

Essentially any episode of cardiac or circulatory arrest and/or respiratory arrest interrupts oxygen supply to the organism and its various organs and tissues. Since the causes are myriad, a classification of the various forms of asphyxia has been undertaken.

Asphyxia is defined as an interruption of any stage of the supply or transport of oxygen between the environment and the organs, e.g., by obstructing the airways or respiration in general, the oxygen-transporting medium blood, blood circulation, and the environment.

Firstly, a distinction is made between external and internal asphyxia:

External asphyxia: Due to insufficient atmospheric oxygen, oxygen is unable to reach the pulmonary alveoli. Possible causes include:

- Generally insufficient levels of atmospheric oxygen (referred to as environmental asphyxia).
- The respiratory excursions of the chest are obstructed (e.g., in the case of burking or positional asphyxia).
- The afferent airways are obstructed, either due to external mechanical compression, as in manual strangulation, or due to obturation, e.g., food aspiration (“bolus death”).

Internal asphyxia: Although atmospheric oxygen supply and oxygen transport to the pulmonary alveoli are both intact, oxygen-hemoglobin binding and/or the delivery of hemoglobin-bound oxygen to the organs and tissues is impaired, e.g., due to carbon monoxide or cyanide poisoning.

Signs of external asphyxia include facial congestion and cyanosis, petechiae in the facial skin and conjunctivae, reddening and swelling of the base of the tongue and pharyngeal wall, as well as rapid and intensive onset of dark blue-violet livor mortis.

Signs of internal asphyxia include hyperemia of internal organs, small bloodless spleen, pulmonary hyperinflation, dilatation of the right ventricle, as well as ecchymosis beneath the serous membranes of the visceral pleura, referred to as Tardieu spots when occurring beneath the pleura. Fluidity of postmortem blood is often seen due to increased fibrinolysis, while microscopic investigations have described vacuoles and fat droplets in hepatocytes, blood extravasation into the heart, kidneys, and liver, as well as ganglion cell necrosis, all of which depend on the type and duration of the mechanism causing death.

Important: None of the signs of asphyxia in internal organs is conclusive for asphyxia— they merely have indicative value!

Any differential consideration of the pathophysiology of asphyxia sets knowledge of a number of terms as a prerequisite; these terms are defined in Table 14.1.

Mechanical trauma involving occlusion of the airways usually occurs in the case of external asphyxia:

1. Occluding the external airways with a soft cover, e.g., infants or elderly care-dependent individuals unable to put up resistance.
2. Aspiration of fluids, in particular water, i.e., drowning, but possibly also aspiration of blood in the case of a skull base fracture with loss of gag reflexes following craniocerebral trauma.

Table 14.1 Terms and pathophysiology of asphyxia

Term	Definition
Dyspnea	Difficulty in breathing, respiratory distress
Apnea	Respiratory arrest
Hypoxia	Diminished oxygen concentration in blood, organs, and tissues
Anoxia	Absence of oxygen
Hypercapnia	Increased carbon dioxide partial pressure (considerable respiratory stimulus)
Asphyxia	Simultaneous hypoxia and hypercapnia
Ischemia	Restricted blood supply resulting in a lack of oxygen, increase in, e.g., carbon dioxide and lactate levels

- Aspiration of solid material resulting in airway occlusion, e.g., aspiration of sand after submersion in sand.
- Placing a tightly fitting plastic bag over the head, seen in an accidental context in children and an accidental autoerotic context in adults.
- Foreign body airway obstruction (“bolus death”), e.g., a piece of sausage occludes the laryngeal inlet. However, in the case of bolus death, death due to a vasovagal reflex has also been discussed.
- External obstruction of respiratory excursions of the chest and simultaneous airway obstruction (nostrils and mouth), referred to as “burking.”
- Death due to positional asphyxia (or postural asphyxia), particularly in individuals in a highly excited state due to psychiatric disease or following drug abuse (excited delirium), restrained in a prone (or “hog-tied”) position; deaths while in police custody are seen.
- Gagging, i.e., introducing a foreign object into the mouth and throat, thereby causing either airway obstruction due to displacement of the posterior airway space by the gag or by simultaneously occluding the external airways.

Cases of accidental strangulation, for example, in children (Fig. 14.1a, b) or belt-restrained patients in hospitals or psychiatric units, are also seen. Cases of this kind usually demonstrate varying degrees of strangulation-related injury to the neck, while hospital fatalities may additionally

give rise to an autopsy investigation on the grounds of allegations of errors in treatment or care.

