Compression asphyxia and other clinicopathological findings from the Hillsborough Stadium disaster

Jerry P Nolan ,[1] Jasmeet Soar,[2] Nathaniel Cary,[3] Nigel Cooper,[4] Jack Crane,[5] Ashley Fegan-Earl,[3] William Lawler,[6] Philip Lumb,[7] Guy Rutty[8]

Handling editor Jason E Smith

[1]Warwick Clinical Trials Unit, University of Warwick, Coventry, UK
[2]Anaesthesia and Intensive Care Medicine, Southmead Hospital, Bristol, UK
[3]Forensic Pathology Services, Unit 12, The Quadrangle, Wantage, UK
[4]Department of Cellular Pathology, Royal Victoria Infirmary, Newcastle upon Tyne, UK
[5]Institute of Forensic Medicine, Queen's University Belfast, Belfast, UK
[6]Dyke Farm, Penrith, UK
[7]Pathology, Royal Oldham Hospital, Oldham, UK
[8]East Midlands Forensic Pathology Unit, University of Leicester, Leicester, UK

Correspondence to
Professor Jerry P Nolan, Warwick Clinical Trials Unit, University of Warwick, Coventry CV4 7AL, UK; jerrynolan@me.com

Received 15 March 2020
Revised 1 July 2020
Accepted 4 July 2020
Published Online First 3 September 2020

To cite: Nolan JP, Soar J, Cary N, *et al*. *Emerg Med J* 2021;**38**:798–802.

ABSTRACT

Ninety-six people died following a crowd crush at the Hillsborough Football Stadium, Sheffield, UK in 1989. The cause of death in nearly all cases was compression asphyxia. The clinical and pathological features of deaths encountered in crowds are discussed with a particular focus on the Hillsborough disaster.

INTRODUCTION

Ninety-six people died following a crowd crush at the Hillsborough Football Stadium, Sheffield, UK, on 15 April 1989. As expert witnesses to the Hillsborough Inquests, we reviewed extensive written, photographic and video evidence related to events on that day. The cause of death in nearly all cases was compression asphyxia, an infrequently encountered form of mechanical asphyxia akin to traumatic and crush asphyxia but one considered more befitting for the nature of the disaster. Large crowd crushing events have also occurred in association with other sporting events and in large religious festivals such as the Hajj.[1] We discuss the clinical and pathological features of deaths encountered in crowds, specifically compression asphyxia in relation to the Hillsborough disaster based on those facts in the public domain.

BACKGROUND TO THE HILLSBOROUGH DISASTER

On 15 April 1989, a Football Association Cup semifinal between Liverpool and Nottingham Forest football clubs was played at Hillsborough stadium. A crowd crush at the beginning of the match resulted in the deaths of 95 spectators; a 96th spectator died in 1993 as a direct result of the severe hypoxic–ischaemic brain injury sustained in the crowd crush. The spectators who died were standing in two of the pens (Pen 3 and Pen 4) in the terraces in the West Stand of the stadium (figure 1). The pens in the West Stand were enclosed by radial metal barriers to the sides and by high fences at the front; there were also horizontal barriers within the pens that were designed to control crowd surges. After a large exit gate had been opened to relieve a crowd crush developing at the entrance to the turnstiles, spectators entered the back of Pens 3 and 4 from a tunnel that led under the West Stand. There was a small narrow gate at the front of each pen.[2]

In 1989, crowd surges were commonplace on the terraces of football stadia—spectators would be carried forward in the crowd, sometimes being lifted off their feet—any severe pressure on their bodies because of the crowd crush would typically ease off between surges. However, at Hillsborough on 15 April, just before the game started at 15:00, there was an influx of spectators through the West Stand tunnel, which intensified the crowd surges to the extent that a continuous and severe pressure was applied to many spectators. The situation in Pen 3 was compounded by the collapse of one of the horizontal crush barriers, which led to many spectators being thrown forward.

