Forensic Pathology Reviews
Series Introduction

Over the last decade, the field of forensic science has expanded enormously. The critical subfield of forensic pathology is essentially based on a transverse, multiorgan approach that includes autopsy, histology (comprising neuropathological examination), immunohistochemistry, bacteriology, DNA techniques, and toxicology to resolve obscure fatalities. The expansion of the field has not only contributed to the understanding and interpretation of many pathological findings, the recognition of injury causality, and the availability of new techniques in both autopsy room and laboratories, but also has produced specific new markers for many pathological conditions within the wide variety of traumatic and nontraumatic deaths with which the forensic pathologist deals.

The Forensic Pathology Reviews series reflects this expansion and provides up-to-date knowledge on special topics in the field, focusing closely on the dynamic and rapidly growing evolution of medical science and law. Individual chapters take a problem-oriented approach to a central issue of forensic pathology. A comprehensive review of the international literature that is otherwise difficult to assimilate is given in each chapter. Insights into new diagnostic techniques and their application, at a high level of evidential proof, will surely provide helpful guidance and stimulus to all those involved with death investigation.

It is hoped that this series will succeed in serving as a practical guide to daily forensic pathological and medicolegal routine, as well as provide encouragement and inspiration for future research projects. I wish to express my gratitude to Humana Press for the realization of Forensic Pathology Reviews.

Michael Tsokos, MD
Preface

A 2003 editorial in the well-renowned journal Science was entitled "Forensic Science: Oxymoron?." An oxymoron is a rhetorical figure in which an epigrammatic effect is created by the conjunction of incongruous or contradictory terms. This short article questioned both the reliability and validity of forensic sciences, alleging a lack of such criteria as error rate, adequate testing, regular standards and techniques, as well as a general lack of acceptance within the field. The will of those involved in death investigation was also questioned, calling for an improvement in the quality of their work in a scientific setting by noticing "Both these public interests—security and justice—would be furthered by a more scientific and reliable technology for analyzing crimes. The mystery here is why the practitioners don't seem to want it!"

From this editor's point of view it is generally impossible to quantify the pain of tortured victims and, without a doubt, the violation of human rights cannot be measured by biostatistical methods. However, police investigators and forensic pathologists have evidenced and documented ethnic cleansing in war zones and thus testified against war criminals in order to continually protect human rights over the past decade. Physical evidence of torture is properly analyzed where based in a scientific setting. Following regular standards and techniques for the identification of human remains, forensic pathologists and anthropologists are able to identify those who are killed by atrocious acts as opposed to civilian deaths. The components of weapons of mass destructions are analyzed by means of modern forensic science techniques. A harmonization of autopsy rules has gained worldwide popularity, providing highly scientific international standards. However, it is probably relatively easy to doubt the methods used in forensic death investigation when one has no insight into real forensic casework. But to doubt the will of those practitioners doing the field work is beyond any serious discussion.

One year after the appearance of the first volume of Forensic Pathology Reviews, this series has gained considerable attention within the forensic and medicolegal scientific community worldwide, which is, among other things, reflected in the efforts of 25 researchers from nine different nations representing four continents who have contributed to this third volume of the series. Most of the authors are the leading authorities in their particular fields of research. The
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chapters in this volume, once again, provide the reader with a profound scientific and practical knowledge on a broad variety of different topics.

Chapter 1 gives the reader a thorough insight into the medicolegal investigation of bodies found in water, focusing not only on victim identification, evaluation of postmortem submersion time, and determination of the cause and manner of death, but also in depth analysis of the pathophysiology of drowning. Chapter 2 devotes attention to human immunodeficiency virus (HIV)-1 infection of the central nervous system in the forensic pathological setting. The forensic pathologist is frequently confronted with HIV-1 infection, especially in the context of drug abuse. In particular, the sampling of specimens for histological examination during autopsy, the neuropathological examination, and the related findings of diagnostic relevance, including the macroscopic and microscopic appearance of opportunistic infections, cerebrovascular complications, and neoplasms associated with the disease, are emphasized. Chapter 3 deals with rare events such as deaths in a head-down position, which most often occur accidentally. The author examines the phenomenology and pathological features of such fatalities, providing new insight into the pathophysiology of inverse body position based on human and animal experiments under true and simulated microgravitational conditions.

Chapter 4 deals with forensic bitemark analysis, giving a comprehensive outlook on promising new areas of research in this field (e.g., the retention of DNA on skin over time and the newly described bacterial fingerprinting technique).