If neck compression is caused by a wide, soft ligature or a wide belt, findings in the neck area may be absent or only discrete. Neck compression persisting after death combined with a particular position of the body can lead to compression-related sparing from livor mortis, e.g., due to the pressure of the compressing collar (Fig. 14.2). For a more detailed discussion of compression trauma to the neck, see Chap. 11.

14.1 General Pathophysiology of Asphyxia

External asphyxia refers primarily to an interruption in external oxygen supply, which—in the absence of mechanical obstruction—includes lack of oxygen in small enclosed spaces, inhalation of gases, and high-altitude death. In the majority of these cases, asphyxia is associated with hypercapnia and an extremely unpleasant sensation of dyspnea (difficulty in breathing, temporary tachycardia, high catecholamine release, and fear of suffocation). However, if carbon dioxide can still be inhaled, one refers to hypoxic asphyxia with a final reduction in respiratory drive, an euphoric state, and an acute loss of consciousness.

The question of how long the process of asphyxia lasts is often raised in forensic practice; this is given as 3–5 min and subdivided into several phases, each lasting 1–2 min. Depending on the concrete pathomechanism or intermittent free respiration in the case of neck compression, asphyxia can last significantly longer. The general stages of asphyxia are shown in Table 14.2.

Findings in external asphyxia may include petechiae in the sclerae, conjunctivae, face, and oral mucosa on external autopsy examination and beneath the serous membranes above the diaphragm, i.e., under the thymus capsule, as well as subpleural and subepicardial petechiae on internal autopsy examination (Figs. 14.3 and 14.4).

If asphyxia is caused by strangulation, congestive petechiae and fine hemorrhages on the inner surface of the epicranial aponeurosis and in the

Fig. 14.1 Accidental strangulation of an infant with a thin cord: in its cot (a) and following reconstruction with a soft toy constricted between the neck and ligature (b)

