12 Traumatic Cardiac Arrest

Matthew O'Meara and Peter Lax

- Epidemiology of traumatic cardiac arrest
- Reversible causes of cardiac arrest in trauma
- Managing medical cardiac arrests in trauma patients
- Resuscitative thoracotomy in traumatic cardiac arrest
- Human factors in traumatic cardiac arrest
- Other causes of arrest relevant to trauma

Introduction

Traumatic cardiac arrest is the final common pathway of several pathologies, often resulting in high mortality.

M. O'Meara (✉)
EMRTS Wales, Llanelli, UK

Deprtment of Anaesthesia and Critical Care Medicine, University Hospital of North Staffordshire, Stoke, UK
e-mail: Matthew.OMeara@doctors.org.uk

P. Lax
Tactical Medical Wing, Royal Air Force, Brize Norton, Oxfordshire, UK

The Air Ambulance Service (TAAS), Rugby, Warwickshire, UK

Epidemiology

Office of National Statistics data highlights that trauma causes approximately 20,000 deaths per year in the UK, out of 500,000 deaths overall (4% of all deaths), but that it disproportionately affects certain age groups (10% of all deaths in those of working age) [1]. Survival rates of up to 4% have been reported in traumatic cardiac arrest, but may be higher in specific situations and for specific underlying conditions.

Out-of-hospital cardiac arrest accounts for 0.5% of all calls to the UK ambulance service, of which traumatic aetiology accounts for 10% of these (1:20,000) calls overall. There are approximately ten million calls to the service each year, yielding the total number of traumatic arrests as 5000 per annum. Up to 50% of these patients will be beyond help and will not have resuscitation commenced, in line with ambulance service guidelines [2].

If these patients were distributed equally across the trauma system, each network would see one potentially treatable patient in traumatic cardiac arrest every 4 days. Thus, it is a comparatively rare event for most healthcare practitioners across the system [3].

Presentation

Reversible traumatic cardiac arrest may occur in the presence of the team caring for the patient or

P. Lax (ed.), *Textbook of Acute Trauma Care*, https://doi.org/10.1007/978-3-030-83628-3_12

on hospital arrival of a patient who has been pre-alerted as being peri-arrest by prehospital teams.

It is possible for patients to be assumed to be peri-arrest but to have actually arrested. This emphasises the importance of situational awareness, especially during critical phases of care, such as during handover. Pulse checks are therefore vital, especially in those with agonal breathing.

Management

Cardiopulmonary Resuscitation

Two theoretical principles exist concerning closed chest compressions during CPR. The cardiac pump theory purports that compression between the bony thorax expels blood into the aorta, as would be the case if the heart was beating natively. The thoracic pump theory relates changes in intra- and extrathoracic pressure to changes in pressure in the vascular system [4].

The role of closed-chest massage has been questioned in the management of traumatic cardiac arrest. Evidence to support or refute its use is weak on both sides of the argument. Therefore, CPR should be initiated and performed (especially if there is a possibility of a medical cause of cardiac arrest) unless it is impeding the performance of other procedures of a higher priority, such as intubation, thoracic decompression, resuscitative thoracotomy (where indicated) or the administration of resuscitative fluids [5–9].

Trauma patients in cardiac arrest are much more likely to present in non-shockable rhythms [10, 11]. In patients who are extremely hypovolaemic but have arterial pressure monitoring in situ, there may be no palpable pulse, but an arterial waveform remains. This indicates a severely compromised but present cardiac output. However, under traditional ALS teaching, this state would be considered Pulseless Electrical Activity (PEA) and managed according to standard cardiac arrest guidelines. Most patients will not have arterial lines in situ unless they arrest in hospital. Still, echocardiography in prehospital care and emergency departments has been used to demonstrate "low-flow" as opposed to "no-flow" states. If echocardiography indicates effective myocardial contraction but severe hypovolaemia, the correct treatment would be aggressive resuscitation with blood and source control of bleeding instead of the administration of adrenaline and CPR [6, 11]. If there is an inadequate circulating volume to the point where pulses cannot be palpated, even the most effective CPR will not correct this.

Reversing the Reversible

The reversible causes of traumatic cardiac arrest vary from those of medical arrest subtly. They can be remembered using the mnemonic HOTT (Hypovolaemia, Oxygenation, Tension pneumothorax and tamponade) [5, 6].

Hypovolaemia

Where traumatic cardiac arrest occurs in the context of hypovolaemia, the chances of survival are reduced as compared with traumatic cardiac arrest from other mechanisms (except for severe neurotrauma) [11].

Hypovolaemia in trauma must always be assumed to be secondary to bleeding. This can occur externally or internally. External blood loss should be aggressively treated with direct pressure, dressings, tourniquets, staples or sutures as mentioned in the massive haemorrhage chapter. Internal haemorrhage from fractures should be managed with manual traction and splintage before surgical fixation. The pelvis should be bound to limit further bleeding into the pelvic cavity. Non-compressible thoracic or abdominal haemorrhage is only controllable surgically, or via interventional radiological means in select cases.

Surgical methods to control haemorrhage include the need to perform a thoracotomy or laparotomy, with packing, clamping or repair of bleeding vessels. In the short term, a thoracotomy performed in the resuscitation room with aortic cross-clamping may buy valuable time to assess and address distal bleeding sites. This procedure should only be performed in the right environment with the right equipment and team and with a viable exit strategy for the next phase of care as discussed later in this chapter.

Current guidelines recommend resuscitative thoracotomy in the event the resuscitation team witnesses the patient arrest, or the patient has had signs of life and CPR within the previous 10 min (blunt trauma) or 15 min (penetrating trauma), with a suspected or confirmed cause that may be amenable to surgical treatment [12]. The indications for resuscitative thoracotomy may change depending on available expertise and resources, as non-specialist surgeons or non-surgical practitioners are able to treat fewer pathologies in comparatively austere environments compared to trained trauma surgeons in an operating theatre [5, 12].

Endovascular techniques have been employed since the 1950s to control excessive haemorrhage in trauma [13, 14]. Modern balloon technology allows for an endovascular balloon to be placed into one of the three zones of the aorta to arrest haemorrhage distal to that site. The occlusion is thought to be temporising while the balloon is inflated. However, it may also be temporarily therapeutic in itself since a reduction in pressure in bleeding vascular beds may facilitate clot formation. Zone three REBOA is reserved for pelvic/lower extremity bleeding. Zone one occlusion (above the diaphragm) is likely to be more appropriate for cases of witnessed or near-witnessed traumatic cardiac arrest where haemorrhage is thought to be the primary aetiology. Once the balloon is inflated, it is imperative to have strict protocols to minimise inflation time, and the patient should be transferred for definitive management to either the interventional radiology suite or operating theatre. When source control has been achieved, effective communication is needed between the surgeon/interventionalist and anaesthetist regarding deflating the balloon to manage the cardiovascular effects of reperfusion. This may lead to cardiovascular instability as cold, acidic and potassium-rich blood is returned into the central circulation at the same time as the volume of the circulation is increased.

Vascular access can be achieved by several methods. The need for short, large bore vascular access for aggressive and ongoing fluid resuscitation is essential. Peripheral access may be possible and adequate, but is frequently challenging (if possible at all) in severe hypovolaemia. Failure of the rapid establishment of adequate peripheral access dictates the placement of central access, ideally with a Swan sheath introducer or another large-bore device (e.g. Vascath or MAC line). Standard central lines are too narrow to provide adequate flow rates for resuscitation (Fig. 12.1). If peripheral access is going to be placed, it should be 16G or larger in the first instance. However, devices such as the Rapid Infusion Catheter (RIC) sets can be used to upsize smaller cannulae via a Seldinger technique.

