Review

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Traumatic cardiac arrest

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Summary

Traumatic cardiac arrest is known to have a poor outcome, and some authors have stated that attempted resuscitation from traumatic cardiac arrest is futile. However, advances in damage control resuscitation and understanding of the differences in pathophysiology of traumatic cardiac arrest compared to medical cardiac arrest have led to unexpected survivors. Recently published data have suggested that outcome from traumatic cardiac arrest is no worse than that for medical causes of cardiac arrest, and in some groups may be better. This review highlights key areas of difference between traumatic cardiac arrest and medical cardiac arrest, and outlines a strategy for the management of patients in traumatic cardiac arrest. Standard Advanced Life Support algorithms should not be used for patients in traumatic cardiac arrest.

Keywords

traumatic cardiac arrest, major trauma

Introduction

Traumatic cardiac arrest is known to have a poor outcome, and some authors have stated that attempted resuscitation from traumatic cardiac arrest is futile. However, advances in damage control resuscitation and understanding of the differences in pathophysiology of traumatic cardiac arrest compared to medical cardiac arrest have led to unexpected survivors.[1] Recently published data have suggested that outcome from traumatic cardiac arrest is no worse than that for medical causes of cardiac arrest, and in some groups may be better.[2] In one military study, 18 of 78 (24%) patients who underwent resuscitation for traumatic cardiac arrest survived.[1]

The initial cardiac rhythm for most patients in survivable traumatic cardiac arrest is pulseless electrical activity (PEA),[3] i.e. the heart is beating but a pulse cannot be felt. traumatic cardiac arrest PEA is often a very low output state, rather than a true cardiac arrest as understood for medical patients with a ventricular fibrillation (VF) arrest (when there is no output at all). When a trauma patient is critically hypovolaemic and has an arterial line *in situ*, it is very apparent that when they lose their palpable pulse (at the onset of traumatic cardiac arrest) they still have an arterial trace and are in a very low output state. Once the conceptual leap has been made to traumatic cardiac arrest being a low output state rather than a true cardiac arrest state, the treatment rationale is easier to understand. This review highlights key areas of difference between traumatic cardiac arrest and medical cardiac arrest and outlines a strategy for the management of patients in traumatic cardiac arrest.

Methods

Our literature review was conducted by searching the Medline database, using the Ovid interface, with the search terms 'traumatic cardiac arrest' and 'traumatic arrest' as keywords. Google Scholar was also searched using the same terms and the first 100 references were scrutinised. A hand search of reference lists of relevant papers was also carried out. We searched guidelines on cardiac arrest management from the UK and European Resuscitation Councils. Most evidence is in the form of observational studies and case series (level II and III), with little in the way of randomised controlled trial evidence to support practice.

Basic life support

The purpose of basic life support is to maintain oxygenation and critical perfusion. While patients with medical (non-traumatic) cardiac arrest are mostly euvolaemic, and often have a primary cardiac cause for their disease process, the pathophysiology of traumatic cardiac arrest is different; the commonest reversible cause is hypovolaemia from blood loss. Whereas the management of VF may be early basic life support and early defibrillation, the management of critical hypovolaemia is urgent and rapid volume replacement. Reversible causes of traumatic cardiac arrest should be sought and

Table 1. Reversible causes of traumatic cardiac arrest.

Cause	Treatment	Intervention
Hypoxia	Oxygenate	Give high flow oxygen
Tension pneumothorax	Decompress chest	Perform thoracostomy
Cardiac tamponade	Decompress tamponade	Perform thoracotomy
Hypovolaemia	Rapid infusion of blood and blood products; damage control resuscitation (including damage control surgery)	Large bore intravenous access (central or peripheral) and transfusion of blood/blood products via rapid infusion device

treated, such as hypoxia treated by oxygenation, tension pneumothorax treated by chest decompression and hypovolaemia treated by rapid infusion of fluid (preferably blood and blood products). The role of basic life support is therefore not as clear as in non-traumatic cardiac arrest.

As a rule of thumb, if basic life support has been commenced, it should be continued until advanced life support skills are available to perform more advanced procedures to address reversible causes (Table 1).

