Chapter 17: Resuscitative Thoracotomy

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KEY POINTS

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- Resuscitative thoracotomy (RT) refers to an emergent thoracotomy, most commonly performed in the emergency department for patients arriving in extremis; RT may also be performed in the operating room or intensive care unit within hours after injury for physiologic deterioration.
- The primary objectives of RT are to release pericardial tamponade, control cardiac or intrathoracic hemorrhage, evacuate bronchovenous air embolism, perform open cardiac massage, and temporarily occlude the descending thoracic aorta.
- The critical determinants of survival following RT include the injury mechanism, anatomic location of injury, and the patient's physiologic condition at the time of thoracotomy.
- The highest survival rate following RT is in patients with penetrating cardiac wounds, especially when associated with pericardial tamponade.
- Based on the literature to date, RT should be performed for (1) penetrating nontorso trauma with cardiopulmonary resuscitation (CPR) of less than 5 minutes, (2) blunt trauma with CPR of less than 10 minutes, and (3) penetrating torso trauma with CPR of less than 15 minutes.
- RT is unlikely to yield productive survival when patients (1) sustain blunt trauma and require more than 10 minutes of prehospital CPR, (2) have penetrating wounds and undergo more than 15 minutes of prehospital CPR, (3) have isolated extremity trauma with more than 5 minutes of prehospital CPR, or (4) manifest asystole without pericardial tamponade.
- Outcome following RT in the adolescent population is largely determined by injury mechanism and physiologic status on presentation to the emergency department; for patients under 15 years of age, there are only isolated cases of survival following penetrating trauma and no apparent survival benefit following blunt trauma.

INTRODUCTION

The number of patients arriving at hospitals in extremis, rather than dying in the prehospital setting, has increased due to the maturation of regionalized trauma systems (see Chapter 4). Salvage of individuals with imminent cardiac arrest or those already undergoing cardiopulmonary resuscitation (CPR) often requires immediate thoracotomy as an integral component of their initial resuscitation in the emergency department (ED). The optimal application of a resuscitative thoracotomy (RT) requires a thorough understanding of its physiologic objectives, technical maneuvers, and the cardiovascular and metabolic consequences. Although resuscitative endovascular balloon occlusion of the aorta (REBOA) has been advocated as a resuscitation maneuver that should replace RT, there is a paucity of objective data to clarify the precise role of REBOA versus RT. In fact, aortic occlusion can be achieved quicker with RT than REBOA in patients arriving with CPR in progress.[1] Furthermore, RT is indicated for life-threatening thoracic injuries. However, we believe REBOA is ideal for unstable pelvic fractures with advanced hemorrhagic shock (see Chapter 39), and we have placed a REBOA in patients who have recovered perfusion following RT when it is evident that a pelvic fracture is the source of acute blood loss. This chapter reviews the features of RT and highlights the specific clinical indications, all of which are essential for the appropriate use of this potentially lifesaving yet costly procedure.

HISTORICAL PERSPECTIVE

Emergent thoracotomy came into use for the treatment of heart wounds and anesthesia-induced cardiac arrest in the late 1800s and early 1900s.[2] The concept of a thoracotomy as a resuscitative measure began with Schiff's promulgation of open cardiac massage in 1874.[2] Block first suggested the potential application of this technique for penetrating chest wounds and heart lacerations in 1882.[3] Following use of the technique in animal models, the first successful suture repair of a cardiac wound in a human was performed at the turn of the century.[4] Subsequently, Igelsbrud described the successful resuscitation of a patient sustaining cardiac arrest during a surgical procedure using emergent thoracotomy with open cardiac massage.[2] The utility of the emergent thoracotomy was beginning to be tested in a wide range of clinical scenarios in the early 1900s.

With improvement in patient resuscitation and an ongoing evaluation of patient outcomes, the indications for emergent thoracotomy shifted. Initially, cardiovascular collapse from medical causes was the most common reason for thoracotomy in the early 1900s. The demonstrated efficacy of closed-chest compression by Kouwenhoven et al[5] in 1960 and the introduction of external defibrillation in 1965 by Zoll et al[6] virtually eliminated the practice of open-chest resuscitation for medical cardiac arrest. Indications for emergent thoracotomy following trauma also became more limited. In 1943, Blalock and Ravitch[7] advocated the use of pericardiocentesis rather than thoracotomy as the preferred treatment for postinjury cardiac tamponade. In the late 1960s, however, refinements in cardiothoracic surgical techniques reestablished the role of immediate thoracotomy for salvaging patients with life-threatening chest wounds.[8] The use of temporary thoracic aortic occlusion in patients with exsanguinating abdominal hemorrhage further expanded the indications for emergent thoracotomy.[9,10] In the past two decades, critical analyses of patient outcomes following postinjury RT has tempered the unbridled enthusiasm for this technique, allowing a more selective approach with clearly defined indications.[11-16]

