CRITICAL CARE

Algorithm for the resuscitation of traumatic cardiac arrest patients in a physician-staffed helicopter emergency medical service

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Abstract

Survival rates following traumatic cardiac arrest (TCA) are known to be poor but resuscitation is not universally futile. There are a number of potentially reversible causes to TCA and a well-defined group of survivors. There are distinct differences in the pathophysiology between medical cardiac arrests and TCA. The authors present some of the key differences and evidence related to resuscitation in TCA, and suggest a separate algorithm for the management of out-of-hospital TCA attended by a highly trained physician and paramedic team.

Introduction

Survival rates following traumatic cardiac arrest (TCA) are known to be poor from both civilian and military data [1,2]. However, the common misconception that resuscitation is futile for all patients is not well founded. Lockey and colleagues demonstrated that a well-trained physician and paramedic team attending out-of-hospital TCAs could achieve a 7.5% survival rate to hospital discharge, comparable with out-of-hospital cardiac arrest from any cause [1]. Survival rates are highly variable depending on the aetiology, and traumatic pathologies associated with an improved chance of successful resuscitation include hypoxia, tension pneumothorax and cardiac tamponade [1-3]. TCA is a unique disease in which clinicians are frequently confronted by a healthy heart that has arrested as a result of hypoxia, haemorrhage or obstructive shock [1-3]. Consequently, there are different management priorities in TCA and potential deviations from existing non-TCA algorithms. The

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authors developed an algorithm (Figure 1) to address these issues based on their own current practice and available published evidence.