The role of chest compressions

External cardiac chest compressions were first described in 1960 by Kouwenhoven and are the cornerstone of treatment of medical cardiac arrest; indeed there is recent evidence to suggest that compression-only cardiopulmonary resuscitation is as beneficial as combined ventilations and compressions in the pre-hospital environment.[4–7]

However, this evidence is based on patients with non-traumatic medical cardiac arrest. The applicability and transferability of these studies is limited, as they all excluded patients in traumatic cardiac arrest, which as already mentioned has different pathophysiological mechanisms to medical cardiac arrest. The one study that has addressed a traumatic cardiac arrest model used only three baboons and developed a model of cardiac tamponade, followed by hypovolaemia, and eventually pharmacologically induced cardiac arrest, to compare haemodynamic parameters.[8] They found that the improvements in blood pressure with chest compressions in normovolaemic cardiac arrest were not reproduced in hypovolaemic arrest, and indeed there was a further depression of diastolic pressure during chest compressions. This could adversely influence coronary artery perfusion.

The difficulties of undertaking resuscitation of a patient in traumatic cardiac arrest while performing chest compressions should not be underestimated. Resuscitation usually includes intubation, performing bilateral thoracostomies, establishing large bore venous access – often central access via a subclavian vein – instituting rapid infusion of blood and blood products, and obtaining ultrasound and plain X-rays. Performing chest compressions can impair and hinder all of these interventions. It is possible that in patients with some chest injuries, for example multiple bilateral rib fractures, external chest compressions may cause significant harm. Anecdotal reports suggest that compressions may impair flow through rapid infusion devices, slowing transfusion and inhibiting volume replacement.

There is currently no definitive animal or human evidence to support the use of external chest compressions in patients with traumatic cardiac arrest secondary to hypovolaemia, and further work is underway to try to answer this question. External chest compressions should therefore not delay critical interventions in traumatic cardiac arrest (listed in Table 1), unless it is suspected that there is an underlying medical cause for the cardiac arrest.

Use of adrenaline

There is no evidence to support the use of intravenous adrenaline in patients with traumatic cardiac arrest. The use of adrenaline in non-traumatic cardiac arrest has recently been brought into question, with a lack of evidence from randomised controlled trials for either the use of high dose[9] or standard dose adrenaline.[10,11]

The hypovolaemic traumatic cardiac arrest state does not usually occur immediately after the traumatic insult. There is a period of deterioration until output is lost during which time the patient will undergo maximal catecholamine release and vasoconstriction. Giving the patient vasopressors may worsen tissue perfusion. Patients in neurogenic shock are an exception to this rule (having lost their own sympathetic vascular tone).[12,13] There is an increased risk of mortality in adults with blunt trauma when vasopressors are used.[12] One case study suggested that vasopressin may be more beneficial but has not been followed up

with any further published evidence other than animal models.[14,15]

Management of reversible causes

Tension pneumothorax. Traditional teaching is that when a patient exhibits signs and symptoms of a tension pneumothorax, they should undergo needle thoracostomy in the second intercostal space, and in the midclavicular line, to decompress the tension. However, depending on the needle or cannula used, this is often ineffective at relieving the tension, as the needle may not be long enough to reach the pleural space, and it may dislodge or kink, rendering it useless, potentially allowing the tension pneumothorax to recur.[16,17]

Thoracostomy is therefore recommended over needle decompression.[18–20] Thoracostomy is achieved by making a skin incision followed by blunt dissection with forceps or finger through the intercostal muscles and pleura in the same location that an intercostal drain is usually placed (fourth or fifth intercostal space, in the mid-axillary line). The tension pneumothorax is relieved as the pleura is breeched. In patients undergoing positive pressure ventilation, the underlying lung usually inflates and so the insertion of the intercostal drain (through the same hole) can be deferred.[19]

In one series of 37 cases of pre-hospital traumatic cardiac arrest, 18 patients underwent chest decompression, resulting in return of spontaneous circulation (ROSC) in four patients with presumed tension pneumothorax.[18] In a larger series of 909 patients, six had ROSC following decompression of a tension pneumothorax.[2] Another registry study found that from 757 patients with traumatic cardiac arrest, 43 (5.7%) had tension pneumothoraces. On scene chest decompression was recommended for all patients with traumatic cardiac arrest.[21]

It is therefore recommended that patients in traumatic cardiac arrest undergo bilateral thoracostomy, and needle decompression should not be performed if there is someone competent to perform thoracostomies present.

Rapid fluid resuscitation. When patients have critical hypovolaemia, and pulses are no longer palpable, the priority in management once obvious external catastrophic haemorrhage has been controlled (for example, with the use of tourniquets to control exsanguinating limb haemorrhage) is to gain large bore intravenous access and initiate rapid transfusion of blood and blood products. The circulation is empty and needs to be filled.