DEFINITIONS

The literature addressing RT appears confusing, likely due to widely varying terminology. As a result, there is a lack of agreement among physicians regarding the specific indications for RT as well as the definition of "signs of life."[17] In this chapter, RT refers to an emergent thoracotomy, most commonly performed in the ED for patients arriving in extremis. At times, an RT is performed in the operating room (OR) or intensive care unit (ICU) within hours after injury for delayed physiologic deterioration or an acute anatomic disruption or physiologic change. The value of RT for acute resuscitation may also be confusing because of the variety of indices used to characterize the patient's physiologic status prior to thoracotomy. Because there have been a wide range of indications for which RT has been performed in different trauma centers, comparisons in the literature are difficult. The authors define "no signs of life" as no detectable blood pressure, respiratory or motor effort, cardiac electrical activity, or pupillary activity (ie, clinical death). Patients with "no vital signs" have no palpable blood pressure but demonstrate electrical activity, respiratory effort, or pupillary reactivity.

PHYSIOLOGIC RATIONALE FOR RESUSCITATIVE THORACOTOMY

The primary objectives of RT are to (1) release pericardial tamponade, (2) control cardiac hemorrhage, (3) control intrathoracic bleeding, (4) evacuate bronchovenous air embolism, (5) perform open cardiac massage, and (6) temporarily occlude the descending thoracic aorta. Combined, these objectives attempt to address the primary issue of cardiovascular collapse from mechanical sources or extreme hypovolemia.

Release Pericardial Tamponade and Control Cardiac Hemorrhage

The highest survival rate following RT is in patients with penetrating cardiac wounds, especially when associated with pericardial tamponade (See Atlas Figure 25).[8] Early recognition of cardiac tamponade, prompt pericardial decompression, and control of cardiac hemorrhage are the key components to successful RT and patient survival following penetrating wounds to the heart (see Chapter 30).[18] The egress of blood from the injured heart, regardless of mechanism, results in tamponade physiology. In acute conditions with a nondistendable pericardium, only 100 to 150 mL of pericardial blood results in tamponade. Rising intrapericardial pressure produces abnormalities in hemodynamic and cardiac perfusion that can be divided into three phases.[19] Initially, increased pericardial pressure restricts ventricular diastolic filling and reduces subendocardial blood flow.[20] Cardiac output under these conditions is maintained by compensatory tachycardia, increased systemic vascular resistance, and elevated central pressure (ie, ventricular filling pressure). In the intermediate phase of tamponade, rising pericardial pressure further compromises diastolic filling, stroke volume, and

coronary perfusion, resulting in diminished cardiac output. Although blood pressure may be maintained deceptively well, subtle signs of shock (eg, anxiety, diaphoresis, and pallor) become evident. During the final phase of tamponade, compensatory mechanisms fail as the intrapericardial pressure approaches the ventricular filling pressure. Cardiac arrest ensues as profound coronary hypoperfusion occurs.

The classic description of clinical findings, Beck's triad (muffled heart sounds, distended neck veins, and hypotension), is rarely observed in the ED; therefore, a high index of suspicion in the at-risk patient sustaining penetrating torso trauma is crucial, with prompt intervention essential. In the first two phases of cardiac tamponade, patients may be aggressively managed with volume resuscitation to increase preload and pericardial drainage (via pericardiocentesis, pericardial window, or median sternotomy). The patient in the third phase of tamponade, with profound hypotension (systolic blood pressure [SBP] <60 mm Hg), should undergo RT rather than pericardiocentesis as the management for evacuation of pericardial blood. Following release of tamponade, the source of tamponade can be directly controlled with appropriate interventions based on the underlying injury (see later section titled Technical Details of Resuscitative Thoracotomy).[21]

Control Intrathoracic Hemorrhage

Life-threatening intrathoracic hemorrhage occurs in less than 5% of patients following penetrating injury presenting to the ED, and in an even lower percentage of patients sustaining blunt trauma.[22] The most common injuries include penetrating wounds to the pulmonary hilum and great vessels; less commonly seen are torn descending thoracic aortic injuries with frank rupture or penetrating cardiac wounds exsanguinating into the thorax through a traumatic pericardial window. There is a high mortality rate for injuries to the pulmonary hilar vasculature or thoracic great vessels due to the lack of hemorrhage containment by adjacent tissue tamponade or by lack of vessel spasm (see Chapters 29 and 30). Either hemithorax can rapidly accommodate more than half of a patient's total blood volume before overt physical signs of hemorrhagic shock occur. Therefore, a high clinical suspicion is warranted in patients with penetrating torso trauma, particularly in those with hemodynamic decompensation. Patients with exsanguinating wounds require RT with rapid control of the source of hemorrhage if they are to be salvaged.

