Jerome H. Modell,[1] *M.D.; Monique Bellefleur,*[2] *M.D.; and Joseph H. Davis,*[3] *M.D.*

Drowning Without Aspiration: Is This an Appropriate Diagnosis?

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ABSTRACT: It has been reported that 10–15% of drowning victims do not aspirate water. We have revisited the original studies quoted to reach this conclusion and find it is without foundation. Sudden cardiac standstill is known to occur on land and, therefore, may also occur when the victim is in water. In the absence of the common finding of significant pulmonary edema in the victim's respiratory system, to conclude his or her death was caused by "drowning without aspiration" is unwise. All causes of sudden death that might occur in which respiration may not take place should receive serious consideration when examining bodies with such findings that are found in water.

KEYWORDS: forensic science, forensic pathology, aspiration, autopsy, drowning, near-drowning

For the past several decades, it frequently has been written that approximately 10–15% of drowning victims drown without aspirating water (1–4). The presumed reason for this is that the victim experiences laryngospasm, chest-wall spasm, or both when submerged and dies without taking a breath (1,4,5). The original studies referenced for this phenomenon are by Cot, who reported his work in the French literature in 1931 (6).

Acceptance of this premise leads investigators to attribute all deaths to drowning when the body is found in water and there is no obvious other anatomical cause of death at autopsy. This assumption precludes the possibility that death could have occurred from any other cause such as sudden cardiac standstill, a lethal cardiac arrhythmia, or foul play in the absence of obvious trauma. We have reviewed coroners' reports that attribute death to drowning in the face of a negative autopsy and list the evidence as "body found in water," "skin of feet wrinkled," or "drowning by exclusion."

Unfortunately, there is no specific, conclusive test to diagnose drowning as a cause of death. In 1921, Gettler proposed using a discrepancy in the concentration of chloride in blood between the right and left ventricles of the heart as a conclusive diagnostic test (7). As early as 1925, Palmer and Doherty expressed some doubt about Gettler's original conclusions (8). Moritz pointed out that variation of blood chloride concentrations occurred as a function of the post-mortem interval alone (9).

Durlacher, Freimuth, and Swann concluded that the plasma specific gravity difference between the two sides of the heart was more reliable than the chloride changes in diagnosing death by drowning (10). Modell and Davis, subsequently demonstrated in their examination of 118 victims of presumed drowning and 24 persons who died of other causes, that neither difference in chloride concentration nor specific gravity between the right and left heart were reliable tests for drowning (11). They found the specific gravity of plasma to be less in the left ventricle than the right in 91% of fresh water drowning victims, 79% of salt water drowning victims, and 75% of nondrowned persons. These tests, more than likely, are positive when a large quantity of water is aspirated and when the circulation persists for a short period of time. This short period of circulation permits fluid to transfer across the alveolar capillary interface in the lung, but circulation is not long enough to permit equilibrium to occur once the fluid is absorbed in the case of fresh water aspiration (12), or hypertonic aspirate is diluted by pulmonary edema in the case of sea water aspiration (13).

Thomas, Van Hecke, and Timperman believed that diatoms found in the body were of great diagnostic value in suspected death by drowning (14). However, Spitz and Schnieder have reported that air contamination could lead to diatoms in the tissues of nondrowned persons (15). Contaminated drinking water and swallowing water that has passed through diatomaceous earth, which is prevalent in swimming pool filters, can also produce diatoms in the viscera of humans.

Obviously, one cannot subject humans to submersion to see if any will die without aspiration of water. Likewise, it is inappropriate today to subject awake animals to such experiments. Therefore, we are left with re-examining the studies that subjected awake animals to submersion before current day ethics were applied to animal research, and also studies that quantitated fluid shifts across the alveolar capillary interface after water aspiration.

Review of the Literature

We began our review by performing a word-for-word translation from French to English of Cot's 1931 report (6). In that report, he reviews the classic experiments, which he states were accepted by the Medical Surgical Society of London. These included the studies of Bergeron and Montana, Paul Bert, Brouardel and Vibert, and Brouardel and Loye. From his review of those experiments, Cot reports that there are five phases to drowning. In these studies, dogs were submerged in their cages and remained underwater until death. Cot reports the first phase as one of surprise; during the first 5–6 seconds of submersion the animals fight in a "poorly active way." The second phase occurs rapidly thereafter and the animal

[1] Professor of Anesthesiology; Associate Vice President for Health Affairs, University of Florida, Health Science Center, Gainesville, Florida.
[2] Assistant Professor of Anesthesiology and Pediatrics, University of Maryland School of Medicine, Baltimore, Maryland.
[3] Professor of Pathology, Emeritus, University of Miami, Director (retired), Medical Examiner Department, Metropolitan Dade County, Florida.

