Reanimating Patients After Traumatic Cardiac Arrest

A Practical Approach Informed by Best Evidence

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KEYWORDS

• Trauma • Cardiac arrest • Resuscitation • Resuscitative thoracotomy • Emergency thoracotomy

KEY POINTS

- Patients arriving at the emergency department with signs of life and/or evidence of cardiac contractility on point-of-care ultrasonography deserve aggressive resuscitative efforts.
- Chest compressions are unlikely to be effective in traumatic cardiac arrest and resources are better directed at addressing treatable causes of the cardiac arrest.
- Empiric bilateral chest decompression should be performed in all traumatic cardiac arrests, preferably via open thoracostomy.
- Simple, temporizing hemorrhage control measures to be considered in all patients include digital pressure, the use of a tourniquet, and empiric pelvic binding.
- Resuscitative thoracotomy should be considered for all patients with traumatic cardiac arrest with signs of life or point-of-care ultrasonography evidence of cardiac contractility, so long as the provider is competent in the procedure and the institution has an established protocol and the required resources.

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INTRODUCTION

Traumatic cardiac arrest (TCA) is not the same as cardiac arrest from coronary ischemia. Although this statement seems obvious, a clear distinction between the origins of cardiac arrest is essential to reorder and change management priorities. The 2015 International Liaison Committee on Resuscitation (ILCOR) makes this distinction.[1] However, in our experience, health care professionals who infrequently care for patients with TCA often follow standard resuscitation protocols that do not effectively address the pathophysiology of TCA. Management goals for medical cardiac arrest resulting from coronary ischemia are to support coronary perfusion to promote transition from a circulatory to electrically responsive phase to facilitate effective defibrillation.[2] In contrast, the management goals for TCA are to address massive hemorrhage and relieve obstructive causes of shock.

This article synthesizes the best available evidence to guide the management of TCA. Where the evidence is imprecise, and if appropriate, the article describe the authors' practice. This article compliments the 2015 ILCOR guidelines, providing more helpful detail and description of practice to aid health care professionals who infrequently care for patients with TCA.

This article emphasizes 5 key principles to guide management. Although these principles are arranged in a hierarchical fashion (a function of a traditional manuscript layout), the authors are not providing an algorithm. Algorithms can be helpful as memory aids in situations of high cognitive load.[3] They can also help structure learning for novices encountering complex tasks. However, algorithms are simplistic representations of patient management and do not account for the tacit knowledge required of expert trauma management. Most importantly, algorithms ignore natural decision-making processes, in which experts reorder management priorities in a dynamic fashion, responding to patient context and the unique complexity of each situation.[4] The authors encourage health care professionals to regularly consider these principles, prioritize them for action, and pause implementation when appropriate (**Fig. 1**). These principles should not be considered as a series of consecutive steps toward a linear conclusion of a trauma resuscitation.

A 54 -year-old woman was the restrained driver in a high-speed, rollover, motor vehicle collision. She is rapidly transported to the closest community hospital by

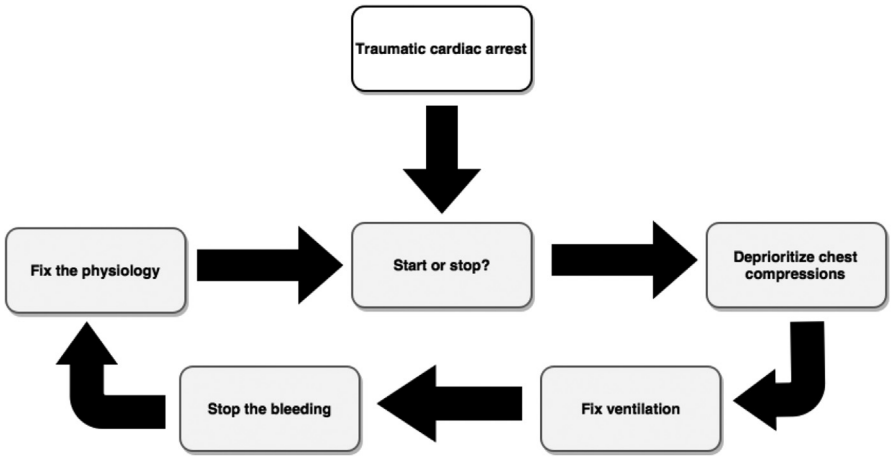

Fig. 1. Principles of traumatic cardiac arrest resuscitation.

emergency medical services (EMS) because of gross hemodynamic instability. She presents immobilized in spinal precautions, receiving supplementary oxygen. Intravenous access has not yet been established in an effort to prioritize transportation from the scene of the accident. On arrival she is obtunded. While vital signs are being determined and an initial assessment is initiated, her pulse can no longer be palpated and her respirations become erratic and gasping.

PRINCIPLE: START OR STOP?

Overall, rates of survival from traumatic arrest are low, although arguably not significantly different from the 5% to 10% range reported for out-of-hospital medical cardiac arrests.[5] A recent study including 2300 patients from North American Resuscitation Outcomes Consortium sites found a 6.3% overall rate of survival for prehospital traumatic arrest and more favorable outcomes among patients with blunt compared with penetrating mechanisms of injury.[6] Comparable rates of survival have been reported from a range of settings, including combat zones,[7] the prehospital, physician-staffed London Air Ambulance program,[8] and a systematic review examining outcomes of more than 5000 patients.[9] Thus, although rates of survival remain low, health care professionals should guard against inappropriate pessimism toward patients with TCA until further prognostic information from the initial bedside assessment is available.