Rationale for the traumatic cardiac arrest algorithm

Effective oxygenation is pivotal to good trauma care given that hypoxia has significant implications for morbidity and mortality [1,4]. Up to 44% of TCA survivors arrest primarily as a result of asphyxia, and consequently correcting hypoxia is essential [1]. There is much controversy over optimal airway management in the prehospital trauma patient, with a paucity of evidence for the benefit of advanced airway interventions over basic interventions [5]. The placement of a cuffed tracheal tube allows the delivery of high functional inspired oxygen concentrations, optimal positive pressure ventilation, prevention of gastric insufflation and protection against tracheal aspiration. Prehospital drug-facilitated tracheal intubation in a physician-led service has been shown to have a high success rate of between 98.8 and 100%, with minimal complications [6-8]. If tracheal intubation is to be used during TCA the tube should be placed by experienced clinicians, with appropriate manual inline stabilisation and mandatory confirmation by waveform capnography.
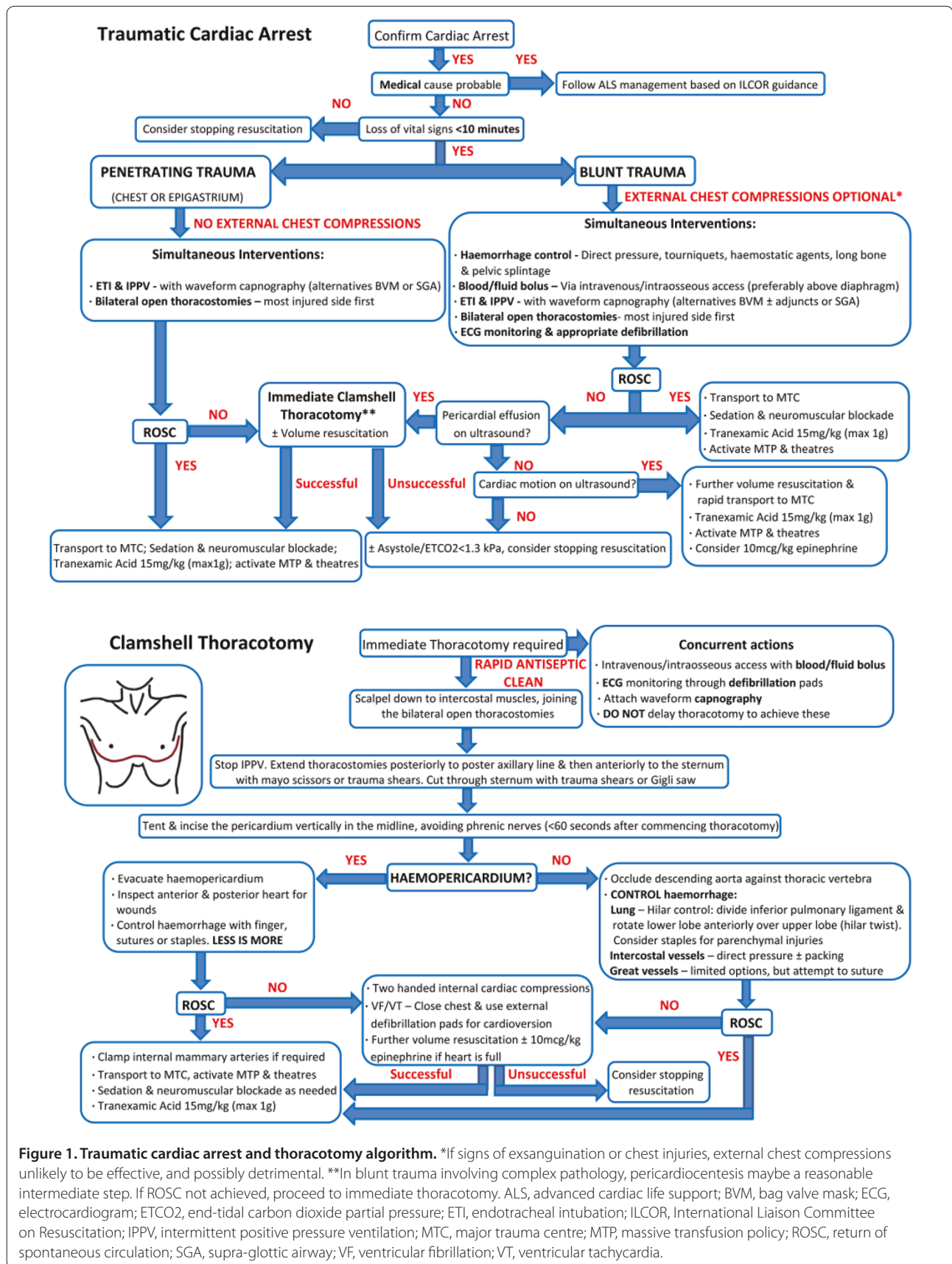
Once patients are intubated, attempts should be made to achieve adequate oxygenation while minimising minute ventilation and intrathoracic pressures. This avoids further reductions in venous return in patients with a shocked or arrested circulation [9]. There is much controversy over the impact of hyperoxia on ischaemic–reperfusion injury and mortality in traumatic brain injury and cardiac arrest survivors [10-12]. If return of spontaneous circulation (ROSC) is achieved, it would seem prudent to titrate the functional inspired oxygen concentration and ventilation to achieve an arterial oxygen saturation of 94 to 98% and normocapnia [4].

Proactive exclusion of tension pneumothoraces is an essential component of ensuring good oxygenation in TCA. Commonly taught needle thoracostomy decompression can potentially fail due to mechanical

Traumatic Cardiac Arrest

Confirm Cardiac Arrest

YES → **YES** → Follow ALS management based on ILCOR guidance

Medical cause probable

NO → **NO**

Consider stopping resuscitation ← Loss of vital signs **<10 minutes**

YES

PENETRATING TRAUMA
(CHEST OR EPIGASTRIUM)

BLUNT TRAUMA

EXTERNAL CHEST COMPRESSIONS OPTIONAL*

NO EXTERNAL CHEST COMPRESSIONS

Simultaneous Interventions:

· **ETI & IPPV** - with waveform capnography (alternatives BVM or SGA)
· **Bilateral open thoracostomies** – most injured side first

Simultaneous Interventions:

· **Haemorrhage control** - Direct pressure, tourniquets, haemostatic agents, long bone & pelvic splintage
· **Blood/fluid bolus** – Via intravenous/intraosseous access (preferably above diaphragm)
· **ETI & IPPV** - with waveform capnography (alternatives BVM ± adjuncts or SGA)
· **Bilateral open thoracostomies** - most injured side first
· **ECG monitoring & appropriate defibrillation**

ROSC

NO → **YES** → · Transport to MTC
· Sedation & neuromuscular blockade
· Tranexamic Acid 15mg/kg (max 1g)
· Activate MTP & theatres

Immediate Clamshell Thoracotomy* ** ± Volume resuscitation

YES ← Pericardial effusion on ultrasound?