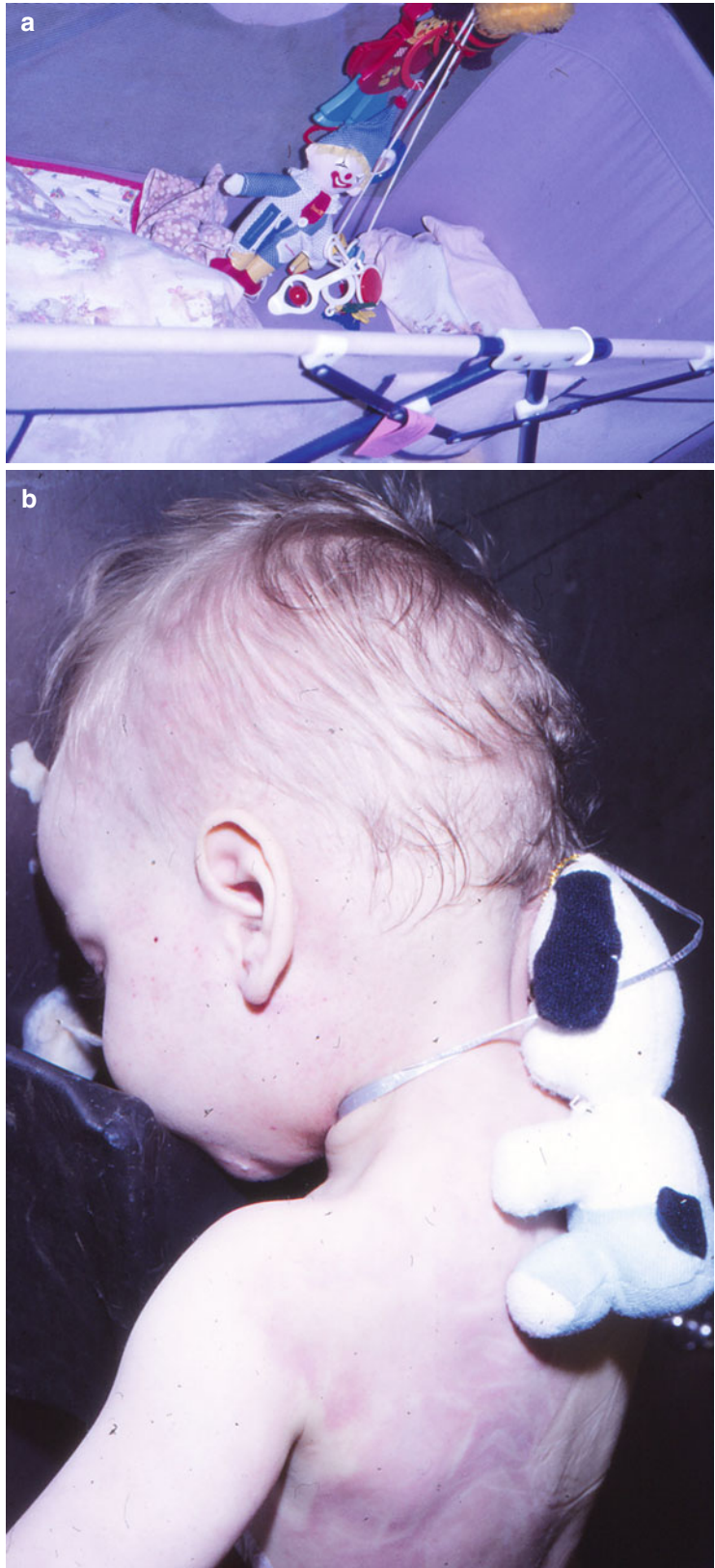


Fig. 14.2 Compressive force of the collar persisting post-mortem with sparing from livor mortis in a case of asphyxial death otherwise presenting scant findings

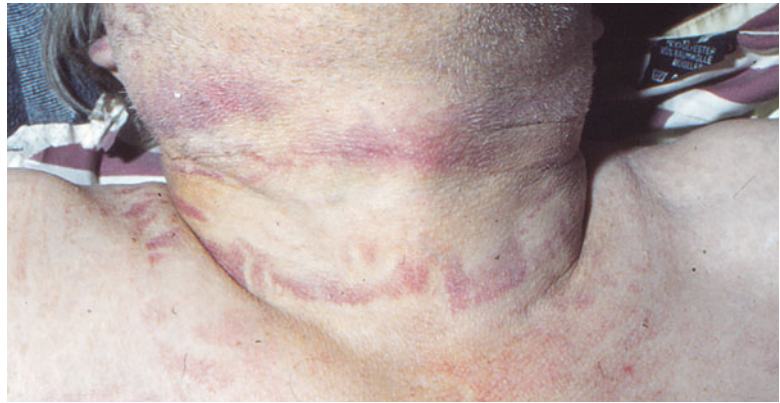


Table 14.2 General stages of asphyxia

Stage	Pathophysiology
Dyspnea	Increased respiratory activity, inspiratory dyspnea (stridor), increasing cyanosis (e.g., if a cushion is pressed against the mouth causing incomplete airway obstruction)
Neurological effects of oxygen deficiency	Tonic–clonic convulsions (asphyxial convulsions), tachycardia, high release of catecholamines from the adrenal medulla (particularly in asphyxial suffocation with hypercapnia), hypotension, loss of consciousness (“blacking out”), possibly also involuntary defecation and urination, ejaculation (e.g., in ligature or manual strangulation until loss of consciousness)
Preterminal respiratory pause	Apnea, hypotension, continued tachycardia
Terminal apnea	Gasping with no effective movement of air, final respiratory arrest

The heart can continue beating for a number of minutes following respiratory arrest; increasing bradycardia!

temporal muscles may be found in addition to petechial hemorrhage (Fig. 14.5). However, petechiae of this kind may also be seen when the body is in a head-down position, making it important to interpret this particular finding in the context of the situation in which the body was found.

Assuming autopsy is carried out shortly after death, acute epiglottitis, a rare cause of asphyxial death, produces findings of severe epiglottic swelling with distinct narrowing of the laryngeal

lumen (Fig. 14.6). If a finding of epiglottitis is made, its inflammatory etiology should be confirmed histologically.

14.2 Particular Constellations in Asphyxial Deaths

In addition to the general pathophysiological process of asphyxial death, there are—more or less irrespective of the type of asphyxia—certain autopsy findings that are indicative of asphyxia. Taken in isolation, these findings are insufficiently characteristic and need to be consistent with a concrete sequence of events, such as gagging, aspiration death, placing a plastic bag over the head, suffocation with a soft cover, bolus death, and positional asphyxia.