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becomes violently agitated and makes some apparently purposeful efforts to exit his cage. During the second phase he keeps his mouth closed and no respiratory movement is observed although there are a few bubbles at the surface suggesting a small amount of exhalation occurs, but not inhalation. The second phase is reported to last around 1 min. This is followed by a third phase where there are profound respiratory movements and a few seconds later, white foam appears at the surface of the water. The agitation disappears and general movement stops with the dog lying on his side, immobile and performing some swallowing movements. This phase is reported to last approximately 1 min. The fourth phase is characterized by the arrest of the circulation with an absolutely immobile thorax, corneal reflex disappears, and the pupils become dilated. One minute later the fifth phase occurs in which there are three to four respiratory movements and then no further evidence of life except for fibrillating contractions of the lips and jaw. The exact time periods vary somewhat from study to study, however, he summarizes this work by stating the time necessary to produce death by the process of abrupt submersion is about 3½ to 4 min.

Cot also mentions that some investigations have shown that cardiac activity exists even beyond this time and the exact timing of the phases is species dependent. He reports that in the case of the duck, the period of apnea can last 4–5 min and also that the heart of a young dog can continue to have some activity for up to an hour. Cot also reports that Brouardel immersed tracheotomized dogs and observed that in these dogs, a period of apnea persisted, thus proving that contraction of the respiratory muscles occurs, not just closure of the glottis.

Cot then proceeded to subject 18 animals to submersion and removed them from the water at various time periods. The animals that he kept submerged for 3 min presented all of the phases and signs of drowning reported above. Upon exit from the water, these animals did not have residual respiratory or cardiac movements. Cot attempted to resuscitate these animals by using an intra-cardiac injection of coramine, 1 cc, applying the artificial respiration method of Schäfer, rubbing them with warm blankets, applying hot air to the bulbar area, and injecting subcutaneous camphor oil at the fifth minute. Resuscitation using these methods was not successful. Likewise, he could not resuscitate dogs that were immersed for 2 min.

For 11 of his animals, he confined the submersion period to 1 min and 30 s. He reported that the average time observed for the first two phases in his animals was 50–75 seconds. In that time, the third phase started characterized by marked agitation, violent convulsions and profound inspiratory movements. Resuscitation was successful in two of the animals that were submerged for this shorter time period, which Cot attributed to the fact that the inspiratory phase did not have time to occur.

Cot reports that autopsy of animals, which died by submersion, revealed a foamy fluid coming out of the trachea, present in the bronchi and pulmonary alveoli, and dripping from the cut surface of the lung. He also described this as "bloody spume" (foam or froth). He reports that the quality of the spume was very different from that of the immersion fluid. The foamy fluid was rich in albumin and suggested that aspirated liquid per se accounted for only about 10% of the total amount of foamy fluid present. He concluded that the spume is formed uniquely by a mixture of air and blood plasma. Cot also concluded that it was not the presence of the aspirated fluid in the lung per se that caused death from submersion, because only small amounts could be recovered, but rather the animal's reaction to this process and the characteristic bloody frothy fluid that was present in the alveoli, bronchioles, bronchi, and trachea.

To investigate whether inhalation of water was crucial in this process, he submerged two dogs in which he had ligated their trachea, thus preventing the inspiration of water. He reports that in these lungs they did not observe any notable congestion. There was no dripping of plasmatic fluid, or presence of the characteristic foamy material that he observed in the animals that he had drowned in the classic sense.