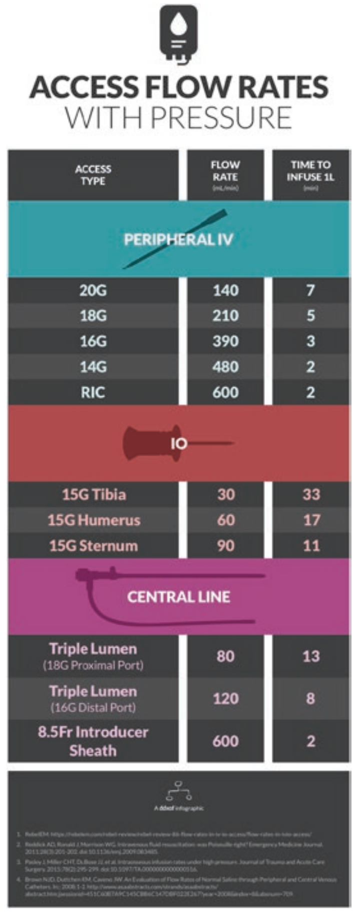

Fig. 12.1 Flow rates through various vascular access devices (from ddxof.com [15])

Central access can be achieved in the internal jugular, subclavian or femoral veins [16, 17]. The subclavian vein is often preferred for several reasons. Logistically, the chest is accessible for intervention without impeding the airway operator if a concurrent procedure such as intubation is ongoing. There is also no requirement to move the neck, which may also have a cervical collar applied, rendering the internal jugular veins inaccessible. Anatomically, the subclavian vein is held open by the clavipectoral fascia and therefore easier to cannulate in hypovolaemic patients, allowing large volume resuscitation straight into the superior vena cava. Compared to other central access sites, subclavian lines carry the lowest risk of infection [18–21], and when secured is more comfortable for the patient than an internal jugular or femoral line [22]. The subclavian route additionally avoids the risk of fluids or drugs not getting to the central circulation due to an interrupted vascular supply (e.g. iliac vein or IVC injury) in the case of femoral lines.

Intra-osseous (IO) access can be helpful for drugs administration and can be used for fluid resuscitation; however, if not appropriately guarded, they are easily displaced. Even when working well, IO lines often require high pressure to ensure flow. This makes them appropriate for drug bolus administration, but for ongoing large volume resuscitation they are impractical and central (or large peripheral) access is preferred.

Fluid resuscitation for bleeding trauma patients is covered in the Circulation Chap. 10 and should follow principles of haemostatic and blood resuscitation where possible. Whole blood, initially used in World War One, is a promising solution as compared with mixed ratios of blood, plasma and platelets. Fibrinogen levels fall early in hypovolaemic shock (especially in traumatic cardiac arrest) and should be replaced with cryoprecipitate or reconstituted fibrinogen [23, 24]. Electrolytic abnormalities, especially hypocalcaemia and hyperkalaemia, are a feature of large volume product transfusion and should be actively sought and excluded in the event traumatic arrest occurs on the operating table [25–28]. All blood products should be warmed to mitigate against hypothermia caused by the administration of refrigerated blood products.

In the context of a traumatic arrest, universal donor blood products should be brought from the blood bank as part of an ongoing major haemorrhage protocol. This ensures a steady and smooth flow of products to the patient. Early tests of coagulation and coagulopathy should be sent, including the use of thromboelastography.

Oxygenation

Hypoxia occurs in traumatic cardiac arrest from a variety or combination of pathologies. Asphyxia occurs when the airway is occluded from any cause—in blunt trauma, the most common cause is a head injury with secondary airway obstruction. There may be a direct injury to the structures of the airway or the neck as a result of blunt or penetrating trauma, which may entirely or partially occlude the airway or limit ventilation and contribute to asphyxia.

Failure to deliver oxygen occasionally occurs due to depletion of portable cylinders or supply lines in a medical context, and constant surveillance for low FiO_2 should be kept whilst managing patients suffering from a traumatic cardiac arrest.

The inspiration of oxygen-deplete air occurs with smoke inhalation and will be associated with toxicity, for example of carbon monoxide or cyanide and will result in histotoxic hypoxaemia.

Failure of the respiratory system to adequately oxygenate the blood can occur due to indirect or direct injury. High cord injuries result in paralysis of the phrenic or intercostal nerves resulting in failure of nerve impulse transmission to the diaphragm or intercostal muscles. Ventilation is impaired and may cease altogether with resulting hypoxia and hypercarbia. Classic mechanisms include those resulting in high cervical spinal injuries from neck hyperflexion or hyperextension, such as diving into shallow water or cycling injuries. Once hypoxia is corrected, a return of spontaneous circulation (ROSC) usually follows. Spinal precautions are essential in these incidents.

Thoracic injury causes damage to the lungs or pleura which may impede ventilation, resulting in hypoxic cardiac arrest—see the Breathing Chap. 9 for more information.

Airway Management

The airway should be managed according to standard protocols. This usually means that the patient should be intubated when possible and the practitioner appropriately trained. This affords optimal ventilation, oxygenation and airway protection, though as described in the fundamentals of airway management chapter, it has not been associated with an increased rate of ROSC in cardiac arrest from medical causes. If a well-seated supraglottic airway is providing effective ventilation and there is no other immediate advantage, there is no immediate need to convert to an endotracheal (ET) tube. This may change if a resuscitative thoracotomy is to be performed, or there is significant chest trauma which may mean that a supraglottic device is inadequate or ineffective to ensure oxygenation and ventilation. Where a cervical spinal injury is thought to co-exist, the patient should be intubated with manual in-line stabilisation of the cervical spine wherever possible, acknowledging that securing the airway takes priority over ensuring spinal immobilisation. Videolaryngoscopy offers some advantage in terms of reduced spinal movement; however, it may be difficult if the airway is significantly contaminated with blood or other liquid debris. Locally agreed protocols should limit intubation attempts, and a failed intubation drill should be followed in the event it proves impossible.

Ventilation

Once intubated, the cuff of the ET tube should be inflated and the patient connected to a ventilator delivering 100% oxygen in the first instance. The exact mode of ventilation matters less than the observance of the principles of lung-protective ventilation, in which the volumes the lungs are exposed to are limited to 6 ml tidal volume per Kg body weight to reduce volutrauma and barotrauma. End-tidal CO_2 monitoring via waveform capnography is mandatory to confirm ET tube placement. It may give a gross marker of cardiac output or adequacy of CPR in the peri-arrest or arrested patient [29–31]. If the patient is successfully resuscitated then a low-normal end-tidal value (approximately 4.5 kPa/34 mmHg) should be targeted to minimise secondary brain injury.

Traumatic Asphyxia

If the patient has been crushed in the thoracic area, ventilation will be impaired, often to the point of cardiac arrest. Patients usually appear plethoric, with petechial haemorrhage visible to the upper torso/face and subconjunctival haemorrhages. Oxygenation will reverse cardiac arrest if delivered early enough [32, 33] (Fig. 12.2).

Impact Brain Apnoea

Traumatic arrest may present secondary to impact brain apnoea syndrome [34]. In this condition, historically referred to as commotio medullaris, the respiratory centre is transiently paralysed by a

Fig. 12.2 Features of traumatic asphyxia include cervicofacial cyanosis with multiple petechiae and subconjunctival haemorrhage (From Lee et al. [32])

blunt force injury in the absence of demonstrable structural brain injury. When laypeople call the emergency services, they usually describe the patient as not conscious and not breathing after a traumatic event (e.g. assault with a head injury). CPR advice is usually given, and the patient may present to the emergency services or hospital as a traumatic arrest. Current dispatcher-advised CPR advises compression-only CPR, which will not reverse the underlying pathology, namely the need to ventilate and oxygenate the patient. CT scans and postmortem data from these patients reveal a hypoxic brain injury pattern without other parenchymal injuries. Many of these patients may have survived with basic airway management and effective oxygenation during the apnoeic period.

Spinal Injury

High (C1–C3) spinal cord injury can result in traumatic cardiac arrest at the scene by the mechanism described above and is often reversed by oxygenation. Following the return of spontaneous circulation, the patient may develop some respiratory effort, but this will be significantly limited by the injury. Neurogenic shock and bradycardia are usually features, and doses of vasopressors or inotropes are required to raise blood pressure. Cord perfusion and cerebral perfusion (and hence oxygen delivery) are reliant on an appropriate blood pressure. A comprehensive search for other injuries is essential to avoid missing concomitant hypovolaemic shock. Excessive fluid resuscitation in these patients will result in pulmonary oedema, and cardiac output monitoring may be helpful in the early phases of management (see circulation chapter for further discussion).