14.2.1 Positional Asphyxia (Physical Restraint)

This includes fatalities in abnormal physical positions that cause causing constriction of breathing and cardiac circulation. In addition to positional asphyxia, fatalities caused while restraining highly excited individuals, known as restraint asphyxiation in excited delirium, are seen, e.g., in police custody, in hospitals, particularly among psychiatric patients, and in care facilities. Death in an upright position, on hyperflexion of the neck and obstruction of the airways, in a head-down position, and in a crucifixion

Fig. 14.3 “Asphyxial petechiae.” Localization of primarily petechial or fine hemorrhages

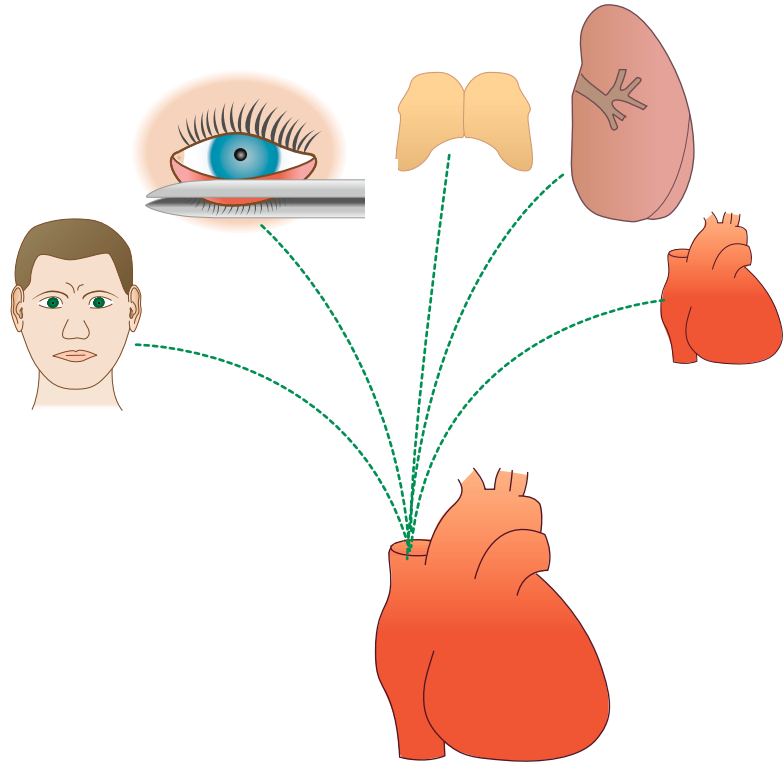
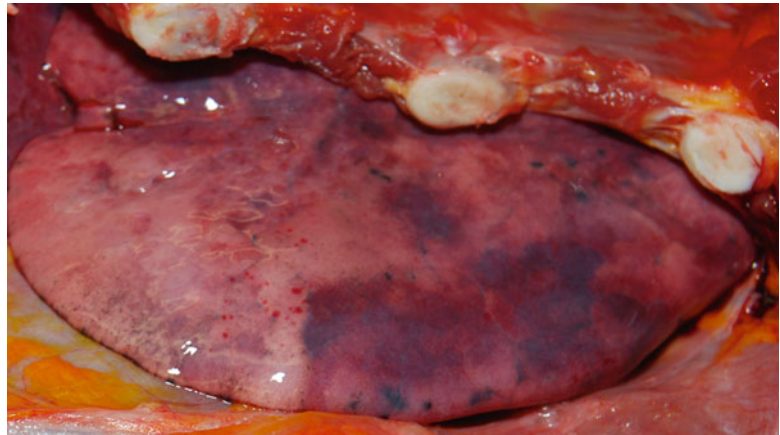


Fig. 14.4 Asphyxial death with multiple subpleural “asphyxial petechiae,” also referred to as Tardieu spots



position all belong to the category of positional asphyxia. Cases of restraint-related compression of the chest have been reported in the context of accidents with wheelchairs.

Therefore, positional asphyxia can affect, among others, highly excited and/or intoxicated individuals restrained in a prone position while partial or complete obstruction of respiratory

excursions of the chest is caused by compression (e.g., a knee pressed against the back!) as well as partial covering of the airways, while simultaneously a maximum release of catecholamines takes place. In cases such as these, acute cardiac and respiratory arrest may ensue, particularly following prior cocaine and amphetamine consumption. Positional asphyxia is seen in

Fig. 14.5 Strangulation- and congestion-related petechiae and fine hemorrhages on the inner surface of the epicranial aponeurosis and in the temporal muscles



Fig. 14.6 Acute epiglottitis with severe mucosal swelling and narrowing of the laryngeal lumen

association with individuals being apprehended by the police and restrained in a prone position due to resistance, in restrained patients in psychiatric and geriatric units and care facilities, as well as in highly intoxicated individuals. Accidental positional asphyxia is rarely seen in children.

