause of death as "consistent with drowning". On review, there were numerous factors that could have contributed to the diver's final demise. This is a plea to those who present evidence in courts or those who perform post-mortems and may not be familiar with diving fatalities to consult diving physicians or forensic pathologists who are experienced with diving fatalities. Guidelines for performing specialized post-mortem on diving fatalities are available.[2-6]

Case review

The deceased was an experienced diver, a 55-year-old man with over 200 dives; he passed as fit to dive by a physician in 2004, and was a PADI Open Water Certificate holder. The incident dive was the second dive of the day in calm seas to a depth of 54 fsw (17 msw), underwater visibility was 65 feet (20 meters), water temperature was 73 °F (23 °C). The deceased decided to go for a dive on his own. His previous dive prior to that day was in April 2011.

His son, shortly after boarding their boat 115 feet (35 meters) away, immediately responded to the call for help. When the son reached the deceased, he was face down, not breathing, with the BCD inflated but weights not ditched. Resuscitation was unsuccessful (initially in water, then he was taken onboard and CPR continued; eventually, medical and nursing staff arrived to assist with the resuscitation but to no avail).

All dive equipment was retrieved by the water police and tested. All was found to be operational, but the content gauge was defective. It showed 20 bars when retrieved (which on subsequent testing showed the actual content to be 5 bars), and when empty, it stayed at 12 bars (Table 1, Figure 1).

Table 1. Comparison of the true air content with the defective content indicator.

ACTUAL (Bar)	100	50	20	10	5	0
Deceased (Bar)	110	60	35	25	20	12

Autopsy was performed two days later, and a post-mortem CT was also performed (date unknown), which showed extensive intravascular gas, especially within the cerebral arterial circulation, the aorta, the coronary arteries, the carotid arteries etc. a pneumothorax was also noted.

The diagnosis made by the pathologist was that of drowning. His reasoning was that:

1. There was no indication of rapid ascent (I am not aware where such evidence was obtained).
2. The heart was opened under water and there was no evidence of significant gas at that time (In the post mortem report, it was stated that the left atrium was opened underwater, and no gas bubbling or frothy fluid was identified).
3. The lungs were heavy (902 g right; 944 g left) and edematous.
4. There was no evidence of injury or natural disease to account for the death.

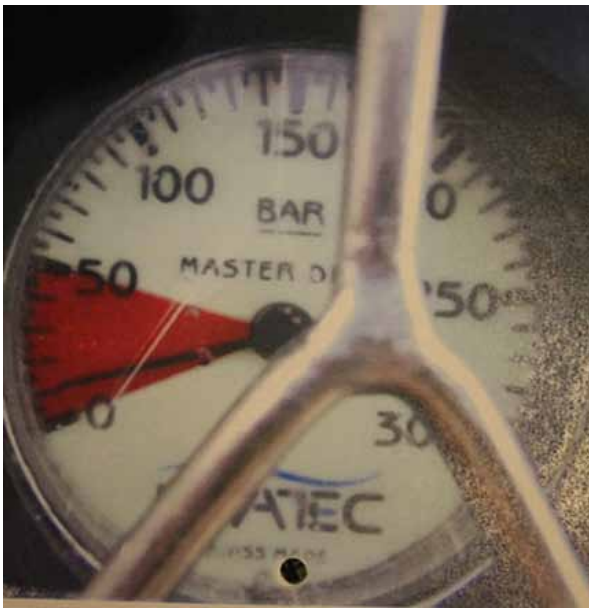

Figure 1. Defective content gauge.

The extensive intravascular gas being present was a fact that was unclear to the pathologist. He stated it was not possible to exclude "off-gassing" from diving; he stated further that this is usually associated with death occurring at depth rather than on the surface. No tests were done on the gas. There was no mention of searching for signs of mediastinal/subcutaneous emphysema over the chest and neck area which would lend support to the possibility of pulmonary barotrauma. Caruso described two approaches commonly used to check for a pneumothorax.[4]

A pneumothorax on CT was noted, however. A search for the presence of patent foramen ovale (PFO) was not made which could have given rise to paradoxical air embolism. There was a 40% stenosis of the left anterior descending coronary artery. There was no mention of an examination of the ears, whether there was evidence of barotrauma of the ear.