Although the narrow front gates to Pens 3 and 4 were opened, it was difficult to access the pens and get spectators out quickly. Many spectators were unconscious or in respiratory or cardiorespiratory arrest. First aid, including in many cases, cardiopulmonary resuscitation (CPR), was started by spectators (some of whom were off-duty doctors, nurses, ambulance personnel or trained in first aid), police, fire and rescue, and ambulance personnel. Eighty-one spectators were pronounced dead at the Hillsborough Stadium; others were transported to hospital with ongoing CPR (figure 2). Five of the spectators who received CPR at the stadium had a sustained return of spontaneous circulation (ROSC) before transfer to hospital; one of these received mouth–mouth rescue breathing en route to hospital but started breathing spontaneously just before hospital arrival. Two of the spectators who received CPR en route to hospital achieved sustained ROSC in the ED but died later in the intensive care unit (ICU) (figure 2).

TERMINOLOGY: TRAUMATIC, CRUSH AND COMPRESSION ASPHYXIA

Traumatic asphyxia and crush asphyxia are forms of so-called mechanical asphyxia and the terms are generally used interchangeably. Breathing is impaired or completely prevented by pressure applied to the chest. Pressure applied to the chest also compresses the heart—compression of the right side of the heart impairs venous return with resulting back pressure causing congestion, cyanosis and petechial haemorrhages in the face and neck.

Some authors have distinguished between traumatic and crush asphyxia: they suggest that traumatic asphyxia is caused by the sudden application of severe pressure (such as being pinned under a heavy object) to the chest of a person whose glottis is closed (voluntarily or involuntarily).[2–4] This is thought to generate a particularly high intrathoracic pressure, which is transmitted to the superior vena cava (SVC). In contrast, it has been suggested that crush asphyxia is caused by a gradually increasing

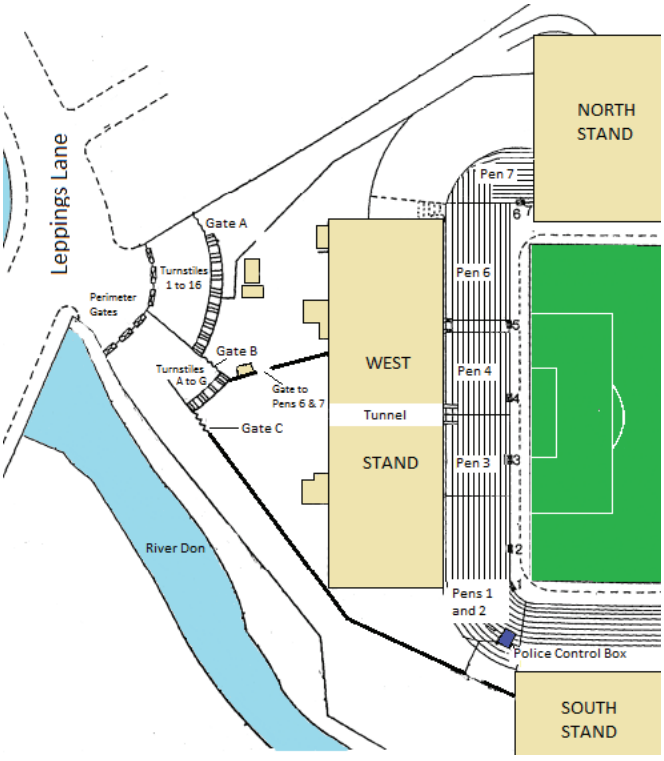

Figure 1 Map of the west terraces at Hillsborough Stadium on 15 April 1989 (Attribution Lord Mauleverer at English Wikipedia—reproduced under the terms of GNU Free Documentation License (https://commons.wikimedia.org/wiki/Commons: GNU_Free_Documentation_License,_version_1.2)).

and sustained pressure on the chest, often in the presence of an open glottis.[2] However, this distinction between traumatic and crush asphyxia is not convincing: given the lack of competent valves in the SVC, severe pressure applied to the chest is likely to generate sufficient back pressure to cause petechiae whether or not the glottis is closed.