Chapters 5 and 6 are devoted to taphonomic changes of human bodies and their remains, namely the underlying biological processes and resultant postmortem changes that a corpse undergoes during the early postmortem interval. The broad range of variables influencing the morphological picture under which distinctive postmortem changes present, as well as elaborate findings that can serve as a basis for the macromorphological exclusion of a forensically relevant lay time of soil-embedded skeletal remains are provided.

Chapter 7 concerns arrhythmogenic ventricular dysplasia, a disease that plays a significant role that should not be underestimated in daily forensic pathological autopsy practice in cases of sudden death. The illness can lead to lethal cardiac arrhythmia and usually manifests during the third decade of life. Interestingly, regionally higher frequencies of the illness in some countries at least suggest a genetic disposition to the disease. Chapter 8 concerns the postmortem diagnosis of death in anaphylaxis. The authors provide the reader with an up-to-date overview concerning morphological, biochemical, and
immunological investigations toward the diagnosis of anaphylaxis and give helpful guidelines for practical casework.

Chapter 9 takes a comprehensive look at gross, microscopical, and genetic findings in the forensic pathological evaluation of fatal pulmonary thromboembolism and the potentially involved medicolegal issues. Chapters 10 and 11 cover aspects of suicide. A profound look at the trends of suicide in the United States during the twentieth century is given in Chapter 10. These trends have altered drastically, especially within the past century and most specifically in the United States. Chapter 11 addresses problems that may arise in the medicolegal investigation of murder–suicides, uncommon events that require careful investigation.

Chapter 12 deals with the investigation of iatrogenic deaths that constitute a substantial forensic contribution to injury prevention, medical audit, and continuing improvement in health care. Iatrogenic injuries such as perioperative hemorrhage, sepsis, trauma, embolic phenomena, cardiovascular and cerebrovascular events, complications associated with anesthesia, interventional radiology and radiotherapy, as well as adverse drug events and reactions are considered in detail. In Chapter 13, thorough information about the use of radiology in medicolegal investigations (e.g., for the location of foreign bodies within the body, documentation of mechanical injuries, identification purposes, or elucidation of child abuse) is provided.

Again, I owe great thanks to my contributors for making their practical and scientific knowledge available.

Michael Tsokos, MD
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Death From Environmental Conditions
Macroscopical, Microscopical, and Laboratory Findings in Drowning Victims

A Comprehensive Review*

Philippe Lunetta, MD and Jerome H. Modell, MD, DSc (Hon)

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SUMMARY

The medicolegal investigation of bodies found in water focuses on victim identification, evaluation of postmortem submersion time, and determina-

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tion of the cause and manner of death. In any given case the circumstances surrounding death, environmental factors, victim’s preexisting diseases, and autopsy findings must be appropriately considered in reaching a diagnosis of the cause and manner of death. In addition to drowning, injuries, intoxications, or natural conditions are all among the potential causes of death in bodies found in water or the factor that may have contributed to the fatal outcome. The interpretation of autopsy findings in putative drowning requires a basic knowledge of the pathophysiology of drowning. Hypoxemia plays a primary role in death by drowning, whereas serum electrolyte changes may be observed in experimental models but have little or no clinical significance in humans. The volume of liquid inhaled depends on factors such as the duration of laryngospasm, the number and depth of respiratory movements before death, and the time of onset of cardiac arrest. Recent studies suggest that the actual incidence of drowning without liquid inhalation is much lower than previously estimated. The most important morphological changes associated with drowning are those related to liquid penetration into the airways: external foam, frothy liquid in airways, and lung overexpansion. However, these changes are not specific to drowning. The diagnostic value given to microscopic pulmonary changes varies significantly and is limited mostly by their heterogeneous distribution within the lung parenchyma. Laboratory methods for the diagnosis of drowning have their rationale in the shift of liquid and electrolytes across the pulmonary air–blood barrier, which may cause blood volume and electrolyte changes. Although some methods have been reappraised recently, their usefulness is greatly hampered by factors such as the variable volume of drowning liquid penetrating the airways, the differing duration of the drowning process, and postmortem biochemical instability. Contributions on the reliability of the diatom method for the diagnosis of drowning have yielded widely divergent opinions, of which the most critical often rely on studies lacking a rigorous methodology. Until standardized protocols and reliable separation values for diatoms between control and drowning cases are established, the diatom method cannot be accepted in definitively proving a diagnosis of drowning in the courtroom, but rather represents a useful supportive tool for the diagnosis of death by drowning.