Tension Pneumothorax

Air in the pleural cavity can build up due to blunt or penetrating trauma, particularly if the patient is undergoing positive pressure ventilation. It is essential to decompress the pleural cavity of all patients presenting with suspected traumatic cardiac arrest. Chest examination is not reliable enough to rule out this treatable and straightforward injury [35].

Decompression should be done according to skill-set and may be by needle or surgical techniques. However, needle decompression has a high failure rate, and surgical methods are strongly preferred (see breathing chapter for further discussion). In an emergency, it is not necessary to place chest drains; simple finger thoracostomies will adequately release tension pathophysiology; intercostal drains can be placed later in the patient care pathway.

Significant air leak may reveal the diagnosis of injury to the bronchial tree. Selective right or left main stem bronchial intubation may be required. This can be done with a bronchial blocker, double-lumen endotracheal tube, or simply advancing the endotracheal tube further, ideally under bronchoscopic guidance in the first instance. In cases of acute massive air leak, pleural air will re-accumulate unless adequately drained.

Tamponade

The heart sits in a tough, fibrous sack (the pericardium) that provides separation from other thoracic structures, allows protection against ventricular dilatation, and facilitates ventricular interdependence and atrial filling. There is usually a small volume of pericardial fluid present to allow lubrication and low friction movement of the heart within the pericardium. Because the volume is so small (usually 10–50 ml in health), the lack of distensibility of the pericardium does not cause a problem with compression of the heart.

Cardiac tamponade occurs when blood fills the pericardium. The thin-walled right atrium and ventricle will become compressed by blood or other fluid between the pericardium and the heart. This impedes the filling of the right side of the heart and forward flow of blood into the lungs and left ventricle. Cardiac output is reduced, and ultimately the patient will suffer a cardiac arrest. The condition is a dynamic and physiological diagnosis, i.e. cardiovascular compromise / traumatic arrest in the face of known or suspected pericardial fluid (usually blood). The pericardium is rich in tissue factor and blood quickly clots. Volumes of blood as small as 200 ml can cause cardiac tamponade if they accumulate rapidly enough. Patients stabbed through the pericardium may bleed into it from a hole in one of the heart's chambers, from the myocardium itself or a pericardial vessel. Similarly, blunt traumatic injuries to the chest may result in a laceration to the pericardium.

Treatment consists of the evacuation of pericardial blood via a thoracotomy and haemostasis of bleeding vessels or chambers; surgical haemostasis can be achieved with sutures or staples. If performed expeditiously, survival rates of 10–35% have been reported (far higher than those from medical cardiac arrest), most of whom will be neurologically intact [11, 36].

For medical causes of cardiac tamponade where fluid other than blood may accumulate (pericarditis, auto-immune or infective causes commonly), needle pericardiocentesis may be an option. This involves inserting a needle into the pericardium under ultrasound guidance and draining the accumulated fluid. This is not appropriate in trauma for two main reasons; firstly, blood will clot quickly, and a needle-based technique will not remove clotted blood. Secondly, there will still be an underlying defect that may still be bleeding and require repair. Without exploration and repair via a thoracotomy, bleeding may continue and the tamponade may re-accumulate.

Resuscitative Thoracotomy in Traumatic Cardiac Arrest

Several international guidelines exist concerning the performance of resuscitative thoracotomy in traumatic cardiac arrest. There is broad agreement that this aggressive intervention should be performed if the patient suffers a cardiac arrest in front of the assembled trauma team, where the skill-set exists. Contraindications to this procedure in this context are primarily relative, and would include excessive time since cardiopulmonary arrest or the presence of other unsurvivable injuries (such as massive head injury).

Risk to Clinicians

Resuscitative thoracotomy is a high-stakes, low-occurrence procedure with significant risks to staff and bystanders, primarily due to potential exposure to bloodborne viruses or psychological sequelae [37]. Patient selection is paramount when deciding to perform this procedure, not only in terms of patient benefit but also in justifying risk to practitioners. Historical US data suggests a 4–8 times higher rate of HIV, hepatitis B or C virus infection risk in the urban trauma population, compared to the trauma population as a whole [38]. With more community awareness and better treatments, new diagnoses have dropped. However, patients live longer with these diseases, so the absolute number of cases has increased [39, 40]. A more recent study has confirmed that approximately 9% of patients who sustained penetrating trauma in one US centre were positive for one of these diseases, many without knowing themselves [38]. This is further supported by a Canadian study that confirmed a three-fold higher rate of hepatitis C prevalence in trauma patients than the background population [41]. Considering that the rates of sharp and needlestick injuries are higher during emergency procedures than elective surgery [42, 43], and that the risk of seroconversion of these diseases is higher with deeper contamination or injury from hollow needles or broken ribs [44], this is a procedure with significant risk to the provider. Prospective multi-centre data shows 7.2% of participants (22 staff members) from 305 emergency department thoracotomies suffered a sharps injury—but only 15 patients survived [45]. The procedure mandates universal precautions to make it as safe as possible. However, there must be a realistic *a priori* prospect of the patient benefitting from the intervention to justify performing it due to the risk to staff.

Patient Selection

Survival is dependent on indication (blunt mechanisms have a worse outcome than penetrating trauma) and time to performance. Again, for patients presenting with penetrating thoracic trauma in cardiac arrest, survival rates up to 10% have been recorded in traumatic cardiac arrest, with most of these patients leaving hospital neurologically functional [36]. The rate may further be increased if resuscitative thoracotomy is undertaken for patients before they arrest, and with specific injury patterns (up to 35% in some case series of patients with penetrating cardiac injuries [12, 46]).

As previously described, the decision to proceed with resuscitative thoracotomy is dependent on a multitude of factors, including the operator, location, likely pathology and time since patient arrest. If a patient is not about to arrest immediately and can be transferred to the operating theatre for the procedure, they should be. This frequently may not be possible, and patients in cardiac arrest already (with appropriate indications) should have a resuscitative thoracotomy in the Emergency Department rather than being transferred to the operating theatre in cardiac arrest with ongoing CPR.

Indications to perform a resuscitative thoracotomy are a penetrating injury to the chest or epigastrium, with the arrest occurring in the presence of the treating team or a thoracotomy commencing within 15 min of arrest. This is also predicated on having a team with appropriate experience and an appropriately permissive environment with good all-round access (either in the emergency department or prehospital). Factors that are associated with better outcomes in Emergency Department (ED) thoracotomy are the presenting pathophysiology causing arrest and timing [47–50]:

- Treatment of tamponade has the best outcome of all causes of traumatic arrest
- Cardiac causes are more amenable to treatment than lung injuries
- Right ventricular injuries are more accessible and treatable than left ventricular injuries
- Single chamber injuries have better outcomes than multiple chamber injuries
- Prehospital arrests survive less frequently than those who arrest during transfer, and the highest survival is in those who arrive at the ED with some vital signs and then subsequently arrest.

There is not universal mortality amongst blunt chest trauma patients who undergo thoracotomy. While blunt mechanisms have poorer outcomes, the increased use of ultrasound scanning in prehospital medicine and emergency departments has meant that pathologies amenable to a resuscitative thoracotomy may be more easily identified. If there is an obvious tamponade following blunt trauma, a resuscitative thoracotomy would be justified, and there are now case reports of survivors with this pathology in the published literature [51]. Some would perform resuscitative thoracotomy for blunt trauma (even if the cause is not clear) on the grounds that it would allow compression of the aorta and internal cardiac massage whilst replacing blood. This depends on local practice, experience and logistics, and a blanket recommendation for or against this is not possible based on current evidence.

