ASPHYXIA

A Walker and C M Milroy, University of Sheffield, Sheffield, UK
J Payne-James, Forensic Healthcare Services Ltd, London, UK

Introduction

The etymological meaning of asphyxia as derived from the Greek means "absence of pulsation." It has come to be used in modern forensic medical practice to refer to death or other clinical sequelae due to lack of oxygen. There are many different ways of classifying asphyxial death, and perhaps the simplest is to consider the possible practical effects by which asphyxia may occur. Asphyxial death can be applied to a number of mechanisms of death where respiration/ventilation is affected, including:

1. suffocation, where death is associated with deprivation of oxygen, either from a lack of oxygen in the surrounding environment or obstruction of the upper airway
2. smothering, more specifically where there is external obstruction of the upper airway
3. strangulation, which may occur by ligature or by manual pressure using one or both hands, when the term throttling may be used
4. choking, e.g., inhaling food
5. garrotting (commonly used to describe application of a ligature)
6. chest compression (due to fixation of the external chest wall as a result of crushing)
7. hanging (by another, deliberate self-harm, or autoerotic asphyxia).

It can be seen that several of these terms, and the mechanisms associated with them, may interrelate. Not all may be due to asphyxia alone; other factors, such as vasovagal stimulation, may contribute. Other terms such as neck compression may be used to cover external pressure on the neck, such as by an arm lock during restraint.

Overly rigorous use of terminology can however be misleading and the emphasis should be on having a clear description of the cause of deprivation of oxygen or prevention of respiration/ventilation and the findings associated with each episode. This article focuses on two areas of asphyxia – that of death in closed and confined spaces and the clinical findings in those who have survived assaults that could have caused asphyxial death.

Asphyxial Death

The investigation of such deaths has traditionally been founded on the so-called "classic signs of asphyxia." These signs have included congestion, cyanosis, edema, petechiae, and fluidity of blood. None of these signs is reliable or indeed diagnostic as an indicator of asphyxia as a mode of death. Petechiae are pinhead-sized hemorrhages seen in the skin, the sclera, and conjunctivae, and on mucosal surfaces in the mouth as well as the organs of the chest. Most characteristically they are seen on the outer and inner surfaces of the eyelids. In cases of manual strangulation petechiae may be florid. They are often absent in hanging and may occur in many nonasphyxia deaths. The proposed mechanisms of causation have included raised venous pressure and increased vascular fragility due to hypoxia. The latter explanation is not supported by their absence in many hypoxic deaths. They can appear rapidly in coughing bouts or retching and the most logical view is that they occur due to raised venous pressure. However, because they are common, when appropriately looked for, and occur in many different mechanisms of death, their presence must be interpreted with caution and in the context of the death with the surrounding circumstances.

The other "classic" signs are congestion, cyanosis, and edema, which are such common findings in many different modes of death as to have no specific diagnostic value. Fluidity of blood has more to do with the rapidity of death than the mode of death and has diagnostic value in determining the cause of death. It has been appropriately stated by Knight that abnormal fluidity of blood is part of forensic mythology and has no relevance in the diagnosis of asphyxia. In all cases of possible asphyxial death physical external and internal evidence of asphyxia may be present and must be taken into account with the known history and background prior to death.

Additional nonspecific features that may rarely be present include frank hemorrhage from orifices such as the nose and ear, and spontaneous evacuation of feces and urine.

Asphyxia with Confined and Enclosed Spaces

Deaths associated with confined and enclosed spaces present problems to the investigating pathologist, as there may be few or no diagnostic features at autopsy. Although the deaths are typically classified

as asphyxial, there may be no features to make such a diagnosis. As with all deaths, but in particular with these deaths, the findings and surrounding circumstances need to be taken into account in determining the cause and ultimately the manner of the death.