There is no single variable that can be used to distinguish salvageable from unsalvageable traumatic arrest cases.[10] Overall, there are several clinical variables that have consistently been shown to be associated with a favorable prognosis following TCA (**Box 1**).[6,10–15]

Patients without at least 1 of the prognostic factors discussed earlier have an extremely poor (<1%) probability of survival. In these cases, resuscitation efforts should be considered futile. Various guidelines directing resuscitative efforts in TCA are shown in **Table 1**.

The Spectrum of Output States in Traumatic Cardiac Arrest

Patients with TCA represent a heterogeneous patient population with a spectrum of physiologic states, ranging from having no signs of life to being severely hypotensive, but with detectable electrical cardiac activity and contractility on point-of-care ultrasonography (**Table 2**). Occasionally, patients present after the return of spontaneous circulation (ROSC) physiology following prehospital resuscitative interventions.

Considering patients with TCA along this physiologic spectrum allows physicians to integrate multiple prognostic variables from the bedside assessment and does not solely rely on imprecise estimates of time since cardiac arrest. This framework also emphasizes TCA as a being a critical low-flow state",[7,18,19] in which point-of-care

Box 1
Favorable prognostic factors following traumatic arrest

Penetrating mechanism of injury, particularly thoracic
Vital signs at any time since first medical contact
Signs of life (any spontaneous movement, respiratory efforts, organized electrical activity on electrocardiogram, reactive pupils) at any time since first medical contact
Short duration of cardiac arrest (<10 minutes)
Cardiac contractility on point-of-care ultrasonography

Table 1
Guidelines on withholding and terminating resuscitation or performing resuscitative thoracotomy in traumatic arrest

	Context	Recommendation	Strengths and/or Limitations
National Association of EMS Physicians and American College of Surgeons Committee on Trauma[16]	Withholding resuscitation efforts	Withhold resuscitation in patients with (1) injuries incompatible with life[a]; (2) signs of prolonged cardiac arrest (dependent lividity, rigor mortis); (3) patients with blunt trauma who are apneic, pulseless, and who have no organized electrocardiographic activity; or (4) patients with penetrating trauma who are apneic, pulseless, have no organized electrocardiographic activity, and no other signs of life[b]	Extensive literature review Multidisciplinary perspective Prehospital focus
European Resuscitation Council[1]	Withholding resuscitation efforts	Consider withholding resuscitation efforts if (1) massive trauma incompatible with survival, or (2) no signs of life in the preceding 15 min	International panel Applies to prehospital and in-hospital settings Considers point-of-care-ultrasonography findings
Western Trauma Association[17]	Indication for RT	Consider RT in (1) patients with blunt trauma with <10 min of prehospital chest compressions, (2) patients with penetrating torso trauma with <15 min of chest compressions, (3) penetrating neck or extremity trauma with <5 min of prehospital chest compressions, or (4) patients with profound refractory shock	Straightforward stratification based on mechanism and time since cardiac arrest Time since cardiac arrest can be difficult to accurately estimate

Eastern Association for the Surgery of Trauma[11]	Indication for RT	Strong recommendation for RT in pulseless patients with signs of life[b] after penetrating thoracic trauma Conditional recommendation for RT in pulseless patients without signs of life[b] after penetrating thoracic trauma, present or absent signs of life[b] after penetrating extrathoracic injury, or present signs of life[b] after blunt injury Conditional recommendation against RT in pulseless patients without signs of life[b] following blunt trauma	Rigorous methodology More than 10,000 patients, from 72 studies, included Patient-oriented outcomes

Abbreviations: EMS, emergency medical services; RT, resuscitative thoracotomy.

[a] Decapitation, hemicorpectomy, exposed brain matter.

[b] Signs of life include reactive pupils, spontaneous movement, agonal respiratory efforts, organized electrocardiographic activity.

Table 2
Physiologic spectrum of patients with traumatic cardiac arrest

	(4) Dead	(3) PEA	(2) Pseudo-PEA	(1) Spontaneous Circulation
Cardiac Output	None	None	Very low	Variable[a]
Palpable Pulses	None	None	None	Present
Signs of Life	Absent	Absent	± Present	Present
ECG Rhythm	Asystole	Nonsinus	Nonsinus	Often sinus tachycardia
Bedside US	No contractility	No contractility	Contractility present	Contractility present (may be hyperdynamic)
End-tidal CO_2	Low/ undetectable	Low/ undetectable	Low	Low to moderate

Abbreviations: ECG, electrocardiogram; PEA, pulseless electrical activity; US, ultrasonography.
[a] Depends on a volume status, heart rate.

ultrasonography plays a critical role in differentiating patients in so-called pseudo-pulseless electrical activity from true pulseless electrical activity (PEA) states.[20] Distinguishing these two groups is important, because their prognoses are significantly different.

Patients in pseudo-PEA are in severe, end-stage shock. They have cardiac output that is not detectable by palpation of the pulse, but they may have other signs of life, including weak respiratory efforts, reactive pupils, or occasional spontaneous movement. In contrast, patients with true PEA have no cardiac output. Cardiac contractility on point-of-care ultrasonography has been reported to have 100% sensitivity for identifying survivors following TCA[12,21,22] and should be used early during resuscitation for its prognostic information.