We cannot find anywhere in his paper where Cot states that 10–15% of his experimental animals drowned without aspiration. We did observe, however, that two of Cot's 18 experimental animals (11%) were successfully resuscitated and lived in an apparent normal condition when removed from the water 1 min and 30 s and 1 min and 50 s after being submerged. These animals did not appear to breathe under water nor did they exhibit the fulminating pulmonary edema such as he described in those animals that breathed water and died. Because these animals were removed from the water before inhalation occurred and, therefore, all phases of drowning were not complete, the term "drown" is not appropriate here. A more appropriate term to describe Cot's two dogs would be "near-drowned." These animals are similar to the patients reported by Modell, Graves, and Ketover where, based on an arterial blood oxygen tension while breathing room air of 80 mmHg or greater, they concluded that 11 of the 91 patients (12%) that they studied had "near-drowned without aspiration" (16). None of these patients had suffered a cardiac arrest while submerged and, therefore, had not experienced the full course of events necessary to "drown."

In 1933, Karpovich reported on his experiments in which rats, guinea pigs, and cats were submerged. He described the phenomenon of drowning as being divided into five stages: 1) an immediate struggle for freedom, sometimes a surprise inhalation; 2) suspension of movement, exhalation of a little air and frequent swallowing; 3) violent struggle for freedom; 4) convulsions, expiration of air and spasmodic inspiratory efforts with the mouth wide open, disappearance of the reflexes; and 5) death (17). Subsequently, in 1939, Loughead, Janes, and Hall submerged dogs and described a period of breath-holding and struggling for approximately one and one-half minutes, which was followed by swallowing large amounts of water, gasping with aspiration, spasmodic struggling resembling tetanic convulsions, and violent vomiting. Finally, there was a loss of all movement and, within five min, apparent death (18).

Cot was apparently intrigued by the fact that in many instances of experimental drowning, aspirated water, per se, could not be retrieved from the lungs of the animal. This might suggest that water is not aspirated. However, he reports that when dogs are drowned in a liquid colored with methylene blue, this liquid penetrates abundantly in the lungs and is seen under the pleura in the subpleural alveoli. Also, when dogs were drowned in bismuth milk and radiographs were performed, the substance was equally distributed in the entire lung. However, in the cadaver immersed after death, the deposition of the opaque substance occurs only in the most dependent area. This substantiates the theory that for water to enter the lungs in any substantial quantity and with diffuse distribution active ventilation is required. Thus, individuals who might be dead before entering the water or who die of sudden death in the water, but without concurrent respiratory effort, would not have diffuse distribution of aspirated water and its associated pathophysiologic response and it would be inappropriate to record their diagnosis as drowning.

Cot does quote Brouardel and Vibert—that absorption of water introduced in the lungs fairly rapidly dilutes the blood. Brouardel

and Vibert reported that the dilution of blood that occurred could sometimes reach one-third of the total mass of blood, particularly when submersion occurred very slowly and where the animal was able to come up from time to time to breathe air at the surface of the water. Being very hypotonic, when fresh water is aspirated during the drowning process, it is absorbed very rapidly into the circulation. This fact, perhaps, made it difficult for Cot to reconcile his inability to retrieve aspirated water from the lungs of his animals that had remained submerged until death. This was apparently known at the time, but the correlation of quantity of water aspirated with resulting changes in blood volume was not fully appreciated until the experiments of Swann et al. (19) and Modell et al. (12,13).

When water is aspirated into the lungs, there is rapid absorption of hypotonic fresh water into the circulation and, in the case of seawater aspiration, there is rapid movement of plasma into the lungs to dilute the hypertonic seawater. This was clearly shown in the whole blood density experiments performed by Swann and his colleagues on dogs who were totally submerged in either fresh water or seawater in the 1940s (19). Under controlled conditions in anesthetized dogs, Modell and his colleagues quantitated the movement of fluid across the alveolar capillary interface by studying the changes in blood volume that occurred with known quantities of aspirated water. With quantities of aspirated fresh water as low as 2.2 mL/kg, blood volume increased within 3 min of aspiration by approximately 5%. There was a linear response to increase in blood volume with increasing volumes of fresh water aspirated so that 3 min post-aspiration of 11 mL/kg, the average increase in blood volume in their anesthetized animals was 115% of the pre-aspiration value, with 22 mL/kg it was 130% of the pre-aspiration level, at 44 mL/kg it was 150% and at 66 mL/kg it was approximately 160% of what it had been prior to aspiration (12). These investigators also quantitated the decrease in whole blood volume that occurred rapidly after aspiration of seawater, with the peak effect being demonstrated 1 min post-aspiration (13). The methodology used in the 1960s to determine blood volume in these studies was the radio-iodinated serum albumin technique. Because pulmonary edema occurs very rapidly after seawater aspiration, there is loss of the radioactive albumin tracer by 3 min into the lung, thus precluding appreciation for the full impact of the fluid shift.