Mechanism of Death

Suffocation is the most common term used for deaths associated with reduced availability of oxygen. It is also used in cases where other nontoxic irrespirable gases are encountered. In a person confined within an enclosed environment a number of factors may contribute to the collapse and death of the victim, including the lack of oxygen and the build-up of carbon dioxide. The presence of other irrespirable gases should be considered, especially in an industrial setting, such as mining. One well-recognized situation in which a hypoxic death occurs is where a young child, often playing a game trying to hide from others, enters a self-locking fridge or box and then has no mechanism of escape. No specific signs are seen and petechiae and other asphyxial signs are absent. Homicide victims may be placed in confined spaces. In one such case seen by one of the authors, the victim had been placed in a box trunk and left tied up but without any obstruction of the airway. The victim had abrasions where he tried to move in the box in his desperate struggle to try and get out. He had no asphyxial signs at autopsy.

Carbon dioxide may play an important part in deaths in enclosed spaces. In environments such as submarines a mechanism to remove carbon dioxide is present. Otherwise an increase in carbon dioxide will result in central nervous system depression and respiratory collapse. The normal concentration of carbon dioxide in air is 0.1%. If the concentration of carbon dioxide is raised but oxygen remains at 20%, death will still occur from breathing such a mixture of gases. This is illustrated when rapid entrance into an environment high in carbon dioxide results in almost immediate collapse. Carbon dioxide poisoning has also been postulated as a cause of death in infants placed in appropriate sleeping positions, when they are at risk of rebreathing carbon dioxide. Carbon dioxide is not *per se* directly toxic, unlike carbon monoxide, cyanide, and hydrogen sulfide. However, even nontoxic gases can be asphyxiating if too high a concentration is present in an enclosed space and rapid collapse may occur on entering such an environment. When multiple deaths are encountered it may be because colleagues go to the aid of a victim and are then overcome by the same gases.

One well-recognized situation where carbon dioxide poisoning occurs is in grain silos. The grain in the silo gives off carbon dioxide and, if a worker enters the silo before the carbon dioxide has been vented the worker may collapse on breathing the carbon dioxide.

Similar episodes of collapse and sudden death may be encountered where workers enter ships' holds or when cleaning or inspecting fuel tanks of ships. Some industrial tanks contain high concentrations of nitrogen. The mechanism of death in these cases appears to be a rapid cardiorespiratory arrest mediated through central brainstem receptors rather than slower hypoxic death. This mechanism of death has been compared to people dying of plastic-bag asphyxia. In these deaths no asphyxial signs are seen and death in these cases appears very rapid. In some planned suicides the placing of the plastic bag over the head has been accompanied by the use of helium, another irrespirable gas.

Methane in an enclosed atmosphere may be hazardous and can be encountered in a number of situations including mining, where methane production has long been recognized as a problem because of its explosive qualities (firedamp). It may also kill by oxygen deprivation. Methane, along with other gases, including hydrogen sulfide, may be produced in sewers.

Use of solvents in enclosed spaces may result in exposure, collapse, and death. Many different solvents are used in industry, many being halogenated hydrocarbons, which are also used as degreasers, cleaners, propellants, and for chemical synthesis. They have properties similar to general anesthetics.

Exposure to methylene chloride (dichloromethane) may result in the production of carboxyhemoglobin in the absence of combustion. In car-exhaust suicides, the typical finding is of very high carboxyhemoglobin concentrations. Lower concentrations may be seen in people with preexisting natural diseases where death takes place before higher levels are reached. However, with emission control technology being introduced on modern motor vehicles, carbon monoxide concentrations in exhaust fumes are significantly reduced. This has resulted in unsuccessful attempts at suicide, and in successful cases the absence of raised carboxyhemoglobin concentrations. In these cases death has been attributed to carbon dioxide poisoning. It has been proposed that the cause of death be given as "inhalation of automobile exhaust gases." Leakage of gasoline (petrol) into the vehicle cabin following automobile accidents has been reported as a contributory factor in deaths, where the victims were trapped, but had not sustained major injury.

Postmortem Investigation

For most of these types of asphyxial deaths there are typically no specific findings. Asphyxial signs are

characteristically absent. The autopsy should be directed at excluding other nonnatural causes and the presence of natural disease, that may have contributed to death. Toxicological examination should be undertaken to exclude other causes and gases that can be measured. In confined spaces where machinery is being used products of combustion may contribute to death and carboxyhemoglobin should be measured. Solvents should be sought where appropriate, in consultation with a toxicologist where unusual gases may be suspected to obtain appropriate samples for analysis. No specific microscopic features are seen. Biochemical markers of hypoxia have not proven their value in casework.