ROSC **NO**

YES

Successful **Unsuccessful** **NO** → · Further volume resuscitation & rapid transport to MTC

Cardiac motion on ultrasound? **YES** → · Tranexamic Acid 15mg/kg (max 1g)
· Activate MTP & theatres
· Consider 10mcg/kg epinephrine

NO

Transport to MTC; Sedation & neuromuscular blockade; Tranexamic Acid 15mg/kg (max1g); activate MTP & theatres

± Asystole/ETCO2<1.3 kPa, consider stopping resuscitation

Clamshell Thoracotomy

Immediate Thoracotomy required

RAPID ANTISEPTIC CLEAN

Scalpel down to intercostal muscles, joining the bilateral open thoracostomies

Concurrent actions

· Intravenous/intraosseous access with **blood/fluid bolus**
· **ECG** monitoring through **defibrillation** pads
· Attach waveform **capnography**
· **DO NOT** delay thoracotomy to achieve these

Stop IPPV. Extend thoracostomies posteriorly to poster axillary line & then anteriorly to the sternum with mayo scissors or trauma shears. Cut through sternum with trauma shears or Gigli saw

Tent & incise the pericardium vertically in the midline, avoiding phrenic nerves (<60 seconds after commencing thoracotomy)

YES **HAEMOPERICARDIUM?** **NO**

· Evacuate haemopericardium
· Inspect anterior & posterior heart for wounds
· Control haemorrhage with finger, sutures or staples. **LESS IS MORE**

· Occlude descending aorta against thoracic vertebra
· **CONTROL haemorrhage:**
  Lung – Hilar control: divide inferior pulmonary ligament & rotate lower lobe anteriorly over upper lobe (hilar twist). Consider staples for parenchymal injuries
  Intercostal vessels – direct pressure ± packing
  Great vessels – limited options, but attempt to suture

ROSC **NO** → · Two handed internal cardiac compressions
· VF/VT – Close chest & use external defibrillation pads for cardioversion
· Further volume resuscitation ± 10mcg/kg epinephrine if heart is full

NO **ROSC**

YES

· Clamp internal mammary arteries if required
· Transport to MTC, activate MTP & theatres
· Sedation & neuromuscular blockade as needed
· Tranexamic Acid 15mg/kg (max 1g)

Successful **Unsuccessful** Consider stopping resuscitation **YES**

Figure 1. Traumatic cardiac arrest and thoracotomy algorithm. *If signs of exsanguination or chest injuries, external chest compressions unlikely to be effective, and possibly detrimental. **In blunt trauma involving complex pathology, pericardiocentesis maybe a reasonable intermediate step. If ROSC not achieved, proceed to immediate thoracotomy. ALS, advanced cardiac life support; BVM, bag valve mask; ECG, electrocardiogram; ETCO2, end-tidal carbon dioxide partial pressure; ETI, endotracheal intubation; ILCOR, International Liaison Committee on Resuscitation; IPPV, intermittent positive pressure ventilation; MTC, major trauma centre; MTP, massive transfusion policy; ROSC, return of spontaneous circulation; SGA, supra-glottic airway; VF, ventricular fibrillation; VT, ventricular tachycardia.