After either fresh water or seawater aspiration, Modell et al. demonstrated a sudden onset of foamy/frothy liquid in the alveoli, bronchioles, bronchi, and trachea of their experimental animals (12,13,20). The etiology of this characteristic type of pulmonary edema fluid after fresh water drowning was later shown to be secondary to rapid temporary fluid overload (21) and the alteration of the normal surface tension properties of pulmonary surfactant (22). The pulmonary edema is frothy due to the presence of albumin and mixing with air and, it is pink in color from free hemoglobin, which results from rupture of red blood cell membranes in the presence of a hypotonic media and hypoxia (23).

After seawater aspiration, however, the osmotic gradient from the plasma to the aspirated seawater is likely the cause, because seawater contains a concentration of electrolytes approximately three times that of blood. Seawater being hypertonic, pulls plasma from the circulation into the lung, also resulting in an impressive amount of frothy fluid in the alveoli and conducting airways. Because red blood cells do not lose the integrity of their membrane as readily when sea water is aspirated (compared to when fresh water is aspirated), the frothy or foamy fluid may be white in color as it is not colored by free hemoglobin.

Swann was also concerned with the question of whether humans drowned without aspirating water. In 1962 he wrote (4):

> "Another matter, which is frequently discussed, is the possibility of death after submergence from simple asphyxia, with little or no water being aspirated. It is suggested that reflex spasm of the glottis prevents water aspiration. Or perhaps death occurs so rapidly from a postulated complete vagal arrest (from which the heart never escapes) that there is not time for water aspiration. This discussion has been going on for 200 years (24) and it is not settled yet. Most modern writers guess that it sometimes occurs, setting the incidence at 10 to 15 percent. The guesses are based primarily on those of Cot (6) and of Moritz (9). But none can claim to speak with much assurance on the point. In most experimental drownings, much water is aspirated, and it is difficult to see why man should be an exception to this rule. It must be borne in mind that aspirated fresh water is absorbed from the lungs with great rapidity, so rapidly in fact that the lungs may "dry up" in a very short time if the animal happens to cease aspiration of water in the last moments of life. Both Schäfer (25) and Tartulier (26) have seen this phenomenon. We have observed two such cases in detail in dogs; in both, no fluid could be drained from the lungs at autopsy, but both experienced strong hemodilution, rapid entrance of H_2O into the blood, and ventricular fibrillation (27). It is occurrences like this, with the lungs "dry" at autopsy, which, in our opinion, prompt the view that men may drown without aspirating much water. Actually, if blood were obtained for analysis in these cases immediately after death, as was done in the dogs, it would probably be found that a large quantity of water had entered the blood stream."

Understandably, controlled observations in humans are lacking. Noble and Sharpe have attempted to reconstruct the events that occur for human victims of drowning. These authors report that the victim panics, struggles violently, then makes automatic swimming movements. Apnea or breath-holding occurs and the victim then swallows large amounts of water; vomiting and gasping ensues and water is aspirated. Blood stained froth appears in the airways, the patient convulses and finally dies (3). This reconstruction by Noble and Sharpe is not from direct observation but rather extrapolated from animal experiments such as those reported above. For the individual who suddenly finds himself submerged and unable for whatever reason to break the surface, this reconstruction is reasonable. However, our experience is that this course of events is rarely described by observers who witness a drowning. In over 120 patients that we have had the opportunity to treat after rescue and over 230 victims who died, in which we have reviewed statements made by persons who were at the scene (bystanders, rescuers, paramedical personnel and the like), more often than not, the body is found either underwater or floating at the surface of the water without an eyewitness as to the immediately preceding events. Some of these victims, undoubtedly, suffered what is frequently termed as shallow water blackout. In this situation the victim hyperventilates, thereby lowering their arterial carbon dioxide tension and suppressing the stimulus to breathe. They, then, can breathhold underwater for a longer period of time than they could at eucapnia. Their oxygen tension, however, falls below the critical level to maintain consciousness and they then begin to breathe underwater and drown without expressing panic and struggling, which Noble and Sharpe presumed to occur and that others have found in various animal species (28–30).