Key Words: Bodies found in water; drowning; pathophysiology; laryngospasm; dry lungs, long QT syndrome; morphology; diatoms; electrolytes; manner of death; body disposal.

1. Introduction

The focus and aim of any medicolegal investigation concerning a body found in water is victim identification, evaluation of postmortem (PM) sub-
mersion time, and determination of cause and manner of death. Localization of the site of death, which can be close to the place where the body is found, a remote aquatic setting or, in the case of the cadaver’s disposal, far away on dry land, represents an important element of these investigations.

The diagnosis of cause and manner of death relies on accurate assessment of autopsy findings, the victim’s individual characteristics, the environment, and circumstances surrounding death. A wide range of possibilities must be considered. The sequence of events leading to death in water or to a body being found in water can be complex: drowning, injuries, intoxications, or natural conditions are all among the potential causes of death.

Pathological processes and traumatic lesions, even trivial, sustained before entering or while in water as well as toxicological findings must be thoroughly considered to reconstruct the events that led the victim into the water because any of them may have triggered or contributed to fatal outcome. Death can occur accidentally during recreational or occupational activities or be the result of an intentional action, for example, either suicide or homicide. Even if the cause of death is determined, the manner may, however, remain difficult to assess.

2. Historical Aspects

Medicolegal problems related to drowning were already mentioned in the Chinese Hsi Yuan Chi Lu (1247 AD), the oldest existing textbook of forensic medicine (1). The chapter on drowning stresses the importance of determining the actual cause of death in bodies found in water. Although the chapter includes popular beliefs devoid of any scientific basis (e.g., position of the victim’s hand, eyes, hair to determine the manner of death, different floating positions for males and females), it also stresses the value of frothy liquid in the victim’s nose and mouth and of water in the stomach as evidence of in vivo submersion, as do modern studies.

In Europe, the first works of forensic medicine appeared during the Renaissance, after the Bamberg Code (1507) and the Constitutio Criminalis Carolina (1530) had highlighted the role of medical experts in the evaluation of injuries and causes of death in court. Textbooks by Paré, Fidelis, de Castro, Platter, Zacchia, Bohn, and Valentini are among the most representative and all contain passages concerning drowning.

Ambroise Paré’s Les Oeuvres (1575), in France, listed signs that prove the “vitality” of drowning as water in the stomach and abdomen, nasal secretions and foam protruding from the mouth, excoriations on the forehead and fingers owing to violent movement, and scraping against the bottom before
death (2). Fortunatus Fidelis, in Italy, wrote in the *De Relationibus Medicorum* (1602) that investigation of the drowned is usually not difficult: the drowning victim has a swollen abdomen; mucous secretion appears from the nostrils, whereas the secretion that protrudes from the mouth is foamy; the fingertips are excoriated. The tumefaction of the victim's body is not caused by swallowed water but instead is derived from the steam produced by warming up of liquids during putrefaction (3). Roderigo de Castro, in Portugal, also underlined in his *Medicus-Politicus* (1614) the dilatation of the abdomen, mucous secretion from nostrils, and foam from the mouth as signs of drowning, which are not present if the body is thrown into water after death. De Castro implicitly admits that excoriations on fingertips may be present even in bodies thrown into water after death. Moreover, the author maintains that the buoyancy of the body may be the result of its content in water or to PM gases (4). In Switzerland, Felix Platter wrote on drowning in his *Observationes* (1614) and later in the *Quaestionum Medicarum Paradoxarum* (1625). In the former contribution, Platter describes four cases of women condemned to drowning for infanticide who were thrown from a bridge into the River Rhine, but who were recovered from the water after a variable interval of being still alive (5). In the latter, he stresses that the stomach of drowning victims contains only a very limited volume of water and that the cause of death is asphyxia consequent to penetration of water into the airways (6).

Paulus Zacchia's work, *Quaestiones Medico-legalis* (1726), contains two passages on drowning. In the Libri Quinti, Titulus II (De Vulneribus), Quaestio XI, Zacchia highlights the difficulties in distinguishing whether a person has drowned or was killed before submersion. He reminds us that this issue has been addressed by Paré, Fidelis, and de Castro (mentioned previously) who have unanimously agreed on the following signs: swollen abdomen full of water, mucous secretion protruding from the nostrils, and foamy secretion protruding from the mouth; the nasal secretion is mucous because the cerebral ventriculi are obstructed by water as a consequence of respiratory arrest, and the mouth secretion is foamy because air is violently extruded from the lungs and respiratory organs. Respiratory arrest is the cause of death rather than the swallowing of water. The third sign is represented by the excoriations on the fingertips because of attempts to avoid death by grasping stones and sand on the bottom of the sea (7).