Perform Open Cardiac Massage

External chest compression provides approximately 20% to 25% of baseline cardiac output, with 10% to 20% of normal cerebral perfusion.[23] This degree of vital organ perfusion can provide reasonable salvage rates for 15 minutes, but few normothermic patients survive 30 minutes of closed-chest compression. Moreover, in models of inadequate intravascular volume (hypovolemic shock) or restricted ventricular filling (pericardial tamponade), external chest compression fails to augment arterial pressure or provide adequate systemic perfusion; the associated low diastolic volume and pressure result in inadequate coronary perfusion.[24] Therefore, closed cardiac massage is ineffective for postinjury cardiopulmonary arrest. The only potential to salvage the injured patient with ineffective circulatory status is immediate RT with subsequent open cardiac massage.

Achieve Thoracic Aortic Cross-Clamping

The rationale for temporary thoracic aortic occlusion in the patient with massive hemorrhage is twofold (See Atlas Figure 24). First, in patients with hemorrhagic shock, aortic cross-clamping redistributes the patient's limited blood volume to the myocardium and brain.[10] Second, patients sustaining intra-abdominal injury may benefit from aortic cross-clamping due to reduction in subdiaphragmatic blood loss.[9] Temporary thoracic aortic occlusion augments aortic diastolic and carotid SBP, enhancing coronary as well as cerebral perfusion.[25] Experimental work has shown that the left ventricular stroke-work index and myocardial contractility increase in response to thoracic aortic occlusion during hypovolemic shock.[26] These improvements in myocardial function occur without an increase in the pulmonary capillary wedge pressure or a significant change in systemic vascular resistance. Thus, improved coronary perfusion resulting from an increased aortic diastolic pressure presumably accounts for the observed enhancement in contractility.[27]

These experimental observations suggest that temporary aortic occlusion is valuable in patients with either shock due to nonthoracic trauma or continued shock following the repair of cardiac injuries or other thoracic exsanguinating wounds. Indeed, occlusion of the descending thoracic aorta appears to increase the return of spontaneous circulation following CPR.[28] Reports of successful resuscitation using RT in patients in hemorrhagic shock exist, including patients sustaining cardiac arrest due to exsanguinating extremity or cervical injuries.[29] In these situations, RT may be a temporizing measure until the patient's circulating blood volume can be replaced by blood product transfusion. However, once the patient's blood volume has been restored, the aortic cross-clamp should be removed. Thoracic cross-clamping in the normovolemic patient may be deleterious

because of increased myocardial oxygen demands resulting from the increased systemic vascular resistance.[30] Careful application of this technique is warranted as there is substantial metabolic cost and a finite risk of paraplegia associated with the procedure.[31-33] However, in carefully selected patients, aortic cross-clamping may effectively redistribute the patient's blood volume until external replacement and control of the hemorrhagic source are possible. Complete removal of the aortic cross-clamp or replacement of the clamp below the renal vessel should be performed within 30 minutes; the viscera's tolerance of normothermic ischemia is 30 to 45 minutes. As mentioned previously, there may be a role for REBOA in the patient with ongoing pelvic bleeding from either pelvic fracture or gunshot wound.

Evacuate Bronchovenous Air Embolism

Bronchovenous air embolism can be a subtle entity following thoracic trauma and may be much more common than is recognized.[34-36] The clinical scenario typically involves a patient sustaining penetrating chest injury who precipitously develops profound hypotension or cardiac arrest following endotracheal intubation and positive-pressure ventilation. Traumatic bronchovenous communications produce air emboli that migrate to the coronary arterial systems; any impedance in coronary blood flow causes myocardial ischemia and resultant shock. The production of air emboli is enhanced by the underlying physiology—there is relatively low intrinsic pulmonary venous pressure due to associated intrathoracic blood loss and high bronchoalveolar pressure from assisted positive-pressure ventilation. This combination increases the gradient for air transfer across bronchovenous channels.[37] Although more often observed in penetrating trauma, a similar process may occur in patients with blunt lacerations of the lung parenchyma (see Chapter 28).

Immediate thoracotomy with pulmonary hilar cross-clamping prevents further propagation of pulmonary venous air embolism. Pericardotomy provides access to the coronaries and cardiac ventricles; with the patient in the Trendelenburg position (done to trap to air in the apex of the left ventricle), needle aspiration is performed to remove air from the cardiac chamber. Additionally, vigorous cardiac massage, done by running a finger along the coronary vessel, may promote dissolution of air already present in the coronary arteries.[36] Aspiration of the aortic root is done to alleviate any accumulated air pocket, and direct needle aspiration of the right coronary artery may be effective.

