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Challenging the dogma of traumatic cardiac arrest management: a military perspective

J E Smith,[1,2] S Le Clerc,[1,3] P A F Hunt[1,3]

▸ Additional material is published online only. To view please visit the journal online (http://dx.doi:10.1136/emermed-2015-204684).

[1]Academic Department of Military Emergency Medicine, Royal Centre for Defence Medicine (Research & Academia), Birmingham, UK
[2]Emergency Department, Derriford Hospital, Plymouth, UK
[3]Emergency Department, James Cook University Hospital, Middlesbrough, UK

Correspondence to Professor Jason E Smith, Academic Department of Military Emergency Medicine, Royal Centre for Defence Medicine, Institute of Research and Development, Vincent Drive, Birmingham B15 2SQ, UK; jasonesmith@nhs.net

Received 23 January 2015
Revised 11 June 2015
Accepted 28 September 2015
Published Online First 22 October 2015

To cite: Smith JE, Le Clerc S, Hunt PAF. *Emerg Med J* 2015;**32**:955–960.

ABSTRACT
Attempts to resuscitate patients in traumatic cardiac arrest (TCA) have, in the past, been viewed as futile. However, reported outcomes from TCA in the past five years, particularly from military series, are improving. The pathophysiology of TCA is different to medical causes of cardiac arrest, and therefore, treatment priorities may also need to be different. This article reviews recent literature describing the pathophysiology of TCA and describes how the military has challenged the assumption that outcome is universally poor in these patients.

INTRODUCTION
Attempts to resuscitate patients in traumatic cardiac arrest (TCA) have, in the past, been viewed as futile. In our experience, patients in TCA are an emotive source of anxiety and stress for resuscitation teams, rivalled only by patients with severe burns, and critically ill and injured children.

Recent military operational experience has resulted in large numbers of patients with TCA being delivered to deployed resuscitation facilities. The frequency and volume of such casualties has led to significant improvements in their management. While some of these evolving management strategies currently lack definitive evidence, the production of such evidence is ongoing. Historically, and despite a paucity of clear evidence to date, it is clear from our experience in the military that a significant proportion of patients deemed to be in TCA by personnel at scene are in fact in a critically low cardiac output state (LCOS) rather than true cardiac arrest. This distinction is extremely important as patients in an LCOS could be salvageable if offered aggressive, timely and appropriate treatment. As recently as 2006, it was thought that patients with TCA secondary to hypovolaemia were not salvageable,[1] but recent military experience has proven this to be overly pessimistic.[2] The experience of clinicians in some civilian trauma systems is also challenging the previously held belief that cardiopulmonary resuscitation (CPR) is a futile intervention in TCA.[3]

This article attempts to share the accumulating evidence on the pathophysiology of TCA, the interventions that can work and those that do not, and the military experience of managing TCA, to enable others to provide a pragmatic and targeted approach in this important group of patients.

METHODS
We searched the MEDLINE database, using the Ovid Interface, using the terms 'traumatic cardiac arrest' and 'traumatic arrest' as keywords. In addition, we used the keywords 'military' and 'combat' to refine the search. Reference lists of relevant papers were hand-searched to identify further evidence. Guidelines on cardiac arrest management from the UK and European Resuscitation Councils were included in the review. Most evidence is in the form of observational studies and case series (levels II and III), which limits the robustness of the evidence supporting some of the observations made in this paper. Some of the evidence comes from military experience, and this should be borne in mind when considering external validity.