In our practice, pulseless patients with no signs of life, asystole, and no cardiac contractility on bedside ultrasonography (group 4, dead; **Table 3**) do not receive

Table 3
Comparison of traumatic versus medical cardiac arrest

	Traumatic Cardiac Arrest	Medical Cardiac Arrest
Common causes	Hypovolemia/hemorrhage Tension pneumothorax Cardiac tamponade Hypoxia/respiratory failure Severe central nervous system injury	Dysrhythmia Myocardial infarction Pulmonary embolism Stroke/intracerebral hemorrhage Electrolyte disturbances (eg, hyperkalemia) Sepsis Drug/toxin
Effective treatments	Oxygenation/ventilation Chest decompression Blood transfusion Control of hemorrhage Resuscitative thoracotomy	Oxygenation/ventilation Electrical cardioversion Chest compressions Targeted temperature management

further resuscitative efforts. These patients do not survive. Further attempts at resuscitation would waste scarce resources (eg, blood products), divert resources from other patients, and risk exposing staff to blood-borne pathogens. It is our practice to aggressively resuscitate all other patients, particularly those in pseudo-PEA states.

The physician team leader performs a rapid assessment. The patient does not have palpable pulses. The patient has gasping irregular respirations and reactive pupils. A single point-of-care ultrasonography cardiac window reveals a hyperdynamic heart. Additional point-of-care ultrasonography information is not sought. The physician team leader indicates that the patient is in pseudo-PEA and indicates to the team that they should begin their resuscitative roles.

PRINCIPLE: DEPRIORITIZE CHEST COMPRESSIONS

It is critical for lead physicians to take a step back, reminding themselves and the other resuscitation team members that patients with TCA cannot be resuscitated using standard advanced cardiac life support (ACLS) algorithms. In most settings, especially outside the emergency department or trauma suite (eg, in the prehospital setting), chest compressions are considered standard of care regardless of the cause of the cardiac arrest.[23] Although current ACLS algorithms that prioritize chest compressions are important in medical cardiac arrest, they can impede timely interventions to correct blood loss and alleviate obstructive causes of shock in TCA. Deprioritizing chest compression early in TCA resuscitation is key.[19,24,25] Later in the resuscitation, chest compressions may be beneficial to support cerebral and cardiac perfusion, while intravascular blood resuscitation is ongoing.

A comparison of the causes and treatments of traumatic versus medical cardiac arrest is provided in **Table 3**.

There are several physiologic as well as logistical reasons to consider withholding chest compressions, at least initially, in the resuscitation of patients with TCA. First, unlike patients in medical cardiac arrest, who are presumed to be euvolemic, most patients with TCA are profoundly hypovolemic because of severe hemorrhage or functionally hypovolemic because of impaired preload from either a tension pneumothorax or cardiac tamponade. Although well-performed external chest compressions may be able to deliver close to a third of the normal cardiac output in the euvolemic state, in animal models with tamponade or hypovolemia this is not true.[23] In animal models with tamponade physiology, chest compressions seem to increase intrapericardial pressures and worsen cardiac output.[23] Meanwhile, in severe hypovolemic states, external chest compressions do not increase output.[23] Establishing intravascular blood volume and relieving any obstruction to cardiac filling must take precedence over chest compressions.

In our experience, team members are often more comfortable withholding chest compressions when the physician team leader is able to articulate to the team why chest compressions do not work in TCA and why they might be harmful. Most often, performing chest compressions impedes the team from performing the procedures that address the cause of TCA. Other downsides to chest compressions potentially include iatrogenic injuries to thoracic and abdominal organs, worsening existing injuries, and slowing the flow of blood via rapid infuser devices.[19]

Of special note, if the patient's presentation and mechanism of injury do not fit with a primary traumatic arrest but are more consistent with a primary medical arrest followed by a trauma (eg, elderly patient with minor trauma, single-vehicle collisions, ventricular fibrillation or ventricular tachycardia as the presenting rhythm, paucity of physical findings of trauma on the patient), the resuscitation is best approached using standard ACLS strategies.[1]

With EMS prehospital notification, the physician team leader has assembled and briefed the health care team on their roles. Included in the team are a respiratory therapist, 2 experienced emergency nurses, and a second emergency physician. The blood bank has been informed about the need for blood. Anticipating the potential for TCA, the physician team leader has reminded the team that cardiopulmonary resuscitation (CPR) is not the first priority. CPR is not performed and the members quickly prepare to perform other, time-sensitive, procedures.

PRINCIPLE: FIX VENTILATION

All patients with traumatic arrest require early airway control to relieve airway obstruction, deliver oxygen, optimize ventilation, and prevent aspiration.[24] Airway obstruction may be managed temporarily with oral or nasal airways and bag-mask ventilation, but ventilation through a bag-mask device may prove difficult in patients with facial trauma, especially with midface fractures and significant bleeding. The need for a definitive airway should be anticipated.

Airway Management

The authors suggest initially providing 100% oxygen through a bag-mask device to ensure adequate oxygenation. If ROSC is obtained, oxygen levels should be titrated to avoid hyperoxemia, which may worsen traumatic brain injury.[26,27]

A cuffed endotracheal tube in the trachea remains the gold standard for airway management, because it allows precise titration of oxygen, protection from aspiration, and controlled ventilation. Most patients with TCA do not require any sedation or paralysis for laryngoscopy and intubation. However, some patients with minimal cardiac output (group 2, pseudo-PEA; see **Table 2**) may retain muscle tone and protective airway reflexes, requiring a dose of a short-acting paralytic. The low cardiac output state requires a doubling of the standard paralytic dose. Sedation is not necessary in this clinical context. Confirmation of endotracheal placement with an end-tidal carbon dioxide detector is standard of care.