In Germany, Johannis Bohn in 1711 critically reviewed in his *De Renunciatione Vulnerum* the signs of the drowning mentioned by Paré, Fidelis, Castro, and Zacchia and stressed that these signs also may be absent in definite drowning and how, in some cases, the volume of water in the stomach or airways may be negligible (Fig. 1 [8]). Michaelis Bernhardt Valentini’s *Cor-
p³s Iuris Medico-legalis (1722) also contains passages on drowning, including case reports on infanticide and suicide as well as correspondence between the author and Johannes Conrad Becker on drowning without inhalation or swallowing of water (9).

3. Pathophysiology

During the 1940s and 1950s, Swann and associates performed experimental studies on the pathophysiology of drowning that have influenced modern views. Swann's works with dogs stressed the effects of drowning media of differing osmolarity on blood volume and serum electrolyte concentrations. In freshwater drowning, the hypotonic liquid penetrated into the circulation, causing hypervolemia, hemodilution with decrease of serum electrolytes, especially sodium (Na) and chloride (Cl), hemolysis with potassium (K) release from red blood cells, and death by ventricular fibrillation (VF) within 3 to 5 minutes. In seawater drowning, conversely, the hypertonic media pulled liquid from the circulation into the alveoli, causing hypovolemia and hemoconcentration with an increase in the concentration of serum Na, Cl, and magnesium (Mg), whereas no hemolysis and VF occurred, and dogs survived for 5 to 12 minutes (10–12).

During the 1960s, studies demonstrated that liquid penetration into an organism may cause no clinically significant electrolyte imbalance because the volume of aspirate may be small and that in death by drowning hypoxemia plays a primary role. In 1966, Modell et al. (13) evaluated, in anesthetized canines, the effects of inhalation of varying volumes of freshwater, from 2.2 mL/kg to 66 mL/kg body weight. Volumes greater than 11 mL/kg were needed to cause a significant alteration in blood volume, greater than 22 mL/kg to observe significant electrolyte changes, and 44 mL/kg or greater to cause VF. The inhalation of such volumes is unlikely to occur in humans because, using the magnitude of serum electrolyte changes found in human drowning victims and comparing these with animal experiments that have a known quantity of water aspirated, it has been calculated that 85% of human drowning victims aspirate only 22 mL or less of water per kilogram body weight (14). Accordingly, serum electrolyte concentrations of resuscitated drowning victims usually fail to reveal significant changes (15).

It is generally agreed that although pathophysiological differences between drowning in freshwater or saltwater may be observed in experimental models, these have little or no clinical significance in human drowning (16,17). The main physiological consequence of drowning is prolonged hypoxemia with resultant metabolic acidosis (18,19). Yet, in peculiar envi-
environments, significant electrolyte changes have been observed, for instance hypercalcemia and hypermagnesemia in the Dead Sea (20) and hypercalcemia in polluted water (21).

### 3.1. Sequence of Events

In humans, the drowning process has been described as a continuum that begins when the victim’s airways are located below the surface of the liquid,
which leads to voluntarily breath-holding and then laryngospasm triggered by the local effects of liquid on the upper airways (22). During this period, the victim does not breathe, which causes hypoxemia, hypercapnia, and respiratory and metabolic acidosis. The victim also may swallow water into the stomach. In human volunteers, the breath-holding breaking point varies from 87 seconds at rest to up to 146 seconds when preceded by hyperventilation (23). Once breath-holding breaks, the victim breathes and allows liquid to enter his or her airways. The respiratory efforts intensify, producing more intense negative airway pressure against a closed glottis, or the liquid column overdistends and ruptures lung alveoli. At this point, different authors believe one of two courses can occur. In 85 to 90% of the cases, as the arterial oxygen tension drops further, laryngospasm abates, and the victim actively inhales a variable volume of liquid (so-called “wet-drowning”). In the remaining 10 to 15%, the victim does not present evidence of water aspiration. Some attribute this to severe laryngospasm causing hypoxia, convulsions, and death before taking a breath (so-called “dry-drowning”) (16,24). These later cases led some researchers to question whether these victims actually die of drowning or of other causes (25).

