Electricity, Lightning, and Gases 13

Case Study

 The lifeless, fully clothed body of a 78-year-old female was found in the middle of winter by the mobile nursing service on the bathroom floor of the apartment in an old building where the victim lived alone. The bathtub had been filled with water; next to it lays an intact, connected hairdryer, while an old oil heater stood at the side of the room. The emergency physician called to the scene confirmed death (bright red livor mortis, advanced rigor mortis), detected a pacemaker subcutaneously beneath the left clavicula, and found several packs of medication, some opened, some empty: diazepam, a β-blocker, an antidepressant, as well as antithrombosis stockings. The victim's nail beds were also bright red, and an old, healed scar running lengthways along the sternum suggested previous cardiac bypass surgery. Since no further medical intervention was called for and once external examination was complete, the emergency physician filled out all the necessary forms, giving the cause of death as unclear. The subsequent police investigation, as part of which a forensic autopsy was performed, concluded the cause of death to be carbon monoxide poisoning (COHb = 32%). A technical expert later determined the oil heater to be defective.

 Domestic accidents involving electricity, as well as other types of accident involving power lines, the use of electricity with suicidal intent, and high voltage are seen in medical forensic practice. Electricity is rarely used with homicidal intent. Knowledge about the effects of electricity has been gained from its use in executions (the "electric chair"). Electricity is also used as a weapon to incapacitate an individual for the purposes of detainment (the Taser gun).

 Death by electrocution is primarily accidental or work-related; it is occasionally seen in a homicidal context (sometimes falsely interpreted as alleged accidental or suicidal death). In the case of suicide (e.g., hairdryer in the bathtub) and accidental death resulting from autoerotic activity, live conductors (cables) are attached to the genital or anal region, for example, or to the extremities. Incorrectly installed electrical devices and cables or poorly maintained devices can cause death; in such cases, charges of manslaughter are often brought against the responsible party. Moreover, evidence of a fatal electrical accident is relevant in terms of insurance law. Individuals with some knowledge of electricity have been known to use it as a means to commit suicide. Fatal injury due to mobile phones exploding—a rare phenomenon seen mainly in Asia—has been reported; the cause here is believed to be the use of cheap batteries, possibly in combination with extreme heat. Electric cushions and blankets, e.g., in a child's bed, can cause burns.

13.1 Fatal Electrocution

 Contact with current-carrying conductors can have various effects on the body:

- Electrical injury due to electrical stimulation: Muscles, nerves, and the cardiac conduction system
- Electrothermal injury due to local currentinduced heat damage, e.g., local charring or cutaneous metallization
- Indirect injury, e.g., fall injuries following electrocution

 In practice, a distinction is made between four voltage ranges: extra-low, low, high, and extrahigh voltage. Household voltage is usually 220 V (USA, 110 V) with a frequency of 50 Hz (USA, 60 Hz). The low-voltage range goes up to 600 V, while the high-voltage range is between 600 and 1,000 V and higher.

However, the duration of current flow (contact time) and the strength of current passing through the body are the crucial factors in electrical injury. The current (I) is determined by the voltage (U) and the body's resistance (R) according to Ohm's law $((U = R \times I))$. If the hand comes in contact with a current-carrying conductor, mild muscle contraction will ensue at a current of 1 mA; from 5 mA, the lower arm will be affected; up to 15 mA it is still possible to break contact, which is no longer the case from 25 mA. Currents of between 25 and 80 mA cause a rise in blood pressure and possibly also cardiac arrhythmias.

 Skin resistance at the current entry and exit points can vary depending on corneal thickness, skin moisture (reduced resistance due to perspiration!), or protective clothing: gum boots serve a protective function, while walking barefoot on wet ground is particularly hazardous. In individual cases, the question of injury or fatal cardiac arrhythmia (generally ventricular fibrillation) depends on several factors. In addition to the current itself, its path and duration, the level of skin resistance, and the age of the victim, the crucial factor in electrocution is the point in time of the current surge in relation to the vulnerable cardiac phase. The "let-go current" is also a crucial factor: firstly, the current excites nerves causing sustained muscle contraction. Once local muscle control is lost, the conducting object can no longer be actively released, and the current continues to flow through the body. The "let-go" threshold is exceeded beyond 15 mA, the risk of fatal cardiac arrhythmia is increased from 50 mA, and values >80 mA cause loss of consciousness and ventricular fibrillation.