Fatalities during and shortly after police restraint play a quantitatively significant role in the USA. Although highly excited individuals under the influence of cocaine are the most commonly affected group, the widely used form of restraint involving a prone position with hands and feet all tied together (“hog-tying”)—and which is banned in some states in the USA—generally increases the risk of positional asphyxia (positional restraint). The cases of positional asphyxia described in the literature show a number of characteristics; these are listed in Table 14.3.

Positional asphyxia affects almost exclusively men, in particular those under the influence of alcohol, drug abusers, and individuals suffering from psychosis. Victims demonstrate highly aggressive and threatening behavior, often necessitating that several law enforcement officers are required in the restraining process. Even the deployment of aids such as tear gas or Taser guns produces no radical effect. During the process of apprehension or restraint, considerable pressure may be exerted on the subject’s back as he is pressed to the ground. In the case of violent resistance, this process will be repeated until cardio-pulmonary and muscle exhaustion result in the usually heavily perspiring subject pausing due to exhaustion. Evidently, this “exhaustion pause” is sometimes misinterpreted as a conscious cessation of resistance, only to be shortly followed by

Table 14.3 Positional asphyxia: risk factors and symptoms

State of high excitement
Significant alcohol and/or drug intoxication, perhaps involving states of confusion or disorientation
Restraint in a prone position
Respiratory obstruction due to partial occlusion of the airways
Constriction of respiratory excursions of the chest
Massive perspiration
Increased/excessive body temperature
Strong respiration with the mouth opened widely
Unusual respiratory sounds
Gasping
Targeted and untargeted resistance
Abrupt cessation of resistance
Pallor or bluish skin discoloration
Sudden circulatory arrest
Impaired consciousness
Convulsions
Involuntary defecation and urination
Increased salivation

a further bout of violent resistance. The next time resistance ceases, the law enforcement officers assume the subject to be feigning submission and continue restraint, including compression of the chest and possibly also at least partial external airway obstruction. Even the subject's claims that he is experiencing respiratory distress, as well as indications of imminent asphyxia—increasing cyanosis, audible stertorous breathing, salivation, etc.—are interpreted as a renewed attempt by the subject to feign more passive behavior rather than as a warning sign of a deterioration in the subject's condition. In cases such as the one described here, respiratory and circulatory arrest follow; if these are falsely interpreted as a “simulated pause in fighting” and the subject's vital parameters (heart rate, respiration, blood pressure) remain unchecked, cardiac arrest will be recognized too late and resuscitation will be unsuccessful. Although a few cases of successful resuscitation have been reported, the victims had already suffered hypoxic cerebral damage with, in some cases, significant neurological deficits and even apallic syndrome.

From a pathophysiological perspective, this represents a stress situation with maximum

catecholamine blood concentrations in the highly excited delinquent. At the same time, increased muscle activity to the point of exhaustion means high oxygen requirements, while respiration or oxygen uptake may be significantly impaired due to chest compression and possibly also partial airway occlusion. A situation such as this causes lactate concentrations to rise and possibly also causes carbon dioxide levels in blood to increase, causing acidosis. If the cardiac muscle has already been sensitized by additional local catecholamine release in the myocardium itself, this will be compounded by the negative effects of acidosis. At the same time, an association between intense muscle activity and increased heat production is suspected, while hyperthermia is more likely to occur in high ambient temperatures.

Important: Acute psychiatric patients under mechanical restraint while in an excited delirious state have been known to suffer cardiac arrest even when no medication has been previously administered. Restraint does not always produce the expected immobilization, exacerbating instead an existing state of excitement.

In the majority of cases reported in the literature, sudden respiratory and cardiac arrest occur directly during police intervention; subsequent resuscitation efforts are unsuccessful. However, there are also reports in which subjects in a prone position experienced respiratory and cardiac arrest once police intervention had ceased and following a certain interval, thereby additionally causing delayed detection. One must assume that leaving a subject in a prone position in itself represents an unacceptable risk factor. Thus, the following applies:

Important: Once actual restraining measures have ceased, highly excited detainees should be immediately moved out of a prone position into a lateral or upright position permitting unrestricted respiration. Thereafter, vital parameters (respiration, pulse, facial color, responsiveness, and reactions) should be closely monitored in those subjects where adverse restraining measures have been used.

Table 14.4 Measures to avoid positional asphyxia

Provide training for police, police physicians, physicians, and care personnel
Avoid the prone position
Fix subjects in a stable lateral position, where possible in an upright position
Avoid any form of respiratory obstruction
Note warning signs: difficulty in breathing, sudden loss of muscle strength (tone), and perspiration
Monitor to check: the jugular vein for pulse, respiratory activity, facial color, and responsiveness

Attention should be paid to preexisting conditions considered to represent risk factors; quite apart from the adverse effects of intoxication, these may include preexisting heart damage, either as a result of coronary artery arteriosclerosis, chronic drug abuse, the effects of cocaine (cocaine cardiomyopathy), as well as cardiac hypertrophy, e.g., due to undetected or untreated hypertension. This type of preexisting damage, particularly in combination with catecholamines or cocaine, is believed to be a predisposing factor to life-threatening cardiac arrhythmia.