The investigation of deaths in confined spaces often presents the pathologist with the task of proving the impossible as carbon dioxide, nitrogen, and similar gases cannot be measured at autopsy. The ultimate identification of the cause of death will involve other agencies such as the Health and Safety Executive (in the UK) carrying out investigations at the scene of the death to identify excess gases in the environment. Other cases, such as a young child found in a closed box, may be self-evident, though investigations should be directed at excluding other causes and criminal and civil proceedings may still be brought, necessitating a thorough inquiry.

Survivors of Asphyxia

The term might appear to be an oxymoron but there are a substantial number of cases in the clinical forensic setting where near-death has occurred as a result of some impairment of respiration and ventilation. In general the survivors are those who have been subject to a physical assault where breathing may have been impaired by pressure on the thorax (e.g., some restraint deaths) or on the neck, or by occlusion of the upper airways such as the mouth and nose. As with asphyxial deaths, additional mechanisms may come into play (e.g., physical damage to local structures such as the hyoid bone or thyroid cartilage) which may account for some of the physical signs seen. Additionally, unlike the decedent, the survivor's injuries will evolve over a period of time.

If the survivor was aware of the attack at the time (i.e., was conscious or not intoxicated through drugs and alcohol) there is the likelihood that signs of a struggle have taken place with the possibility of bruising, scratching, and other signs of struggle on both victim and assailant.

In many cases when an asphyxial mechanism is applied for only a short time the findings may be absent or minor. Figure 1 illustrates the neck of a 40-year-old female subjected to two-handed manual pressure to the neck sufficient to render her unconscious. No petechiae, cyanosis, or swelling was observed. The only findings confirming the account (which had been witnessed) were two small curvilinear superficial abrasions with associated "skin lifts" caused by the assailant's fingernails.

Figure 2 shows the neck of a 38-year-old male around whose neck a ligature had been placed. The linear scratch overlaid with dried blood was caused

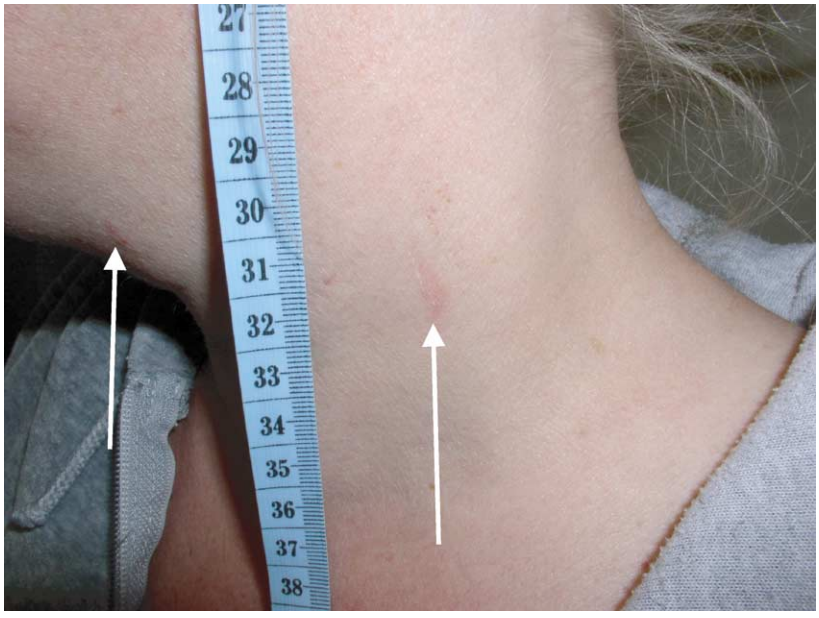

Figure 1 Victim of two-handed manual pressure applied to neck – only visible injuries are two curvilinear superficial abrasions – caused by fingernails. Photo courtesy of Dr Jason Payne-James.

Figure 2 Residual red ligature mark – scratch caused by victim's own fingernails when trying to remove the ligature. Photo courtesy of Dr Jason Payne-James.