The human victim of drowning may also have suffered trauma, such as hitting their head on the side or bottom of the pool during

swimming or diving and suffering a concussion or spinal cord injury, both of which would render them incapable of spontaneously exiting from the water. Others will partake of alcohol, which has been called the "ubiquitous catalyst" or drugs, both of which will alter their response to submersion and their ability to recognize when they are in trouble (31). Others still may be the victim of foul play or may have suffered a medical event such as a lethal cardiac arrhythmia, sudden cardiac standstill, coronary artery spasm, or a seizure and just happen to be in the water at the time.

Davis and Wright in their discussion of the very sudden cardiac death syndrome report that when ventricular fibrillation or very sudden heart stoppage occurs, little to no pathology is found in the lungs at autopsy. Conversely, if slower heart stoppage occurs, the lungs may be relatively heavy, congested, edematous, and demonstrate petechial hemorrhages of the pleura. They go on to point out that the structurally normal heart of adolescents and young adults may be prone to ventricular fibrillation, as is the case with persons with Ramono-Ward disorder with a prolonged QT interval (32).

In a study of prehospital ventricular fibrillation and sudden cardiac death, Liberthson et al. documented ventricular fibrillation in 52 of 70 patients and other terminal rhythms in the remaining 18 patients who had on-site electrocardiography monitoring by first responding emergency medical technicians. Of these individuals, 10% showed no coronary stenosis or other cardiac abnormality that might serve as a predisposing factor to ventricular fibrillation or sudden cardiac death. If these individuals had been found in water, the coroner who is prone to making a diagnosis of drowning by exclusion may have erroneously attached the "drowning without aspiration" label to cases of this type (33).

Conclusions

If the submersion episode is witnessed, this will usually provide evidence to support whether drowning actually occurred. Establishing a conclusive diagnosis of death by drowning in the unwitnessed situation may be difficult and all surrounding evidence and circumstances should be considered in determining the cause of death (34,35). If observers were able to describe the events as Noble and Sharpe believe them to be and not inconsistent with the animal experiments reported above, one can be reasonably comfortable with the diagnosis. For the victim who is not observed, however, and is found motionless in the water, one needs to be cautious and not a priori assume that the victim's death was caused by drowning rather than by some other event. Clearly, specific tests such as the chloride test, specific gravity test, or presence of diatoms cannot be relied upon for a definitive diagnosis. There may be a variety of other findings at autopsy of the drowned victim that are non-specific and are not universally present; such as hyperinflated lungs (36), middle ear congestion and hemorrhage, bloody watery fluid in the sinuses, and engorgement of solid organs, such as the liver.

The absence of free water in the lungs of a person that is found dead in the water is not uncommon. We believe that not all of these persons drowned. The presence of pulmonary edema per se is also found in persons with a variety of other conditions such as pulmonary infection, chronic pulmonary disease, cardiac failure, and trauma from cardiopulmonary resuscitation. This type of edema, however, is of a much finer quality and does not exhibit the frothy, foamy variety that is seen in the alveoli, bronchioles, bronchi and/or trachea of someone who drowns. We believe that one is on shaky ground by making a diagnosis of drowning at autopsy in the absence the characteristic foamy/frothy fluid seen after water aspiration.

Because all animal experiments we have been able to find in the literature that are descriptive of the drowning process include a period of ventilation underwater prior to death, and because we are unable to find evidence in animals subjected to the complete drowning process that the lungs are void of the characteristic frothy/foamy material characteristic of water aspiration, we question whether the concept of "drowned without aspiration" is valid. Unless there is evidence of the characteristic frothy/foamy edema of drowning as described above, we believe that all of the causes of sudden death that might occur in the water in which respiration might not occur should receive serious consideration when examining such cases found in water. Because causes of sudden death, such as cardiac standstill, lethal arrhythmias, or coronary artery spasm can not be confirmed anatomically after death, to ascribe drowning as the cause of death to a body found in water without some evidence of the effect of having aspirated water is risky. In this situation, it may be more accurate to list a differential diagnosis rather than a specific cause of death.

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Additional information and reprint requests:
Jerome H. Modell, M.D.
Department of Anesthesiology
PO Box 100254
Gainesville, FL 32610-0254
Telephone: 352-395-8076
Fax: 352-395-8013