CLINICAL RESULTS FOLLOWING RESUSCITATIVE THORACOTOMY

The value of RT in resuscitation of the patient in profound shock who is not yet dead is unquestionable. Its indiscriminate use, however, renders it a low-yield and high-cost procedure.[15,38-40] In the past three decades, there has been a significant clinical shift in the performance of RT, from a nearly obligatory procedure before declaring any trauma patient dead to very few patients undergoing RT. During this swing of the pendulum, several groups have attempted to elucidate the clinical guidelines for RT. In 1979, we conducted a critical analysis of 146 consecutive patients undergoing RT and suggested a selective approach to its use in the moribund trauma patient, based on consideration of the following variables: (1) location and mechanism of injury, (2) signs of life at the scene and on presentation to the ED, (3) cardiac electrical activity at thoracotomy, and (4) SBP response to thoracic aortic cross-clamping.[40]

To further delineate these clinical parameters, we established a prospective study in which these data were carefully documented in all patients at the time of thoracotomy. In 1982, the first 400 patients were analyzed.[39] In 1998, the second analysis of 868 patients was reported.[41] The most recent review has summarized the data on 1708 patients who have undergone RT at the Denver Health Medical Center.[42] Of these, 1289 (78%) were dead in the ED, 272 (16%) died in the OR, and 41 (2%) succumbed to multiple organ failure in the surgical ICU. Ultimately, 106 patients (6%) survived. Although, this yield may seem low, it is important to emphasize that thoracotomy was done on virtually every trauma patient delivered to the ED in the early years of the study period. Additionally, 79% were without vital signs in the field and underwent prehospital CPR. In contrast, it is equally important to stress that patients without signs of life at the scene but who responded favorably to resuscitation were excluded from this analysis because they did not require RT; these patients remind the practitioner that prehospital clinical assessments may not always be reliable in triaging these severely injured patients. Indeed, the authors have salvaged a number of individuals sustaining blunt and penetrating trauma who were assessed to have no signs of life at the scene of injury. The importance of guidelines for RT is evident in the improved survival rate in recent years: in 1974 to 1979, survival was only 5%, compared to 14% survival in 2010 to 2014.[42]

The survival rate and percentage of neurologic impairment following RT vary considerably, due to the heterogeneity of patient populations reported in the literature. As previously discussed, critical determinants of survival include the mechanism and anatomic location of injury and the patient's physiologic condition at the time of thoracotomy.[43-46] We have attempted to elucidate the impact of these factors in ascertaining the success rate of RT

by collating data from a number of clinical series reporting on 50 or more patients (Table 17-1). Unfortunately, inconsistencies in patient stratification and a paucity of clinical details limit objective analysis of these data. Although some reviews provide a specific breakdown of the injury mechanism and clinical status of patients presenting to the ED, others combine all injury mechanisms. We believe it is crucial to stratify patients according to the anatomic location (cervical/extremity vs torso) and mechanism of injury (penetrating vs blunt), as well as the presence of signs of life (ie, blood pressure, respiratory effort, cardiac electrical activity, and pupillary activity).

TABLE 17-1:

Outcome Following Emergency Department Thoracotomy in Adults

Injury pattern	Shock	No vital signs	No signs of life	Total
Cardiac				
Denver[91]	3/9 (33%)	0/7 (0%)	1/53 (2%)	4/69 (6%)
Detroit[92]	9/42 (21%)	3/110 (3%)		12/152 (8%)
Johannesburg[93]				13/108 (12%)
Los Angeles[94]	2/5 (40%)	6/11 (55%)	2/55 (4%)	10/71 (14%)
New York[95]	7/20 (35%)	18/53 (32%)	0/18 (0%)	24/91 (26%)
San Francisco[45]	18/37 (49%)	0/25 (0%)		18/63 (29%)
Seattle[96]	4/11 (36%)	11/47 (23%)		15/58 (26%)
Overall	43/124 (35%)	47/254 (19%)	4/126 (3%)	96/612 (16%)
Penetrating				
Denver[41]	19/78 (24%)	14/399 (4%)		33/477 (7%)
Detroit[92]	9/42 (21%)	3/110 (3%)		12/152 (8%)
Houston[97]	14/156 (9%)	18/162 (11%)		32/318 (10%)
Indianapolis[98]	3/7 (43%)	1/50 (2%)	0/80 (0%)	4/137 (3%)
Johannesburg[93]	31/413 (8%)	10/149 (7%)	1/108 (1%)	42/670 (6%)
Los Angeles[94]	2/5 (40%)	6/11 (55%)	2/55 (4%)	10/71 (14%)
New York[99]	8/32 (25%)	8/77 (10%)	0/25 (0%)	16/134 (12%)
Oakland[100]	8/24 (33%)		2/228 (1%)	10/252 (4%)
San Francisco[45]				32/198 (30%)

Seattle[96]	4/11 (36%)	11/47 (23%)		15/58 (25%)
Washington[101]	7/13 (54%)	3/47 (6%)		10/60 (17%)
Toronto[15]				3/96 (3%)
Denver[42]				11/88 (12.5%)
Overall	145/1007 (14%)	100/1252 (8%)	6/615 (1%)	297/3170 (9%)
Blunt				
Denver[41]	4/86 (5%)	4/311 (1%)		8/397 (2%)
Houston[97]	0/42 (0%)	0/27 (0%)		0/69 (0%)
Johannesburg[93]	1/109 (1%)	0/39 (0%)	0/28 (0%)	1/176 (1%)
San Francisco[45]				1/60 (2%)
Seattle[102]				1/88 (1%)
Denver[42]				13/89 (15%)
Overall	5/237 (2%)	4/377 (1%)	0/28 (0%)	24/879 (2.7%)