Outcomes from resuscitative thoracotomy can be better than cardiac arrest in the general population in some subgroups (e.g. penetrating chest injury, treated immediately before, or just after arrest). This may reflect differences in pathophysiology, resource investment and baseline population differences. As is often highlighted, trauma tends to affect younger people disproportionately and is the leading cause of death in adults under 40 years old. Extrapolating from this, one may theorise that the majority of patients who suffer a traumatic cardiac arrest from penetrating injuries are likely to be younger. Consequently, they may have more physiological reserve, less cardiorespiratory comorbidity and be thought more salvageable than a patient who is elderly and has had a "medical" cardiac arrest. Extrapolating this line of thought further, the younger trauma patient potentially may have a higher level of prehospital or in-hospital healthcare resources allocated to them. Any individual or combination of these variables may account for the differences in survival, though there is currently a paucity of data looking at this phenomenon.

Surgical Technique

The principles of resuscitative thoracotomy are to access the thoracic cavity, assess for reversible pathology and address it. A secondary aim may be to compress the descending aorta. This has two benefits: Firstly, it ensures maximal perfusion of coronary and cerebral circulations. Secondly, it decreases or removes perfusion pressure to subdiaphragmatic organs. This will reduce or stop bleeding in these structures (for example, major pelvic bleeding) in addition to temporarily decreasing the effective volume that is attempting to be perfused. However, from the moment of applying aortic compression, distal organs become ischaemic and this will rapidly cause significant physiological derangement. This ischaemic burden will continue to accumulate with time until reperfusion. Surgical control of any bleeding must be achieved urgently, transferring the patient rapidly to theatre for ongoing damage control resuscitation and surgery.

All other aspects of resuscitation must be performed simultaneously as part of a well-led trauma response to achieve success—airway management, oxygenation & ventilation, vascular access and blood resuscitation. Good lighting and suction are essential.

Clamshell vs Anterolateral Approach

In the resuscitative situation, the thoracic cavity can be accessed by a left lateral or clamshell incision. There has been much discussion regarding the “best” approach to resuscitative thoracotomy, with proponents of both the left anterolateral and clamshell approach. Ultimately, the “best” procedure is the one that works and will primarily be dependent on the skill of the individual operator and experience within the care system. For trained thoracic or trauma surgeons, the anterolateral approach may be more familiar and safer in their hands. For non-thoracic surgeons or non-surgeons, a clamshell approach provides optimal access quickly for the potentially reversible causes that they can treat and is easily taught [52, 53]. A recent prospective study in US trauma centres has indicated that the clamshell approach does not cause additional systemic or thoracic complications when compared to an anterolateral approach [54].

Further, a recent prospective randomised crossover trial of emergency physicians performing both procedures on cadavers showed a higher success rate using the clamshell approach [55]. This trial also showed a lower rate of procedural iatrogenic injuries and greater physician preference and is congruent with other published research [56]. When considering the need to perform this procedure pre-hospitally, the clamshell approach may be preferable as the environment is more austere compared to the emergency department. The superior exposure afforded by a clamshell incision may mitigate against difficulties caused by less than perfect lighting or space afforded to the practitioner.

Clamshell Thoracotomy

A 2004 article by Wise et al. provides a succinct stepwise guide to performing a clamshell thoracotomy [52]. Preparation for thoracotomy should begin with pre-alert information, and the potential (or actual) need for a resuscitative thoracotomy should be discussed before first seeing the patient. Roles should be allocated, and the procedure discussed to minimise time wasted and prepare the team mentally before proceeding [57]. Once the decision made to proceed to thoracotomy, the patient should be positioned supine with their arms abducted to 90° whilst the airway and vascular access are secured. Bilateral finger thoracostomies should be performed in the fifth intercostal space. An assessment made at this point to see if there has been any return of circulation following any of the interventions so far (Fig. 12.3). These are IV fluid challenge to correct **h**ypovolaemia, intubation and correction of hyp**o**xia, and thoracostomies to treat **t**ension pneumothorax—**HOT**. If the assessment reveals the return of circulation, based on the effective intervention and physiological state, the decision must be made regarding the next step. This may be to continue to thoracotomy (Figs. 12.4, 12.5, 12.6, and 12.7) if there is pathology requiring immediate treatment, or may be transferring the patient to the CT scanner or operating theatre (or hospital if the procedure is carried out in prehospital care).

If there is no return of circulation, the next step is to join the two thoracostomies across the chest in a "swallowtail" shape cut following the line of the rib in the intercostal space (Fig. 12.4).

Fig. 12.3 Finger thoracostomy. (Figures 12.3, 12.4, 12.5, 12.6, and 12.7 reproduced from Voiglio EJ, Coats TJ, Baudoin YP, Davies GD, Wilson AW. Thoracotomie transverse de réanimation. Annales de Chirurgie. 2003 Dec;128(10):728–33)

Fig. 12.4 Joining of thoracostomies

The skin and underlying fat should be cut down to the muscle layer. Scissors can then be placed through the thoracostomies, deep to the innermost intercostal muscles. The muscle layers can then be cut through, following the rib line up to the sternum. This is performed bilaterally, and the sternum is subsequently divided with a large pair of scissors or a Gigli saw (Fig. 12.5).

Once access is achieved, it should be maintained using rib spreaders in the right hemithorax (if available) or an assistant to retract the sternum superiorly (Fig. 12.6).

This incision allows full access to the thoracic structures. On opening the chest, the anterior pericardium may be adherent to the posterior sternum and can be liberated by simple blunt finger dissection. The superior pericardium should

Fig. 12.5 Cutting through the sternum with trauma shears

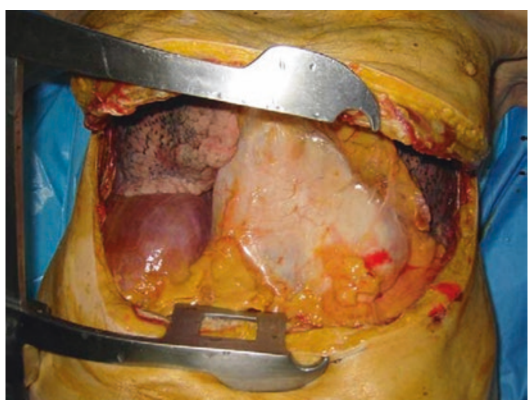

Fig. 12.6 Rib spreader in situ with good visualisation of thoracic structures from above

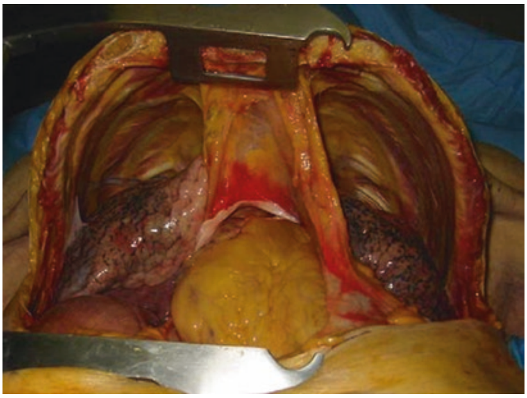

Fig. 12.7 The delivered heart. Note the incised pericardium at the top of the heart and the right atrial appendage in view

be held up with heavy forceps and cut open with a pair of scissors, starting at the cranial aspect and continuing caudally. It may be necessary to extend this incision laterally at the caudal aspect in the so-called "inverted T" incision. This avoids damage to the phrenic nerves, which run laterally along the pericardium, and care should be taken to avoid injuring the coronary vessels. The heart should then be delivered through the pericardium and inspected for wounds, even if there is no overt evidence of tamponade on first opening the chest (Fig. 12.7).

In the event of penetrating trauma, wounds can be temporarily occluded with a finger, secured with mattress sutures or staples, or in extremis, a Foley catheter can be inserted into the defect. If a Foley is used, then the lumen should be occluded to prevent bleeding, and the balloon gently inflated to occlude the hole. This is not the first choice intervention as it may cause further damage to the myocardium if overinflated, and the balloon may decrease chamber volume and impair cardiac output if circulation is returned. For non-surgeons, cardiac staples may be challenging to place, and mattress sutures may cut through muscle if they are pulled too tight. A small section of the pericardium may be used as a pledget or buttress to guard against this in the acute setting. Again, care should be taken to avoid inadvertently suturing any of the coronary vessels. The posterior aspect of the heart should be inspected, but care should be taken to avoid lifting the heart and kinking the great vessels as this will compromise cardiac output. Swabs can be inserted behind the heart (starting from the apex and moving superiorly) to lift the heart anteriorly for inspection without kinking the vessels. Internal cardiac massage should be performed using a two-handed technique. One hand is inserted behind the heart and kept flat while the other lies anteriorly. These then compress the heart in a "clapping" manner from the apex to the superior aspect in an attempt to mimic normal cardiac ejection. This is an efficient manner to improve cardiac output and allows a degree of assessment of circulating volume. Fluid resuscitation (ideally with blood) should continue at this point.