 The alternating current usually found in households has a frequency of 50 Hz. The likelihood that a current alternating direction 50 times/s triggers fatal cardiac arrhythmia (by reaching the heart during the vulnerable phase) is significantly higher compared to the effects of direct current. Very high-frequency currents, however, penetrate the organism only slightly, while the thermal effects at the interface to the current-carrying conductor or electrode are greater. For this reason, high-frequency surgery is carried out in the 300- to 2,000-kHz range. Frequencies over 100,000 Hz are harmless, since nerves are no longer excited; at even higher frequencies, the current runs off body layers close to the surface and internal body structures remain unaffected.

 The effects of current strength and voltage on the heart are shown in Table [13.1](#page-2-0) .

 Skin resistance determines current strength in the body, whereby a distinction is made between various alternating-current ranges (Table 13.2). Alternating-current frequencies between 40 and 150 Hz are hazardous. Ventricular fibrillation is generally the cause of death in electrical accidents. The risk of fatality is greater when the current path between contact sites goes via the heart (arm–arm or arm–foot). However, the direction of the current is also relevant; in the case of a transverse current flowing from hand to hand, a lower proportion of the current flows through the heart compared with a longitudinal current flow. Current flow to other sites, such as between two fingers, usually only causes local thermal injury.

Electrical Burns. In the case of electrical fatalities, electrical burns at the point of contact with the conductor need to be sought on the body. Points of contact can be extremely small, uncharacteristic in appearance, or completely absent. Broad and moist contact surfaces in particular often develop no electrical burns at all. If electrical

 Table 13.1 The effects of current strength and voltage on the heart

 Table 13.2 The effects of alternating current

 Note: In the case of direct current, limit values can be increased by a factor or four

burns are present, the following findings of electrothermal injury are usually made:

- Crater-like elevation of the skin around a central depression (sunken center) (Fig. 13.1).
- Pale, porcelain-like, or alabaster-colored borders.
- Particles of metal from the conductor may be "burnt into" the skin (so-called metallization), although this is not always the case. Metal particles can be detected with the help of spectrographic or histochemical analysis.
- Blister formation in the raised border of the electrical burn (honeycomb pattern) can be seen histologically.
- Fishbone-like deformation of epidermal basal cells and nuclear elongation can also be detected histologically (Fig. 13.2).

Although electrical burns are often specific for electrocution, they are not a sign of vitality unless

there is microscopic evidence of a cellular reaction and/or local hemorrhage. From a differential diagnostic point of view, it may be necessary in individual cases to distinguish between a burn mark (contact burn) and an electrical burn. One important criterion here is the formation of an elevated border, as well as possible discoloration at the center of the lesion (Fig. 13.3).

 Depending on the intensity of the local current, blackish charred contact sites can be seen on the skin, frequently on the fingers, and involving metallization that is often only detectable using microscopic analysis (Fig. 13.4). However, additional skin samples taken adjacent to the affected area should also be investigated, since metal particles (e.g., copper) in the skin of electricians' hands, for example, are found relatively often even in the absence of electrocution.

Fig. 13.1 Electrical burn. Trough-like electrical burn on the palm of the hand with multiple sunken areas

Fig. 13.2 Histological finding in an electrical burn: basal cell deformation, elongation, as well as palisade and fishbone-like arrangement of cell nuclei

 Alongside electrical fatalities in the context of occupational accidents (subject to reporting requirements), electrocution in the bath merits particular attention in forensic medical practice.

Electrocution in the Bath. Occasionally, a fine linear reddening of the skin can be seen at the level of the water in the case of electrocution in the bath (Fig. 13.5).

Even in the absence of any findings, a fatality in the bath should always arouse the suspicion of electrocution. Since electrical burns can be incurred post-mortem, they do not represent a sign of vitality. Thus the diagnosis "electrocution" is often a *diagnosis of exclusion* . In equivocal cases, a technical expert needs to be consulted, not least to prevent further cases of electrocution. Acute asystole may occur up to some minutes following contact with a current. Even when electrocution is initially survived, electrothermal injury to the skeletal and heart muscles (infarctlike pattern of damage) can cause late fatalities in the setting of multiorgan failure (MOF), including kidney failure ("crush kidneys"). Whenever an individual is found lifeless in a filled or unfilled bathtub, particular attention should be paid to finding and identifying an electrical burn. Suspicious skin lesions should be investigated histologically. All conductors and current sources at the scene need to undergo technical inspection, while a careful record of the scene itself needs to be made. Cases are known where bodies have been "cooked" in the bathtub due to bathwater being heated by a hairdryer falling into the water.