Survival following TCA
Survival from TCA has previously been considered extremely poor. The European Resuscitation Guidelines from 2010 included a review of 18 articles with a total of 3032 patients presenting with TCA following blunt trauma, of whom 94 (3.1%) survived, and 1136 patients with TCA following penetrating trauma, of whom 37 (3.3%) survived.[4]

However, more recently, there have been several reports of higher survival rates in various series, particularly from the military. In a prospective study of 52 adult casualties (mean age of 25 years) suffering TCA presenting to a military field hospital in Afghanistan, Tarmey *et al*[5] recorded a rate of return of spontaneous circulation (ROSC) of 27%, with 8% surviving to discharge. Of the survivors, all achieved a good neurological recovery. The principal mechanism of injury was noted to be blast from improvised explosive device with exsanguination as the most common cause of cardiac arrest. In another paper describing military outcomes, Russell *et al*[2] found that 18 of 78 (24%) patients with TCA survived. The remarkable survival in this group was put down to the advanced resuscitation strategies employed by the UK military, and in particular, the ability to effectively stop and treat catastrophic haemorrhage following combat trauma. In another paper looking specifically at 65 patients with TCA who underwent resuscitative thoracotomy, Morrisson *et al*[6] describe ROSC in 33 patients (51%) and survival in 14 patients (21%), with survival in 11 of 26 patients who underwent thoracotomy in the emergency department (ED). A further detailed analysis of all patients with TCA included on the UK Joint Theatre Trauma Registry is underway.

Recent civilian data have also shown an improvement in survival following TCA. Leis *et al*[7] reported an ROSC of 49% in 167 patients with TCA, similar to the rates quoted above. There was an eventual survival rate of 6.6% with intact neurology, which was higher (9.4%) when the responding ambulance included a physician. Another study from Australia of 89 patients with TCA reported a survival rate of 4.5%.[3]

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Novel concepts in TCA management

Patients with TCA have different pathophysiological processes to those suffering cardiac arrest from medical causes. Three algorithms have been described recently, all highlighting the difference between medical and TCA management.[8–10] Patients suffering cardiac arrest due to medical conditions will usually have a full circulating volume, and therefore, the standard elements of resuscitation such as external chest compressions (ECCs) can restore a proportion of normal cardiac output. In contrast, the majority of patients with reversible TCA will have either critical hypovolaemia, tension pneumothorax or cardiac tamponade, the physiology of which cannot be reversed by delivering CPR alone; in the absence of cardiac injury, it is likely that the heart is healthy, but the circulation is the issue that needs to be addressed. The concept of a critically low-flow state is one that has been demonstrated in the military trauma context.[5] Although a central pulse may not palpable, organised cardiac electrical activity (described as pulseless electrical activity (PEA)) may still be present. In these instances, the ventricles demonstrate appropriate hyperdynamic contractility but appear underfilled when viewed with ultrasound.[11] This reversible cause may, therefore, be targeted by aggressive resuscitation.

Distinguishing between cardiac mechanical 'standstill' and a critically LCOS is a key factor when considering the *platinum 10 minutes,* often described as the window when interventions will determine the survivability or otherwise of a trauma patient.[12] According to the European Resuscitation Guidelines, the diagnosis of TCA is a clinical one in trauma victims presenting unresponsive, apnoeic and pulseless, with or without organised (electrocardiographic) cardiac activity. If the concept that LCOS exists is accepted, it becomes extremely difficult to ascertain the start point of true cardiac arrest (although ultrasound can guide this, as described below). The distinction also needs to be made between TCA and commotio cordis,[13] or medical causes of cardiac arrest that may lead to secondary trauma, in which case standard ALS care, including CPR, may be appropriate.[4] The challenge for the trauma team is to rapidly determine which of the trauma victims arriving in suspected TCA still have residual cardiac output with a potentially reversible cause, and therefore, could benefit from aggressive resuscitation.

One potential method is to visualise the rate and quality of myocardial contractility and cardiac filling using ultrasound. In military trauma teams, a consultant radiologist will usually be available to provide this, although all senior emergency physicians in the UK are now trained in the ultrasound skills necessary to assess cardiac motion, detect free fluid in the abdomen, pericardial effusion and pneumothoraces.

Irreversible causes of TCA

Typically, the causes of TCA are divided into those deemed potentially reversible and those that are not. Examples of cases where patients are identified by the Joint Royal Colleges Ambulance Liaison Committee (JRCALC) as irreversible and where resuscitation should not be attempted include massive cranial and cerebral destruction; hemicorporectomy; massive truncal injury incompatible with life, including decapitation; decomposition or putrefaction; incineration; hypostatis (blood pooling) and rigor mortis.