obstruction or kinking, can cause iatrogenic injury, can be incorrectly sited in 40% of patients and is expected to fail in 42.5% of patients due to the chest wall thickness [13-16]. Conversely, tube thoracostomy has been shown to be more effective at evacuating tension pneumothoraces during cardiac arrest [13], and prehospital placement is a predictor of survival in TCA (odds ratio = 0.3, 95% confidence interval = 0.13 to 0.8) [17]. The survival benefit of tube thoracostomy must be balanced against the notable risk of tube malposition in both the hospital and prehospital setting [18,19]. Prehospital open thoracostomy avoids the risk of tube malposition, and has been shown to be rapid, safe and effective during positive pressure ventilation [20-22]. An open thoracostomy also allows for complete lung re-expansion and easy thoracic reassessment. Cuffed tracheal tube insertion into the open thoracostomy should be considered when there is difficulty in maintaining patency due to excessive soft tissues [23]. The authors' opinion is that all TCA patients with chest injuries should have bilateral open thoracostomies to proactively exclude tension pneumothoraces.

Uncontrolled haemorrhage is the commonest cause of preventable death in trauma [24], and survival from exsanguination and true TCA is very rare [1,25]. The early control of catastrophic external haemorrhage with pressure dressings, haemostatic agents or tourniquets, combined with the traditional primary survey and damage control resuscitation, is rapidly becoming a standard of care [25,26]. In TCA, early control of catastrophic external haemorrhage should be accompanied by long bone splintage and application of a pelvic binder for blunt trauma patients.

Damage control resuscitation is a package of care that is composed of damage control surgery, haemostatic resuscitation and permissive hypotension [25]. Haemostatic resuscitation is the provision of balanced volume replacement in the haemorrhaging patient that targets trauma-induced coagulopathy [25]. This involves the early transfusion of packed red blood cells, fresh frozen plasma and platelets accompanied by tranexamic acid, while limiting the use of crystalloids and vasopressors [25,27]. The ideal ratio of fresh frozen plasma to packed red blood cells remains contentious; a ratio of 1:1 or greater may not confer additional benefit over ratios of 1:2 to 3:4 [28].

Prehospital blood transfusion has been infrequently described [29], and concerns regarding expense, transfusion reactions and difficult storage have limited the interest of prehospital providers. The authors recently reviewed 5 years of prehospital blood transfusions carried out by their own service. A total of 382 units of packed red blood cells were transfused to 147 trauma patients, with no documented transfusion reactions and acceptable wastage (presently unpublished data). The

authors contend that blood is currently the resuscitation fluid of choice in TCA patients and should be utilised pre hospital if available.

Volume resuscitation in TCA should ideally be achieved through large-bore peripheral intravenous access above the diaphragm. This allows desirable flow characteristics, low complication rates and reduced risk of extravasation through injured pelvic or abdominal vessels. However, intraosseous access has been shown to have a better first-pass success rate and time to vascular access in out-of-hospital cardiac arrest [30]. If intravenous access is not immediately available in TCA, the authors' current practice is to obtain proximal humerus intraosseous access given the desirable flow rates over other sites below the diaphragm [31].

Early use of aggressive haemorrhage control, haemostatic resuscitation and damage control surgery in TCA may produce a number of unexpected survivors [2]. The British military have demonstrated that 75% of their TCA survivors had arrested as a result of haemorrhage [2]. This patient group demonstrates the importance of differentiating patients with true mechanical cardiac standstill (classical pulseless electrical activity or asystole) from those with cardiac motion but impalpable pulses due to profound hypotension (pseudo-pulseless electrical activity). The overwhelming majority of survivors present organised electrical activity on electrocardiogram or cardiac motion on ultrasound, and belong to the latter group [2,32,33].

Prehospital ultrasound has a multitude of applications in the unstable trauma patient, extending from simple thoracic ultrasound through to an extended focused assessment with sonography in trauma [34-36]. In TCA, the use of ultrasound to assist in the assessment of cardiac tamponade [37], pulse presence [37,38] and cardiac motion [2,32,33,37] forms an integral part of the authors' current practice.