Intubation may be difficult in patients with traumatic arrest for several reasons, including facial trauma, blood or emesis obscuring laryngoscopy, and the need to maintain cervical spine precautions. If difficulties are encountered or there are not adequate personnel to intubate the patient and perform other time-sensitive procedures, a supraglottic airway device can be placed. Multiple intubation attempts have the potential to distract the team from other important tasks that need to be performed simultaneously. A supraglottic device provides adequate oxygenation and ventilation for the duration of the resuscitation. With ROSC, conversion of a supraglottic device to an endotracheal tube should be prioritized based on other tasks to be performed and the complexity of airway injury.

Chest Decompression

Tension pneumothorax is notoriously difficult to diagnose in patients with blunt traumatic arrest. The authors agree with existing guidelines suggesting that empiric bilateral chest decompression be performed on all patients with blunt and penetrating thoracic TCA to avoid missing a tension pneumothorax.[19,24,25,28] In addition, tension physiology may develop during the course of resuscitating a patient with blunt chest trauma and bilateral chest decompression also prevents this complication.

Our practice is to perform open thoracostomies (ie, sharp and blunt dissection of the chest wall at the anterior axillary line, fourth to fifth intercostal space, to facilitate internal palpation of the hemithorax)[29] rather than needle thoracostomies for chest

decompression. The rationale for this approach is that it ensures that a potential tension pneumothorax is fully decompressed (the clinician can palpate the lung). The second advantage of the open thoracostomy is in diagnosing massive hemothorax as a cause of the arrest. The open thoracostomy can be converted to a chest tube once the patient has stabilized.

If the physician performs a needle thoracostomy, the authors recommend placement in the anterior axillary line at the fourth to fifth intercostal space (where the chest wall is thin). Data from a meta-analysis suggests a 13% failure rate at this site, compared with a 38% failure rate at the traditional landmark of the second intercostal space in the midclavicular line when using a 4-cm (1.5-inch) needle.[30] Use of a 6.44-cm (2.5-inch) needle will penetrate the chest wall and decompress the pleura in 95% of the population.[31]

The physician team leader directs the respiratory therapist to insert a supraglottic airway and ventilate the patient by hand using 100% oxygen. An excessively rapid ventilation rate that impedes venous blood return to the heart is avoided, as is an excessively slow ventilation rate that does not correct hypoxia or respiratory and metabolic acidosis. The plan is to intubate the patient after the initial set of procedures is performed. The second physician performs bilateral open thoracostomies using a clean technique (ie, sterile gloves, rapid skin cleaning, and the use of a sterile towel for local draping). No air or blood returns from the right hemithorax, whereas on the left side there is a large return of air and ongoing, oozing blood loss.

PRINCIPLE: STOP THE BLEEDING

Control of hemorrhage can be divided into temporizing and definitive procedures. In TCA caused by massive hemorrhage, the clinician must identify and provide temporizing control of hemorrhage while the patient's intravascular volume is simultaneously restored via blood transfusion. To this end, there are several potential options to achieve temporary hemorrhage control, including use of manual pressure and topical hemostatic agents for external hemorrhage, tourniquets for peripheral vascular hemorrhage, pelvic ring closure for pelvic hemorrhage, and thoracotomy for control of cardiac or major vascular hemorrhage. These options are all bridges to definitive hemorrhage control, which occurs in an operating theatre, angiography suite, or a hybrid operating theater that combines both capabilities. Institutional processes for accessing local or regional resources must be developed and not spontaneously developed in an ad hoc fashion.

Manual Pressure and Topical Hemostatic Agents

Manual pressure is the basis for control of all surgical bleeding. The ability to occlude a bleeding source is reliant on the ability to effective apply pressure to it (ie, can it be pinched between fingers or compressed against something firm like a bony structure?) and on the size of the area of hemorrhage (ie, is it a vessel or the entire surface of an organ?).

Topical hemostatic agents can be divided into mechanical hemostats, active hemostats, flowable hemostats, and fibrin sealants. They are often used in combination.[32] External topical hemostatic agents include gauze bandages impregnated with a hemostatic agent. These bandages can be applied to the surface of a bleeding area in combination with pressure or potentially packed into bleeding open wounds. Long used in the military, hemostatic bandages, such as HemoCon, Quikclot, are now being deployed for civilian trauma as part of a national American effort to optimize care provided by immediate responders (ie, the public) (http://www.bleedingcontrol.org). The

application of manual pressure using gauze or hemostatic gauze to any bleeding site is the first maneuver to obtain temporary hemorrhage control.[33]

Tourniquets for Peripheral Vascular Hemorrhage

The use of tourniquets has waxed and waned for decades but, based on valuable experience from recent military conflicts[34,35] and in civilian trauma,[36–38] the use of tourniquets is now standard care.[39] Tourniquets are indicated for significant extremity hemorrhage if direct pressure is ineffective or impractical. Commercially produced windlass, pneumatic, or ratcheting devices that occlude arterial flow are preferred, whereas the use of narrow, elastic, or bungee-type devices is not recommended. Preferred tourniquets include the Combat Application Tourniquet and the pneumatic tourniquet. Although some junctional hemorrhage devices have been developed to control bleeding from the groin (the so-called Black Hawk Down injury) or axilla, they are not typically available outside of the military setting and their application is not straightforward.[40] The application of an improvised tourniquet should only be considered if a commercial device is unavailable. Tourniquets placed in the prehospital setting should not be released until the patient has reached definitive care.[33] The time of placement of a tourniquet should be recorded, preferably on the patient or tourniquet.