The patient may develop ventricular fibrillation—this can be treated by either "flicking" the heart or by internal defibrillation paddles set to deliver a 10 J shock. If these are not available, the chest can be closed, and standard external pads can be used at normal energy settings.

Once haemostasis is achieved and massage has continued, the patient may develop a return of spontaneous circulation and even wake up. The team should be prepared for this and administered sedation/anaesthesia as required. Dividing the sternum also divides the internal mammary arteries, which should be clamped or tied off at both ends to prevent further bleeding once a cardiac output is restored. The patient should be rapidly transferred to theatre for definitive surgical control of wounds/bleeding / other injuries.

Other Manoeuvres

In the event of damage to lung structures, bleeding can be controlled by performing hilar twisting or clamping. The inferior pulmonary ligament (anchoring the posterior aspect of the lung and the hilum to the posterior thoracic cavity) must be divided in order to twist the lung, bronchial and pulmonary vascular structures around the hilum. An endotracheal tube tie or foley catheter can be used to tie the whole of the hilum off temporarily.

Control of the descending aorta can be achieved by following the posterior ribs on the left side and manually placing the hand against the soft structures lateral to the spinal column and pressing back against it. Surgical cross-clamping requires dissection of the descending aorta from within its fascial plane—this is best done by those specifically trained to do so. Once dissected, a Satinsky clamp can be applied to ensure temporary hands-free control of defending aortic blood flow. Occlusion of the aorta may be a temporising measure to buy time in hospital. However, it may not be feasible to maintain following prehospital thoracotomy during transfer depending on distance and transport modality. The use of REBOA in patients with non-thoracic haemorrhage is discussed more fully in the dedicated chapter.

Resuscitation should continue until reversible causes have been reversed. In the event the patient does not achieve a return of spontaneous circulation, resuscitation should stop. As a dramatic and rarely performed procedure, the team should debrief where possible.

Medical Confounders

When accidents are reported to emergency services, the full history of antecedent events is rarely known. Patients often crash cars or fall down stairs due to co-existing medical conditions such as cardiac, respiratory or renal disease. The key is usually in eliciting a history from bystanders or family members, which is not always possible in the early phases of arrest. These patients must be treated according to standard resuscitative guidelines, with uninterrupted CPR, defibrillation and drugs as appropriate to the situation.

Human Factors in Traumatic Cardiac Arrest

The nature of resuscitation often involves an impromptu formed team of varying levels of experience and understanding. Thus, good followership and leadership are essential components of a well-functioning team, which will be crucial in the optimal treatment of a patient having suffered a traumatic cardiac arrest [58]. Resuscitative thoracotomy is a damage control procedure, and the decision making around it is more fully explored in Chap. 29.

Given the comparative rarity of traumatic arrest as presented to healthcare teams, teams must regularly rehearse and practice together where possible. Most centres have pre-drawn up algorithms/standard-operating procedures (SOPs) to assist in managing traumatic arrest, including when and when not to perform aggressive resuscitative procedures. Cognitive aids such as these assist in decision-making and the expeditious execution of a traumatic cardiac arrest algorithm. It is helpful that each team member understands their own and others roles in the management of traumatic cardiac arrest. Various courses, such as the American College of Surgeons Advanced Trauma Life Support (ATLS) or the European Trauma Course (ETC) provide guidance and help individuals prepare for managing a patient in traumatic cardiac arrest.

In hospital practice, the Trauma Team Leader (usually an emergency medicine consultant in the UK) has a critical role. The role of the TTL is to remain "hands-off" the patient to retain overall situational awareness while coordinating several other clinicians. This leadership role begins even before the patient arrives with pre-briefing based on the ambulance pre-alert, assignment of roles and setting expectations of likely courses of action [57]. Whilst the TTL is an experienced clinician who should be able to perform resuscitative thoracotomy if required, they should limit themselves to directing others rather than get involved in the procedure itself. Should they be the only person capable of performing the procedure, they should formally hand off the role of TTL to another clinician who can retain oversight and situational awareness while the ED consultant performs the procedure.

Given the high mortality rate and the dramatic nature of presentation and treatment, the welfare of the team should be considered in the aftermath of managing a patient with traumatic cardiac arrest, in addition to that of the patient and their family. Debriefing is an essential part of processing such events and should be offered, both hot and cold. Hot debriefs are conducted as near to the incident as possible, in time terms, whereas cold debriefs are conducted some time later. They offer a crucial opportunity for questions to be raised, issues addressed and individual and organisational learning.

Other Causes of Arrest Relevant to Trauma

The following special circumstances of traumatic cardiac arrest are discussed since they are included in most trauma registries as traumatic death.

Commotio Cordis

Commotio Cordis is a condition in which the sudden application of blunt force to the chest, usually over the manubrium, results in the transmission of that force to the mediastinum, pericardium and the heart within [59]. The putative mechanism is that mechanical energy is converted into electrical energy within the heart's conducting system. If the energy is delivered in such a way to interfere with normal sinus rhythm (for example, by inducing an R on T phenomenon), it may put the patient into a malignant arrhythmia such as ventricular fibrillation or pulseless ventricular tachycardia. Much of this mechanism is speculative or derived from animal studies since it is impossible to contrive a situation in which a patient suffering blunt chest trauma is ECG monitored at the time of injury.

Treatment consists of standard resuscitation protocols, including the use of defibrillation and anti-arrhythmic drugs as appropriate. If successfully resuscitated, the patient should be ECG monitored, have myocardial enzymes or troponin levels sent and be admitted for observation. Discussion with cardiology should be initiated as appropriate. The patient rarely requires any long term follow up. The diagnosis is more often made postmortem if a sudden blunt force to the chest has caused cardiac arrest in the absence of any underlying post mortem findings. A slight myocardial contusion may be apparent, but this is unusual.

Lightning

Several thousand people are struck by lightning worldwide each year. Some bodies provide advice about what to do if caught in a storm, but most of this is based on expert opinion and conjecture. Electrical injuries are more fully considered in the Burns section of this textbook (Chap. 40).

Lightning causes injuries due to the passage of high voltage, high current electricity through the body. The electricity passes through the body for fractions of a second, which may go some way to explain the high survival rate. The body may be thrown some distance, causing secondary blunt force injury, and burns over the skin may also be visible. There is usually an entry and an exit wound apparent, as with all electrical injuries. Where the skull is struck, severe cranial injury is likely reducing the chance of survival. If the patient is standing, the exit wound is often on the foot. Traumatic cardiac arrest usually follows respiratory arrest, most probably as a result of paralysis of the respiratory centre. Consequently, cardiac arrest will ensue in the absence of artificial respiration. Most authorities comment on the need for prolonged resuscitative attempts, with some case reports advocating the use of extracorporeal support. A full trauma response, appropriate diagnostic workup and transport to critical care are crucial to survival.

The science of lightning injuries, "Keraunomedicine" (from the Greek for lightning/thunderbolt), flags up some interesting clinical phenomena. Keraunoparalysis is a transient clinical syndrome observed in most lightning strike victims. The patients often report being unable to get up/move after the event and of paraesthesia and paralysis of the limbs. It bears all the hallmarks of an acute spinal cord injury, with lower motor neurone findings on examination and a spinal level. The limbs may appear acutely pale and pulseless as if suffering from severe vascular compromise. This finding has led most authors to conclude that the pathophysiology of the neurological findings are caused by acute vasospasm to the cord. As with other forms of vasospasm, the syndrome is reversible, and in most patients it resolves over the next 12 h.