 When the diagnosis "death by electrocution" appears plausible only as a diagnosis of exclusion, the goal of any criminal investigation is to establish whether an accident, a suicide, or a homicide has taken place. In forensic medical practice, electrocution is usually accidental in nature; electricity is rarely used to commit suicide. It is essential to weigh up findings carefully while taking the background history, findings at the scene of death, as well as autopsy results into consideration. This may provide information about tetanic muscle cramps or a hand "getting stuck" on a conductor. Emitting a sudden scream followed by loss of consciousness and pulse is consistent with electrocution. Finding a body in

 Fig. 13.3 Differential diagnosis between a burn mark (*contact burn*) and an electrical burn: skin burn mark *without* a raised border, electrical skin burn *with* a raised border

 Fig. 13.4 Blackish charred electrical contact site on the skin of the finger

 Fig. 13.5 Fine linear reddening of the skin at the level of the water in the case of electrocution in the bath

the vicinity of a power source should suggest the possibility of electrocution.

High-Voltage Accidents. In the case of high voltage, no direct contact is needed for current to pass through the body—being at a certain distance from the conductor is sufficient, e.g., a few centimeters from a 10,000-V conductor and several decimeters from a 100,000-V conductor. An electric arc with high current, extreme heat, and intense light is formed between the conductor and the body. Victims show charring on the conducting extremities and convulsive contortion of the facial muscles; eyelids may also be closed. When there is no electric arc and *flashover* occurs, multiple brownish-blackish, densely arranged skin perforations can be seen.

 Most high-voltage accidents occur in an occupational setting, almost always affecting males (working on overhead power lines, substations, transformers, and tall cranes that come into contact with high-voltage current, etc.). Brief contact with a current is often followed by a fall and associated fall injuries, which are sometimes erroneously interpreted as the cause of death. In the majority of cases, high-voltage accidents cause immediate death—direct contact with a power line is not necessary for this to occur, since an electric arc can be formed from an electric arc flashover. Temperatures within electric arcs of this kind reach several thousand degrees Celsius. Such cases are usually accidental, where the victim came too close to high-voltage power lines. The flashover distance for 100 kV is approximately 3.5 cm, increasing with increasing voltage. Contact may also be caused, for example, by urinating from a bridge onto highvoltage power lines. Suicide should be considered in individual cases (climbing up a high-voltage mast). Although most high-voltage accidents produce clear findings on the body and clothing, short but intense contact with a highvoltage conductor has been known to cause fatalities with only scant findings. A small electrical burn, as seen in low- voltage accidents, is often the only finding, and even this may be overlooked if localized on feet covered by shoes. However, high-voltage accidents generally produce the following classic constellation of findings, the intensity of which depends on the duration of current flow:

- 1. Mostly third-degree burns to the face and other exposed sites (Fig. 13.6). The formation of "crow's feet" (see Fig. [12.8\)](http://dx.doi.org/10.1007/978-3-642-38818-7_12#8) is possible, i.e., lines of spared skin at the canthi of the eyes in otherwise burned facial skin. Other lines of skin spared from burning suggest a final contraction of the facial mimic muscles. Linear sparing of this kind is also interpreted as a sign of vitality in high-voltage and electric arc accidents.
- 2. Possible metallization along skinfolds similar to "crow's feet." This finding is not necessar-

 Fig. 13.6 High-voltage accident with facial burns. Protrusion of the tongue and singed hair

ily a sign of vitality in high-voltage accidents, since electrically induced muscle contraction can also cause increased skinfold formation.

- 3. Scalp hair, eyebrows, eyelashes, and facial hair are often singed.
- 4. Depending on the duration of current flow, both direct and indirect burn injuries (Fig. 13.7) including extensive charring, articular dehiscence, and an "overcooked" appearance of skeletal muscles have also been described.
- 5. An electric arc can cause "molten beads" to form on bone from molten calcium phosphate.
- 6. In addition, petechial hemorrhage of the conjunctiva, facial skin, mucosa of the upper respiratory tract, as well as beneath the pleura and epicardium, are occasionally seen. These findings are suggestive of current-induced tetanic respiratory muscle contraction and an intrathoracic pressure increase ("electrical asphyxia").

 Extensive skin burns running lengthways over the body are seen in electrical arc accidents, the

 Fig. 13.7 Extensive blackish charring of the skin and subcutaneous soft tissue at the point of current entry on the knee

burns sometimes ending abruptly where they meet insulating clothing, e.g., sparing of the skin of the foot if shoes are worn (Fig. 13.8).