Additionally, according to guidance from the JRCALC, resuscitation is deemed futile when there has been asystole >15 min without bystander CPR or submersion for >1 h in adults, or 1.5 h for children.[14]

In practice, some patients who present with TCA have devastating neurological injury. In this group of patients, no intervention will reverse the primary neurological injury, and therefore, TCA in this group is also irreversible.

Reversible causes of TCA

Some causes of cardiac arrest are potentially reversible as described in traditional resuscitation guidelines as the 'the 4Hs and 4Ts'.[15] Those specific to TCA are hypoxia, hypovolaemia, tension pneumothorax and cardiac tamponade. These pathological processes are potentially amenable to aggressive early treatment.

Control of haemorrhage and fluid resuscitation

Given the likelihood of a LCOS state in TCA, initial resuscitation measures should focus on aggressive control of haemorrhage, and addressing hypovolaemia and hypoxia. Although a detailed review of these measures is beyond the scope of this paper, current standard of care includes pressure dressings, novel haemostatic agents and/or tourniquets to control bleeding. Mitigation of non-compressible haemorrhage can be initiated with pelvic binder placement for blunt trauma patients where suspicion of injury is high, and long bone traction for identified fractures. Hypovolaemia is addressed with rapid volume replacement using blood products in order to maximise oxygen-carrying capacity, circulating volume and treatment of trauma-induced coagulopathy. The use of crystalloids can exacerbate traumatic coagulopathy and should be avoided unless there is no alternative and emergency transport is prolonged.[4]

Obtaining large vessel circulatory access is paramount in order to facilitate rapid infusion of blood products. However, if there is a delay in obtaining venous access, rapid infusion can still be achieved through an intraosseous needle.[16] As resistance to flow is higher, an appropriate infusion system is required to deliver blood products; this could involve a three-way tap and 50 mL syringe in the circuit with which to draw and infuse through the intraosseous needle system. Although concern regarding the potential for haemolysis has been raised,[17] there is no scientific evidence to suggest this is a limitation.[18]

Military practice includes routine near-patient testing for haemoglobin, $[Ca^{2+}]$, blood gas analysis and rotational thomboelastometry (ROTEM) as a guide to administration of blood products including PRC, FFP, platelets and cryoprecipitate in a bespoke, targeted fashion.[19] This approach has been supported by evidence from a civilian study of patients requiring more than five units of red blood cells in a 24 h period.[20] However, a recent Cochrane systematic review failed to find convincing evidence in support of the use of ROTEM for trauma-induced coagulopathy and called for further research before it is instituted into routine clinical practice.[21]

Airway

As in medical cardiac arrest, intubation improves oxygen delivery to the tissues and should be performed early. There is some animal evidence that permissive hypoventilation can optimise venous return.[22]

Drugs in TCA

It is currently accepted practice that during the management of medical cardiac arrest intravenous adrenaline is administered in accordance with ALS guidelines. It is believed that this supports an increase in venous return by increasing systemic vascular resistance, and consequently, improves cardiac output; however, there is limited evidence for the efficacy of any drugs during

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cardiac arrests of medical cause. A recent randomised control trial has questioned the role of adrenaline; although associated with increased incidence of ROSC, there was no improvement in survival to hospital discharge.[23] In the TCA patient, adrenaline may adversely affect cerebral microvascular flow, worsening cerebral ischaemia in an already severely acidaemic and maximally vasoconstricted victim. The role of other vasopressors in TCA is unclear, with a limited evidence base involving mainly case reports. However, results from a prospective cohort study of patients in severe haemorrhagic shock suggest that early vasopressors may worsen outcome and result in an 80% higher risk of mortality.[24]

Drugs that may be indicated in TCA include tranexamic acid for those with life-threatening haemorrhage[25] and administration of parenteral calcium to offset the effects of citrate in stored blood for those receiving massive transfusion.[26 27]