Educating police trainees on the risk factors for sudden positional asphyxia, as well as on preventive measures, should form an integral part of police training (Table 14.4).

If police restraining measures are used in the presence of a physician, the latter should be appropriately qualified and call for measures to reduce the risk of positional asphyxia and encourage appropriate monitoring to be carried out subsequently. In the absence of these measures, the police officers and physician could face prosecution for manslaughter or bodily injury in the case of fatality or hypoxic brain damage, respectively.

14.2.2 Autoerotic Accidents

In the case of autoerotic accidents, the circumstances under which the body is found may initially be misinterpreted as those of a suicide or more rarely a homicide. Fatal electric shock, fatal hanging and strangulation, death following toxic substance (narcotics) abuse, as well as induced asphyxia all come into consideration depending



Fig. 14.7 A death scene following an autoerotic accident involving a plastic bag placed over the head

on the mechanism of accidental death. In general, these cases are clearly of an accidental nature. Victims are generally alone and certain circumstances can be reconstructed that indicate a mechanism of sexual gratification (Fig. 14.7). Fatalities during masturbation with no other adverse circumstances are not classified as autoerotic accidents.

When examining the place where the body was discovered, attention should be paid to paraphernalia indicating exhibitionistic, masochistic, or sadistic tendencies (whips, chains, barbed wire, fetters, leather clothing, etc.); transvestite and fetishistic components may also be seen. Bondage (Fig. 14.8) may include “hog-tying” in either a horizontal or vertical position.

Pornographic material, nude photographs, women’s erotic underwear, or apparatus for the purposes of self-photography are frequently found. The lower body or genitals are often exposed and signs of ejaculation or condoms are present. If electrical current has been used, wires, clamps, or metal foil serving as electrodes may be attached to the genitals or erogenous zones (nipples, anal region). In cases such as these, attention should be paid to electrical marks.

Fig. 14.8 Fatal autoerotic accident involving prior self-bondage



Perforations in the intestinal wall may be present if objects have been inserted in the rectum; this, however, can only be established at autopsy.

A situation becomes life-threatening when (supposedly carefully metered) asphyxial techniques are applied in the context of autoerotic activity, such as strangulation or placing a plastic bag over the head or airways. Although without pathophysiological basis, a certain degree of hypoxia is believed to be sexually arousing. The plastic bag placed over the head is sealed around the neck; asphyxia ensues as oxygen is consumed and carbon dioxide builds up. Findings consistent with external asphyxia are often scant and uncharacteristic. In cases where toxins are inhaled, these generally include acetones, alcohols, benzene, chloroethyl, chloroform, ether, nitrous oxide, or carbon tetrachloride, sometimes in products such as paint thinner, adhesives, or nail polish remover.

Autoerotic activity of this kind has been classified to a certain extent under the umbrella term “sexual preference disorder” or “paraphilia.” The vast majority of victims are male. Case reports predominate in the literature and a high number of unreported cases are assumed. Strangulation deaths and asphyxia involving plastic bags are both relatively frequent; fatal mechanical injury is rarely seen.

14.2.3 Aspiration

Foreign material aspiration, in particular chyme, but also blood (craniofacial or skull base fractures!), can cause partial or complete airway displacement. Impaired swallowing and gag reflexes, either due to alcohol (or some other form of) intoxication or neurological deficit following craniocerebral trauma, often represent a causal factor here. Fatal amniotic fluid aspiration is occasionally seen in neonates, as is foreign substance aspiration in occupational accidents. Finally, vomiting followed by chyme aspiration during the agonal phase is possible, whereby in this case chyme aspiration would not represent the cause death. In some cases, the (iatrogenic) spread of, e.g., chyme to the peripheral branches of the bronchial tree during resuscitation should be considered. Vegetable matter, among others, can be detected microscopically in the peripheral bronchi following chyme aspiration.