 In high-voltage accidents, damage to clothing needs to be correlated with findings on the body. Clothing can be badly torn (Fig. 13.9) and charred black, while particles of molten metal may be detected. Damage to clothing and shoes may help to identify the current entry point.

 The feet or soles are the most commonly seen point of current exit, demonstrating extensive or patchy charring of the skin (Fig. 13.10).

13.2 Lightning

 Although rare, lightning-related accidents occur and are associated with a mortality rate of up to 40 % in cases of extremely high voltage delivered within less than a millionth of a second. A lightning discharge can carry a current of several million volts. Fluid in moist objects vaporizes along the lightning pathway, while mechanical and thermal injury can occur.

 Fig. 13.8 Shoes spare the feet from burning in the case of an electric arc accident

 Fig. 13.9 Badly torn and partially blood-soaked clothing following a high-voltage accident involving an electric arc

 Fig. 13.10 Patchy blackish charring at the point of current exit on the skin of the foot sole

Lightning-Induced Injury. The mechanisms by which a bolt of lightning may cause injury are differentiated as follows:

- Direct stroke.
- Step voltage: A lightning bolt initially strikes the ground or possibly a tree.
- Flash discharge: A lightning bolt "jumps" from one object to another.
- Contact strike: The victim comes in contact with an object hit by lightning.
- Blunt trauma: Secondary injury, e.g., a victim falls as a result of a bolt of lightning.

As with high-voltage accidents, findings on the body and clothing should be identified and compared.

Fernlike Injury. Fernlike injury (arborescent erythema or feathering burns; Fig. 13.11), which forms part of the injury pattern typically seen in lightning strikes, is due to small-vessel hyperemia in the corium and can disappear within hours post-mortem and with increasing postmortem interval. In addition, epidermal cells in an elongated and palisade-like arrangement can sometimes be seen at the same level. Singed hair along the lightning pathway, charred and torn clothing, metal particles with signs of melting, as well as torn shoes are also observed. The entry point of lightning in the case of fatality is typically located on the head (crown area). Although not always the case, extensive linear skin burns may be observed along the trunk and on the lower extremities. Contact burns and metallization may occur wherever the skin comes in contact with metal objects. Clothing is subject to varying degrees of mechanical and thermal damage, including hole formation and melting of metal parts (belt buckles, wristwatches, earrings, buttons, etc.). As in high-voltage accidents, holes and tearing may be seen in footwear (shoes, socks/stocking).

Fig. 13.11 (a, b) Two cases with fernlike injury in lightning strikes

Ground Strike. The term "ground strike" or "step voltage" describes the partial flow of current from a lightning bolt through the body (from leg to leg) of an individual or animal located near the point at which a lightning bolt strikes the ground. Ground strikes of this kind form a discharge voltage pattern, whereby voltage reduces as it moves out from the center towards the periphery. Thus when the legs are in a striding position, the potential difference on the ground may be picked up ("stride potential") and cause death. In this way, bolts of lightning are able to kill entire herds of cattle.

 Although immediate resuscitation following a lightning strike may be successful, the extent of burns as well as injury to internal organs will determine the further course. Neurological deficits of varying degree and duration dominate the clinical picture in the case of survival. In addition to acute cardiac and cerebral death following a lightning strike, myocardial necrosis or fulminant bronchopneumonia may lead to late fatality despite initially successful resuscitation. Acute kidney failure may develop depending on the degree of tissue injury. Survivors of lightning strikes often demonstrate neurological deficits, including aphasia, temporary blindness, swallowing and speech impairments, spastic paraplegia affecting the legs, cerebellar injury that is reversible for up to 1 year following the insult, as well as temporary paralysis.

13.3 Electric Shock Devices and Taser Guns (Stun Guns and Remote Electrical Discharge Weapons)

Electric Shock Devices. Electric shock devices are gun- or prod-like devices designed to incapacitate a subject by means of an electrical discharge—without causing significant injury. When pressed against the skin, the high-voltage circuit is reduced by skin resistance to the extent that the current at the electrodes drops significantly. Commercially available devices produce an unpleasant perception of pain without impairing consciousness. Skin reddening (erythema) in a paired arrangement, approximately 5 mm in diameter and at intervals corresponding to the distance between the electrodes, are possible; local pain has been reported. Local erythema is temporary and fades within several hours. The paired arrangement of erythema is the most significant indication of the use of an electric shock device.