For patients in traumatic cardiac arrest secondary to hypovolaemia, the most appropriate immediately available fluid in most developed world healthcare systems is packed red cells. A starting volume is typically 4–6 units of O-negative (universal donor) blood. Depending on the patient response to initial rapid infusion the packed red cells may be followed by plasma, platelets and cryoprecipitate (rich in fibrinogen) as well as more packed red cells. Contrary to traditional teaching, resuscitation with large volumes of crystalloid in trauma patients has now been replaced by targeted blood and blood product transfusion.[22,23]

In terms of rapid fluid resuscitation, in one retrospective study, those patients surviving from traumatic cardiac arrest had significantly more fluid resuscitation than those non-survivors (although blood products were not used in this study).[24]

The role of thoracotomy. Emergency thoracotomy, performed either in the pre-hospital environment or in the emergency department, has been directed at one pathological mechanism of traumatic cardiac arrest, namely pericardial tamponade secondary to low energy penetrating chest trauma. This is usually caused by a stab wound to the chest, causing myocardial injury and subsequently accumulation of blood in the pericardial space. In one series, there were eight survivors from 93 pre-hospital thoracotomies performed at scene (8.6% survival rate).[2]

Outcome from ballistic penetrating chest trauma with traumatic cardiac arrest was thought to be universally poor, but a recent retrospective series of patients in traumatic cardiac arrest seen in a military field hospital, with a combination of blast and gunshot wounds, had a 21.5% survival rate following emergency thoracotomy.[3] Patients were more likely to survive if they arrested in the emergency department and underwent thoracotomy there. Most survivors were in PEA (93%) and most (86%) required vascular control of their thoracic aorta. The role of thoracotomy in patients following blunt trauma is less well defined, but there is increasing evidence that thoracotomy may:

1. Provide access to the heart to facilitate closure of simple wounds, e.g. left auricular appendage lacerations
2. Enable vascular control of the thoracic aorta, which can be achieved by simple compression, to reduce bleeding below the diaphragm
3. Provide access to the hemithoraces to enable simple measures to be performed to control bleeding from one lung, e.g. by collapse or twisting of a lung
4. Permit internal cardiac compression to be performed.[25,26]

Ultrasound. The role of ultrasound to diagnose cardiac tamponade as part of the extended focused assessment

with sonography in trauma is well established.[27,28] However, it can also be used to rapidly identify patients with PEA, by detecting cardiac motion in patients with traumatic cardiac arrest that may give an indication of severe hypovolaemia, which may be amenable to rapid transfusion of blood and blood products. This reinforces the concept that some traumatic cardiac arrest states, where there is no palpable pulse, are in fact low output states with an empty circulation rather than true cardiac arrest. Ultrasound may also be useful in identifying pneumothorax and in assessment of volume status by measurement of inferior vena cava diameter.

Use of a traumatic cardiac arrest standard operating procedure (SOP). Many centres use an SOP for the management of patients in traumatic cardiac arrest (example SOP uploaded as supplementary file for online resource). The management of these patients is complex, stressful and uncommon, and therefore prone to error unless a reproducible system is used to facilitate management. The key elements of the resuscitation care bundle included in the SOP are summarised in Box 1. The use of an SOP in these circumstances should be encouraged, along with training and familiarisation with protocols for all members of a trauma team, in order to optimise human factors and team performance.[29]

Box 1. Key elements of the resuscitation care bundle, designed to identify and treat the pathologies causing traumatic cardiac arrest: hypoxia, tension pneumothorax, exsanguination and cardiac tamponade.

Exclude:

- underlying medical cause for cardiac arrest (ALS algorithm applies)
- clinically unsurvivable head injuries
- long downtime without pulse

For all others (usually with PEA as initial rhythm) apply resuscitation bundle:

- no external chest compressions
- no vasopressors
- oxygenate
- bilateral thoracostomies (unless certain of no tension pneumothorax; caution – can be bilateral)
- rapid infusion of warmed blood and products via large catheter and infusion device
- ultrasound the heart
- ultrasound the abdomen
- X-ray chest and pelvis

Summary points

- The management of patients with traumatic cardiac arrest is not always futile
- Reversible causes such as hypoxia, tension pneumothorax, cardiac tamponade and hypovolaemia should be actively sought and excluded or treated
- A SOP for the management of these patients is provided
- Standard Advanced Life Support algorithms should not be used for patients in traumatic cardiac arrest.
- Ongoing research is described in Box 2.

A patient's story (1)

Sergeant Nigel Lithgow Royal Marines was on a vehicle patrol in Helmand Province, Afghanistan in 2008, when he was injured by an improvised explosive device blast. He was brought to the emergency department of the field hospital in Camp Bastion by the medical emergency response team, where he suffered a traumatic cardiac arrest shortly after arrival. He underwent damage control resuscitation including massive transfusion of blood products and damage control surgery to control intraperitoneal haemorrhage from a liver laceration. He was transferred

Box 2. Ongoing research.