Pelvic Binders for Pelvic Hemorrhage

Bleeding from pelvic fractures continues to be a leading cause of preventable traumatic death in major trauma centers.[41] The bleeding from pelvic fractures is often multifocal (ie, arterial, venous, and bony hemorrhage), diffuse, and difficult to compress. Minimizing pelvic bleeding requires reapproximation of the bony pelvic architecture to prevent further injury to the myriad of pelvic vessels.[42] Our practice is to use a folded sheet positioned over the greater trochanters and anterior iliac spines and held snug with a simple square knot or pair of large, straight, Kelly hemostats. In our experience, this approach is preferable to commercially available devices for both fiscal reasons and more readily accessible groin access (by cutting a hole in the sheet without releasing the pelvic binder) if subsequent angiography or resuscitative endovascular balloon occlusion of the aorta (REBOA) is required. Our practice is to empirically apply a pelvic binder to patients with TCA with blunt abdominal or pelvic injury. It is a fast and simple procedure with insignificant side effects, whereas the physical examination of a mechanically unstable pelvis can be unreliable.

Another maneuver to control pelvic hemorrhage is preperitoneal packing.[43] However, such a procedure requires a skilled operator, typically a trauma surgeon or orthopedic trauma surgeon, as well as the necessary surgical equipment. This maneuver is typically performed in the operating theater, sometimes concurrently with abdominal exploration. This maneuver is not our standard practice in blunt TCA.

Thoracotomy for Control of Intrathoracic Cardiac or Vascular Hemorrhage

Resuscitative thoracotomy (RT) is the ultimate invasive procedure to attempt reanimation of patients with TCA. This procedure is controversial, especially when accounting for the risks of infectious disease exposure to health care providers and the futility of the procedure when applied without appropriate patient selection.[13,44] There are consensus guidelines on indications to perform an RT (see **Table 1**). The best outcomes occur when RT is performed for patients with thoracic stab injuries who arrive with signs of life in the emergency department.[10]

The primary goals of RT are to release pericardial tamponade and to control intrathoracic bleeding. Evacuation of bronchovenous air embolism, elimination of a bronchopleural fistula, performing open cardiac massage, and temporary occlusion of the descending

thoracic aorta to optimize brain and cardiac perfusion or control abdominal or pelvic hemorrhage are also indications for RT.[45,46] However, these are rarely indications for performing an RT in a center without significant experience and appropriate surgical support.

Historical indications for RT have changed because comparisons of open cardiac massage with closed-chest compressions for superior hemodynamics[47,48] have failed to show improved patient-oriented outcomes.[49,50] Patients requiring occlusion of the descending thoracic aorta to restore perfusion of the brain and heart have shown mortalities greater than 90% in some studies.[51]

RT is, at best, a temporizing maneuver.[52] To have any chance of a successful outcome, the patient needs to be rapidly transported to a well-equipped and staffed operating theater with a surgeon immediately available who is capable of repairing the underlying injury.[52] Regardless of clinical discipline,[53,54] the physician performing the RT must be competent in the procedure with the ability to successfully address the cause of the patient's TCA. The hospital must identify a priori a process to perform an RT and to provide definitive care if the patient is reanimated.

The steps to performing an RT are well described elsewhere.[45] Although typically started as a left anterolateral thoracotomy, extension across the sternum into a bilateral thoracotomy or clamshell incision may be required for adequate exposure to the heart, superior mediastinum, or right pulmonary hilum.[55] Cadaver-based experiments suggest that access time may be equivalent for these incisions, but time to control of a cardiac injury may be shorter for the clamshell incision.[56]

A primary objective of an RT is releasing pericardial tamponade by opening the pericardium. Any bleeding from a ventricular injury may be temporized with judicious finger pressure by the pads of the finger, which move with the beating of the heart. Larger defects not controlled by finger pressure may be closed using a skin stapler and/or occluded via the inflated balloon of a Foley catheter. Of note, a novel device has recently been described that provides temporary hemorrhage control for ventricular injuries without the negative effects on cardiac function that Foley balloon catheters often create.[57] Atrial ruptures are best controlled with application of a clamp to close the defect. Although suturing of cardiac injuries is required for definitive control, this requires a skilled operator and equipment not typically available in the emergency department. It is best accomplished in the operating theater. However, cardiac tamponade secondary to trauma is poorly treated by percutaneous pericardiocentesis, although it may be used as a temporizing measure in centers without the ability to perform an RT.[58]

The second goal of controlling intrathoracic hemorrhage can be accomplished by inspecting for bleeding sources. Chest wall bleeding can be packed and focal bleeding from the lung or major vessels can be controlled with focal pressure. If this is ineffective, clamping of the pulmonary hilum or a pulmonary hilar twist may also be used for severe bleeding, although the latter maneuver requires division of the inferior pulmonary ligament.[59] Occlusion of the hilum with a clamp or external compression is simpler to perform than a hilar twist. If significant blood loss is present from a right-sided open thoracostomy, a bilateral thoracotomy is necessary to identify and control the source of bleeding.

Our practice is to perform an RT if a physician competent in the procedure is present, the patient in TCA has signs of life in the emergency department, or there is cardiac contractility on point-of-care ultrasonography. Our practice is to perform a left anterolateral thoracotomy, only extending to a clamshell incision if initial decompression of the right hemithorax reveals significant blood loss. If ROSC is achieved, our practice is to cover the incision with dry, sterile surgical towels in anticipation of rapid transportation of the patient to an operating theater. The open thoracostomy of the right hemithorax is then converted to a right-sided chest tube. Our practice is to continue the resuscitation until all reversible causes of TCA have been treated.[1]

Resuscitative Endovascular Balloon Occlusion of the Aorta for Control of Abdominal or Pelvic Hemorrhage

The use of REBOA, although first described in the Korean War, is being rapidly adopted in the care of civilian patients with trauma.[60–62] The greatest utility of REBOA is as an alternative to RT for the occlusion of the thoracic descending aorta to stop abdominal or pelvic hemorrhage.[63] Its use is covered in more detail in Steven Skitch and colleagues' article, "Acute Management of the Traumatically Injured Pelvis"; and Megan Brenner and Christopher Hicks's article, "Major Abdominal Trauma: Critical Decisions and New Frontiers in Management," in this issue. Our current practice does not include REBOA in the management of TCA.