The Taser Gun. "Taser" is an acronym of "Thomas A. Swift's electric rifle" from the book by V. Appleton (1911). In contrast to electric shock devices, two projectiles (Taser darts) are fired out of a cartridge from long range and at a speed of up to 50 m/s. The darts are fitted with insulated wires that deliver electrical impulses from the Taser gun to the subject. The wires are several meters long, while the darts have small

barbs capable of penetrating several layers of clothing. The device has a range of up to 10 m. Although each cartridge can only be fired once, the operator is able to deliver several electric shocks to a subject by pressing the trigger. Taser guns are considered appropriate weapons for use at an escalation level below that of firearms and are supposed to confer a lower risk of mortality. At the same time, Taser guns are used by police officials as a means of self-defense. They should only be used when all other means (physical force, baton, pepper spray) have proved unsuccessful; moreover, the subject should be given prior warning of their use. In practice, the effects of the gun depend on various factors:

- The point at which the electrodes penetrate the body
- The distance of the electrodes from one another
- The length of the current path between the electrodes
- The nerves and muscles affected by the current path (is the heart affected?)
- The penetration depth of the electrodes
- The current intensity delivered (should be 3 A, but can go up to 5 A)

 According to the manufacturers of the Taser gun, which can fire two or four barbed darts, an electric shock of approximately 17,500– 500,000 V (open-circuit voltage) is delivered via the barbed hooks attached to the wires only when the trigger is activated or for up to 5 s. The current surge delivered by the Taser gun causes immediate loss of neuromuscular control, i.e., the subject is immediately incapacitated or defenseless and can be apprehended by police officials. The Taser gun is now available worldwide and has been in use by British and US police authorities since 2001. Although the immediate electric shock causes brief, intense pain, local skin findings at the point of impact of the live mini-darts show scant morphological changes.

 From a medical perspective, however, the electric shocks delivered in this context are not entirely without hazard. In principle, electric shocks of this kind may trigger fatal ventricular fibrillation if delivered during the vulnerable phase of the cardiac cycle. Thus, there are an

increasing number of reported fatalities associated with the use of Taser guns. The recommendation that police officials keep a defibrillator ready for use when deploying Taser guns has been discussed. However, from a critical point of view, it must be borne in mind that a proportion of the subjects to be apprehended—as we know from experiences with so-called positional asphyxia during or following police apprehension—are in a psychologically disturbed state, highly agitated due to psychiatric disease (schizophrenia, psychosis, delusions, etc.), and/or under the influence of alcohol or drugs. Long-term abusers of neuroleptics as well as numerous types of drugs (in particular cocaine, amphetamines, heroin) may have occult cardiac damage that predisposes them to sudden fatal ventricular fibrillation triggered by the electric shock delivered by a Taser gun. An inflammatory cardiac response is not impossible, whether due to chronic infection, e.g., hepatitis, or due to an often undetected yet acute viral infection with mild cardiac involvement, which would normally follow an unremarkable and often symptom-free course before resolving. Given the known risk of preexisting cardiac damage in drug abusers and individuals with psychiatric disease, it is to be expected that the wider availability and use of Taser guns will be accompanied by a corresponding increase in related fatalities. Precisely for this reason, police officials in some countries are obliged to summon emergency medical services to examine victims following deployment of Taser guns. There is also a regulation specifying that Taser darts should only be removed from the body of the victim by a physician.

 In many countries where Taser guns are in use, particularly in the USA, detailed forensic investigations or autopsies are only rarely performed in the case of Taser-related deaths. From a forensic point of view, investigations are required and should include microscopic analysis of the heart and cardiac conduction system. Of 167 individuals who died following the use of Taser guns between 1999 and 2005, coroners stated that Taser deployment was the cause of death, contributed to death, or could not be excluded as the cause of death in 27 cases (from

a report by "Arizona Republic," [http://www.](http://www.raidh.org/RAIDH-devoile-la-liste-des-167.html) [raidh.org/RAIDH-devoile-la-liste-des-167.html \)](http://www.raidh.org/RAIDH-devoile-la-liste-des-167.html). Since June 2001, Amnesty International has reported more than 330 fatalities associated with Taser deployment in the USA alone. In more than 50 cases, coroners maintained that the electric shocks were the direct or indirect cause of death. In many cases, alone the proximity in time of death to the use of a Taser gun suggests a causal link. For this reason, Taser guns should be classified at best as "less lethal weapons" than firearms. Particular caution should potentially be exercised when using Taser guns in the vicinity of flammable materials or fumes, such as gasoline fumes.