The data summarized to date confirm that RT has the highest survival rate following isolated cardiac injury (Table 17-1). An average of 35% of adult patients presenting in shock, defined as an SBP less than 70 mm Hg, and 20% without vital signs were salvaged after isolated penetrating injury to the heart if RT was performed. In contrast, 1% to 15% of blunt trauma patients undergoing RT survive. Following penetrating torso injuries, 14% of patients requiring RT are salvaged if they are hypotensive with detectable vital signs, whereas 8% of those who have no vital signs but have signs of life at presentation and 1% of those without signs of life are salvaged. These findings are reiterated by a recent report incorporating all patients undergoing RT for either blunt or penetrating mechanism from 24 separate studies[44]; survival rates for patients undergoing RT for penetrating injuries and blunt mechanisms were 8.8% and 1.4%, respectively. Additionally, more patients survive RT for isolated cardiac wounds (19.4%), followed by stab wounds (16.8%) and gunshot wounds (4.3%).

Although there is a clear role for RT in the patient presenting in shock but with measurable vital signs, there is disagreement about its use in the patient population undergoing CPR prior to arrival in the ED. Although there have been multiple reports with low survival rates and dismal outcomes following prehospital CPR, termination of resuscitation in the field should not be performed in all patients.[47] Our most recent evaluation, spanning 40 years of experience, indicates that RT does play a significant role in the critically injured patient undergoing prehospital CPR.[42] The majority of patients arriving in extremis who survived to discharge sustained a stab wound to the torso, consistent with previous reports. Additionally, the majority of patients were neurologically intact at discharge.

To further define the limits of RT, a prospective, multicenter trial was performed by the Western Trauma Association (WTA).[13] The WTA data substantiate that injury mechanism alone is not a discriminator of futility. Specifically, with the exception of an overtly devastating head injury, blunt trauma does not prohibit meaningful survival, even with requirements for CPR. This multicenter experience, combined with prior reports, suggests current indications for RT (Table 17-2). RT should be performed for penetrating nontorso trauma with CPR of less than 5 minutes, blunt trauma with CPR of less than 10 minutes, and penetrating torso trauma with CPR of less than 15 minutes. RT is unlikely to yield productive survival when patients (1) sustain blunt trauma and require more than 10 minutes of prehospital CPR, (2) have penetrating wounds and undergo more than 15 minutes of

prehospital CPR, (3) have isolated extremity trauma with more than 5 minutes of prehospital CPR, or (4) manifest asystole without pericardial tamponade. We recognize, however, that there will invariably be exceptions to the recorded literature.[29,43,48,49]

TABLE 17-2:

Current Indications and Contraindications for Emergency Department Thoracotomy

Indications:
Salvageable postinjury cardiac arrest
Penetrating thoracic trauma patients with <15 min of prehospital CPR
Penetrating nonthoracic trauma patients with <5 min of prehospital CPR
Blunt trauma patients with <10 min of prehospital CPR
Persistent severe postinjury hypotension (SBP <60 mm Hg) due to:
Cardiac tamponade
Hemorrhage: intrathoracic, intra-abdominal, extremity, cervical
Air embolism
Contraindications:
CPR >15 min following penetrating injury and no signs of life (pupillary response, respiratory effort, or motor activity)
CPR >10 min following blunt injury and no signs of life
Asystole is the presenting rhythm and there is no pericardial tamponade

CPR, cardiopulmonary resuscitation; SBP, systolic blood pressure.

Emerging data indicate the clinical results in the pediatric population mirror those of the adult experience for penetrating injury (Table 17-3). One might anticipate that children would have a more favorable outcome compared to adults; however, this has not been borne out in multiple studies.[50-56] A recent review of RT in the pediatric population encompassed eight published reports with 2336 pediatric trauma patients[57]; 269 RTs were performed in this study population. Overall survival rate was 1.7% in blunt trauma and 14% following RT for penetrating trauma. According to this review, in the past 25 years, no published series of RT has demonstrated a survivor following blunt trauma in the pediatric age group. In the published literature over the past four decades, only two blunt trauma patients are reported to have undergone RT and survived; each of these patients was over the age of 15. Beaver et al[52] reported no survivors among 27 patients, from 15 months to 14 years of age, undergoing postinjury RT at Johns Hopkins Hospital. Powell et al,[55] at the South Alabama Medical Center, described an overall survival of 20% (3 of 15 patients) in patients ranging from 4 to 18 years old. A review using the National Trauma Data Bank of all pediatric blunt trauma patients with no sign of life in the field identified a 1.3% survival rate for those undergoing RT.[56] In a study at Denver Health Medical Center, encompassing an 11-year experience with 689 consecutive RTs, we identified 83 patients (12%) who were under 18 years old.[53] Survival by injury mechanism was 9% (1 of 11 patients) for stab wounds, 4% (1 of 25 patients) for gunshot wounds, and 2% (1 of 47 patients) for blunt trauma. Among 69 patients presenting to the ED without vital signs, only 1 patient (1%) survived (with a stab wound). This contrasted to a salvage of 2 (14%) of 14 patients with vital signs. The outcome in blunt trauma, the predominant mechanism of lethal injury in children, was disappointing, with only 2% salvage, and there were no survivors when vital signs were absent. A more recent analysis spanning 40 years identified a similar rate of RT in the pediatric population[58]; of 179 trauma patients undergoing RT, there were 6 survivors (3.4%). In the adolescent age group (15–18 years old), survival rate was 4.8%, whereas in the group younger than age 15 years, there were no survivors. Thus, as in adults, outcome following RT in the adolescent population is largely determined by injury mechanism and physiologic status on presentation to the ED; for patients under 15 years of age, there are only isolated cases of survival following penetrating trauma and no apparent survival benefit following blunt trauma.

