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Review article

The physiology of sudden violent death

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Abstract

Background and purpose: The physiology behind sudden violent death is considered in the light of information from a wide variety of clinical, experimental, forensic and veterinary sources. Physiological causes can be classified under the headings of blood loss; asphyxia; electrocution; tissue loss; destruction of brain; disconnection of brain; and poisoning. Death, although sudden, is not necessarily instantaneous; asphyxia and electrocution take some time to kill the subjects. *Conclusions:* The chemistry of dying, death and changes post mortem, needs more research in order to design further rational resuscitation procedures.

Keywords: Blood loss; Asphyxia; Electrocution; Tissue loss; Sudden death

Resumo

Contexto e objectivo: A fisiologia subjacente à morte súbita violenta é considerada à luz da informação de uma grande variedade de fontes quer clínica, experimental, forense e veterinária. As causas fisiológicas podem ser classificadas como perdas sanguíneas; asfixia; electrocussão; perda de tecidos; destruição do cérebro, desconexão cerebral e envenenamento. A morte, embora súbita, não é necessáriamente instantânea; a asfixia e a electrocução demoram algum tempo a matar. *Conclusão:* A química da morte, a morte e as alterações pós-morte necessitam de mais pesquisas, de forma a desenhar procedimentos de reanimação mais racionais.

Palavras chave: Perdas sanguíneas; Asfixia; Electrocussão; Perda de tecidos; Morte súbita

Resumen

Antecedentes y propósito: Se considera la fisiología detrás de la muerte súbita violenta, a la luz de la información de una amplia variedad de fuentes clínicas, experimentales, forenses y veterinarias. Las causas fisiológicas pueden ser clasificadas bajo los encabezados de pérdida sanguínea; asfixia; electrocución; pérdida de tejido; destrucción cerebral; desconección cerebral; y envenenamiento. La muerte, aunque súbita, no es necesariamente instantánea; la asfixia y la electrocución toman tiempo en matar a los sujetos. *Conclusiones*: La química del morir, muerte y cambios post mortem, necesitan de investigación, para poder diseñar procedimientos ulteriores racionales de resucitación.

Palabras clave: Pérdida de sangre; Asfixia; Electrocución; Pérdida de tejido; Muerte súbita

1. Introduction

This paper reviews the pathophysiology of acute violent death, due to murder, war, execution and accident. It does not deal with starvation, dehydration,

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torture, or medical or surgical conditions, all of which may contribute to or cause death. There is a vast literature relevant to this subject in military medicine, emergency practice, forensic pathology, and animal experiments.

2. The nature of death

The final cause of death is usually assumed to be hypoxia of the tissues, and ultimately of cells. However, it can also be caused by protein (including enzyme) denaturation, high or low pH in tissues, or poisons, including metabolic inhibitors. Death can be understood as a descending loss of organisation from death of the whole patient, to deterioration of function of the individual organs, to rigor mortis, to bacteraemia, to death of tissues, to collapse of homeostasis in cells [1]. From a clinical view point, the stages of death may be defined [2]. Firstly, the agents causing death produce their own effects, such as haemorrhage, blast, or tissue damage. At this stage, the subject is hyperactive due to stress and pain. Secondly, homeostatic systems supporting respiration, blood pressure, temperature and the immune systems collapse, unless resuscitation is initiated rapidly. The third stage is chemical; chemical changes occur, such as the disruption of metabolic pathways, the denaturation of proteins, and the cessation of biosynthesis. Bacterial breakdown of tissues and proteolysis occurs. The body putrefies, giving off nitrogen and sulphur-containing gases. These changes can be slowed down by climatic cold or refrigeration. However, they all start when unconsciousness and zero blood pressure become irreversible.