Compression asphyxia, although less commonly encountered, can be viewed as synonymous with traumatic and crush asphyxia, as the underlying mechanism for all three is one of compression of the chest and/or abdomen. Traumatic asphyxia and crush asphyxia are terms typically used when the pressure is sustained. However, pressure applied to the chest and abdomen need not be continuous, but rather, for example as in the pressure waves experienced within crowds, may be intermittent in

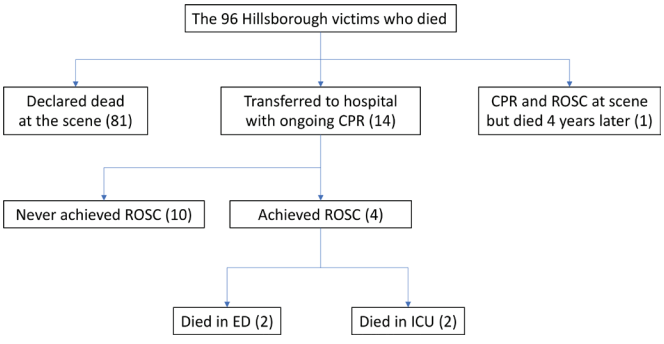

Figure 2 Flow chart showing the 96 Hillsborough victims who died. CPR, cardiopulmonary resuscitation; ICU, intensive care unit; ROSC, return of spontaneous circulation.

nature and yet still have a life-threatening effect on an individual. For this reason, in the 2014–2016 Hillsborough Inquests the term 'compression asphyxia' was agreed to best describe the mechanical asphyxia experienced by those who lost their lives.[5] Unlike in classic traumatic and crush asphyxia, where the pressure applied to the chest and resulting obstruction to venous flow is continuous, in a crowd situation several different mechanisms, including compression asphyxia, can be encountered, which explains why some individuals will show classic features of asphyxia whereas others will not.

MECHANISMS FOR INJURY AND DEATH IN CROWD-RELATED INCIDENTS

Several mechanisms of injury and death may be encountered in a crowd-related incident, which accounts for the variety of subsequent clinical and pathological presentations and findings.

Compression asphyxia: mechanisms for loss of consciousness (LOC) and cardiac arrest

Compression force applied in the anteroposterior plane restricts the ability of the lungs to expand and therefore impairs breathing—this causes progressive hypoxaemia. This positional impairment to breathing (ie, in the front-to-back position and not side-to-side) has been described by survivors of crushes in crowds.[6] If breathing is completely prevented by the compression force, consciousness will be lost when the oxygen concentration in the arterial blood reaches 56% (normal values are approximately 96%–99%).[7] The time taken to reach this concentration is very difficult to predict but is likely to be of the order 1–2 min.[8 9] The time taken is most likely to be at the lower end of this range because compression of the chest will decrease lung volume and therefore the oxygen reservoir in the lungs. Oxygen values in the blood continue to decrease until cardiac arrest occurs. Based on animal experiments, the time taken for cardiac arrest to occur following complete cessation of breathing (asphyxia) is 4–11 min after respiratory arrest.[9 10]

Severe compression of the chest and abdomen will also increase the intrathoracic pressure, which will reduce venous return to the heart. This dramatically reduces the cardiac output and the blood pressure decreases. Extreme pressure on the chest could cause loss of cardiac output independently from hypoxaemia and this could occur very rapidly (we estimate less than a minute).[11] The relative contribution of hypoxaemia and high intrathoracic pressure to cardiac arrest in the victims of the Hillsborough disaster is unknown. The additional complicating factor in the Hillsborough disaster was that the crushing episodes experienced by individuals were variable in force and duration (cited by survivors)—this made it difficult to predict the time to LOC and to cardiac arrest in any individual. Some individuals in close proximity to those who died seemed to sustain little or no injury.

Smothering

Smothering is a form of mechanical asphyxia caused by occlusion of the nose and the mouth. Conceivably, pressure on the neck could also be a factor. A crowd will typically include adults and children of different physical builds. In a stadium, tiered terraces may enable smothering to occur. Those of small build may find themselves compressed between people with their face forced into another person's body and clothing (figure 3), occluding their mouth and nose. Smothering as a mode of death may explain the limited signs of compression asphyxia in some of those involved in a crowd crush.