- Research is ongoing into the utility of chest compressions in patients in traumatic cardiac arrest.
- Novel therapeutic options such as the use of Resuscitative Endovascular Balloon Occlusion of the Aorta and thoracic aortic compression in patients with hypovolaemic traumatic cardiac arrest are currently being investigated.
- Research is ongoing into emergency preservation resuscitation (formerly known as suspended animation) whereby profound hypothermia to around 10°C is induced rapidly to slow the metabolic rate of all tissues in order to extend the time window to undertake haemostatic surgery. This may be of particular use in patients bleeding from multiple sites.
- The appropriateness of the use of the principles outlined in this paper when dealing with children in traumatic cardiac arrest is currently being explored.

to the Royal Centre for Defence Medicine in Birmingham for ongoing treatment and rehabilitation.

> I have no recollection of the incident or the treatment I received in the field hospital. I was in the ICU [intensive care unit] in Birmingham for about a week in an induced coma. After another week I had to walk the length of the ward in order to prove I could be moved, then after a few more days had to prove I was able to climb stairs before going home.
> At home I needed help to get out of bed and shower, and this would totally wear me out. I spent my time split between the Defence Rehabilitation Centre at Headley Court and home. I would spend my afternoons asleep after exercising and would still sleep at night. I went through a long period of physiotherapy and fitness training, but after approximately 18 months I completed a half marathon and since have trekked to Everest base camp twice. I do still have periods of fatigue but have learnt to manage it. I was discharged from the Royal Marines in 2013.

He now lives with his wife and children in Cornwall, where he works as a part-time tree surgeon.

A patient's story (2)

Stephen Lloyd was 63 years old when he sustained severe crush injuries to his left leg during an incident in 2012 and was transported to the emergency department at Derriford Hospital, Plymouth.

> I was hit by my own car and my leg was crushed between the car and the garage wall. The paramedics arrived and gave me oxygen. That was the last thing I remember before waking up while being moved from the Intensive Care Unit (ICU) to an isolation room.

He suffered a hypovolaemic traumatic cardiac arrest en route to hospital, and after arrival underwent massive transfusion of blood and blood products, followed by vascular surgery to repair the blood supply to the injured limb.

> I returned home after about 8 weeks in hospital. I was able to walk unaided, but it was difficult, and I had a lot of pain at the start. I underwent 10 sessions of physiotherapy over about 10 weeks. My limit is about a quarter of a mile, even after 15 months.

He has now been able to return to a near-normal life, enjoying overseas holidays, and he recently spent a spring day fixing his septic tank.

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References

1. Russell RJ, Hodgetts TJ, McLeod J, Starkey K, Mahoney P, Harrison K, et al. The role of trauma scoring in developing trauma clinical governance in the Defence Medical Services. *Phil Trans R Soc B* 2011; 366: 171–191.
2. Lockey D, Crewdson K and Davies G. Traumatic cardiac arrest: who are the survivors? *Ann Emerg Med* 2006; 48: 240–244.
3. Morrison JJ, Poon H, Rasmussen TE, Khan MA, Midwinter MJ, Blackbourne LH, et al. Resuscitative thoracotomy following wartime injury. *J Trauma* 2013; 74: 825–829.
4. Kouwenhoven WB, Jude JR and Knickerbocker GG. Closed-chest cardiac massage. *JAMA* 1960; 173: 1064–1067.
5. Bohm K, Rosenqvist M, Herlitz J, Hollenberg J and Svensson L. Survival is similar after standard treatment and chest compression only in out-of-hospital bystander cardiopulmonary resuscitation. *Circulation* 2007; 116: 2908–2912.
6. SOS-KANTO Group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study. *Lancet* 2007; 369: 920–926.
7. Iwami T, Kawamura T, Hiraide A, Berg RA, Hayashi Y, Nishiuchi T, et al. Effectiveness of bystander-initiated cardiac-only resuscitation for patients with out-of-hospital cardiac arrest. *Circulation* 2007; 116: 2900–2907.
8. Luna GK, Pavlin EG, Kirkman T, Copass MK and Rice CL. Hemodynamic effects of external cardiac massage in trauma shock. *J Trauma* 1989; 29: 1430–1433.
9. Jacobs IG, Finn JC, Jelinek GA, Oxer HF and Thompson PL. Effect of adrenaline on survival in out-of-hospital cardiac arrest: a randomised double-blind placebo-controlled trial. *Resuscitation* 2011; 82: 1138–1143.
10. Perkins GD, Cottrell P and Gates S. Is adrenaline safe and effective as a treatment for out of hospital cardiac arrest? *BMJ* 2014; 348: g2435.
11. Ong ME, Tan EH, Ng FS, Panchalingham A, Lim SH, Manning PG, et al. Survival outcomes with the introduction of intravenous epinephrine in the management of out-of-hospital cardiac arrest. *Ann Emerg Med* 2007; 50: 635–642.
12. Sperry JL, Minei JP, Frankel HL, West MA, Harbrecht BG, Moore EE, et al. Early use of