3. Method of study

A large range of methods can be used to study dying and death, by observing human beings, and observing and experimenting on animals in a variety of different conditions (Table 1). However, it should be stressed that the acute process of dying cannot be studied using procedures in which the tissues are killed during the examination. The reason is, that fixation kills the tissue, so that histology, histochemistry and electron microscopy, cannot be used to study the acute processes of dying or death [3]. Of course, the latter may properly be used to examine long standing histological changes [4]. Dying can only be studied by observation on whole human beings or animals, and gross pathology at post mortem. So far the biochemical changes making death irreversible are not known.

Table 1
Publications relevant to the understanding of acute death

Evidence from other sources	
Evidence from gunshot wounds to animals and patients	[20–23]
Forensic pathology	[24,25]
Experiments on slaughter and dying in animals	[2,26,27]
Clinical observations on burns and electrocution	[28–30]
Effects of blast	[31,32]
Clinical observations on drowned animals and persons	[33,34]
Effects of haemorrhage and shock	[5,35,36]
Post mortem reports on executed persons	[17,37,38]
Neurological observations on patients with high spinal cord injuries	[39–41]
Volunteers who have been made hypoxic or stimulated with electricity	[42–44]
Observations of executions	[45–47]

4. Physiological classification of the causes of death

These are summarised in Table 2, and the different categories will be discussed separately.

4.1. Blood loss

Blood is lost when a large artery or vein is severed, or the heart is penetrated. A small loss is compensated by local vasoconstriction, and sometimes external pressure from clothes or dressings. One or 2 l can easily be lost into the limbs, thorax or abdomen, without causing much observable swelling. However, if more than 20–25% of the blood is lost acutely, the vasomotor system collapses, probably because the synapses involved in the baroreceptor reflexes are blocked by hypoxia [5]. Death supervenes within an hour, unless the bleeding is stopped by pressure, a tourniquet, or the artery is tied off, and fluid is replaced by saline, serum or blood.

Table 2
Physiological and pathological causes of acute death

Causes of death	Circumstances of death
(A) Blood loss	Bullet wounds, road, air or rail accidents, cutting the throat, shrapnel from bombs, suicide by cutting wrists, death by firing squad, stoning
(B) Asphyxia	Drowning, strangulation, hanging, judicial or suicide, gassing, anaesthetic accident, blast injuries to lungs
(C) Electrocution	Accidental electrocution, electrocution by the electric chair
(D) Tissue loss	Explosion, decapitation or loss of limb, road, aeroplane or rail accidents
(E) Destruction of brain	Short range execution by shooting, bullet wound to vital centres in brain
(F) Disconnection of brain and spinal cord	Execution by hanging, axe or guillotine, fall from height
(G) Poisoning	Intravenous lethal injection

Bullets and shrapnel are usually pieces of metal, although, nowadays, they may be plastic. They are usually very hot, and this causes the tissue fluids to boil and vaporise rapidly. This immediately produces a cavity with a volume that may be 300 times that of the bullet, but the cavity contracts considerably subsequently, when the tissue cools and shrinks around the cavity [6,7]. At the beginning of the entry of the missile, the expansion of gas may tear blood vessels and capillaries.

In view of the difficulty of measuring the blood loss rapidly, the signs of shock are regarded as the first clear evidence of considerable loss; these are a fast thin pulse, air hunger, pallor or cyanosis, and coldness of thready skin. Later on, the haematocrit and red cells can be measured, if the patient is still alive.

Death by the firing squad is carried out by ten to 20 soldiers at a distance of 10–20 m. Therefore, the subjects die soon after the bullets penetrate the heart or sever large blood vessels. However, when subjects are shot by a single bullet at short range, death is probably due to destruction of vital centres of the brain (please see below).

4.2. Asphyxia

In drowning, water obstructs the respiratory tract. During hanging and strangulation, obstruction is caused by mechanical compression of the trachea. Asphyxia may also result from bilateral collapse of the lungs due to bullet wounds. The rise of pCO_2 and collapse of pO_2 result in the subject attempting to hyperventilate, but this is prevented by the obstruction. This is extremely distressing, as is the paralysis of the respiratory muscles after the current is switched on in the electric chair (see below). In experiments on human volunteers, it was found that if arterial pO_2 was lowered rapidly, consciousness was lost within 20 s [8].