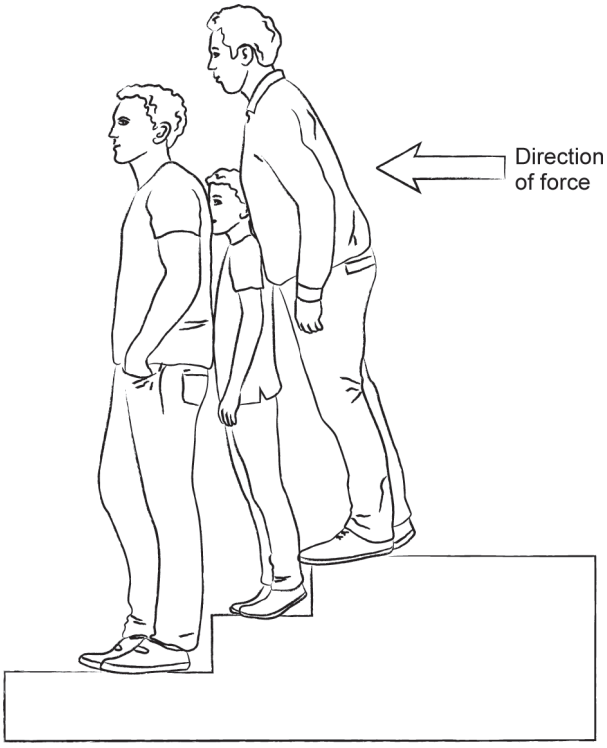

Figure 3 A smaller individual may be 'smothered' when compressed between larger people. Illustration by Vicky Eves.

Aspiration of gastric contents

The finding of gastric contents within the upper and lower airways of the victim of a crush incident is not unexpected and should not be taken as the cause of death without careful consideration. An individual transfixed by the pressure of the crowd may vomit, an active process requiring involuntary but coordinated muscle activity to expel stomach contents forcefully up the oesophagus and out of the mouth. This process requires adequate circulation of blood to the brain and does not normally occur after cardiac arrest. If vomit is not expelled, then it may occlude the upper airway or, if the gag reflex is compromised in the unconscious individual, it can enter the lungs. Massive aspiration with total blockage of the airways that prevents adequate oxygenation will cause LOC and then cardiac arrest. It may also prevent CPR being successful. Immediately after cardiac arrest, the gastro-oesophageal sphincter relaxes and stomach contents can pass passively up the oesophagus and out of the mouth.[12 13] Although this is a passive process, chest compressions may eject the contents forcibly from the victim's mouth. Thus, during chest compressions, it can be difficult to tell the difference between passive regurgitation and active vomiting (implying the person is not in cardiac arrest). An unprotected airway is vulnerable to gastric contents entering it passively, hence being found at later examination. The independent contribution of aspiration to a victim's death after cardiac arrest is unknown. Among those who survive to hospital, but who subsequently die, the mode of death is most commonly brain injury or failure of the heart and circulation.[14 15] Although aspiration is associated with the development of pneumonia, the presence of pneumonia is not associated with increased mortality.[16] Finally in the dead, body movement through recovery and storage can result in passive regurgitation and stomach contents entering the pulmonary airways; this can mimic true aspiration of gastric contents.[17]

Positional asphyxia

If a person is rendered unconscious from compression asphyxia and is held upright by the crowd surrounding them, their head will tend to slump forward which, if not corrected, will cause airway obstruction. Under these circumstances, even if the crowd crush eases sufficiently to enable breathing movement, the continuing unconsciousness and airway obstruction will cause progressive hypoxaemia leading to cardiorespiratory arrest. If an individual who is rendered unconscious from compression asphyxia falls to the floor, depending on their position, their airway may become or remain obstructed. This may be even more likely if they end up underneath other bodies (as was the case for many of the victims of the Hillsborough disaster). Forced flexion of the torso, particularly if applied over a barrier, for example, will compromise ventilation. Finally, if an unconscious person is being recovered from an incident area, failure to ensure that their airway is open could lead to worsening hypoxaemia. This could occur, for example, if an unconscious breathing individual was carried in the supine position without maintaining basic airway manoeuvres such as a chin lift and/or head tilt.

Trauma

There are several life-threatening organ injuries that can be caused when a person is involved in a crowd crush. A severe head injury may be caused as a person rendered unconscious by compression asphyxia falls to the ground or by trampling. Rib fractures may cause a tension pneumothorax, haemothorax or lacerations to the lungs or abdominal organs. Severe compression of the chest can rupture the right atrium and severe compression of the abdomen can rupture intra-abdominal viscera. Compression of the abdomen against a rigid object such as a barrier can injure the abdominal aorta or its branches—traumatic rupture of the abdominal aorta was the cause of death in one of the Hillsborough victims.