TABLE 17-3:

Outcome Following Emergency Department Thoracotomy in Children

Injury pattern	Shock	No vital signs	No signs of life	Total
Penetrating				
Baltimore[52]		0/2 (0%)		0/2 (0%)
Denver[53]	1/3 (33%)	1/5 (20%)	0/28 (0%)	2/36 (6%)
Mobile[55]	0/1 (0%)	3/9 (33%)		3/10 (30%)
Sacramento[54]	1/4 (25%)	0/4 (0%)		1/8 (13%)
Overall	2/8 (25%)	4/20 (20%)	0/28 (0%)	6/56 (11%)
Blunt				
Baltimore[52]		0/15 (0%)		0/15 (0%)
Denver[53]	1/11 (9%)	0/6 (0%)	0/30 (0%)	1/47 (2%)
Mobile[55]		0/5 (0%)		0/5 (0%)
Sacramento[54]	0/6 (0%)	0/9 (0%)		0/15 (0%)
NTDB[56]			6/499 (1.3%)	6/499 (1.3%)
Europe[50]	1/17 (6%)	0/10 (0%)		0/10 (0%)
Overall		0/35 (0%)	6/529 (1%)	7/591 (1.2%)

NTDB, National Trauma Data Bank.

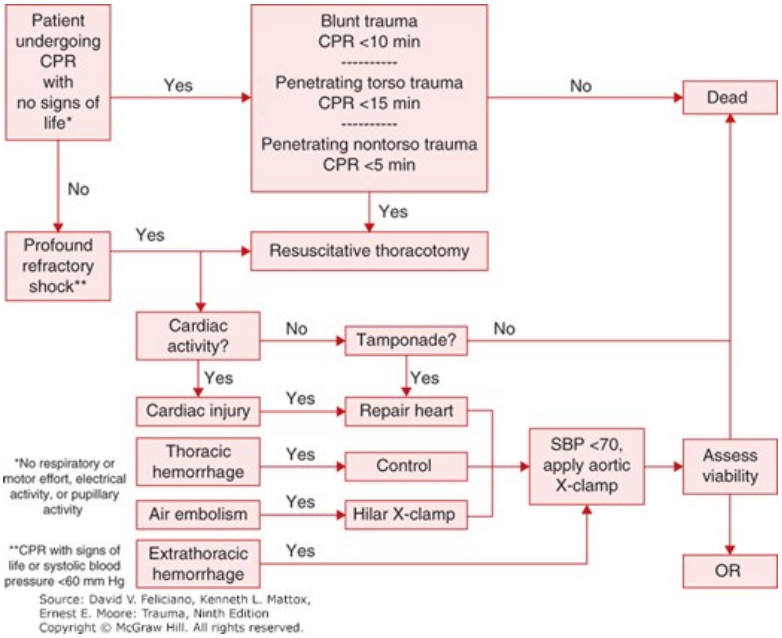
In sum, overall analysis of the available literature indicates that the success of RT approximates 35% in the patient arriving in shock with a penetrating cardiac wound and 15% for all penetrating wounds. Patients undergoing CPR upon arrival to the ED should be stratified based on injury and transport time to determine the utility of RT. Conversely, patient outcome is relatively poor when RT is done for blunt trauma: 2% survival in patients in shock and less than 1% survival with no vital signs. A clear understanding of the indications for RT combined with available expertise will hopefully improve compliance with commonly accepted clinical practice and result in reduced mortality rates.[59]

INDICATIONS FOR RESUSCITATIVE THORACOTOMY

Based on our successive years of RT prospective analysis[42] and the recent WTA multicenter trial,[12] we propose current indications for RT (Table 17-2). Clearly, the specific application of these guidelines must include signs of life, mechanism of injury, and logistic issues such as qualified personnel. Our current decision algorithm for resuscitation of the moribund trauma patient and use of RT was formulated based on these analyses (Fig. 17-1). At the scene, patients in extremis without electrical cardiac activity are declared dead.[60] Patients in extremis but with electrical cardiac activity are intubated, supported with cardiac compression, and rapidly transported to the ED.

