Traumatic asphyxia in children

Gregor Campbell-Hewson, Conor V Egleston, Andrew R Cope

Abstract
Two cases of traumatic asphyxia in young children are reported. The first was a 2 year old child run over at low speed by the front wheels of a delivery van. He made an uncomplicated recovery. The second child was pinned to the floor by an empty chest of drawers in an unattended accident. He was discovered in cardiac arrest and resuscitation was unsuccessful. The outcome following traumatic asphyxia is a product of duration of compression and the weight involved. Considerable weight can be tolerated for a short period, whereas a comparatively modest weight applied for a longer period may result in death.

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The syndrome of traumatic asphyxia has been reported regularly in medical publications since its initial description by D’Angers Ollivier following his observations on the cadavers of people trampled upon during crowd disturbances in Paris on Bastille day 1837. It has been defined as cervico-facial cyanosis, subconjunctival haemorrhage, and cutaneous petechial haemorrhages following thoraco-abdominal compression.

It was recently brought to prominence by the Hillsborough Stadium disaster where the victims bore some of the stigmata of traumatic asphyxia, although there were marked differences in presentation and outcome.

The purpose of this paper is to report two cases of traumatic asphyxia in young children which show important and contrasting features of the pathophysiology of this condition, and to review the relevant published reports.

Case 1
The patient, a healthy 2 year old boy, chased a ball onto a road. He fell and his torso was run over by the front wheel of a delivery van (Ford Transit, unladen weight 1533–2075 kg). The van had been driving slowly and stopped before the back wheels had reached the child. He did not lose consciousness and remained motionless under the van on the instructions of his parents.

On arrival in accident and emergency (A&E) he was alert but jittery. His blood pressure was 105/55, pulse 115, respiratory rate 18, and he was well perfused. He complained of abdominal pain which was poorly localised. His facial appearance was striking, with cervico-facial cyanosis and swelling, widespread petechiae, and bilateral subconjunctival haemorrhages (figure). There were no marks of compression on the torso. On fundoscopy there were bilateral retinal haemorrhages and exudates. Otherwise neurological examination was normal. There were no fractures or other abnormalities on radiographs of the skull, cervical spine, or chest. A plain abdominal radiograph showed acute gastric dilatation. He was initially treated with oxygen, intravenous fluids, urinary catheterisation, and insertion of a nasogastric tube to decompress the stomach. Arterial blood gases, haematology, and biochemistry profiles were all normal. A further chest radiograph 18 hours after the accident was also normal. An ultrasound of the abdomen failed to show any abdominal injury. Clinically he made good progress, although his dramatic facial appearance persisted. He was discharged 8 days later, by which time his appearance had almost returned to normal with only the resolving subconjunctival haemorrhages remaining. The main reason for his prolonged inpatient stay was the development of a swinging pyrexia on day 4 caused by an urinary tract infection, presumably secondary to catheteri-
sation. At the time of his review one month later examination was entirely normal.

Nine years after the accident he is healthy without any cognitive impairment or focal neurological deficit.

Case 2
The patient, an 18 month old boy, was one of twins and had no significant past medical history. His parents were profoundly deaf but had equipped their house with an intercom system using a sound activated LED (light emitting diode) display.

He had been put to bed in the late afternoon as usual and was found approximately one hour later trapped under an overturned empty chest of drawers. It was not possible to ascertain the duration of the entrapment because the parents had not heard the chest being overturned. The parents began mouth to mouth resuscitation when they found the child unresponsive and apnoeic. On arrival of the ambulance crew, cardiac arrest was confirmed. He was intubated and ventilated with 100% oxygen and chest compressions were continued. On arrival in A&E he was given boluses of adrenaline, atropine, and sodium bicarbonate. He remained asystolic throughout. Arterial blood gases were analysed and showed a pH of 6.367, Pco₂ 17.94 kPa, Po₂ 5.7 kPa, and a base excess of −39.1. He was not hypothermic. He remained asystolic and after 25 minutes of full resuscitation he was pronounced dead.

On necropsy examination there were widespread petechial haemorrhages over the front and sides of the face and neck extending on to the upper part of the trunk. There were pressure marks from the chest of drawers on the lower chest and neck. There was also minor bruising of varying ages in keeping with the "rough and tumble" of childhood. There were no features of deliberate physical abuse. The cardiovascular, gastrointestinal, and genitourinary systems were all normal. The respiratory system was normal apart from mild oedema and congestion of the lungs. There was congestion on the external surface of the brain but no focal abnormality. A skeletal survey showed no bony injury. The findings at necropsy, taken in conjunction with the clinical history, were entirely consistent with death due to traumatic asphyxia.

Discussion
The syndrome of traumatic asphyxia is an uncommon one and presents with the classical triad of craniofacial cyanosis, petechiae, and subconjunctival haemorrhages. Motor vehicle accidents are the most common cause of traumatic asphyxia in the paediatric population. It is typically associated with transient ischaemic neurological deficit and injuries to the thorax and abdomen. Despite the dramatic appearance of the masque ecchymotic the outcome is generally good if the patient survives the initial few hours following injury. In 1908 Bolt suggested that there were four principal factors in the pathogenesis of traumatic asphyxia.11 These are deep inspiration, closure of the glottis, splinting of the thoracic and abdominal musculature, and thoracic or upper abdominal compression. He postulated that in order to produce the classical features of traumatic asphyxia there must first be a "fear response" in which the victim, apprehensive of imminent injury, takes and holds a deep breath. This allows the generation of far higher thoracic pressures than compression alone. The pathogenesis of the cutaneous features of traumatic asphyxia has yet to be fully elucidated. Animal studies to date have yet to reproduce all the clinical features associated with the condition. The most widely accepted explanation is that following a compressive force to the chest or upper abdomen positive pressure is transmitted to the mediastinum, forcing blood out of the right atrium into the innominate and jugular veins. This leads to an increase in pressure in the small venules and capillaries of the face and head, causing petechial haemorrhages. The small vessels become atonic following...
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