3.2. Respiratory System

The primary target organ for submersion injury is the lung. The respiratory disturbance depends more on the volume of water aspirated than on its osmolarity. In animal experiments, the aspiration of 2.2 mL of water per kilogram body weight decreases the arterial $O_2$ partial pressure to approx 60 mmHg within 3 minutes (13). In humans, it seems that as little as 1 to 3 mL/kg produces profound alterations in pulmonary gas exchange and decreases pulmonary compliance by 10 to 40% (13,26,27).

Freshwater, which moves rapidly across the alveolar–capillary membrane into the circulation, produces disruption and denaturation of surfactant, which leads to an increase in surface tension and a decrease in compliance, atelectasis, and intrapulmonary shunts with marked ventilation/perfusion mismatching (26). In these conditions, as much as 70% of the cardiac output may be shunted past perfused but unventilated alveoli (18). Because of the liquid shift across the alveolar–capillary interface, the freshwater drowning victim may develop acute hypervolemia. In saltwater drowning, the hypertonic liquid draws protein-rich liquid from the vascular space into the pulmonary alveoli, causing damage to the basement membrane, dilution and washout of surfactant, and reduction of compliance (26). Pulmonary edema occurs rapidly, and usually within a few minutes the liquid-filled alveoli are incapable of normal gas
exchange, which leads to intrapulmonary shunting and a perfusion/ventilation mismatch (28,29). The shift of liquid into the alveoli results in hypovolemia. Systemic hypoxemia, in fresh- and saltwater drowning, causes myocardial depression, reflex pulmonary vasoconstriction, and alteration of pulmonary capillary permeability, all of which contribute to pulmonary edema.

### 3.3. Cardiovascular System

The effects of liquid penetration on the circulation have been studied in detail in animal experiments. Significant hypervolemia occurs in dogs after aspiration of at least 11 mL/kg of freshwater; within 2 to 3 minutes, a linear relationship occurs between the volume of water aspirated and the increase in blood volume (30). Blood volume increases by 1.4% for every milliliter of liquid/kg until 44 mL/kg of water is aspirated. At this value, the blood volume reaches a plateau, likely caused by the cessation of circulation (31). The absorption of large quantities of freshwater can result in a dramatic decrease in blood density (12). When the victim survives, the hypervolemia after aspiration of freshwater is transient, with blood volume returning to normal levels within 1 hour (32). This readjustment in blood volume is likely the result of redistribution of the liquid into other body compartments and to plasma transudation into the lungs. When significant quantities of seawater are aspirated, the reverse is seen, with hypovolemia and elevated concentrations of serum Na and Cl (28).

Cardiac dysfunction during drowning is predominantly secondary to changes in arterial oxygen tension and acid–base balance. The acute hypoxemia results in catecholamine release, leading to tachycardia and hypertension, which are transient and are followed by bradycardia and hypotension as hypoxemia intensifies. In addition, hypoxemia may directly reduce myocardial contractility. Hypoxia and acidosis elevate the risk for arrhythmias, including ventricular tachycardia, fibrillation, and asystole. A variety of electrocardiographic abnormalities have been reported after drowning, such as a decrease in the amplitude of the P-wave, disappearance of the P-wave, widened PR interval, complete atrioventricular dissociation, depression of the ST segment, widening of the QRS complex, frequent premature ventricular contractions, increase in amplitude of the T-wave, auricular fibrillation, and VF, among others (31). As previously discussed, early studies in the 1950s suggested that in freshwater death was caused by VF and in seawater by pulmonary edema (12), but several studies since then have shown that VF as an immediate cause of death is uncommon in human drowning victims (15).
3.4. Central Nervous System

Brain death is the common final pathway of fatal submersion, whether the pathophysiological mechanism is hypoxia attributable to liquid penetration into airways or to laryngospasm or anoxia from vagally mediated cardiac arrest (33). When the brain is deprived of oxygen for more than approx 3 minutes, ischemic damage can occur. It is estimated that a window of up to 4 to 6 minutes may exist before irreversible neuronal damage occurs when the oxygen supply is completely interrupted under normothermic conditions. The central nervous system (CNS) has a selective vulnerability to hypoxic or anoxic events, involving, in decreasing order of vulnerability in adults, the hippocampus, cerebral neocortex, cerebellum, thalamus, basal ganglia, brainstem, and hypothalamus (34). There is, however, no data as to exactly how long a drowning victim can remain submerged, receive cardiopulmonary resuscitation (CPR), and still recover with no sequelae. Among the factors that influence this interval perhaps the most important is the body temperature of the victim and the effectiveness of CPR applied. Generally, under normothermic temperatures, most researchers will agree that if a victim is rescued and effective CPR applied within 3 minutes, the vast majority of victims will successfully be resuscitated. By the time 5 minutes have passed, although return of an effective heartbeat is commonly observed, the majority of persons will show permanent hypoxic encephalopathic damage.