Lichtenberg phenomena occur as a result of charged particles becoming discoloured within the skin. They often occur in the form of unusual patterns (e.g. the appearance of trees or leaves—"arborialisation"), giving the appearance of a photograph of the environment the patient was standing in at the time of the strike. This has led the science of keraunomedicine to be surrounded in mystique and folklore over the ages. These phenomena are usually transient and disappear within 24 h.

The patient may have other acute and severe burns which require consultation with burns and plastics teams. The burns are often full thickness and may involve deeper structures such as muscle and bone, requiring early surgical management and observation for complications such as rhabdomyolysis and compartment syndrome.

The heart may be affected, however in survivors this is usually transitory.

In summary, those presenting to a hospital who have been struck by lighting usually have a good prognosis and, even in traumatic arrest, extensive resuscitative efforts should be offered.

Electrocution

Electrocution occurs when the patient contacts a suitable source. It may be classified as high voltage (usually industrial) or low voltage (domestic).

In the context of high voltage electrocution, as may occur secondary to industrial or railway accidents, the patient usually suffers severe burns, which can be of high percentage. Traumatic arrest is thought to occur secondary to respiratory arrest or disturbance to cardiac rhythm, but incineration of the patient by secondary burning can occur. These patients have a poor prognosis.

Domestic power usually arises from alternating current at a power of less than 400 volts and a frequency of 50–60 Hz. The peculiarity of this frequency is that it the exact frequency to induce tetany in the muscles. Thus, the patient may touch the source and then be unable to let go, prolonging the electrocution injury.

Rescue efforts must focus on safety to the rescuers before extrication of the casualty. Early application of defibrillators alongside conventional life support algorithms is the mainstay of treatment. In the event of a return of spontaneous circulation, the patient should be transferred to critical care for ongoing management. If the patient regains consciousness quickly, they should be ECG monitored for a suitable period before hospital discharge to exclude any underlying cardiac injury or rhythm disturbance.

Hypothermia

Hypothermia is discussed more fully in the immersion and submersion and burns and thermal injuries chapters (39 and 40). Severe hypothermia is classified as anyone with a body temperature of less than 32 °C. The confounding problem is trying to elucidate whether a patient has gone into cardiac arrest because of hypothermia, or whether they have been in cardiac arrest for a while and become cold. As the body cools down, the basal metabolic rate of oxygen consumption decreases (approximately 10% per degree of cooling). As the body cools, physiological functions are affected, respiration decreases, arrhythmias including AF, VF, and bradycardia can all occur, resulting in cardiac arrest.

Treatment should include active and passive rewarming. In the context of cardiac arrest, several points are worth considering. Firstly, resuscitative protocols alter, including the sequence of the delivery of cardioversion and the use of resuscitative drugs. Pulses may be challenging to feel, and the use of ultrasound may elucidate some cardiac activity.

There have been case reports of successful resuscitation with full neurological recovery with aggressive resuscitation depending on the degree and speed of cooling. Extracorporeal membraneous oxygenation (ECMO) and cardiopulmonary bypass may be utilised where facilities are available. These are often only needed for short periods to provide oxygenation to the organs and warm the patient. Some guidelines incorporate potassium as part of the prognostic workup, with patients having serum potassium of greater than 8 mmol/l being beyond help.

Hanging/Strangulation

There are two categories of hanging: Judicial and non-judicial. Whilst traumatic cardiac arrest secondary to judicial hanging is unlikely to present to healthcare practitioners, patients attempting to commit suicide may do so following the same mechanism, i.e. a long drop with a noose tied anteriorly around the neck with a thick rope. This has the effect of the weight of the body pulling on the neck and hyperextending it, injuring the upper cervical vertebrae and brain stem. This results in near-instant cardiac arrest due to high cord lesions, brain stem effects or severe internal vascular damage to the vertebrobasilar and carotid vessels. Careful handling, immobilisation of the neck and imaging for spinal injuries is essential in patients who have unsuccessfully attempted suicide via this mechanism.

Injuries from non-judicial hanging usually occur secondary to an impairment of blood flow to the brain due to compression of the vessels in the neck or hypoxia from occlusion of the trachea. Compression of the veins results in raised ICP as the hanging ensues and may present as petechiae or subconjunctival haemorrhage. The hyoid bone may be fractured, and there may be damage to the carotid vasculature. Ligature marks are often present externally on the neck, and it is good practice to note their presence.

As cerebral hypoxia ensues, the patient often starts to fit. This may be reported by rescuers or be witnessed by emergency medicine services arriving on scene quickly after the event.

If presenting in cardiac arrest, these patients frequently develop a sustained return of spontaneous circulation once oxygenation resumes, for example, following endotracheal intubation. Airway management is usually straightforward in the early phase of this injury. Whilst ROSC may ensue, survival with a favourable neurological outcome is less common. The vessels in the neck should be imaged to exclude injury/dissection. Hanging injuries which present as short drops and asphyxia are incredibly unlikely to have unstable neck injuries. In a 2014 literature review of 2,795 patients, cervical fracture occurred in approximately 2% of all fatal and non-fatal hangings, and more harm may be caused by a blanket rule advocating the application of a cervical collar to these patients [60]. If the patient has had a true "judicial style" hanging, they will suffer an irreversible cardiac arrest almost instantaneously due to severe vascular and or neural trauma. If the patient shows signs of life when prehospital practitioners arrive, it is highly unlikely that they are in this group. Given that the primary pathology is asphyxia, these patients do not require transfer to a major trauma centre and can be well managed at any hospital with intensive care facilities.

Forensic Aspects

A traumatic cardiac arrest will have been caused as a direct result of an intentional or unintentional act by way of assault or accident. In most jurisdictions, there will follow an investigatory process, which will fall to the police and coronial services.

In the UK, all deaths and injuries which occur in the workplace are reportable under the Health hand Safety law via the Reporting of Injuries, Diseases, and Dangerous Occurrences Regulations 2013 (RIDDOR). This places a legal duty on employing organisations to file an initial report if a person dies or is incapacitated for more than 7 days. Whilst it is not the immediate responsibility of the healthcare team to form a report, it can be helpful to advise the patient/relatives or employers of the need to report. In addition, the follow-up investigation is likely to require a report from the healthcare team to help inform the level of harm and risk of the incident to other people in the workplace.

The Coroner is a judicial appointment by the local council (or other unitary area) under the Coroners and Justice Act 2009. The key responsibility of the Coroner is to investigate any death which is unexpected or unexplained. There is a further list of specific situations which must be discussed with or reported to the Coroner. These include deaths that were sudden, violent or unnatural (including accidents and suspected suicides or murders). All traumatic cardiac arrests should be discussed and referred to the coroner. Those occurring outside the hospital will be reported to the police initially, who may take on the role of 'Coroner's Officers', particularly out of hours. Most ambulance services have established reporting relationships with the local coroner to assist in these out of hospital situations. In-hospital reporting is usually undertaken the next working day by a senior member of the healthcare team. The body will have been taken to the mortuary.

After death, "last offices" are administered. This is the process of respectful treatment of the body per the religious or cultural wishes of the patient as and when they are known. There are some crucial forensic aspects to consider in the context of traumatic cardiac arrest. First and practically, injuries or incisions from medical procedures may continue to bleed. Medical procedures may involve the placement of cannulae, catheters, endotracheal tubes, for example. Although coroners vary in the extent to which they mandate drips and lines stay in, it is usually good practice to discuss these requirements with the local coroner, their officer or the pathologist/mortuary who will ultimately carry out the post mortem. If medical devices are removed, this should be carefully documented in the notes to assist in the subsequent post mortem examination.