FIGURE 17-1

Algorithm directing the use of resuscitative thoracotomy in the multiply injured trauma patient. CPR, cardiopulmonary resuscitation; OR, operating room; X-clamp, cross-clamp.



On arrival to the ED, the time from initiation of CPR is reported by the prehospital personnel to the trauma surgeon; blunt trauma patients with greater than 10 minutes of prehospital CPR and no signs of life are declared, whereas penetrating torso trauma patients with greater than 15 minutes or isolated extremity/cervical trauma patients with greater than 5 minutes of prehospital CPR and no signs of life are pronounced. Patients within the time guidelines or those with signs of life trigger ongoing resuscitation and RT. After performing a generous left anterolateral thoracotomy and subsequent pericardotomy, the patient's intrinsic cardiac activity is evaluated. Patients in asystole without associated cardiac tamponade are declared. Patients with a cardiac wound, tamponade, and associated asystole are aggressively treated; the cardiac wound is repaired first, followed by manual cardiac compressions, intracardiac injection of epinephrine, and defibrillation. Following treatment and blood product resuscitation, one should reassess salvageability, typically defined as the patient's ability to generate an SBP greater than 70 mm Hg.

Patients with an intrinsic rhythm following RT should be treated according to underlying pathology. Those with tamponade should undergo cardiac repair, either in the trauma bay or in the OR (see Chapter 30). Control of intrathoracic hemorrhage may entail hilar cross-clamping, digital occlusion of the direct injury, or even packing of the apices for subclavian injuries. Treatment of bronchovenous air embolism includes cross-clamping of the hilum, putting the patient in Trendelenberg position, aspirating the left ventricle and aortic root, and massaging the coronaries. Finally, cross-clamping of the descending aorta is performed to decrease the required effective circulating volume, increase cerebral and coronary blood flow, reduce infradiaphragmatic blood loss, and facilitate resuscitation. In all of these scenarios, reassessment of the patient following intervention and aggressive resuscitation efforts is performed, with the goal SBP of 70 mm Hg used to define salvageability.

TECHNICAL DETAILS OF RESUSCITATIVE THORACOTOMY

The optimal benefit of RT is achieved by a surgeon experienced in the management of intrathoracic injuries. The emergency physician, however, should not hesitate to perform the procedure in the moribund patient with a penetrating chest wound when thoracotomy is the only means of salvage. The technical skills needed to perform the procedure include the ability to perform a rapid thoracotomy, pericardiotomy, cardiorrhaphy, and thoracic aortic cross-clamping; familiarity with vascular repair techniques and control of the pulmonary hilum are advantageous. Once life-threatening intrathoracic injuries are controlled or temporized, the major challenge is restoring the patient's hemodynamic integrity and minimizing vital organ reperfusion injury.

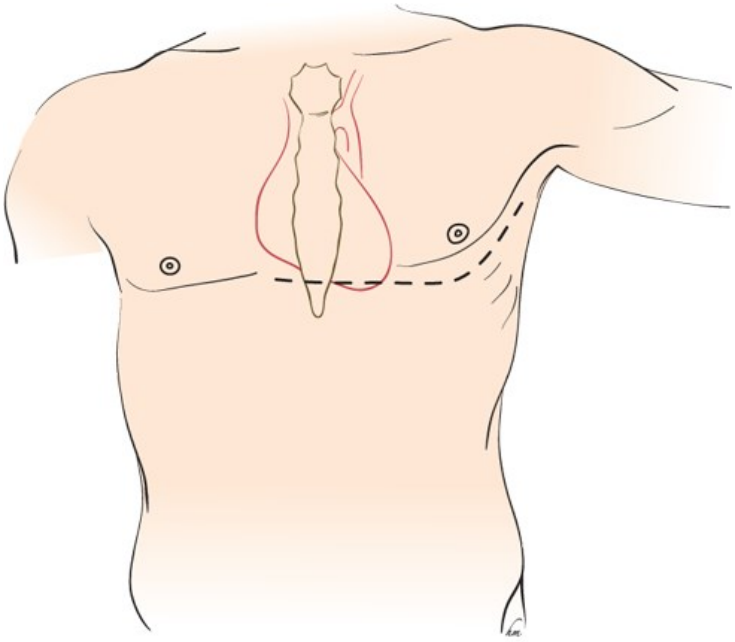
Thoracic Incision

A left anterolateral thoracotomy incision is preferred for RT. Advantages of this incision in the critically injured patient include (1) rapid access with simple instruments, (2) the ability to perform this procedure on a patient in the supine position, and (3) easy extension into the right hemithorax, a clamshell thoracotomy, for exposure of both pleural spaces and anterior and posterior mediastinal structures. The key resuscitative maneuvers of RT, namely, pericardiotomy, open cardiac massage, and thoracic aortic cross-clamping, are readily accomplished via this approach. The initial execution of a clamshell thoracotomy should be done in hypotensive patients with penetrating wounds to the right chest. This provides immediate, direct access to a right-sided pulmonary or vascular injury while still allowing access to the pericardium from the left side for open cardiac massage. Clamshell thoracotomy may also be considered in patients with presumed air embolism, providing access to the cardiac chambers for aspiration, coronary vessels for massage, and bilateral lungs for obliteration of the source.