3.5. Other Organ Effects

Hypoxia secondary to drowning can affect various organs. Many reports point to acute renal and hepatic insufficiency, gastrointestinal injuries, and disseminated intravascular coagulation. Concerning abnormalities in blood-clotting factors, Modell et al. (35) described a child whose platelet count rose to 1.9 million/mm$^3$ after a submersion episode in excess of 20 minutes in cold water and who experienced a complete recovery.

3.6. Delayed Complications

Immediate complications of drowning include cardiac arrhythmias (VF, asystole) and cardiogenic shock caused by myocardial damage secondary to hypoxia and acidosis. At times, the drowning victim appears healthy in the emergency department but develops fulminant pulmonary edema as long as 12 hours after submersion owing to acute respiratory distress syndrome from the primary pulmonary damage by liquid, as a consequence of hypoxia and circulatory failure, or the drowning victim develops neurogenic pulmonary edema attributed to cerebral hypoxia. Acute respiratory distress syndrome also
may develop as a consequence of pulmonary injuries caused by aspiration of gastric contents. Common fatal sequelae of drowning in hospitalized drowning victims are brain death as the result of hypoxic encephalopathy, pneumonia (aspiration, chemical, bacterial), sepsis, and multiorgan failure. Posthypoxic encephalopathy may occur because of hypoxemia sustained during the drowning episode or secondarily to pulmonary damage or to increased intracranial pressure (22).

3.7. Dry-Drowning

The sequence of events that follows the penetration of liquid into an organism has been the subject of considerable speculation that has focused on the volume of liquid penetrating the airways during the drowning process and the concept of drowning without aspiration of liquid ("dry-drowning") (25,36). The volume of liquid aspirated varies considerably from one drowning victim to the next (14) and depends on factors such as the frequency and duration of laryngospasm, the number and depth of respiratory movements before death, and the time of onset of cardiac arrest. Experimental (37) and clinical (15) studies together with the autopsy finding of "dry lungs" in bodies found in water, suggest that death can occur with no significant aspiration of liquid into the lungs in approx 10 to 15% of alleged drowning victims (38–40). Dry-drowning has been variously explained. In addition to laryngospasm, the role of mechanisms, such as vago-vagal cardiac inhibition triggered by contact of the liquid with the upper airways, sudden cardiac arrest, pulmonary reflexes, or absorption of aspirated liquid into the bloodstream after prolonged resuscitation, have been proposed (30,40–42). Brinkmann (43) has listed different potentially life-threatening reflexes, which may occur in human beings during immersion or submersion.

The issue of dry-drowning has recently been reappraised, and the suggestion has been made that its actual incidence may be lower than previously estimated and that human bodies found in water with apparently normal lungs could conceal more natural deaths or body disposal in water than is actually recognized (19,22,25,44). The "laryngospasm" hypothesis has its rationale in the complex innervation and reflexes of the upper airways under various stimuli (45,46). However, no concrete evidence exists that prolonged laryngospasm until death occurs during submersion, whereas experimental evidence suggests that initial breath-holding and/or laryngospasm ceases within two minutes from the onset of submersion (23,48).
3.8. Hypothermia

After immersion in cold water, hypothermia, defined as body temperature below the normal range of 36.8 to 37.7°C (49) can be, especially at high latitudes, a component of drowning by its effects on the heart, lungs, and CNS. When body temperature is less than 33°C, hypoventilation occurs, and muscle rigidity ensues; between 28°C and 30°C, a decrease in heart rate and bradyarrhythmias occur, respiration becomes irregular, and apnea is a common feature; at temperatures less than 28°C, VF, severe bradycardia or asystole can occur, and respiration may be difficult to detect (24,36). As to the CNS, when body temperature decreases to less than 35°C, victims may become confused and disoriented, and at less than 33°C, they are semicomatose, with a substantial percentage of drowning occurring at this time. At temperatures less than 30°C, it may be difficult to distinguish between hypothermia and death, as frank coma supervenes (24,36). Cold-induced anaphylaxis in people with cold urticaria syndrome can be a rare cause of drowning (50).