The role of resuscitative thoracotomy

A meta-analysis of 42 outcome studies including 7035 emergency department thoracotomies (EDTs) demonstrated an overall survival rate of 7.8%, of which 15% survived with some degree of neurological deficit.[28] The outcome following EDT in blunt trauma victims with witnessed TCA is poor, with an estimated survival rate of 1.6%. The survival rate is highest, estimated as 31%, following penetrating cardiac injuries with a short on-scene and transport time, witnessed signs of life or ECG activity.[28]

However, resuscitative thoracotomy should not be seen as purely a last-ditch resort in those who are destined to die[6] and should be *considered* in all those presenting in TCA. Similarly, the time-critical 10 min window from point of witnessed cardiac arrest to thoracotomy[29] is likely to be challenged when current evidence is reviewed and the concept of LCOS gathers wider acceptance.

In the military population, the role of resuscitative thoracotomy was questioned in one early series,[30] but there are later descriptions of successful resuscitation following thoracotomy by the same authors,[5 6] who conclude that resuscitative thoracotomy following combat injury will yield survivors. Best outcomes are in patients who arrest in the ED or on admission to the hospital.[6]

As a body cavity access technique, the practice of thoracotomy is arguably relatively straightforward and can be performed with commonly available, non-specialist equipment in any clinical setting as demonstrated by numerous prehospital helicopter emergency medical services (HEMS) systems operating around the world. However, the key to successful resuscitation is in the subsequent identification and control of bleeding, where possible. As part of a protocolised TCA approach, existing incisions for bilateral thoracostomies can be extended anteriorly to the midline and slightly posteriorly in a clamshell approach.[31] This technique allows clinicians to gain access to the pericardium, myocardium, lungs and great vessels. The primary indication for EDT in the management of TCA is to relieve cardiac tamponade, but it will also facilitate interventions aimed at controlling bleeding from the heart, lungs or great vessels. This can be achieved in a variety of methods including direct pressure, haemostatic compounds, sutures or surgical clips. Thoracotomy also enables cross-clamping of the descending aorta or simply direct pressure applied posteriorly against the vertebrae in order to control ongoing haemorrhage below the diaphragm.

Resuscitative endovascular balloon occlusion of the aorta achieves a similar mechanical effect to aortic cross clamping and is under investigation as a potential option in the management of patients with TCA.[32]

Tension pneumothorax

Ongoing intrapleural accumulation of air from a bronchopleural leak, open pneumothorax or injudicious positive airway pressure will lead to a rise in intrathoracic pressure. If untreated, this will eventually lead to mediastinal shift and reduced venous return, resulting in obstructive shock and subsequent cardiac arrest. Spontaneously breathing patients may tolerate tension pneumothorax for some time before becoming critically compromised by the lack of venous return, leading to a sudden loss of cardiac output. Unlike medical cardiac arrest, chest compressions are unlikely to be of benefit and efforts should instead be focused on proactive decompression of the tension pneumothorax by bilateral open thoracostomies.[33–35] Conventional needle thoracocentesis techniques are prone to failure due to mechanical obstruction or kinking, not reaching the pleural space effectively or causing iatrogenic injury.[36 37] Open thoracostomies are effective in allowing complete lung re-expansion and much quicker to perform than tube thoracostomies.

Cardiac tamponade

Cardiac tamponade is more common in penetrating trauma, particularly secondary to knife-related cardiac wounds, although it can occur following blunt trauma. Compression of the heart by blood building up within the pericardial sac leads to a rise in diastolic pressure, impairing cardiac filling with a resultant fall in stroke volume and consequently cardiac output. The use of ECCs in this group of patients will be ineffective as the underlying physiological process is obstructive, and attempts to mechanically compress the heart will fail to restore stroke volume or cardiac output. There is no evidence of benefit from needle pericardiocentesis, and it may cause myocardial injury and delay other more effective therapeutic measures.[4] The treatment of choice is immediate surgical release of tamponade via thoracotomy.

External chest compressions: a waste of energy?