14.2.4 Gagging

Gagging is a subtype of asphyxia caused by forcing a foreign object, such as a rolled-up cloth or other piece of (usually) fabric material, into the mouth and throat. Depending on the size and position of the gag, the nasopharyngeal space

Fig. 14.9 A case of gagging involving a gag fixed with adhesive plaster



may be displaced, either by the gag itself or by the base of the tongue as it presses upwards. While volumes of up to around 100 ml may be tolerated, anything over 150–200 ml can obstruct breathing by closing the internal airways. A life-threatening situation ensues rapidly if nasal respiration is additionally obstructed. Cases of self-gagging are seen, e.g., in the context of autoerotic activity or with suicidal intent. In cases where the gag has been removed following death, distinct but generally small injuries to the mouth region, including the oral mucosa, may support the suspicion of gagging. In some cases, smear samples taken from the oral cavity yield evidence of textile fibers. If the gag has been fixed with material such as adhesive tape or plaster (Fig. 14.9), imprints running outwards from the corners of the mouth can sometimes be seen on the face.

Distinct signs of asphyxial suffocation are usually found: petechiae, hemorrhages in the auxiliary respiratory muscles and at the base of the tongue, as well as facial congestion. If a gag is placed in the mouth or throat with significant force and then fixed with some form of device such as adhesive tape, perioral abrasions and gag marks at the corners of the mouth and on the cheeks may be visible (Fig. 14.10). Concomitant injuries may be present in the case of violent gagging.

If the victim is unable to remove the gag or gagging material themselves, for example, in the case of infants, fatal gagging without gag fixation using adhesive tape may occur (Fig. 14.11).

In other cases, the gagging material is chosen spontaneously, such as grass and leaves, and pressed into the victim's mouth and throat without fixing the gagging material in any way until

Fig. 14.10 A gagging victim: the gag has been removed and evidence of gag fixation using adhesive tape can be seen at the corners of the mouth and on the cheeks



death occurs. In all cases of gagging, additional signs of trauma, such as strangulation marks or evidence of attempts to cover the airways, should be sought (Fig. 14.12).

14.2.5 Other Forms of Asphyxia

In addition to the asphyxial forms discussed above, there are other constellations that can lead to death by asphyxia; a number of these represent forms of homicide that leave little or no evidence.

Placing a Plastic Bag over the Head. Here anoxic asphyxia occurs following oxygen consumption with a low dead-space volume of air in the plastic bag (Fig. 14.7). Asphyxia can occur even if the plastic bag is not completely sealed around the neck, since sufficient air circulation is

no longer possible. As, in principle, exhalation remains possible, asphyxia symptoms are believed to be less dramatic. If the plastic bag is removed following asphyxia, it is possible that no findings indicating the cause of death can be made. Fatalities of this kind are seen in the context of autoerotic accidents (see above), suicide, and accidental death in children.

Placing a plastic bag over the head in the context of assisted suicide (“exit bag”) or “killing on request” similarly causes asphyxial death. The bag is placed over the head either by the individual wishing to end their life or by an assisting person; oxygen is then prevented from entering the bag by sealing it at the neck with, e.g., an elastic band. Assisted suicide organizations, such as “The Right to Die Society of Canada” or “Exit International,” have been known to work with this type of “exit bag,” leading to strong controversy.

Fig. 14.11 Fatal gagging of a neonate minutes after birth (positive hydrostatic test; lungs only; see Fig. 20.2) without external fixation of the gagging material (toilet tissue), as reported by the perpetrator



Exit bags are considered by the disabled community as a threat to the lives of care-dependent individuals; they argue that “the production and distribution of the Exit Bags directly threatens people with disabilities (...) who are pressured by “caregivers” to commit suicide, or killed without their consent, because they are considered a burden” (from the Canadian journal *Abilities*, 2002, edition 52, p. 9). In some countries, such as Ireland, the use and provision of exit bags is a criminal offense.

Suffocation with a Soft Cover (Smothering). Causing death by obstructing the airways with a soft object, such as a pillow or clothing, is seen primarily where the perpetrator is physically stronger than his/her weak victim (infants and young children, sick, elderly, and care-dependent individuals, as well as unconscious individuals, e.g., following intoxication). There may be no, or

only discrete, findings of asphyxia, as in the case of infants suffocated with a soft cover to simulate sudden infant death syndrome (SIDS). Attention should be paid to discrete petechiae in the facial skin and conjunctivae, fine tears at the corners of the mouth, and discrete lesions to the lips and oral mucosa. Small reddish-brown areas of drying around the mouth and nostrils, as well as lung expansion, may be seen. These scant findings are often—understandably—misinterpreted as the result of intensive resuscitation efforts. It is not unusual for a homicide to be discovered only as a result of the perpetrator’s confession.