No mention was made of the dive equipment nor the various medical history which could have contributed to the final demise. The decedent suffered from anxiety and depression treated with mirtrazapine (tetracyclic antidepressant), cipramil (selective serotonin reuptake inhibitors (SSRI) antidepressant), and fluoxetine (or Prozac for anxiety and panic disorder. In addition, he had eczema and psoriasis treated with corticosteroids; and he was taking Chlorpheniramine (anti-histamine). He also had sleep apnea requiring a Continuous Positive Airway Pressure (CPAP) machine.

In the post-mortem report, it was mentioned that there was evidence of previous neurosurgery with an apparent neurostimulator in situ – this intracranial stimulator was present in the right upper chest. Leads from this were tunneled subcutaneously above the clavicle up to the right side of the neck before emerging over the skull. One of these leads entered the skull through a burr hole in the right frontal bone and the other in the left before penetrating both frontal lobes. The neurostimulator was to treat essential tremor together with propranolol; however, there was no mention that the neurostimulator was in working order, or that all the leads were intact. Should the neurostimulator malfunctioned or one of the leads were detached, it could have led to tremor with resultant anxiety and perhaps panic. There was also an episode of head injury with resultant organized subdural hematoma found in post-mortem CT was done at the time of injury (November 2011), but was not followed up by the pathologist.

According to the Guidelines of Royal College of Pathologists of Australasia, CT should be performed within eight hours of death. Cole et al., using sheep, showed that a simulated dive of 45 mins to 59 fsw (18 msw) could produce large amounts of gas on CT scan due to post-mortem off-gassing at 8 and 24 hours. While decomposition could produce intravascular gas, hydrogen and methane in the recovered gas would be an indication of decomposition. With Pulmonary Barotrauma (PBT) and Cerebral Arterial Gas Embolism (CAGE), gas would be found in the left side of the heart, Circle of Willis, and coronary and retinal arteries, where there is a low probability of post-mortem "offgassing" or decomposition. It has also been suggested to aspirate the chambers of the heart to record the volume of gas in each chamber and correlate with the CT scan. Furthermore, Caruso indicated that when autopsy is performed soon after death, intravascular bubbles present mainly in arteries are suggestive of air embolism.[7] Also, gas present only in the left ventricle (or if analysis shows gas in the left ventricle) has a higher oxygen content than that present on the right side would also lend support for the occurrence of an air embolism. Intravascular gas from decomposition or off-gassing from the dive would contain little oxygen and be made up mostly of nitrogen and carbon dioxide. None of these facts were mentioned or indeed performed in the autopsy of this diving fatality.

The conclusion reached by the pathologist was that there was no indication of speed of ascent (no evidence of rapid uncontrolled ascent – I am not aware how this conclusion was derived) and that the heart was opened under water and there was no evidence of significant gas at that time. A CT was done, which showed extensive intravascular gas especially within the cerebral arterial circulation: the aorta, the coronary arteries, the carotid arteries and some abdominal vessels. A small pneumothorax was also noted but the pathologist commented that the exact significance of this extensive intravascular gas is not clear to him. He added that the dive was only to 39 fsw (12 msw) and there was no indication of speed of ascent. He was obviously not aware that a transpulmonary pressure (difference between intra-tracheal and intrapleural pressure) of 37 inches (95-110 cm) of water is sufficient to rupture the lung parenchyma to cause PBT. [8,9] In vivo, documented cases of PBT have been shown to occur in as little as 3 – 4 fsw (1 – 1.2 msw or 92-120 cm).[3]

The pathologist, in a letter to the coroner, also stated that the case "may represent PBT/CAGE… it is not possible to exclude 'off gassing' from diving" - although this is usually associated with death occurring at depth rather than on the surface. He, however, suggested that there were a few features that would be consistent with PBT/CAGE such as crying out for help, collapsing on the water surface, a pneumothorax and intravascular gas.

The conclusion drawn was "Consistent with Drowning" – because of:

1. Heavy edematous lungs
2. No evidence of injury or natural disease to account for death
3. Heart was opened underwater and there was no significant gas at the time and there was no definite history of rapid ascent

What was missed in the autopsy?

Past medical history was not taken into consideration (dislocated shoulder when he fell in a ditch five months earlier before the incident dive and when he also sustained a head injury). No information on this event was sought e.g. post traumatic amnesia, Glascow Coma Scale, subdural hematoma, post-traumatic epilepsy, depressed skull fracture, rhinnorhea or otorrhoea etc.

Other relevant medical history include:

- Sleep apnea requiring CPAP
- Depression, on mirtrazapine (tetracyclic antidepressant); Cipramil (SSRI); Fluoxetine (anti-depressant)
- Eczema and psoriasis – on corticosteroids
- Allergy on anti-histamine Chlorpheniramine
- Implanted neurostimulator to control essential tremor

CT was done, but was not taken into consideration when making the diagnosis. No radiologist was consulted on the findings of the CT scan.