It is well recognised that early and effective ECCs with minimal interruptions improve outcomes in patients suffering medical cardiac arrest. ECCs are less likely to be effective in hypovolaemic TCA compared with cardiac arrest from other causes, although the only evidence to guide practice is from a small animal study, which showed no haemodynamic benefit in performing ECC.[38]

In patients with normovolaemic cardiac arrest, ECC has been shown to deliver around one-third of normal blood flow to the brain, although this relies on adequate venous return.[39] In a critically hypovolaemic patient, it will be much lower. The authors, therefore, recommend withholding ECC in a patient with clear signs of hypovolaemic TCA, certainly while more urgent interventions are carried out, and until adequate circulating volume has been restored. However, due to the complex nature of severe injuries and often multiple factors leading to TCA, compressions should not be considered as a futile measure in all cases.[3 4]

Ultrasound

Emergency ultrasonography is a proven investigation in trauma resuscitation and is a sensitive test for pericardial tamponade.[40] Other important findings, such as haemoperitoneum and haemothorax or pneumothorax, can also be diagnosed reliably with ultrasound, and it has been shown to reduce time to definitive

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surgery in trauma patients.[41] As a portable bedside technique, it can be integrated unobtrusively into TCA protocols. Prehospital ultrasound includes simple thoracic ultrasound and focused assessment with sonography in trauma.[10] Two of the authors (PAFH and SLC) have practical experience of using prehospital ultrasound successfully and safely in both military and civilian helicopter-based primary retrieval systems, with good image quality and utility for assessment; aiding interventions such as central venous access and regional anaesthesia.

The military approach

The military approach begins with the training of resuscitation teams (box 1). Military resuscitation teams discuss TCA in detail and rehearse decision-making, team-working and individual skills in a simulated environment well in advance of providing trauma care on operations. Criteria for patients in whom we will perform chest compressions or administer adrenaline to (it is possible that neither may be indicated in TCA) are agreed in advance by consensus within clear, defined operating procedures. From a human factors perspective, the team is, therefore, able to function effectively with a shared understanding of key objectives, enabling safe, rapid and decisive treatment within the first few seconds and minutes of the patient's arrival. This is developed during individual and team training in a simulated environment at a whole hospital exercise[42] and the Military Operational Surgical Training course.[43]

There are few differences between military and civilian trauma teams, but some key features should be highlighted. In addition to an airway clinician and assistant, the team includes another senior clinician standing by in order to obtain rapid central venous access; the preferred site being the right subclavian vein using a large bore (usually 8.5F gauge) trauma line. Other personnel are specifically tasked to manage the rapid infusion devices (one device on each side of the patient) to facilitate rapid transfusion of blood products. Within the military system, pre-alerts are reliably passed from prehospital medical platforms to the receiving facility so that units of packed red blood cells and thawed plasma are prepared, checked and ready for immediate transfusion. The rapid infusion devices are run through with crystalloid solution so that they are ready to be used. A consultant radiologist is part of the trauma team and provides rapid diagnostic information with ultrasound, offering the benefit of early visualisation of the heart in order to guide subsequent targeted treatment if required, such as the release of cardiac tamponade at thoracotomy.

Box 1 The military approach

1. Protocols: traumatic cardiac arrest (TCA) protocols are developed to create shared understanding of approaches.
2. Teams: teams rehearse regularly, focusing on trauma team management in a 'real-life' resuscitation setting with full multidisciplinary involvement, including senior representation from:
   - Emergency department personnel
   - Anaesthesia and critical care
   - Surgical teams with general, orthopaedic, cardiothoracic and vascular capability
   - Radiology
3. Initial management: <C>ABC (listed in order but may be performed simultaneously):
   - Control of external haemorrhage
   - Tracheal intubation and ventilation
   - Bilateral open thoracostomies
   - Application of a pelvic binder if appropriate mechanism of injury
   - Rapid blood product administration: (pRBC+FFP in a 1:1 ratio)
   - Consideration of resuscitative emergency department thoracotomy
4. Further measures:
   - Tranexamic acid 1 g (15 mg/kg) intravenous/intraosseous over 15 min should be administered within 3 h of injury to all patients with TCA secondary to blood loss, followed by an infusion of 1 g over 8 h.
   - Calcium replacement should be considered during massive blood transfusion, with monitoring of [Ca^{2+}] levels.
   - Targeted blood product administration should be guided by rapid coagulation testing such as rotational thomboelastometry as soon as possible after return of spontaneous circulation.
   - External chest compressions in TCA are not attempted until underlying causes have been appropriately addressed, including hypoxia, hypovolaemia, tension pneumothorax and cardiac tamponade.