Cardiac tamponade is a very real concern in penetrating trauma, but can also be seen in a small number of blunt trauma patients following cardiac rupture [39]. Physician-performed on-scene thoracotomy following penetrating TCA has been shown to have a survival rate to hospital discharge of 18% [3]. The primary pathology for all survivors in this series was cardiac tamponade secondary to knife-related cardiac wounds [3]. Such survival rates are similar to published data for in-hospital emergency department thoracotomy [40-42]. This dramatic intervention requires adequate training and clinical governance to ensure appropriate patient selection and optimal outcomes [43]. Given that the majority of physicians undertaking this procedure in the prehospital arena will be nonsurgeons, a simplified and standardised approach is required to maximise the chances of success, as described by Wise and colleagues

[43]. As a result of this limited surgical experience and the high prevalence of cardiac tamponade in prehospital TCA survivors [3,43], the primary aim of the developed thoracotomy algorithm was to address this single pathology (Figure 1). Although the management of other thoracotomy findings are discussed, operators should be aware of the dismal prognosis in this group [3,41-43].

End-tidal carbon dioxide partial pressure ($ETCO_2$) measurement has shown much promise in non-TCAs for confirming tracheal intubation, adequacy of chest compressions, ROSC and prognostication [44-46]. The difficulty comes when interpreting $ETCO_2$ in cardiac arrests because it can be affected by the type of airway *in situ*, the presence of good quality chest compressions and the initial rhythm, cause and duration of cardiac arrest [46]. One of the most established and promising applications of $ETCO_2$ is as a predictor of survival in non-TCAs [44-46]. What has proven more challenging has been defining a cutoff point for determining futility, and when it should be applied following cardiac arrest.

In one of the largest studies to date, Kolar and colleagues suggested that $ETCO_2$ ≤1.9 kPa (14.3 mmHg) at 20 minutes in non-TCAs was able to predict ROSC with a sensitivity, specificity, positive predictive value and negative predictive value all of 100% [44]. There are obvious limitations when trying to extrapolate these findings to TCA patients for a number of reasons. The aetiologies clearly vary, and 20 minutes might be considered by some to be an excessive duration of resuscitation for TCA. Eckstein and colleagues showed that an initial $ETCO_2$ >1.3 kPa (10 mmHg) and the absence of a fall >25% from the baseline were significantly associated with ROSC in out-of-hospital cardiac arrest [45].

No single $ETCO_2$ value can be used in isolation in TCA. The authors feel that, following aggressive resuscitation, the combination of a downtime >10 minutes, no organised electrical activity on the electrocardiogram, a lack of cardiac motion on ultrasound and $ETCO_2$ <1.3 kPa (10 mmHg) would suggest futility. Caution should be exercised when extrapolating the use of $ETCO_2$ in TCA given the paucity of evidence in this setting and its reliance on good-quality chest compressions.

A gravid patient in TCA, known to be greater than 20 weeks gestation or with a fundal height higher than the umbilicus, may require a perimortem caesarean delivery (PMCD). PMCD will not only potentially result in a viable neonate, but is also vital to optimise maternal haemodynamics and outcomes [47]. PMCD should ideally be initiated within 4 minutes of TCA to improve maternal and neonatal outcomes; however, neonatal survival is still likely if delivery occurs within 10 to 15 minutes [47]. Prehospital PMCD has been undertaken by our service previously [48], and the authors feel it is an essential skill for prehospital physician–paramedic teams.

Along with these subtle additions to managing TCA patients there are potentially a number of omissions from advanced cardiac life-support management that should be considered. The evidence for the benefit of external chest compressions in TCA is sparse. External chest compressions can deliver approximately one-third of the normal blood flow to the brain in non-TCAs and rely on adequate venous return [49]. In TCA the presence of hypovolaemia, tension pneumothorax or cardiac tamponade will all limit venous return, left ventricular end-diastolic volume and chest compression effectiveness [50]. External chest compressions may also delay or increase the procedural risk of definitive interventions for reversible causes, such as thoracostomy. Furthermore, in the presence of significant thoracic trauma, chest compressions could theoretically exacerbate underlying parenchymal injuries. Given these concerns, consideration should be given to omitting external chest compressions in TCA, or delaying them until preload and obstructive causes have been addressed. Such a decision should be clinically guided and is entirely at the discretion of individual team leaders and organisations.