The earliest response of a conscious person to hypoxia or pain is to show hyperactivity, particularly of the limbs or muscles, and to cry out. These normal physiological reactions are not possible in hanging, because the rope is tied above the vocal cords, and the hands and legs are tied; in the electric chair, the limb muscles and vocal cords are supramaximally stimulated, causing paralysis, and the subject is bound to the chair.

4.3. Electrocution and the electric chair

Death from accidental electrocution, or electrocution by the electric chair, is probably mainly due to asphyxia, as indicated above. However, it does represent unique physiological problems, some of which can be elucidated by comparing the signs with those of other syndromes, and also because of the important experiments carried out by Gregory and Wotton and their colleagues on the procedures of electrical stunning of animals for slaughter [26] (Table 1).

When a sensory nerve is stimulated strongly it produces tingling and then pain. When a motor nerve is stimulated faradically, for example by a physiotherapist, increasing current produces contraction, spasm and then cramp. With the electric chair, a supramaximal stimulus of 2000–2500 V at 8–12 A is used. Therefore, one may assume that all sensory, motor, secretory and autonomic nerves are stimulated. One may then examine the main normal functions of these nerves and thereby deduce the likely effects on the nervous system of accidental electrocution or the electric chair (Table 3).

Table 3
Correlation of the physiological functions of nerves with the signs observed in persons killed by accidental electrocution or by the electric chair

Nerves	Effects	Signs observed in electrocution
Physiological effects of massive electrical stimulation on the nervous system		
Olfactory nerve	Smells	–
Optic nerve	Vision	–
Oculo motor nerve	Eye movements	Rolling eye balls; sunken eyes
Trochlear nerve	Eye movements	Rolling eye balls
Trigeminal nerve	Facial, dental, jaw sensation	Clenching teeth
Abducens nerve	Eye movements	Rolling eye balls
Facial nerve	Facial movements; salivary secretion; taste from tongue and soft palate	Risus sardonicus; drooling; biting tongue
Auditory nerve	Hearing; balance	Tinnitus; feeling of inbalance
Glossopharyngeal nerve	Motor to pharyngeal muscle; salivary secretion; sensory to tongue, larynx and carotid body	Drooling; vomiting;
Vagus nerve	Motor to lungs, heart, digestive tract; sensory to aortic body	Drooling; vomiting; defecation
Accessory nerve	Motor to neck muscles; pharyngeal muscles, larynx, soft palate	Contraction of neck muscles; inability to vocalise
Hypoglossal nerve	Motor to strap muscles of neck and tongue	Contraction of neck muscles
Autonomic nervous system	Fight and flight; rest	Vomiting; defecation; micturition; erection
Peripheral nerves	Motor activities; sensation	Motor paralysis; tingling: pain

Table 4
These signs are nearly always seen

Signs	Cause
Non nervous effects of massive electrical stimulation seen during electrocution or at post mortem	
Air hunger	Paralysis of the respiratory muscles
Brain and viscera hot	Heat dissipated in the body
Erythema	Vasodilation of the skin vessels
Burns at site of electrodes	Heat dissipated at contact with the skin
Charring of skin and brain	Heat and contact with the skin, and conduction to brain
No pulse	Cardiac arrest
Petechial haemorrhages in skin	Probably due to initial hypertension
Haemolysis of blood	Probably due to heat
Gas liberation and swelling of skin	Heat boiling the body fluids
Splitting of skin	Due to sudden expansion of gases in tissue
Considerable sweating	Autonomic stimulation
Pain in muscle and skin	Muscle cramp; burns
Malodors	Burning of tissues and evaporation of body fluids

In addition, it is probable that—as in death by electrocution—the subject is very distressed by being unable to react when he or she feels pain. Some subjects who become unconscious may not remember the sensation, fear or events, as occurs temporarily after electroconvulsive therapy for mental depression.