Post-resuscitation care

Once ROSC has been achieved, access to definitive surgical haemorrhage control is key to optimising outcome. Military experience has emphasised the benefit of having a functional operating theatre adjacent to the resuscitation room. However, physical proximity is only part of the solution—the mindset also needs to be right. Trauma team leaders should recognise when to transfer the patient rapidly to an operating theatre or even to move directly there on arrival—a process termed *right turn resuscitation* in Camp Bastion due to the layout of the resuscitation room.[44] Surgical control of life-threatening haemorrhage is a key component of damage control resuscitation, and the operating theatre should, therefore, be seen as an extension of the resuscitation room in cases of TCA. Whole-body CT scanning should be considered for diagnostic purposes following ROSC, if the patient is stable enough, in order to guide further emergency surgical or radiological intervention and to identify other important or time-critical injuries such as intracranial or spinal injury.

A formal secondary survey should be delayed until the patient is stable enough to allow a more comprehensive clinical examination. However, any other important or significant injuries should be documented clearly for further management as soon as possible.

When to stop resuscitation

Cessation of resuscitation is an emotive subject and represents one of the most difficult decisions to be made in the management of trauma victims. In many cases, the obvious severity of the injuries, the nature of the presentation, comorbidities or lack of physiological reserve allow the decision to be made confidently and appropriately. However, there are also a number of features that may suggest futility in continuing resuscitation for TCA. Guidelines have been published regarding the termination or withholding of out-of-hospital resuscitation for TCA cases,[45] although the general adoption of these remains controversial.

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In a recent military case series of 52 patients, the longest duration of TCA associated with survival was 24 min. All of the survivors demonstrated PEA rhythms during arrest, whereas asystole was universally associated with death. Also, 6 out of 24 of the patients had ultrasound evidence of cardiac activity during arrest; all six with cardiac activity subsequently exhibited ROSC and two survived to hospital discharge.[5] Existing civilian guidelines suggest that the maximum CPR time associated with favourable outcome following TCA is 16 min.[4]

Limitations of this review

It should be highlighted that the patient population, mechanism of injury and clinical trauma teams are different in a military environment. The increasing proportion of elderly major trauma in the UK limits the applicability of some of these recommendations to the UK trauma population,[42] and the end-to-end trauma care that military personnel benefit from on deployed operations[46] is not replicated in a civilian environment.

It should be emphasised that some of the recommendations from this review are, by necessity, experience and opinion based, rather than evidence based.

SUMMARY

The underlying pathophysiology, and therefore, clinical management of patients with TCA, is different to that of patients with medical cardiac arrest, and resuscitative efforts should be directed at providing rapid and proactive interventions designed to treat the potential reversible causes. ECCs should not delay, or interfere with, these interventions. The use of a protocol can ensure rapid, consistent and structured delivery of evidence-based clinical care for the relatively small number of critically injured patients arriving in hospital with TCA. An example of a TCA resuscitation protocol is available as an online resource (supplementary file one).

Contributors JS and SLC initiated the review. All authors contributed to the review of the literature and the development of the manuscript. All authors have approved the final version of the manuscript.

Competing interests All authors are serving members of HM Armed Forces.

Provenance and peer review Not commissioned; externally peer reviewed.

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J E Smith, S Le Clerc and P A F Hunt

Emerg Med J 2015 32: 955-960 originally published online October 22, 2015
doi: 10.1136/emermed-2015-204684

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