Dive equipment was not considered (such as low on air).

Dive profile was not considered.

No attempt to see if there was any significant subcutaneous or mediastinal emphysema.

Tympanic membranes were not examined.

PFO was not sought which might have given rise to air embolism.

Scuba Divers Pulmonary Edema as a diagnosis was not entertained.[10,11]

On analysis,

- Was the diver "fit to dive" on that occasion? Or was he ever fit to dive? Was he psychologically or physically fit to undertake the dive? Although he was "passed fit to dive"by a physician, was the physician trained in diving medicine?
- The diving environment was certainly suitable for diving on that day.
- The equipment was faulty – content gauge was inaccurate.

Conclusion

The triggering event was likely to be low on air (defective content gauge), which would have made it difficult to obtain air from the cylinder; as he has a personality of anxiety and depression, it was very likely that the incident would have led to panic and over breathing of the regulator when he discovered that he was low on air. This could have led to breath-holding and ascent to the surface causing pulmonary barotrauma and cerebral arterial gas embolism.

Despite all the available information, the pathological diagnosis made was "Consistent with Drowning".

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Expert Panel Review of Investigation and Autopsy Findings

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Abstract

Pathologists, diving medicine doctors, equipment experts, scuba instructors, lifeguards, police, Coast Guard personnel, recovery divers, and death investigators each have their own skill sets that can either conflict or muddle a diver death investigation if they do not communicate, or complement each other well to form a thorough investigatory body. A model committee formed in San Diego brings together representatives from all of these groups for review of diver deaths to maximize the knowledge and experience of everyone for the benefit of the investigation and to promote safety for the local diving community.

Introduction

As has already been evidenced, the investigation of a diver death is complex, due to the interplay of the equipment, environment, natural disease, human behavior, and the physics and physiology of the body under pressure. However, few forensic pathologists have much, if any, familiarity with diver deaths. So, they may be unaware of the potential for all of these factors to play a role in the death. They may also suffer from high caseloads, limited time, and lack of resources that prevent them from being able to invest adequate time in such an investigation after the autopsy is complete. Altogether, they develop a sort of tunnel vision that causes them to think that because the diver was in the water (the "hostile environment") and then died, he or she simply must have drowned. In reality, it is never so simple, and the wise death investigator will seek out the expertise of others to figure out not only the cause of death, but what the triggering events were that resulted in that cause of death.

The San Diego Diver Death Review Committee

In most jurisdictions, there are no special protocols for investigation of a diver death. It is only in larger metropolitan areas such as Los Angeles or Miami, or smaller offices with comparatively high numbers of diver deaths (such as the Florida Keys), that will have protocols or at least sufficient familiarity with such cases to ensure that an investigation is completed properly. However, only one jurisdiction (to my knowledge) has a formal review procedure with an expert panel: San Diego, California.

In 2008, based on a group that had functioned in the 1980's or 1990's and at the behest of Captain Nick Lerma of the San Diego Lifeguards, the San Diego Diver Death Review Committee was formed. (Notably, the name is awkward, but the members have been more concerned about their work than the name.) The group consists of members from the San Diego County Medical Examiner's Office, San Diego Lifeguards (and Dive Team members), San Diego Police (also Dive Team members), University of California San Diego (UCSD) Hyperbaric Medicine staff, Scripps Oceanographic Institute, San Diego State University, the United States

Coast Guard (USCG), and select members of the local diving community. Since 2008, every diver death has been formally reviewed by this group.

Cases

From 2007-2013, San Diego County had 18 scuba diver fatalities, with two to three per year. Of these, the manners of death were classified as Accident in 15 and Natural in 3. However, of the 15 deaths with accident as a manner, natural disease played a role in 7. If one reframes these deaths, then natural disease played a role in 10, or over half of the deaths. Certainly this number alone is an indicator of the role that health can play in diver deaths!