Natural causes

In theory, the stress caused by being caught up in a crowd crush might induce a cardiac arrhythmia in a person with ischaemic heart disease. It seems likely that a person with severe chronic lung disease would be more vulnerable to the effects of a crowd crush than a previously healthy person, but we know of no evidence to prove this hypothesis.

IDENTIFYING RESPIRATORY AND CARDIAC ARREST AFTER COMPRESSION ASPHYXIA

Identifying the moment cardiac arrest occurs can be difficult not just for laypeople but also for healthcare staff. Abnormal gasping breathing occurs at the time of cardiac arrest in about one-third of cases and this is frequently mistaken for normal breathing—it is usually short-lived (less than a minute) but can last for a few minutes and can be sustained by CPR if it generates sufficient blood flow to the brain.[18 19] Eye opening, abnormal twitching movements or even convulsions can also occur at the onset of cardiac arrest.[20] These 'signs of life' can return if high-quality CPR is circulating sufficient blood to the brain.[21]

Unless an individual is experienced in feeling for a pulse, pulse checks are highly unreliable as a means of confirming cardiac arrest—there is good evidence that the inexperienced (both laypeople and many healthcare staff) are unable to feel a pulse even when it is normal, and some think they can feel a pulse when it is absent.[22 23] The recommendation for laypeople to perform a pulse check to confirm cardiac arrest before starting CPR was removed from international guidelines in 2000 but it

would have been part of the standard assessment at the time of the Hillsborough disaster in 1989.[24]

Assessment of breathing by laypeople and inexperienced healthcare staff is also unreliable[25] and very slow or shallow breathing could easily be missed, particularly in a noisy football stadium for example. The presence of dilated pupils, or loss of blinking to touching the eye (corneal reflex) is not a reliable method for assessing whether an individual will or will not benefit from a CPR attempt. Dilated pupils and loss of the corneal reflex is common after cardiac arrest. In cardiac arrest survivors, these signs have been observed up to 72 hours after ROSC.[26]

ATTEMPTED RESUSCITATION AFTER CARDIORESPIRATORY ARREST CAUSED BY COMPRESSION ASPHYXIA

There are very few data on the outcome of resuscitation attempts in victims of crush or compression asphyxia but what little there are suggests almost universal poor outcome once cardiac arrest has occurred. Of the 13 patients arriving at the Northern General Hospital (NGH), Sheffield, in cardiac arrest and receiving CPR after the Hillsborough Stadium disaster, resuscitation was continued in 9 and only 2 of these survived to be admitted to the ICU—both of these individuals died.[2]

SYMPTOMS, SIGNS AND OUTCOMES OF THOSE WITH LOC WHO WERE ADMITTED TO HOSPITAL

According to Wardrope *et al*,[2] 159 individuals were taken to the NGH or Royal Hallamshire Hospital in Sheffield. Fourteen were in cardiac arrest and receiving CPR (figure 2). A cardiac output was restored in four: two re-arrested in the ED and died there; two were transferred to the ICU but later died. Thus, no patient admitted to hospital with ongoing CPR survived. A total of 18 patients (including the 2 resuscitated from cardiac arrest) were intubated and admitted to an ICU for mechanical ventilation.[2] Most of these patients were admitted directly from the ED, but five were initially admitted to the ward and then deteriorated requiring intubation and mechanical ventilation. Nine patients were admitted unconscious or convulsing and a further six were confused on admission and then deteriorated, started convulsing and required intubation.[2] A further 15 patients gave a history of losing consciousness at the scene, giving at total of 30 who had a history of LOC in addition to those who were receiving CPR on admission. A total of 16 patients were known to have had had seizures (4 before reaching hospital and 12 after admission). The occurrence of seizures suggests that these individuals had a prolonged period of hypoxaemia. Of the 18 patients who were admitted to ICU, 2 died (both receiving CPR on admission to hospital) and 6 survived but with neurological deficit. Two patients were described as having cortical blindness.[2]

Nine patients were described as having a right heart strain pattern on their ECG.[2 27] In seven of these patients, the initial echocardiogram showed a dilated and impaired right ventricle—these all later improved.[2 27] One patient had mitral valve prolapse thought to be due to compression of the left ventricle causing rupture of the chordae tendineae or papillary muscles, or direct injury to the valve leaflets.[28]

We are aware of four individuals who received CPR at Hillsborough and survived to leave hospital. One of these individuals remained in a persistent vegetative state until their death in 1993. A fifth person received mouth-to-mouth and bag-mask ventilation at Hillsborough but had a pulse; this person also survived to leave hospital.