Figure 3 Red grouped, lined marks, caused by fingers of one hand gripping neck. Photo courtesy of Dr Jason Payne-James.

by a scratch from the victim's own fingernail as he tried (and succeeded) in pulling the ligature off. The ligature mark is clearly seen.

Figure 3 shows the neck of a man who had been throttled by an assailant single-handed. At the time of the assault the victim was intoxicated and was unable to defend himself. Others intervened, with witnesses indicating that the victim appeared cyanosed.

Figure 4A shows the plethoric complexion of a male who had been garroted to the point of near-unconsciousness on perhaps three occasions over an hour or so. The photograph was taken about 3 h after the events. He stated he had been unable to breathe on

Figure 4 (A) Florid plethoric complexion caused by ligature application about three hours after event. (B) Same male approximately five days later with resolution of color. Photo courtesy of Dr Jason Payne-James.

each occasion. Initially it was considered that the color change between his face and neck related to an outside lifestyle. He advised that his skin was normally of normal color. No petechiae were distinguishable.

However, over the course of the next 5 days his face reverted to the color and appearance seen in Figure 4B.

His eyes on presentation (Figure 5A) exhibited marked conjunctival hemorrhages (just visible on this image) with no obvious edema.

By day 5 (Figure 5B), the conjunctival hemorrhages were more extensive with the start of breakdown of pigment. However, marked resolving edema could be seen in the infraorbital region, associated with evolution of periorbital bruising (there had been no blunt impact of any kind).

This series of photographs is consistent (although in extreme form) with the leakage of blood from blood vessels following constriction, and that all physical signs appear above the level of the constricting or obstructing force.

Figure 5 (A) Same person as **Figure 4**, note conjunctival hemorrhages at three hours. (B) At day five conjunctival hemorrhages still present but also has marked infraorbital edema. Photos courtesy of Dr Jason Payne-James.

Finally, **Figure 6** shows the change in appearance of the ligature mark over the same time period.

The effects of arm locks during restraint causing asphyxia can also create florid signs. A young Asian male was stopped by police and he attempted to swallow some drugs. An officer attempted to prevent this happening by placing him in an arm lock. The male subsequently said that he was unable to breathe and thought he was going to die. Examination (about 1 h after the incident) revealed marked petechiae, particularly in the periorbital region (**Figure 7A**). Further examination of the eyes, which initially appeared unremarkable (**Figure 7B**), revealed extensive and expanding conjunctival and subconjunctival hemorrhage (**Figure 7C**).

In addition to the external signs of asphyxia the clinician needs to be aware that some signs of asphyxiation may also be present intraorally. A male who had been held in an arm lock by club security for a number of minutes with only brief respite to gasp for air presented a few hours later with hoarseness and dysphagia. Intraoral examination showed petechial hemorrhage on the uvula associated with marked uvular edema. This took about 24 h to resolve.

Additional soft-tissue and bony injury is well recognized following blunt-force trauma to the upper airways (including hyoid bone and cartilaginous fracture). Although the damage itself may not be truly asphyxial, the subsequent soft-tissue swelling may precipitate upper-airway obstruction and potential asphyxiation. In any individual with persistent or marked anterior neck, throat, or mouth pain after potential asphyxial injury consideration should be given to further management (e.g., hospital admission for observation, administration of steroids) or investigation (imaging of the neck) to exclude or confirm more serious underlying pathology (**Figure 8**).

In particular it is appropriate to reexamine the living victims some days after the initial insult to

Figure 6 Ligature marks from male in **Figures 4** and **5** showing (A) initial and (B) subsequent appearance. Photos courtesy of Dr Jason Payne-James.