If we examine the causes of death listed in these cases, they include drowning (with an injury contributing in one), arterial gas embolism, immersion pulmonary edema, sudden cardiac death, and drowning with cardiac issues contributing. While these causes give some information, they tend to leave one wondering, "But what really happened?" Three case examples are presented and illustrate the importance of the various committee members:

Case 1: This 65-year-old man was on a liveaboard boat and had conducted three unremarkable dives. During a fourth dive, he indicated that he needed to ascend. He was conscious at the surface, but after rescuers reached him, became unresponsive. The rescuers prevented him from ever going below the surface. Autopsy documented over 90% stenosis of three major coronary arteries. Based on the history of a sudden arrest with his face out of water, his cause of death was certified as "Sudden cardiac arrest while scuba diving due to atherosclerotic coronary artery disease." It should be noted that the scuba diving was only included in the cause of death for case tracking purposes, and not because it was felt that diving played a role in the death any more than any other form of exercise would have. The manner of death was classified as natural.

Case 2: This 45-year-old man with almost 300 logged dives liked to dive solo and deep, over 150 fsw (46 meters), on a single cylinder of air. He had stopped by a lifeguard tower to inquire about local dives, stating that he was unfamiliar with the area. Sometime later, he was found floating 150 yards (137 meters) from shore in diving gear, but missing his mask. He was brought to shore and transported to a hospital, and death was pronounced in the emergency department. His dive computer data showed a maximum depth of 211 fsw (64 msw), with some up and down change after. He had risen to 79 fsw (24 msw), at which point his cylinder pressure had fallen to 153 psi (functionally empty at that depth for an 80-ft^3 cylinder). The cause of death was listed as "Drowning while scuba diving" and the manner of death classified as accident.

Case 3: This 54-year-old woman had an unremarkable medical history. The initial information presented to the autopsy pathologist was that she was an "experienced" diver in a class and that she had "surfaced and arrested." She was then taken to a hospital, where she died a few days later. The autopsy documented obesity, but her heart was not enlarged and she had no other natural disease. The other autopsy findings were those going along with anoxic brain injury and intensive care: non-perfused brain, hepatic centrilobular necrosis, and diffuse bowel ischemia. The autopsy did not provide much information as to why she was dead.

Lifeguards and police gathered further history. She was a certified diver, but had not been diving for about six years. In a refresher class, she descended to about 85 fsw (26 msw) with an instructor, but after two minutes indicated that she needed to ascend. She and the instructor began an ascent, but she showed distress at 40 fsw (12 msw). At 15 fsw (5 msw), she tried to remove her regulator, but the instructor kept it in place. They made a rapid ascent from that point. At the surface, she mouthed, "I can't breathe," and became unresponsive. She "seemed to vomit" frothy fluid, which was also noted by arriving medics and other responders. They

noted that the material "never stopped" coming up. After she was taken to a hospital, she was given a full Treatment Table 6 decompression, but had no improvement. Notably, as pointed out by one of the reviewers from UCSD, she had laboratory values upon hospitalization that strongly suggested arterial gas embolism. Examination of her equipment showed no issues.

This follow-up information came about as a result of committee members each doing their parts and coming together to synthesize the information. This allowed determination of the cause of death as "anoxic brain injury due to arterial gas embolism in the setting of immersion pulmonary edema while scuba diving." The manner of death was classified as accident.

Diver safety

In addition to examination of diver deaths, one of the committee's stated purposes is to use information gleaned from reviews to improve the safety of the local dive community. To this end, the committee has conducted a press release shortly before the beginning of the recreational lobster season, with guidelines identified by common trends seen in diver deaths:

- Ensure physical fitness to dive: train for your sport and be sure that you exercise regularly and follow a healthy diet.
- Use the buddy system.
- Follow your training: check your gauges often, respect depth and time restrictions, and do not dive beyond your training limits.
- Weight yourself properly and remember to dump your weights when appropriate.
- Ensure that your skill level and familiarity are appropriate for conditions.
- Have your equipment serviced and maintained regularly.
- Account for all divers (a physical, individual response should be received from every diver before entering/after exiting.
- Avoid overhead environments unless properly trained and equipped.
- Breath-hold divers should remember to use the buddy system and be aware of the dangers of shallow-water blackout.

Committee members also assisted the USCG in developing a diver-safety brochure in response to an increased number of diver deaths in California. Current plans include promoting a diver safety day and development of signage for local dive sites listing safety recommendations for local divers, with the grim admonishment that divers have died at those sites.

Conclusion

Any jurisdiction with one or more diver deaths per year is likely to benefit from a committee such as the one formed in San Diego. However, not all regions will have the volume of diver deaths to justify such a group. In those cases, the underlying point of the committee is still the same: diver deaths are complex and may require the expertise of others besides a death investigator and pathologist. Persons working in low-volume areas should seriously consider reaching out to experts outside of their regions for these cases.

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