PATHOLOGICAL FEATURES OF COMPRESSION ASPHYXIA

The classic description of the external findings in a traumatic/crush/compression asphyxia-related death are craniocervical congestion/cyanosis, petechiae of the head and neck and subconjunctival haemorrhage.[29] The extent to which these changes will occur depends on the individual and the mechanism causing their death. The petechiae are caused by obstruction to venous return in the SVC with back pressure into the capillaries while the arterial system remains patent. This back pressure is readily transmitted because the SVC has no functional valves. This causes capillary rupture in the face, neck and upper chest. The petechiae are uncommon in the lower body because the inferior vena cava collapses in the presence of increased intra-abdominal pressure.[29] Whether petechiae are attributable solely to congestion of the venules and capillaries, or from perivascular blood accumulating in the unsupported structures, such as the mucous membranes, is unclear. Facial oedema, serosanguinous foam from the nose and mouth, nose bleeds and blood from external auditory canals have also been described following compression asphyxia. The classical 'masque ecchymotique' or 'death mask' caused by the florid petechiae in the face and neck with a clear cut-off line between normal and abnormal skin was first described in 1904.[30] Several non-specific changes occur in the internal organs of the head, neck, chest and abdomen secondary to extreme vascular congestion. These include scalp petechiae, cerebral congestion, meningeal petechiae, and intra-cranial bleeds (subdural haematoma and subarachnoid haemorrhage). Laryngeal congestion and petechiae, epiglottic petechiae and oedema of the tongue may all occur. There may be pulmonary oedema and haemorrhage, and blood in the airway. Petechiae may also occur in the thymus, heart and pleura. The subdiaphragmatic organs can show generalised congestion, with intestinal haemorrhage, and in living survivors, haematuria may occur.

Acknowledgements We thank the Hillsborough Families who, through their legal team representatives, gave approval for this manuscript to be written and submitted for consideration for publication. We also thank the Home Office for clarifying that we were in a position to submit this paper for consideration for publication.

Contributors JPN and JS were clinical expert witnesses to the Coroner for the 2014–2016 Hillsborough Inquests. GNR, WL, NC and JC were forensic pathologists to the Coroner during the 2014–2016 Hillsborough Inquests. NC, PL and AF-E were forensic pathologists representing the families during the 2014–2016 Hillsborough Inquests. All authors made substantial contributions to the conception and drafting of this manuscript and revising it critically for intellectual content. JPN is responsible for the overall content as guarantor.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests All authors received payment for their contributions as expert witnesses for the 2014–2016 Hillsborough Inquests.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; externally peer reviewed.

Author note We wish to dedicate this paper in memory of the 96 men, women and children who lost their lives as a direct result of the Hillsborough Football Stadium disaster, 15 April 1989.

ORCID iD

Jerry P Nolan http://orcid.org/0000-0003-3141-3812

REFERENCES

1 Working With Crowds. Crowd disasters by year. Available: https://www.workingwithcrowds.com/crowd-disasters-year/ [Accessed 25 Jun 2020].
2 Wardrope J, Ryan F, Clark G, *et al*. The Hillsborough tragedy. *BMJ* 1991;303:1381–5.
3 Sertaridou E, Papaioannou V, Kouliatsis G, *et al*. Traumatic asphyxia due to blunt chest trauma: a case report and literature review. *J Med Case Rep* 2012;6:257.
4 Williams JS, Minken SL, Adams JT. Traumatic asphyxia--reappraised. *Ann Surg* 1968;167:384–92.
5 Rutty GN, Cary N, Lawler W. Death in crowds. In: Rutty GN, ed. *Essentials of autopsy practice; reviews, updates and advances*. London: Springer, 2017: 43–8.