 In turn, evidence that the use of Taser guns results in fewer fatalities than the use of conventional firearms has hitherto been unconvincing. If anything, there are fears that the purported harmlessness of the Taser gun has encouraged its premature and unnecessary deployment. There is criticism that Taser guns are used in situations where a firearm or even a baton would not have been warranted. In many countries, the Taser gun has not yet been classified according to firearm regulations or remains the subject of controversy. However, it is conceivable that in countries with relatively high numbers of firearm deaths (approximately 30,000/year in the USA), consistently substituting a firearm with a Taser gun indeed results in an overall reduction in fatalities.

 The potential use of Taser guns as a method of torture is cause for concern, given that intense pain can be inflicted in a traceless manner (socalled white torture). The UN Committee Against Torture expressed its concern in this regard in November 2007. Also of concern is the use of Taser guns by laypersons for self-defense purposes or their misuse as a means of intimidation to compel sexual activity, for example.

13.4 Gas Fatalities

Gas fatalities are classified as death by asphyxia. Carbon monoxide (CO) intoxication is the most commonly encountered accident involving gas.

Other gases, such as hydrogen sulfide and putrefaction gases, are found in sewage plants and silos, among others.

Carbon Monoxide Intoxication. Carbon monoxide has a 300-fold greater affinity for hemoglobin than oxygen; as a result, the latter is displaced. Thus even small ambient concentrations are enough to cause fatal intoxication. While COHb values of around 15 % are possible in heavy smokers, COHb values above 40 % are considered a plausible cause of death. Values <40 % combined with a fatal course are suggestive of preexisting (cardiac) damage to the organism or additional intoxication with a poison such as cyanide. The most commonly seen causes include house fires, defective gas pipes, smoldering fires, or poor room ventilation. Suicides are occasionally seen involving the diversion of car fumes into the interior of a car or into a closed garage, resulting in fatal carbon monoxide intoxication. On external forensic examination, bright red livor mortis (Fig. 13.12) and pinkish finger- and toenails are conspicuous, while salmon-colored muscles and cherry-red blood are apparent at autopsy. Internal organs may also show a striking bright reddish color. It is always essential to find the source of carbon monoxide; where necessary, a technical expert should be consulted.

Decomposition and Fermentation Gases. In addition to carbon monoxide poisoning, intoxication by gases that are heavier than oxygen and thus settle and accumulate at the bottom of structures such as silos, septic tanks, and digestion towers—is also possible. Without the victim subjectively perceiving any symptoms, anoxic asphyxiation due to lack of oxygen as well as sudden loss of consciousness may ensue. This is caused by so-called fermentation gases, which have a significantly higher carbon dioxide $(CO₂)$ content than normal ambient air, which is usually around 0.04 %. $CO₂$ is formed during the alcohol fermentation of mash, must, and feeding stuffs. Decomposition gases can build up at the bottom of silos and in wine cellars. Odorless, the gas goes undetected. The risk of symptoms and a fatal course increase according to the concentration of $CO₂$ in ambient air (Table 13.3).

 Fig. 13.12 Bright red livor mortis in the partially charred body of a domestic smoldering fire victim (COHb level in postmortem blood, 52 %)

Table 13.3 $CO₂$ concentrations in ambient air and their effects

Thus, unprotected individuals attempting to help already unconscious victims occasionally become victims of asphyxia themselves.

 There is a danger that individuals entering a room where there is an increased level of $CO₂$ will initially feel a sensation of mild intoxication; however, victims rapidly collapse and lose consciousness, exposing the respiratory organs to even greater levels of fermentation gases due to the higher concentrations of $CO₂$ at ground level and thereby increasing the gas' noxious effects. Since silos need to be entered by a ladder, they often present the additional hazard that, on losing consciousness, the victim falls from the ladder into the silo.

In addition to its destructive force, fire also produces fumes; in addition to carbon monoxide, these fumes often contain other elements in varying concentrations, such as aldehydes, acrolein, cyanides, sulfur dioxide, ammonia, hydrogen chloride, and phosgene. The concentrations of individual elements of smoke depend largely on the type of material burnt. In cases of fatality, samples should be taken from each pulmonary lobe and stored in headspace vials for the toxicological detection of volatile gases.