There is limited evidence for the efficacy of any drugs during non-TCAs and, despite the continued use of epinephrine, a recent randomised control trial has questioned its role [51]. Jacobs and colleagues showed that although epinephrine was associated with increased incidence of ROSC, there was no improvement in survival to hospital discharge [51]. Epinephrine is also known to adversely affect cerebral microvascular blood flow during cardiopulmonary resuscitation, and may worsen cerebral ischaemia [52]. Combined with the fact that hypovolaemic TCA patients are already hypoperfused, severely acidaemic and maximally vasoconstricted, this may limit the role of epinephrine in this setting [53,54].

Post-resuscitation care

If ROSC is achieved, meticulous post-resuscitation care should be accompanied by expediting transport to an appropriate major trauma centre. A pre-alert should be delivered to the receiving major trauma centre with a request for activation of their massive transfusion policy if deemed necessary. Consideration should be given to bypassing the emergency department for the operating theatre or the interventional radiology suite for definitive haemorrhage control for select patients.

Ventilation should target normoxia and normocapnia, while minimising intrathoracic pressures [4]. This limits further secondary neurological insults and any reduction in venous return [4]. To assist with ventilation and neuroprotection, suitable sedation, analgesia and neuromuscular blockade should be titrated to effect. Sedation and analgesia also provide a degree of sympatholysis that allows continued haemostatic resuscitation, while

maintaining permissive hypotension. The associated high-flow/low-pressure state promotes microvascular washout of previously hypoperfused areas with a theoretical improvement in lactate clearance and serum pH [55].

Physiological resuscitation endpoints post ROSC will depend on the underlying pathology and transport times involved [25,56]. The quality of evidence for permissive hypotension or targeted resuscitation is variable. For short transport times, however, the authors utilise the following endpoints for resuscitation: blunt trauma without traumatic brain injury – palpable radial pulse (systolic blood pressure >80 mmHg); blunt trauma with probable traumatic brain injury – systolic blood pressure >100 mmHg; and penetrating torso trauma – palpable central pulse or patient cerebration (systolic blood pressure >60 mmHg). If the transport time exceeds 1 hour, the detrimental effects of permissive hypotension may outweigh the benefits, and there may be a role for novel hybrid resuscitation [57].

The neuroprotective role of mild therapeutic hypothermia in traumatic brain injury and TCA survivors remains unclear [4,58,59]. The adverse impact of hypothermia on a trauma-induced coagulopathy could have catastrophic consequences prior to definitive haemorrhage control [60]. The authors' current practice is to maintain normothermia during transport through sufficient patient packaging to maximise passive warming, and to utilise active warming if necessary.

Conclusion

Greater Sydney Area Helicopter Emergency Medical Services undertakes prehospital and interhospital retrievals of critically ill and injured patients utilising a physician–paramedic team. TCA management by this service is guided by a standard operating procedure [61] that employs the evidence-based principles discussed above, and is summarised by the algorithm shown in Figure 1. This standardised approach for a highly trained physician-led prehospital team aims to maximise the number of neurologically intact survivors in out-of-hospital TCA. Further research is required before such an algorithm is to be more widely adopted.

Abbreviations
$ETCO_2$, end-tidal carbon dioxide partial pressure; PMCD, perimortem caesarean delivery; ROSC, return of spontaneous circulation; TCA, traumatic cardiac arrest.

Competing interests
The authors declare that they have no competing interests.

Authors' contributions
PBS conceived the idea for the paper, and drafted the initial algorithm and manuscript. CR, KH and BJB devised the Greater Sydney Area Helicopter Emergency Medical Services standard operating procedure for TCA management. All authors were involved in the final editing of the algorithm and manuscript.

All authors have made substantial contributions to all of the following: the conception and design of the study, or acquisition of data or analysis and interpretation of data; drafting the article or revising it critically for important intellectual content; and final approval of the version to be submitted.

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