6 Gill JR, Landi K. Traumatic asphyxial deaths due to an uncontrolled crowd. *Am J Forensic Med Pathol* 2004;25:358–61.
7 O'Driscoll BR, Howard LS, Earis J, *et al*. BTS guideline for oxygen use in adults in healthcare and emergency settings. *Thorax* 2017;72:ii1–90.
8 Farmery AD, Roe PG. A model to describe the rate of oxyhaemoglobin desaturation during apnoea. *Br J Anaesth* 1996;76:284–91.
9 Safar P, Paradis NA, Weil MH. Asphyxial cardiac arrest. In: Paradis NA, Halperin HR, Kern KB, *et al*, eds. *Cardiac arrest - the science and practice of resuscitation medicine*. 2nd edn. Cambridge: Cambride University Press, 2007: 969–93.
10 DeBehnke DJ, Hilander SJ, Dobler DW, *et al*. The hemodynamic and arterial blood gas response to asphyxiation: a canine model of pulseless electrical activity. *Resuscitation* 1995;30:169–75.
11 Boback SM, McCann KJ, Wood KA, *et al*. Snake constriction rapidly induces circulatory arrest in rats. *J Exp Biol* 2015;218:2279–88.
12 Bowman FP, Menegazzi JJ, Check BD, *et al*. Lower esophageal sphincter pressure during prolonged cardiac arrest and resuscitation. *Ann Emerg Med* 1995;26:216–9.
13 Gabrielli A, Wenzel V, Layon AJ, *et al*. Lower esophageal sphincter pressure measurement during cardiac arrest in humans: potential implications for ventilation of the unprotected airway. *Anesthesiology* 2005;103:897–9.
14 Laver S, Farrow C, Turner D, *et al*. Mode of death after admission to an intensive care unit following cardiac arrest. *Intensive Care Med* 2004;30:2126–8.
15 Lemiale V, Dumas F, Mongardon N, *et al*. Intensive care unit mortality after cardiac arrest: the relative contribution of shock and brain injury in a large cohort. *Intensive Care Med* 2013;39:1972–80.
16 Perbet S, Mongardon N, Dumas F, *et al*. Early-onset pneumonia after cardiac arrest: characteristics, risk factors and influence on prognosis. *Am J Respir Crit Care Med* 2011;184:1048–54.
17 Knight BH. The significance of the postmortem discovery of gastric contents in the air passages. *Forensic Sci* 1975;6:229–34.
18 Bobrow BJ, Zuercher M, Ewy GA, *et al*. Gasping during cardiac arrest in humans is frequent and associated with improved survival. *Circulation* 2008;118:2550–4.
19 Wolfskeil M, Vanwulpen M, Duchatelet C, *et al*. Detection and quantification of gasping during resuscitation for out-of-hospital cardiac arrest. *Resuscitation* 2017;117:40–5.
20 Breckwoldt J, Schloesser S, Arntz H-R. Perceptions of collapse and assessment of cardiac arrest by bystanders of out-of-hospital cardiac arrest (OOHCA). *Resuscitation* 2009;80:1108–13.
21 Olaussen A, Nehme Z, Shepherd M, *et al*. Consciousness induced during cardiopulmonary resuscitation: an observational study. *Resuscitation* 2017;113:44–50.
22 Bahr J, Klingler H, Panzer W, *et al*. Skills of lay people in checking the carotid pulse. *Resuscitation* 1997;35:23–6.
23 Eberle B, Dick WF, Schneider T, *et al*. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. *Resuscitation* 1996;33:107–16.
24 Marsden AK. Guidelines for cardiopulmonary resuscitation. basic life support. revised recommendations of the resuscitation Council (UK). *BMJ* 1989;299:442–5.
25 Perkins GD, Stephenson B, Hulme J, *et al*. Birmingham assessment of breathing study (BABS). *Resuscitation* 2005;64:109–13.
26 Rittenberger JC, Sangl J, Wheeler M, *et al*. Association between clinical examination and outcome after cardiac arrest. *Resuscitation* 2010;81:1128–32.
27 Channer KS, Edbrooke DL, Moores M, *et al*. Acute right heart strain after crushing injury at Hillsborough football ground. *BMJ* 1989;299:1379–80.
28 Grech ED, Bellamy CM, Epstein EJ, *et al*. The Hillsborough tragedy. *BMJ* 1992;304:573–4.
29 Richards CE, Wallis DN. Asphyxiation: a review. *Trauma* 2005;7:37–45.
30 Beach HH, Cobb F. I. traumatic asphyxia. Report of a recent case, with a study of the minute pathology, and summary of reported cases. *Ann Surg* 1904;39:481–94.