Caisson Disease (Decompression Sickness). The air we breathe is made up of 78 % nitrogen (N), 22 % oxygen (O_2) , and 1 % other gases. All gases dissolve in blood and tissue. Once oxygen has been used up, mainly nitrogen remains in blood and tissue. Just like oxygen dissolved in blood, nitrogen dissolved in blood can also be released and form bubbles at depths of around 13 m. The deeper the diving depth, the more nitrogen is released with each breath due to water pressure. Depending on the temperature and water pressure, blood and tissue absorb nitrogen; the maximum absorbable volume is referred to as the saturation limit, which is reached after a certain length of time at particular depths. As pressure reduces while the diver's return to the surface, the "stored" nitrogen is delivered to the lungs via the bloodstream. In order for the nitrogen to be safely expelled from the lungs, particular ascent rates need to be observed. Failure to

adhere to decompression times, combined with rapid ascent, leads to the formation of nitrogen bubbles in blood and tissue (caisson disease), causing damage to small blood vessels and tissue.

 Caisson disease, which has varying degrees of severity, is not seen at depths of less than 9 m. Early symptoms may appear within minutes or after several hours, with the spectrum ranging from pruritus to death and including skin irritation (formication); fatigue; exhaustion; back pain; joint and extremity pain; sensory, visual, and auditory impairment; speech disorders; and paralysis. In extreme cases, embolic spread of nitrogen bubbles can cause fatal pulmonary artery embolism. At autopsy, attention should be paid to identifying gas bubbles in the capillaries and fatty tissue, in addition to cardiac, pulmonary, and cerebral gas embolism. Osteoarthropathies represent the most common late effect in survivors of caisson disease. However, fatalities associated with caisson disease are rare among divers, with drowning representing a more frequent cause of death, sometimes due to claustrophobia or panic reactions.