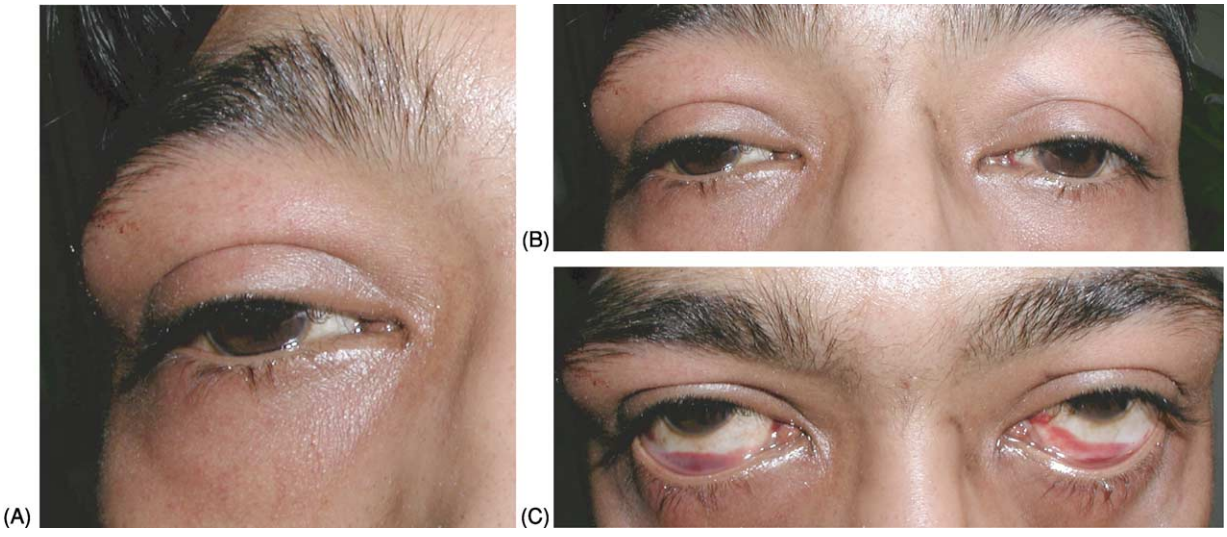

Figure 7 (A), Eye signs, following asphyxia secondary to restraint in neck lock. (B and C) illustrate conjunctival hemorrhage that remains occult until lower eyelash retracted. Photos courtesy of Dr Jason Payne-James.

Figure 8 Uvular edema with petechial hemorrhage following neck compression. Photo courtesy of Dr Jason Payne-James.

record and determine the evolution of any relevant physical findings.

Summary

The diagnosis of asphyxial death or injury requires an understanding of the range of situations that it may encompass. The signs of asphyxial death may be nonspecific and may not conform to classically described patterns. The results of asphyxial injury in the living may overlap with other types of injury, such as blunt force applied to the neck, or the consequences of such injury and assault, such as vasovagal inhibition. In all cases a full and detailed assessment of the circumstances surrounding the death or injury is required and then physical or autopsy findings must be interpreted in the light of those issues.

See Also

Carbon Monoxide Poisoning: Clinical Findings, Sequelae In Survivors; **Postmortem Changes:** Overview

Further Reading

Byard RW (2004) Accidents (with Cohle SD). In: *Sudden Death in Infancy and Childhood and Adolescence,* 2nd edn. Cambridge, UK: Cambridge University Press.

Byard RW, Wilson GWP (1992) Death scene gas analysis in suspected methane asphyxia. *American Journal of Forensic Medicine and Pathology* 23: 42–44.

Byard RW, Gilbert JD, Klitte A, Felgate P (2002) Gasoline exposure in motor vehicle accident fatalities. *American Journal of Forensic Medicine and Pathology* 23: 42–44.

Ely SF, Hirsch CS (2000) Asphyxial deaths and petechiae: a review. *Journal of Forensic Sciences* 45: 1274–1277.

Gill JR, Ely SF, Hua Z (2002) Environmental gas displacement. Three accidental deaths in the work place. *American Journal of Forensic Medicine and Pathology* 23: 26–30.

Gilson T, Parks BO, Porterfield CM (2003) Suicide with inert gases. *American Journal of Forensic Medicine and Pathology* 24: 306–308.

Saukko P, Knight B (2004) Suffocation and "asphyxia." In: *Knights Forensic Pathology,* 3rd edn. London: Arnold.

Schmunk GA, Kaplan JA (2002) Asphyxial deaths caused by automobile exhaust inhalation not attributable to carbon monoxide toxicity: study of 2 cases. *American Journal of Forensic Medicine and Pathology* 23: 123–126.