People who drown in water less than 5°C generally have a better prognosis than those who drown in warm water because with metabolism diminution, O₂ consumption and CO₂ production decrease. For every 1°C decrease in temperature, there occurs a 7–9% decrease in oxygen required (25). Hypothermia is, however, a double-edged sword because it increases the risk for fatal arrhythmias especially below 28°C (51). The rapidity of the temperature fall in a body has a profound influence on the capacity of the brain to withstand hypoxia. In children, the large surface area-to-weight ratio associated with cold water aspiration often causes a rapid core cooling below 30°C during submersion, which gives some degree of cerebral protection during hypoxia (52) and explains remarkable recoveries of children after even more than 30 minutes submersion with return to normal activity (24,35,53,54).

4. Macrossopical Findings

The main macromorphological changes associated with drowning (external foam, frothy fluid in airways, lung overexpansion) are related to the penetration of drowning liquid into the airways. These changes can be valuable for the diagnosis of drowning when interpreted within an appropriate investigative context. However, they are not pathognomonic for drowning and are not always detected because they fade with the onset of putrefaction. Changes involving the body’s systems other than the respiratory system will be briefly summarized in the next sections, although their relevance for the diagnosis of drowning is marginal.
4.1. Upper Airways

The penetration of drowning media into the respiratory system increases airway pressure and causes a reactive pulmonary edema. The mixture of drowning liquid with edema liquid, bronchial secretions, and pulmonary surfactant produces a frothy fluid which, under respiratory efforts during drowning, can reach the upper airways and be extruded from the nostrils and mouth, at times as a mushroom-like foam (Figs. 2 and 3).

The external foam and internal frothy liquid (Fig. 4) are generally white or blood-tinged (especially when freshwater is aspirated) and consist of drowning and edema liquid, mucus, and fine air bubbles, which are relatively resistant to collapse because of surfactant content. Respiratory epithelial cells and CD68+ alveolar macrophages have been isolated from the frothy fluid (55). External foam and frothy fluid may persist up to several days and, after the onset of putrefaction, become red-brown, the fine air bubbles being replaced by larger gas bubbles.

This external foam is considered one of the most valuable findings for the diagnosis of drowning, yet it can be observed also in cardiogenic pulmonary edema, epilepsy, drug intoxication, and electrical shock. Moreover, it is
generally found in a minority of drowning victims (43). In a series of 1590 bodies found in water, Lunetta et al. (2002) found external foam in 17.3% of the cases (56). Some authors underline the greater quantity of frothy fluid in drowning compared with other causes of death (42,57,58); however, no clear demarcation exists between different conditions. Great caution is necessary when interpreting the origin of frothy liquid in any suspicious death, in which the body may have been disposed of on land after homicidal drowning.

4.2. Lungs

The lungs of drowning victims with no putrefactive changes usually are waterlogged and overdistended (“emphysema aquosum”). Lungs occupy most of the pleural cavities with at times imprints of ribs on pleural surfaces and overlapping of the anterior margins on the mediastinal midline (Fig. 5). Lunetta et al. (2002) found overextension of lungs with overlap of the anterior margins in 42.1% of 1590 bodies found in water (56). Pleural adhesions can mask these changes. The lung surfaces usually are pale and mottled, with red and grey areas displaying sometimes marked alveolar overdistension. After their removal from the pleural cavities, the lungs retain their shape and size, and cut sections ooze a variable quantity of foamy liquid. Subpleural hemorrhages (Paltauf’s spots Fig. 6) are found in 5 to 60% of drownings (43), and their blurring aspect is the result of hemolysis within intraalveolar hemorrhages (42).
4.2.1. Lung Weight

Different studies have addressed the weight of lungs in freshwater and saltwater drowning. Lung weight alone has, however, little diagnostic significance because of frequent overlap between drowning and control values. Moreover, wide individual variations exist, as well as marked discrepancies as regards the normal range of lung weight (59–63). De la Grandmaison et al. (64) reported the most detailed data on lung weight based on 684 healthy adults who died of injury after a survival time of less than 1 hour. In males, the right
Fig. 5. Overdistension of the lungs with overlap of their anterior edges over the midline in a drowning victim.