Selected References and Further Reading

- Al-Jarabah M, Coulston J, Hewin D (2008) Pharyngeal perforation secondary to electrical shock from a Taser gun. Emerg Med J 25:378
- Amnesty International: USA (2008) "Less than lethal?" The use of stun weapons in US law enforcement. [http://](http://www.amnesty.org/en/library/asset/AMR51/010/2008/en/530be6d6-437e-4c77-851b-9e581197ccf6/amr510102008en.pdf) [www.amnesty.org/en/library/asset/AMR51/010/2008/](http://www.amnesty.org/en/library/asset/AMR51/010/2008/en/530be6d6-437e-4c77-851b-9e581197ccf6/amr510102008en.pdf) [en/530be6d6-437e-4c77-851b-9e581197ccf6/amr](http://www.amnesty.org/en/library/asset/AMR51/010/2008/en/530be6d6-437e-4c77-851b-9e581197ccf6/amr510102008en.pdf) [510102008en.pdf](http://www.amnesty.org/en/library/asset/AMR51/010/2008/en/530be6d6-437e-4c77-851b-9e581197ccf6/amr510102008en.pdf)
- Andrews C, Cooper MA, Holle R (2009) Section V: Electrical injuries by source of electricity of mechanism of injury. In: Fish RM, Gedes LA (eds) Electrical injuries: medical and bioengineering aspects, 2nd edn. Lawyers & Judge Publishing Company, Tucson, pp 373–397
- Augustin AJ, Koch F, Böker T (1995) Macular damage following lightning strikes. Ger J Ophthalmol 7: 214–216
- Biro Z, Pamer Z (1994) Electrical cataract and optic neuropathy. Int Ophthalmol 18:43–47
- Bozeman WP, Hauda WE, Heck JJ et al (2009) Safety and injury profile of conducted electrical weapons used by law enforcement officers against criminal suspects. Ann Emerg Med 53:480–489
- Carte AE, Anderson RB, Cooper MA (2002) A large group of children struck by lightning. Ann Emerg Med 39:665–670
- Cherington M, Yarnell P, Hallmark D (1993) MRI in lightning encephalopathy. Neurology 43:1437–1438
- Dawes DM, Ho JD, Reardon RF et al (2010) The respiratory, metabolic, and neuroendocrine effects of a new generation electronic control device. Forensic Sci Int 207:55–60
- Dennis AJ, Valentino DJ, Walter RJ (2008) Taser26 discharges in swine produce potentially fatal ventricular arrhythmias. Acad Emerg Med 15:66–73
- Dumas JL, Walker N (1992) Bilateral scapular fractures secondary to electrical shock. Arch Orthop Trauma Surg 111:287–288
- El Faki HMA (1993) High voltage electrical injuries clinical and operative observations. Eur J Plast Surg 16:89–93
- Fieseler S, Zinka B, Peschel O, Kunz SN (2011) Electroweapon Taser®. Function, effects, critical aspects. Rechtsmedizin 21:535–540
- Fontanarosa PB (1993) Electrical shock and lightning strike. Ann Emerg Med 22:378–387
- Han JS, Chopra A, Carr D (2009) Ophthalmic injuries from a Taser. CJEM 11:90–93
- Ho JD, Dawes DM, Reardon RF et al (2011) Human cardiovascular effects of a new generation conducted electrical weapons. Forensic Sci Int 204:50–57
- Jauchem JR (2011) Increased hematocrit after application of conducted energy weapons (including Taser devices) to Sus scrofa. J Forensic Sci 56:229–233
- Jauchem JR, Sherry CJ, Findes DA, Cook MC (2006) Acidosis, lactate, electrolytes, muscle enzymes and other factors in blood of Sus scrofa following repeated Taser exposures. Forensic Sci Int 161:20–30
- Kornblum RN, Reddy SK (1990) Effects of Taser in fatalities involving police confrontation. J Forensic Sci 434–448
- Kroll M (2009) Physiology and pathology of Taser electronic control devices. J Forensic Leg Med 16:173–177
- Le Blanc-Louvry I, Gricourt C, Touré E, Papin F, Proust B (2012) A brain penetration after Taser injury: controversies regarding Taser gun safety. Forensic Sci Int 221:e7–e11
- Lichtenberg R, Dries D, Ward K, Mashall W, Scanlon P (1993) Cardiovascular effects of lightning strikes. J Am Coll Cardiol 21:531–536
- Lifschultz BD, Donoghue ER (1993) Deaths caused by lightning. J Forensic Sci 38:353–358
- Mangus BE, Shen LY, Helmer SD, Maher J, Smith RS (2008) Taser and Taser associated injuries: a case series. Am Surg 74:862–865
- Mellen PF, Weedn VW, Kao G (1992) Electrocution: a review of 155 cases with emphasis on human fractures. J Forensic Sci 37:1016–1022
- Milzman DP, Moskowith L, Hardel M (1999) Lightning strikes at a mass gathering. South Med J 92:708–710
- Mueller B (1975) Gerichtliche Medizin, 2nd edn. Springer, Berlin/Heidelberg/New York
- Nafs FJE, Aromir FC, Carreira IS, Olaso PSC (1993) High tension electrical burns. Eur J Plast Surg 16:84–88
- Ng W, Chehade M (2005) Taser penetrating ocular injury. Am J Ophthalmol 139:713–715
- Sloane CM, Chan TC, Vilke GM (2008) Thoracic spine compression fracture after TASER activation. J Emerg Med 34:283–285
- Sousa W, Ready J, Ault M (2010) The impact of Taser on police use-of-force decisions: findings from a randomized field-training experiment. J Exp Criminol 6: 35–55
- Tropea BI, Lee RC (1992) Thermal injury kinetics in electrical trauma. J Biomech Eng 114:241–250
- Wetli CW (1996) Keraunopathology. An analysis of 45 fatalities. Am J Forensic Med Pathol 17:89–98
- Zack F, Hammer U, Klett I, Wegener R (1997) Myocardial injury due to lightning. Int J Leg Med 110:326–328
- Zack F, Rothschild MA, Wegener R (2007) Blitzunfall Energieübertragungsmechanismen und medizinische Folgen. Dtsch Ärztebl 104:A3545–A3549
- Zack F, Rummel J, Püschel K (2009) Blitzschläge auf Fußballplätzen – Eine unterschätzte Gefahr. Rechtsmedizin 19:77–82
- Zack F, Rammelsberg JO, Graf B, Büttner A (2010) Tod durch Bltizschlag – und wieder unter einem Baum. Rechtsmedizin 20:108–110
- Zack F, Raphael T, Kupfer J, Jokuszies A, Vogt PM, Büttner A, Püschel K, Schalke B, Todt M, Dettmeyer R (2013) Four fatalities due to a lightning on a golf course. Rechtsmedizin 23:114–118
- Zaffren K, Durrer B, Henry JP, Brugger H (2005) Lightning injuries: prevention and on-site treatment in mountains and remote areas: official guidelines of the International Commission for Mountain Emergency Medicine and the Medical Commission of the International Mountaineering and Climbing Federation (ICAR and UIAA MEDCOM). Resuscitation 65: 269–372