Bolus Death. This is seen when a foreign body is lodged at, above, or in the tracheal space adjacent to the laryngeal inlet; the object is usually a fragment of under-chewed food such as a small piece of sausage or meat (Fig. 14.13), very rarely a dislodged or poorly fitting dental prosthesis.

Fig. 14.12 Foliage used as the gagging material and pressed into the mouth and throat by the perpetrator; additional dried-out skin seen periorally is a sign of attempted obstruction of the airways



Extensive occlusion of the airways causes asphyxial suffocation or reflex cardiac arrest (vasovagal reflex evoked by stimulation of the autonomic nerve plexus of the laryngeal inlet). Sudden unresponsiveness, collapse, and rapid death have been reported in the case of a vasovagal episode, thereby precluding the more dramatic symptoms of asphyxia such as respiratory distress, facial cyanosis, convulsions.

Heavily intoxicated individuals as well as craniocerebral trauma patients with organic brain injury and resultant swallowing and gag reflex disorders are commonly affected. However, overly hasty food intake can also play a causal role.

Perthes Syndrome. This describes thoracic compression and fixation, usually in an expiration position, e.g., persons trapped or buried under rubble or due to an occupational accident,

mass panic in a crowd, or a fallen tree; rapid compression-related increase in blood circulation and copious dense petechiae in the head, neck, and shoulder area, as well as in the conjunctivae and possibly also below the level of compression in the lower extremities (around the ankle and sole), are seen. A similar mechanism causes death in the case of burking.

Burking. This form of asphyxial homicide takes its name from the serial offender Burke: the physically stronger perpetrator obstructs respiratory excursions by sitting or kneeling on the victim's chest while simultaneously occluding the external airways (mouth and nose). Burking produces little or no external signs of violent trauma. The mass murderer Burke killed 16 people in this manner in the early nineteenth century, leaving little trace of his crimes. He then sold the bodies

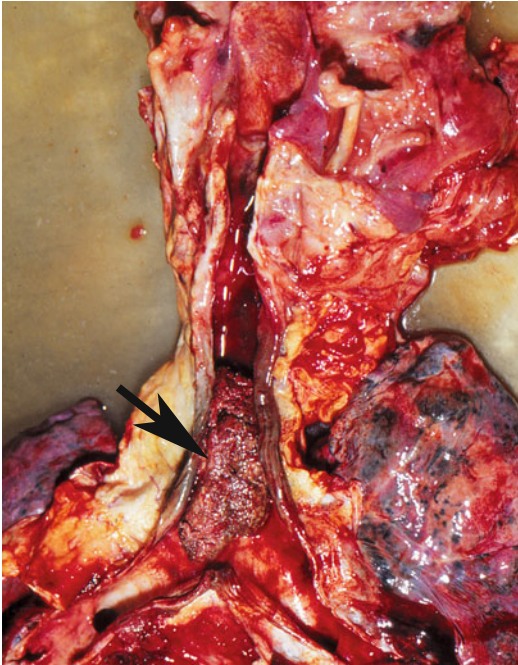


Fig. 14.13 Bolus death after a piece of steak (*arrow*) became lodged in the trachea

to Edinburgh's institute of anatomy. The mechanism of death is similar to that seen in traumatic asphyxia caused by a constricting snake, although this latter form involves no airway occlusion.

High-Altitude Illness (Mountain Sickness)/ High-Altitude Death. The composition of air at approximately 21 % O₂, around 78 % N₂ (+ noble gases), and 0.03 % CO₂ barely changes up to an altitude of 100 m. However, at higher altitudes, air pressure drops and inspiratory pO₂ decreases. Hyperventilation is able to compensate for arterial hypoxia to only a certain degree. Hemoglobin oxygen saturation drops due to the altitude-related reduction in O₂ partial pressure. The following approximate classification applies in this setting:

1,600–2,000 m ASL = mild hypoxia

3,000 m ASL = moderate hypoxia

5,000 m ASL = severe hypoxia

7,500 m ASL = 50 % of unacclimatized individuals lose consciousness within minutes

Acute mountain sickness causes the following symptoms: headache, nausea, dry cough, tachycardia, and chest pain. Altitude adaption primarily

affects erythropoiesis, causing increased erythrocyte production and the development of polycythemia. Acute pulmonary edema is sometimes seen at altitudes above 2,500 m even in individuals with healthy heart and lung parameters. Due to the insufficient supply of oxygen to the brain, altitude illness can lead to symptoms such as euphoria, impaired concentration, languor, convulsions, as well as loss of consciousness, and ultimately to death.

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