As for the values in drowning victims, Copeland (65) found a right lung weight of $744.9 \pm 199.3$ g (SD) and left $655.4 \pm 184.2$ g in saltwater drowning ($n = 95$), whereas the corresponding values for freshwater were $727.7 \pm 210.6$ g and $657 \pm 206.3$ g. Kringsholm et al. (66) reported a combined lung weight of $1411 \pm 396.4$ g in 91 adults with a PM submersion time of less than 24 hours (66). Zhu et al. (67) suggest that differing body structure, pulmonary vital capacity, cardiac function, and survival time in water may account for differences in lung weight (67).
Fig. 6. Paltauf's spots (arrows) located in the upper lobe of the right lung. (Courtesy of Dr. Michael Tsokos, Hamburg, Germany.)

4.2.2. Dry Lungs

Apparently normal lungs with no signs of aqueous emphysema ("dry lungs") have been reported in approx 10 to 15% of all presumed drowning victims. Forensic pathologists have variably interpreted the PM finding of dry lungs. Spitz (41), for instance, stressed the role of liquid reabsorption into the circulation, especially when resuscitative attempts are performed before death. Di Maio and Di Maio (68) mention the potential role of laryngeal spasm, and Saukko and Knight (42) name, in addition to the above mechanisms, also reflex cardiac arrest. Brinkmann (43) has listed several reflexes that may be triggered by contact of the body with water and result in death with no significant liquid inhalation.

Contributions on lung morphology in drowning have, at times, traced a direct correlation between dry lung and lung weight, most using 1000 g as a cut-off value for this definition (65,66,69). This approach is misleading because, as mentioned previously, no consensus exists for normal lung weight, and low-weight lungs are not necessarily "normal" because they may be overdistended or show signs of liquid penetration. Lunetta et al. (44) observed the absence of overdistension or signs of liquid penetration in less than 2% of "low-weight lungs." Copeland (65), using a cut-off value of 500 g per lung, found that in saltwater drowning the percentage of dry right and left lung was 11.5% and
18.9%, respectively, whereas the corresponding values for freshwater drowning were 10.4% and 16.8%. Kringsholm et al. (66) found that 7.7% of 91 drowning victims with a PM interval of less than 24 hours had a combined lung weight of less than 1000 g, but when considering a PM submersion time up to 1 week and the combined lung and pleural liquid weight, 13% of 131 drowning victims had a combined lung weight of less than 1000 g. Morild (69) found that only 6% of 133 drowning cases, most with a PM submersion time of less than 1 week, had a combined lung weight of less than 1000 g. In Copeland’s (65) and Morild’s (69) series, no significant differences are reported between salt- and freshwater drowning, whereas no data on the influence of water salinity are available in the study by Kringsholm et al. (66).

4.2.3. Drowning Lung vs Postmortem Hydrostatic Lung

The PM penetration of drowning media into lungs of bodies submerged and having died from causes other than drowning has been addressed experimentially using high-pressure chambers or lowering human bodies under water to different depths. Reh (70) performed canine experiments in a baro-chamber and observed significant pulmonary overexpansion mimicking drowning lungs at 0.2 atm, whereas subpleural hemorrhages similar to Paltauf’s spots appeared at 0.4 atm. Reh in the same year also published experiments on eight humans in hyperbaric chambers (atm: 0.3–1.35; duration 4 to 65 hours) who died of causes other than drowning (71). Lung changes identical to those of drowning were observed in bodies kept at 0.3 atm for 65 hours, whereas those kept at more than 0.5 atm for at least 45 to 50 hours showed marked pleural exudates as well. Other investigators have reported, based on animal experiments, that active respiration must be present for significant quantities of water to enter the lungs of floating bodies (38).

4.2.4. Pleural Effusion

Pleural effusion is a relatively common finding in bodies recovered from water, as the result of PM diffusion of pulmonary liquids into the thoracic cavity (72). Morild (69) found pleural effusions (mean, 432 mL) in 53.3% of 133 drowning victims older than 16 years of age with no advanced putrefaction. Kringsholm et al. (66) reported an increase in the volume of pleural exudate during the PM interval. Yorulmaz et al. (73), using univariate analysis, investigated the relationship between volume of pleural liquid, circumstances, and autopsy findings in 43 drowning victims and confirmed the link between PM submersion time and pleural effusion. The correlation between the volume of pleural liquid and lung weight varies among studies, with some authors describing a decrease in lung weight parallel to an increase in pleural liquid