There are various types of post mortem examinations carried out In the event of traumatic death. The coroner ultimately decides on whether or not a post mortem examination is required. In the event of a traumatic cardiac arrest, it would be unusual not to request some form of post mortem. In cases of multiple casualty events or major incidents, virtual or computer-reconstructive post mortem examinations have been used to arrive at the cause of death. It is more common for the coroner to request a standard "Virchov" post mortem examination, in which a pathologist reviews the notes and makes an examination of the body before arriving at a cause of death. The cause is often recorded as "multiple injuries" in such cases. If there is a criminal investigation pending, the police may well commission a forensic post mortem. These are undertaken by independent pathologists and comprise a thorough review of injuries, illnesses, interventions and the antecedent history of events. Second post mortem examinations may be requested by a defendant as some of the findings may be subsequently challenged. There is increasing use of scanning technology (CT or MRI) to elucidate injuries after death, and some faith groups favour this. Advantages include an accurate radiographic description of injuries and the ability to carry out limited dissection of the body. Although increasingly popular, their validity compared with classic post mortem techniques is currently the subject of ongoing research. Samples of blood, hair, vitreous humour may be taken for toxicological analysis as part of the post mortem.

When providing evidence concerning those suffering a traumatic injury, whether deceased or alive, several principles are worth bearing in mind. Firstly, the healthcare team providing treatment should stick to simple descriptions of injuries and interventions such that a layperson can understand the information. Excessive medical jargon within statements or verbal evidence is likely to lead to further questioning/extended requests for evidence. Opinion about causation or fault should be avoided. Police and hospitals usually have standard a standard pro forma to help in writing statements. It must be signed and dated. Numbering any paragraphs helps with subsequent requests.

The patient still has a right to confidentiality which extends after death. Each healthcare practitioner must be familiar with the relevant guidance outlining the circumstances in which information can be disclosed, particularly to outside agencies. If information is disclosed, it is good practice to note down what has been revealed, why, and to whom in the patient's notes.

Conclusion

Traumatic cardiac arrest can be from many causes, and the treatment varies depending on the suspected mechanism. Patients may have been in cardiac arrest from medical causes before they suffered trauma, and their treatment is different from patients who have suffered a cardiac arrest *because* of trauma. While some patients with particular injury patterns may benefit from invasive treatments conducted early (e.g. penetrating chest injury with tamponade physiology requiring resuscitative thoracotomy), this is not a universal fact. The presenting pathology and time since arrest are more likely to determine the likelihood of successful resuscitation than a specific mechanism, though certain mechanisms make certain pathologies more apparent. The risks to staff from the procedure are not insignificant, so there must be a reasonable expectation of success rather than performing resuscitative thoracotomies on patients where it is clearly inappropriate.

Questions

1. All patients in traumatic cardiac arrest are candidates for resuscitative thoracotomy.
   (a) True
   (b) False
2. Thoracotomy for penetrating thoracic injuries has a better outcome than for blunt injuries.
   (a) True
   (b) False
3. Flow rates through peripheral 18G cannulae are higher than those through centrally placed 18G cannulae.
   (a) True
   (b) False
4. Hypokalaemia is a commonly encountered cause of traumatic cardiac arrest
   (a) True
   (b) False
5. Patients who present following a hanging must be taken to a major trauma centre as they are highly likely to have a cervical spinal injury.
   (a) True
   (b) False

Answers

1. b
2. a
3. a
4. b
5. b

References

1. Office of National Statistics. Leading causes of death, UK: 2001 to 2018 [Internet]. 2020 [cited 2020 Oct 17]. Available from: https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/causesofdeath/articles/leadingcausesofdeathuk/2001to2018
2. Clinical Audit and Research Unit. Cardiac Arrest Annual Report 2018/19 [Internet]. London Ambulance Service NHS Trust; 2019 [cited 2020 Oct 17]. Available from: https://www.londonambulance.nhs.uk/wp-content/uploads/2020/01/Cardiac-Arrest-Annual-Report-2018-2019.pdf
3. London Ambulance Service NHS Trust - Annual Report & Accounts 2018–19. Annu Rep.:128.
4. Redberg RF, Tucker KJ, Cohen TJ, Dutton JP, Callaham ML, Schiller NB. Physiology of blood flow during cardiopulmonary resuscitation. A transesophageal echocardiographic study. Circulation. 1993;88(2):534–42.
5. Lockey DJ, Lyon RM, Davies GE. Development of a simple algorithm to guide the effective management of traumatic cardiac arrest. Resuscitation. 2013;84(6):738–42.
6. Smith JE, Rickard A, Wise D. Traumatic cardiac arrest. J R Soc Med. 2015;108(1):11–6.
7. Leis CC, Hernández CC, Blanco MJG-O, Paterna PCR, Hernández RE, Torres EC. Traumatic cardiac arrest: should advanced life support be initiated? J Trauma Acute Care Surg. 2013;74(2):634–8.
8. Pickens JJ, Copass MK, Bulger EM. Trauma patients receiving CPR: predictors of survival. J Trauma Injury Infect Crit Care. 2005;58(5):951–8.
9. Sherren PB, Reid C, Habig K, Burns BJ. Algorithm for the resuscitation of traumatic cardiac arrest patients in a physician-staffed helicopter emergency medical service. Crit Care Lond Engl. 2013;17(2):308.
10. Morrison JJ, Poon H, Rasmussen TE, Khan MA, Midwinter MJ, Blackbourne LH, et al. Resuscitative thoracotomy following wartime injury. J Trauma Acute Care Surg. 2013;74(3):825–9.
11. Harris T, Masud S, Lamond A, Abu-Habsa M. Traumatic cardiac arrest: a unique approach. Eur J Emerg Med. 2015;22(2):72–8.
12. Moore EE, Knudson MM, Burlew CC, Inaba K, Dicker RA, Biffl WL, et al. Defining the limits of resuscitative emergency department thoracotomy: a contemporary Western trauma association perspective. J Trauma Injury Infect Crit Care. 2011;70(2):334–9.