Other Direct Pressure Maneuvers

A few specific scenarios worth mentioning include penetrating injuries to zone 1 of the neck. Severe hemorrhage from such wounds often originates from the subclavian vessels or from intrathoracic vessels, both of which are difficult to compress externally. The placement of a Foley catheter into the wound with subsequent inflation may be considered[64] (See Angelo Mikrogianakis and Vincent Grant's article, "The Kids are Alright: Pediatric Trauma Pearls," in this issue).

Penetrating injuries to the face or facial smashes can create severe bleeding that may require facial packing. However, with complete disruption of the bony architecture there is no ability to obtain tamponade, because there is nothing to pack against. In such scenarios, consideration for external bolstering by completely wrapping the face and head circumferentially may be considered.[65]

As a principle, hemorrhage control in patients with TCA must be obtained, at least in a temporizing manner, in order to have any possibility of ROSC. **Table 4** lists temporizing measures to use as appropriate.

Table 4
Temporizing hemorrhage control measures

Source of Hemorrhage	Intervention
Severe scalp laceration	• Closure of wound with skin stapler, clamp, or rapid whipstitch suture
Open soft tissue wounds	• Direct pressure • Packing with hemostatic gauze • Closure of wound with skin stapler, clamp, or rapid whipstitch suture
Mechanically unstable pelvis	• Pelvic binder
Open extremity injury or amputation	• Reduce fracture • Direct pressure • Pack wound • Tourniquet application
Junctional zone (eg, groin, axilla)	• Direct pressure
Zone 1 penetrating neck wound	• Direct pressure • Foley balloon tamponade
Severe facial smash	• Nasopharyngeal packing • Consider circumferential bolster of the head
Massive hemothorax	• Resuscitative thoracotomy
Pericardial tamponade	• Resuscitative thoracotomy

The patient has a partial amputation of the left ankle. Direct pressure with gauze and realignment of the bony anatomy is performed by one of the nurses. Oozing from the wound is ongoing with blood pooling on the floor. The nurse applies a pneumatic tourniquet and blood flow stops. Simultaneously, the pelvis is bound using a folded sheet that was placed on the resuscitation bed with the initial EMS activation. The physician with the most point-of-care ultrasonography experience (the second physician) attempts to identify a pericardial effusion. Significant thoracic injury prevents obtaining a good cardiac view. The physician team leader makes the decision to perform a left anterolateral thoracotomy. The operating room is informed and the thoracic surgeon and on-call anesthetist is paged to the emergency department. The physician team leader begins to prep the chest for a clean procedure (ie, sterile gloves, rapid skin cleaning, local draping with sterile towels). A clamshell incision is not planned, because the right-sided open thoracostomy did not reveal any major bleeding. However, sterile prep includes the right side of the chest. In anticipation of the thoracotomy, the respiratory therapist and second physician intubate the patient. Sedation and paralytic agents are not required. An orogastric tube is also placed to assist in identifying the esophagus from the aorta if compression of the aorta is required.

PRINCIPLE: FIX THE PHYSIOLOGY

Warm the Patient and Keep the Patient Warm

Environmental exposure, blood loss, and resuscitation with hypothermic fluids all contribute to heat loss in patients with TCA. Unlike patients with medical cardiac arrest, who have been shown to benefit from postarrest hypothermia, patients with hypothermic trauma have been shown to have greater complication rates and higher mortality.[66] Hypothermia contributes to worsening coagulopathy in patients with hemorrhaging trauma[66] as a result of reduced enzyme activity, platelet dysfunction, and reduced clotting factor activity.[67] In addition, hypothermia and acidosis compound coagulopathy.[68]

Maintaining normothermia during resuscitation is best achieved through a combination of passive and active warming. Wet clothing must be removed. The ambient temperature of the trauma suite should be increased whenever possible. Although exposure and injury identification are priorities during the physical examination, warm blankets or forced air warming blankets should be placed on the patient after the physical examination is complete.

All resuscitative fluids should be delivered warm and under pressure (with the exception of platelets) via a rapid infuser (Level 1 Rapid Infusor). Rapid infusion devices are capable of warming fluids to 36°C to 40°C depending on the delivery rate. Maximum in vivo delivery rates are listed at 1100 mL/min and vary depending on fluid type, catheter size, and catheter length (Smith Medical).

Intravenous Access

The initial fluid resuscitation in a TCA should be done through multiple large-bore peripheral intravenous catheters (14–18 gauge) secured in the upper limbs. Intravenous access above the diaphragm is necessary because of the possibility of venous obstruction or extravasation from injured venous structures inferior to the right heart. In the event of penetrating injury to the upper extremity, axilla, or lateral neck, peripheral access should be secured on the side contralateral to the injury.