One may also correlate the signs seen with the likely physiological mechanisms causing them (Table 4).

From these tables, one may conclude that: death by accidental electrocution or the electric chair results mainly from paralysis of the respiratory muscles, causing asphyxia. The motor system reacts to the stimulation at the same time as massive sensory stimulation causes pain [18]. Cardiac arrest causes a catastrophic fall in blood pressure, rendering the subject unconscious. Severe pain may also cause unconsciousness; it is impossible to know which of these would be the first cause. The autonomic effects, such as drooling, vomiting, defecation and erection, occur involuntarily, but would not necessarily be seen in the judicial execution, because the prisoners are clothed and masked, and are given napkins before electrocution.

Both with hanging and the use of the electric chair, the important signs of distress cannot be seen; many of them are masked by the paralysis of the subject. This has led to the widespread belief that the subject becomes unconscious immediately the hung person falls, or current is turned on, and feels no pain. This is contrary to the evidence and physiological understanding of the situation.

4.4. Tissue loss

This may result either from an explosion or a mechanical impact, in which the integrity of the body is lost, and the subject dies from blood loss, or decapitation. The police usually keep the tissue for subsequent forensic examination and DNA identification.

4.5. Destruction of the brain

This kills a person if it is extensive enough, or if it destroys the 'vital centres' in the medulla, from where the respiratory drive originates [9,10]. After excision or absence of large parts of the brain other than the medulla, virtually normal physiological and psychological activity may be retained as is usually seen after early hemispherectomy [11,12], lobotomy [13], hydrocephalus [14,15], and brain injury [16]. The medulla is important not only because it contains the respiratory and cardiac centres, but also because it is the route of communication between the higher brain and the spinal cord. Its crucial nature was demonstrated graphically when the assassin's bullet lodged in President Kennedy's medulla in 1964.

4.6. Disconnecting the brain and spinal cord

When a person's head is completely disconnected by axe, sword or guillotine, there is clear discontinuity between the brain and the spinal cord, accompanied by blood loss from the exposed arteries. It was also thought that during hanging without decapitation, fracture of the atlas contributed to the dying. However, in an important study [17] James and Nasmyth-Jones found that fracture had occurred in only six out of 34 cases of hanging. In these cases, death was probably due to asphyxia.

4.7. Poisoning

The use of intravenous lethal injections was designed to minimise the pain of execution [19]. Essentially, the prisoner is killed while anaesthetised. The subject is strapped down supine on a trolley, and the arms and legs are bound. Three intravenous lines are set up in the prisoner's forearm. This is no more painful to the subject, nor difficult for the operator, than a routine intravenous injection in a hospital unless the prisoner fights to resist it, or has sclerotic veins due to intravenous drug abuse.

A short acting anaesthetic, such as thiopentone, is administered through the intravenous cannula. When the subject is anaesthetised, a muscle relaxant is administered paralysing all muscles, including those of respiration, and then a high concentration of potassium chloride to cause cardiac arrest. The procedure takes a few minutes. Intravenous lethal injection is the commonest method of capital punishment in the United States in those states that retain the death penalty. Although contemplation that a person is to be executed is obviously extremely stressful there is very little pain

attached to the execution, except under the circumstances indicated above. There are usually three intravenous lines, and measures are taken so that the technicians carrying out the injections cannot be seen by the prisoner or his or her relatives, so that there is no way that the person who actually administered the lethal drugs can be identified.

5. Conclusion

Although the physiology of the dying process is largely understood, the biochemical mechanisms which trigger the processes of dying, death, and the changes post mortem, require more studies using procedures which do not kill the tissues themselves. A new era in resuscitation would be opened if the biochemical mechanisms could be elucidated.

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