13. Hughes CW. Use of an intra-aortic balloon catheter tamponade for controlling intra-abdominal hemorrhage in man. Surgery. 1954;36(1):65–8.
14. Napolitano LM. Resuscitative endovascular balloon occlusion of the aorta: indications, outcomes, and training. Crit Care Clin. 2017;33(1):55–70.
15. Editor. Differential Diagnosis of Infographic: Access Flow Rates [Internet]. Differential Diagnosis of. 2019 [cited 2020 Oct 17]. Available from: https://ddxof.com/infographic-access-flow-rates/
16. Scalea TM, Sinert R, Duncan AO, Rice P, Austin R, Kohl L, et al. Percutaneous central venous access for resuscitation in trauma. Acad Emerg Med Off J Soc Acad Emerg Med. 1994;1(6):525–31.
17. Joy MA, Marciniak D, Rubin KP. Establishing vascular access in the trauma patient. In: Smith CE, Como JJ, editors. Trauma Anesthesia [Internet]. 2nd ed. Cambridge: Cambridge University Press; 2015. [cited 2020 Oct 17]: [79–92 p.]. Available from: https://www.cambridge.org/core/product/identifier/9781139814713%23c03826-5-0/type/book_part.
18. Nagashima G, Kikuchi T, Tsuyuzaki H, Kawano R, Tanaka H, Nemoto H, et al. To reduce catheter-related bloodstream infections: is the subclavian route better than the jugular route for central venous catheterisation? J Infect Chemother Off J Jpn Soc Chemother. 2006;12(6):363–5.
19. Lorente L, Henry C, Martín MM, Jiménez A, Mora ML. Central venous catheter-related infection in a prospective and observational study of 2,595 catheters. Crit Care. 2005;9(6):R631–5.
20. Ruesch S, Walder B, Tramèr MR. Complications of central venous catheters: internal jugular versus subclavian access--a systematic review. Crit Care Med. 2002;30(2):454–60.
21. Merrer J, De Jonghe B, Golliot F, Lefrant JY, Raffy B, Barre E, et al. Complications of femoral and subclavian venous catheterisation in critically ill patients: a randomised controlled trial. JAMA. 2001;286(6):700–7.
22. Tan BK, Hong SW, Huang MH, Lee ST. Anatomic basis of safe percutaneous subclavian venous catheterisation. J Trauma. 2000;48(1):82–6.
23. Holcomb JB, Tilley BC, Baraniuk S, Fox EE, Wade CE, Podbielski JM, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA. 2015;313(5):471.
24. Janelle GM, Shore-Lesserson L, Smith CE, Levy JH, Shander A. What is the PROPPR transfusion strategy in trauma resuscitation? Anesth Analg. 2016;122(4):1216–9.
25. Webster S, Todd S, Redhead J, Wright C. Ionised calcium levels in major trauma patients who received blood in the emergency department. Emerg Med J. 2016;33(8):569–72.
26. Wilson RF, Binkley LE, Sabo FM, Wilson JA, Munkarah MM, Dulchavsky SA, et al. Electrolyte and acid-base changes with massive blood transfusions. Am Surg. 1992;58(9):535–44. discussion 544–545
27. Sihler KC, Napolitano LM. Complications of massive transfusion. Chest. 2010;137(1):209–20.
28. Clevenger B, Kelleher A. Hazards of blood transfusion in adults and children. Contin Educ Anaesth Crit Care Pain. 2014;14(3):112–8.
29. Touma O, Davies M. The prognostic value of end tidal carbon dioxide during cardiac arrest: a systematic review. Resuscitation. 2013;84(11):1470–9.
30. Kolar M, Krizmaric M, Klemen P, Grmec S. Partial pressure of end-tidal carbon dioxide successful predicts cardiopulmonary resuscitation in the field: a prospective observational study. Crit Care Lond Engl. 2008;12(5):R115.
31. Deakin CD, Sado DM, Coats TJ, Davies G. Prehospital end-tidal carbon dioxide concentration and outcome in major trauma. J Trauma. 2004;57(1):65–8.
32. Lee M-C, Wong S-S, Chu J-J, Chang J-P, Lin P-J, Shieh M-J, et al. Traumatic asphyxia. Ann Thorac Surg. 1991;51(1):86–8.
33. Eken C, Yıgıt O. Traumatic asphyxia: a rare syndrome in trauma patients. Int J Emerg Med. 2009;2(4):255–6.
34. Wilson MH, Hinds J, Grier G, Burns B, Carley S, Davies G. Impact brain apnoea – a forgotten cause of cardiovascular collapse in trauma. Resuscitation. 2016;105:52–8.
35. Roberts DJ, Leigh-Smith S, Faris PD, Blackmore C, Ball CG, Robertson HL, et al. Clinical presentation of patients with tension pneumothorax: a systematic review. Ann Surg. 2015;261(6):1068–78.
36. Lockey D, Crewdson K, Davies G. Traumatic cardiac arrest: who are the survivors? Ann Emerg Med. 2006;48(3):240–4.
37. Søreide K, Petrone P, Asensio JA. Emergency thoracotomy in trauma: rationale, risks, and realities. Scand J Surg. 2007;96(1):4–10.
38. Sloan EP, McGill BA, Zalenski R, Tsui P, Chen EH, Duda J, et al. Human immunodeficiency virus and hepatitis B virus Seroprevalence in an urban trauma population. J Trauma Injury Infect Crit Care. 1995;38(5):736–41.
39. Centres for Disease Control and Prevention. CDC Fact Sheet - Today's HIV/AIDS Epidemic [Internet]. US Department of Health and Human Services; 2016 [cited 2020 Oct 17]. Available from: https://www.cdc.gov/nchhstp/newsroom/docs/factsheets/todaysepidemic-508.pdf
40. van den Berg CHSB, Smit C, Bakker M, Geskus RB, Berkhout B, Jurriaans S, et al. Major decline of hepatitis C virus incidence rate over two decades in a cohort of drug users. Eur J Epidemiol. 2007;22(3):183–93.
41. Xeroulis G, Inaba K, Stewart TC, Lannigan R, Gray D, Malthaner R, et al. Human immunodeficiency virus, hepatitis B, and hepatitis C Seroprevalence in a Canadian trauma population. J Trauma Injury Infect Crit Care. 2005;59(1):105–8.
42. Adams S, Stojkovic SG, Leveson SH. Needlestick injuries during surgical procedures: a multidisciplinary online study. Occup Med. 2010;60(2):139–44.
43. Elder A, Paterson C. Sharps injuries in UK health care: a review of injury rates, viral transmission and

potential efficacy of safety devices. Occup Med. 2006;56(8):566–74.
44. US Public Health Service. Updated US Public Health Service Guidelines for the Management of Occupational Exposures to HBV, HCV, and HIV and Recommendations for Postexposure Prophylaxis. MMWR Recomm Rep Morb Mortal Wkly Rep Recomm Rep. 2001;50(RR–11):1–52.
45. Nunn A, Prakash P, Inaba K, Escalante A, Maher Z, Yamaguchi S, et al. Occupational exposure during emergency department thoracotomy: a prospective, multi-institution study. J Trauma Acute Care Surg. 2018;85(1):78–84.
46. Burlew CC, Moore EE, Moore FA, Coimbra R, McIntyre RC, Davis JW, et al. Western trauma association critical decisions in trauma: resuscitative thoracotomy. J Trauma Acute Care Surg. 2012;73(6):1359–63.
47. Henderson VJ, Smith RS, Fry WR, Morabito D, Peskin GW, Barkan H, et al. Cardiac injuries: analysis of an unselected series of 251 cases. J Trauma. 1994;36(3):341–8.
48. Seamon MJ, Shiroff AM, Franco M, Stawicki SP, Molina EJ, Gaughan JP, et al. Emergency department thoracotomy for penetrating injuries of the heart and great vessels: an appraisal of 283 consecutive cases from two urban trauma centers. J Trauma. 2009;67(6):1250–7. discussion 1257–1258
49. Codner PA, Brasel KJ. Emergency department thoracotomy: an update. Curr Trauma Rep. 2015;1(4):212–8.
50. Molina EJ, Gaughan JP, Kulp H, McClurken JB, Goldberg AJ, Seamon MJ. Outcomes after emergency department thoracotomy for penetrating cardiac injuries: a new perspective. Interact Cardiovasc Thorac Surg. 2008;7(5):845–8.
51. Rogerson T, Efstratiades T, Von Oppell U, Davies G, Curtin R. Survival after prehospital emergency clamshell thoracotomy for blunt cardiac rupture. Injury. 2020;51(1):122–3.
52. Wise D, Davies G, Coats T, Lockey D, Hyde J, Good A. Emergency thoracotomy: "how to do it". Emerg Med J. 2005;22(1):22–4.
53. Puchwein P, Sommerauer F, Clement HG, Matzi V, Tesch NP, Hallmann B, et al. Clamshell thoracotomy and open heart massage—a potential life-saving procedure can be taught to emergency physicians. Injury. 2015;46(9):1738–42.
54. DuBose JJ, Morrison J, Moore LJ, Cannon JW, Seamon MJ, Inaba K, et al. Does clamshell thoracotomy better facilitate thoracic life-saving procedures without increased complication compared with an anterolateral approach to resuscitative thoracotomy? Results from the American Association for the Surgery of Trauma aortic occlusion for resuscitation in trauma and acute care surgery registry. J Am Coll Surg. 2020; S107275152032322X
55. Newberry R, Brown D, Mitchell T, Maddry JK, Arana AA, Achay J, et al. Prospective randomised trial of standard left anterolateral thoracotomy versus modified bilateral clamshell thoracotomy performed by emergency physicians. Ann Emerg Med. 2020; S019606442030439X
56. Flaris AN, Simms ER, Prat N, Reynard F, Caillot J-L, Voiglio EJ. Clamshell incision versus left anterolateral thoracotomy. Which one is faster when performing a resuscitative thoracotomy? The tortoise and the hare revisited. World J Surg. 2015;39(5):1306–11.
57. Reid C, Brindley P, Hicks C, Carley S, Richmond C, Lauria M, et al. Zero point survey: a multidisciplinary idea to STEP UP resuscitation effectiveness. Clin Exp Emerg Med. 2018;5(3):139–43.
58. Marsch SCU, Müller C, Marquardt K, Conrad G, Tschan F, Hunziker PR. Human factors affect the quality of cardiopulmonary resuscitation in simulated cardiac arrests. Resuscitation. 2004;60(1):51–6.
59. Link Mark S. Commotio Cordis. Circ Arrhythm Electrophysiol. 2012;5(2):425–32.
60. Chikhani M, Winter R. Injury after non-judicial hanging. Trauma. 2014;16(3):164–73.