If attempts for peripheral catheterization are unsuccessful, expedient placement of intraosseus catheters in the proximal humerus should be the next step in management. The proximal humerus is the preferred intraosseus location because it is both

located above the diaphragm and has been shown to have comparable flow rates with other intraosseus locations in human studies.[69–71] Sternal placement has been described; however, the authors do not recommend this site because it is more challenging given the thinner cortical bone of the sternum and the risk for mediastinal injury. Sternal catheter location may also interfere with ongoing management and intrathoracic procedures. Intraosseus access has been shown to be faster and to have a greater first-attempt success rate than central venous access in critically ill patients.[72] Intraosseus catheters have also been endorsed for use in TCA.[73]

Attempts to secure central venous access should not be made in the immediate care of arrested patients in whom intravenous or intraosseus access has been established. It may eventually be required, if peripheral or intraosseus access cannot be secured, or to aid in a large-volume fluid resuscitation. In these cases, catheter selection should be limited to 8-French to 9-French percutaneous introducer sheaths. The flow rate from the 16-gauge distal port of a triple-lumen central catheter (15-cm length) has been shown to be approximately 3 times slower than that of a 14-gauge, 5-cm peripheral catheter.[74] These multiple-lumen central venous catheters should not be used.

The subclavian vein is the optimal site for central venous access if required. A rigid cervical collar will invariably be in place in patients with blunt trauma and prevents internal jugular vein access. Femoral vein access is less desirable given that it is below the diaphragm.

Intravascular Resuscitation

Over the past 2 decades, advancements in the resuscitation of patients with trauma have focused on damage control resuscitation. The principles include minimizing the use of crystalloid fluids, transfusing balanced ratios of blood products, permissive hypotension, and damage control surgery (ie, rapid, temporary repair of injuries without definitive surgical correction to minimize physiologic insult during the operation).

Large-volume resuscitation with crystalloid fluid leads to coagulopathy through the dilution of clotting factors, fibrinogen, platelets, and calcium. Crystalloids can worsen acidemia given that Ringer lactate solution (pH 6.5) and normal saline (pH 5.5) are both acidic fluids. In addition, the chloride load in normal saline can precipitate hyperchloremic metabolic acidosis.

The use of crystalloid fluid should mainly be limited to the delivery of medications. Judicious use may be considered in the initial resuscitation of the intravascular space only until blood products become available. It should be recognized that most patients with trauma presenting in shock have likely received a significant volume of crystalloid in the prehospital environment. The amount of unintended crystalloid fluid delivered to the patient in the trauma suite can be significant.

The optimal fluid for the initial resuscitation of the intravascular space in patients with TCA is whole blood (or the components that constitute it). In patients whose primary mechanism of arrest is profound hemorrhagic shock rather than obstructive shock, the anticipated blood volume deficit is likely to require a significant transfusion volume. Almost all of these patients will have assessment of blood consumption (ABC) scores totaling 2 or greater, which has shown to be both a reasonably sensitive and specific prediction score for the need for massive transfusion (MT).[75,76]

The authors recommend the initiation of an institutionally coordinated MT protocol in patients with TCA once a decision to resuscitate the patient is made. Such protocols have been shown to aid in communication and speed of delivery of blood products.[77] Early and frequent communication with the blood bank is necessary for the expedited

release and delivery of blood products, as well as conservation of blood bank resources.

Military[78] and civilian[79–81] trauma studies have shown the benefits of equal ratios of fresh frozen plasma and platelets to packed blood cells in order to best approximate the components of whole blood. There is no current consensus on the most beneficial component ratio. It is our practice to approximate an ongoing resuscitation ratio of 1:1:1 (red blood cells (RBCs)/fresh frozen plasma/platelets) once a decision to initiate MT is made.

A nurse attempts intravenous access in the left antecubital fossa. On completion of endotracheal intubation of the patient, the second physician places an intraosseus cannula in the right humerus. The hospital does not have an MT protocol. However, the blood blank has provided 4 units of packed RBCs. These units are infused under pressure and warmed. Fresh frozen plasma and platelets are being made available. The physician team leader performs a left anterolateral thoracotomy. The pericardium is opened and the heart delivered. A small clot is removed. No large cardiac lacerations are identified. There is significant oozing from the left lung, which stops with direct pressure applied by a nurse. Open cardiac massage is performed using a hinged, clapping motion of 2 cupped hands to avoid fingertip pressure on the heart. With the administration of blood, a carotid pulse is palpated as the heart spontaneously beings to beat.

PRINCIPLE: FIX THE PHYSIOLOGY (AFTER RETURN OF SPONTANEOUS CIRCULATION)

Once ROSC is achieved, monitoring for the complications commonly seen during an MT is important. Hypocalcemia is of particular importance because calcium is a cofactor for several stages of the clotting cascade and plays a role in vascular tone and myocardial contractility. Hypocalcemia results from hemodilution as well as chelation with the citrate contained in stored RBCs.

Addressing Coagulopathy

The ongoing need for component transfusion should also be considered. Additional factor-specific replacement can be considered based on International Normalized Ratio, prothrombin time, platelet count, and fibrinogen. Although not universally available, transfusion may be guided by thromboelastography or rotational thromboelastometry. In addition, it is our practice to administer tranexamic acid to patients with TCA who experience ROSC based on extrapolation of evidence of benefit in bleeding, nonarrested patients with trauma.[82]

Addressing Anticoagulation

Patients treated with antiplatelet agents or anticoagulants (vitamin K antagonists, anti-IIa agents, anti-Xa agents) before trauma provide an additional challenge with hemorrhage control. Reversal of these agents should occur early after ROSC (**Table 5**). Consultation with a hematologist may be helpful, particularly with direct oral anticoagulants, for which empiric reversal therapies have limited evidence.

Blood Pressure Management

After a pulse is restored, the primary goal in resuscitation is to maintain end-organ perfusion while managing injures. The authors recommend the early placement of an arterial catheter for real-time blood pressure monitoring. Automated cuff pressure can be unreliable[83] and rapid changes in blood pressure may be missed by interval measurements.