vasopressors after injury: caution before constriction. *J Trauma* 2008; 64: 9–14.
13. Ristagno G, Tang W, Huang L, Fymat A, Chang YT, Sun S, et al. Epinephrine reduces cerebral perfusion during cardiopulmonary resuscitation. *Crit Care Med* 2009; 37: 1408–1415.
14. Haas T, Voelckel WG, Wiedermann F, Wenzel V and Lindner KH. Successful resuscitation of a traumatic cardiac arrest victim in hemorrhagic shock with vasopressin: a case report and brief review of the literature. *J Trauma* 2004; 57: 177–179.
15. Voelckel WG, Raedler C, Wenzel V, Lindner KH, Krismer AC, Schmittinger CA, et al. Arginine vasopressin, but not epinephrine, improves survival in uncontrolled hemorrhagic shock after liver trauma in pigs. *Crit Care Med* 2003; 31: 1160–1165.
16. Martin M, Satterly S, Inaba K and Blair K. Does needle thoracostomy provide adequate and effective decompression of tension pneumothorax? *J Trauma* 2012; 73: 1412–1417.
17. Inaba K, Ives C, McClure K, Branco BC, Eckstein M, Shatz D, et al. Radiologic evaluation of alternative sites for needle decompression of tension pneumothorax. *Arch Surg* 2012; 147: 813–818.
18. Mistry N, Bleetman A and Roberts KJ. Chest decompression during the resuscitation of patients in prehospital traumatic cardiac arrest. *Emerg Med J* 2009; 26: 738–740.
19. Deakin CD, Davies G and Wilson A. Simple thoracostomy avoids chest drain insertion in prehospital trauma. *J Trauma* 1995; 39: 373–374.
20. Massarutti D, Trillo G, Berlot G, Tomasini A, Bacer B, D'Orlando L, et al. Simple thoracostomy in prehospital trauma management is safe and effective: a 2-year experience by helicopter emergency medical crews. *Eur J Emerg Med* 2006; 13: 276–280.
21. Huber-Wagner S, Lefering R, Qvick M, Kay MV, Paffrath T, Mutschler W, et al. Outcome in 757 severely injured patients with traumatic cardiorespiratory arrest. *Resuscitation* 2007; 75: 276–285.
22. Miller TE. New evidence in trauma resuscitation – is 1:1:1 the answer? *Perioper Med* 2013; 2: 13.
23. Neal MD, Hoffman MK, Cuschieri J, Minei JP, Maier RV, Harbrecht RG, et al. Crystalloid to packed red blood cell transfusion ratio in the massively transfused patient: when a little goes a long way. *J Trauma* 2012; 72: 892–898.
24. Leis CC, Hernandez CC, Blanco MJ, Paterna PC, Hernandez-de E and Torres EC. Traumatic cardiac arrest: should advanced life support be initiated? *J Trauma* 2013; 74: 634–638.
25. Moore EE, Knudson MM, Burlew CC, Inaba K, Dicker RA, Biffi WL, et al. Defining the limits of resuscitative emergency department thoracotomy: a contemporary Western Trauma Association perspective. *J Trauma* 2011; 70: 334–339.
26. Hunt PA, Greaves I and Owens WA. Emergency thoracotomy in thoracic trauma-a review. *Injury* 2006; 37: 1–19.
27. Cureton EL, Yeung LY, Kwan RO, Miraflor EJ, Sadjadi J, Price DD, et al. The heart of the matter: utility of ultrasound of cardiac activity during traumatic arrest. *J Trauma* 2012; 73: 102–110.
28. Press GM and Miller S. Utility of the cardiac component of FAST in blunt trauma. *J Emerg Med* 2013; 44: 9–16.
29. Manser T. Teamwork and patient safety in dynamic domains of healthcare: a review of the literature. *Acta Anaesthesiol Scand* 2009; 53: 143–151.