Table 5
Antiplatelet and anticoagulant reversal therapies

Medication	Treatment
Antiplatelet agents (ASA, clopidogrel, dipyridamole)	Platelet transfusion ddAVP
Anti–vitamin K agents (warfarin)	Vitamin K and Prothrombin complex concentrate
Anti–factor IIa agents (dabigatran)[a]	Idarucizumab or Prothrombin complex concentrate
Anti–factor Xa agents (apixaban, rivaroxaban, edoxaban)[a,b]	Prothrombin complex concentrate

Abbreviations: ASA, acetylsalicylic acid; ddAVP, desamino-D-arginine vasopressin (desmopressin).
[a] Recombinant factor VIIa can be considered in the treatment of refractory bleeding in consultation with a hematologist.
[b] Novel reversal agents for these medications are in development at the time of publication.
Data from Hogg K, Panag A, Worster A, et al. Direct oral anticoagulants: a practical guide for the emergency physician. Eur J Emerg Med 2016;23(5):330–6.

The concept of permissive hypotension (or just-adequate normotension) is intended to preserve end-organ perfusion without disruption of clot formation via increasing intravascular pressure and loss of vasoconstriction. However, the evidence for this practice is not widely generalizable to the emergency department resuscitation of patients with TCA.[84]

The authors suggest that the patient's comorbidities, injury pattern, and clinical response to therapy should be considered in targeting a specific blood pressure. Our recommendations are for a mean arterial pressure goal of approximately 65 mm Hg. Permissive hypotension has not been thoroughly studied in closed head injuries and should not be used in these patients.[85]

Vasopressors should not be routinely used in the resuscitation of patients with trauma. Ongoing shock in the absence of a neurogenic cause (eg, cervical or high thoracic spinal cord injury) should prompt a repeated examination to rule out evolving or a previously missed obstructive cause, continued blood resuscitation, and an aggressive plan to control bleeding. The authors agree with the European Guideline on the Management of Major Bleeding in Trauma parameters for use of a vasoactive medication to support perfusion. Vasopressors should only be used when severe hypotension not responsive to blood product therapy and efforts to control hemorrhage are present.[86] In this context, norepinephrine is a typical first-line agent, unless there is evidence of cardiac dysfunction on point-of-care ultrasonography that may best respond to epinephrine.

Cardiac Dysfunction/Dysrhythmia

Ventricular dysrhythmia in patients after TCA should be treated with electrical cardioversion or defibrillation. Negative chronotropy, negative ionotropy, and vasodilatory properties common to many antiarrhythmic medications are likely harmful in the post-TCA period. Supraventricular tachycardias should not be treated initially, given the likelihood that they maybe a physiologic response.

Sedation and Analgesia

Analgesia and sedation are important to relieve pain and anxiety secondary to traumatic injuries and painful procedures, as well as to aid in the ventilation of intubated

post-ROSC patients with TCA. All commonly used pharmacologic agents for induction of anesthesia and sedation can cause precipitous hypotension by decreasing sympathetic tone. The authors recommend using 25% to 50% of a standard dose and closely monitoring patient response. No consensus exists with respect to the safest sedation and analgesia protocol in the critically ill patients with trauma.

The authors use ketamine as a single agent or with the addition of a short-acting opioid such as fentanyl.[87] Historic concerns for increased intracranial pressure associated with the use of ketamine in brain-injured patients have been reconsidered.[88] Despite the common belief that ketamine is a hemodynamically neutral agent, hypotension is frequently observed in critically ill patients.[89] Etomidate is also a reasonable sedative agent if available. However, guidelines have recommended against etomidate for prolonged sedation via infusion in patients with trauma.[90] The authors recommend against the use of propofol or benzodiazepines as sedative agents in the patients after TCA.

Within 20 minutes the anesthetist and thoracic surgeon arrive in the emergency department and make arrangements to bring the patient immediately to the operating theater. The thoracotomy incision is covered with dry, sterile surgical towels. The right open thoracostomy is converted to a chest tube. Forced air warming blankets cover the patient. An arterial catheter is placed to monitor blood pressure. Spinal immobilization is maintained. Blood products, as available, are infused one at a time, aiming for equal administration of RBC, plasma, and platelets, to maintain a mean arterial pressure of 65 mm Hg. Ketamine boluses of 25 mg are titrated to patient effect. Blood work is sent to guide ongoing resuscitation. A point-of-care ultrasonography scan suggests intra-abdominal free fluid. A pelvic radiograph does not show any fracture. The regional trauma center (15 minutes away by ambulance) is contacted to assist in guiding further management, including timing of imaging the brain. The preliminary plan is to surgically control thoracic bleeding, perform a laparotomy and pack the abdomen, and continue blood resuscitation. Arrangements for transfer of the patient postoperatively to the trauma center are being made.

SUMMARY

TCA is a survivable condition. Recognition and management of TCA as a distinct pathophysiologic process from medical cardiac arrest is essential. Principles of resuscitation include making a rapid decision to proceed with resuscitation based on prognostic information available at the bedside (signs of life and point-of-care ultrasonography evidence of cardiac contractility), deprioritizing chest compressions, correcting dysfunctional ventilation, temporarily controlling ongoing hemorrhage, and initiating a balanced ratio of blood product transfusion as the first step in addressing physiologic derangement. Health care professionals, who appreciate the complexity and nuance of trauma care, prioritize for action these principles, pause implementation when appropriate, and do not assume that an algorithm is a preferred approach in TCA.

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