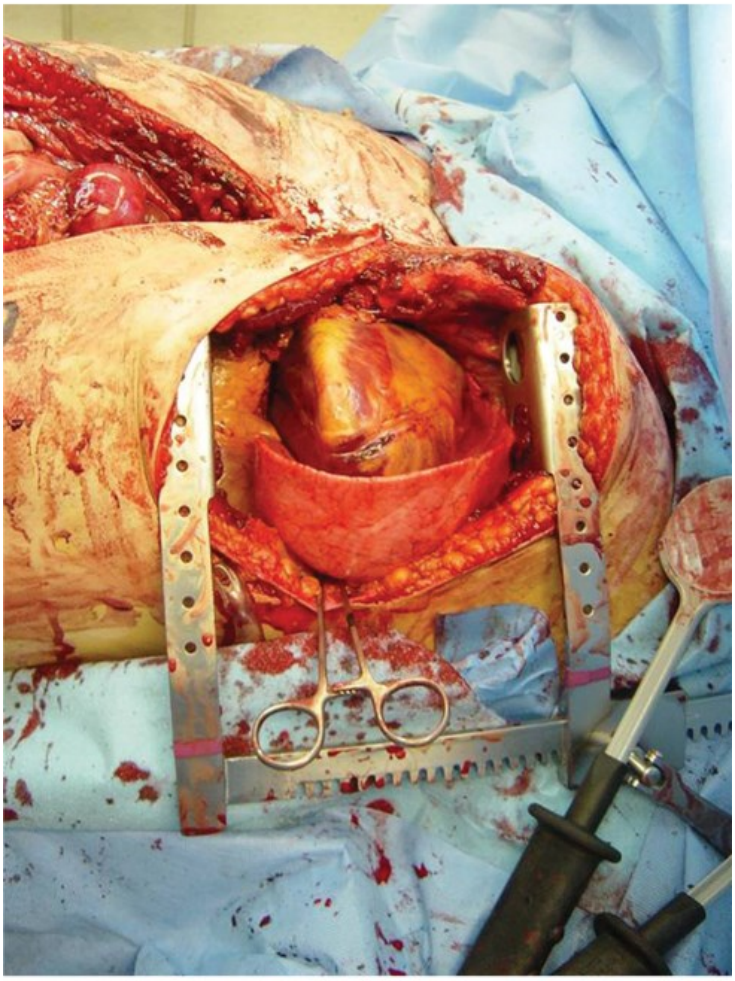
Preparation for RT should be performed well ahead of the patient's arrival. Set-up should include a 10-blade scalpel, Finochietto chest retractor, toothed forceps, curved Mayo scissors, Satinsky vascular clamps (large and small), long needle holder, Lebsche knife and mallet, and internal defibrillator paddles. Sterile suction, skin stapler, and access to a variety of sutures should be available (specifically 3-0 Prolene on a CT-1 needle, 3-0 silk ties, and Teflon pledgets). Upon patient arrival and determination of the need for RT, the patient's left arm should be placed above the head to provide unimpeded access to the left chest. The anterolateral thoracotomy is initiated with an incision at the level of the fifth intercostal space (Fig. 17-2). Clinically, this level for incision corresponds to the inferior border of the pectoralis major muscle, just below the patient's nipple. In women, the breast should be retracted superiorly to gain access to this interspace, and the incision is made at the inframammary fold. The incision should start on the right side of the sternum; if sternal transection is required, this saves the time-consuming step of performing an additional skin incision. As the initial incision is carried transversely across the chest and one passes beneath the nipple, a gentle curve in the incision toward the patient's axilla rather than direct extension to the bed should be performed; this curvature in the skin correlates with the natural curvature of the rib cage. The skin, subcutaneous fat, and chest wall musculature are incised with a knife to expose the ribs and associated intercostal space. Intercostal muscles and the parietal pleura are then divided in one layer with curved Mayo scissors; the intercostal muscle should be divided along the superior margin of the rib to avoid the intercostal neurovascular bundle. Chest wall bleeding is minimal in these patients and should not be a concern at this point in the resuscitation. Once the incision is completed and the chest entered, a standard Finochietto rib retractor is inserted, with the handle directed inferiorly toward the axilla (Fig. 17-2) (see Atlas Figures 22 and 23). Placement of the handle toward the bed rather than the sternum allows extension of the left thoracotomy into a clamshell thoracotomy with crossing of the sternum without replacing the rib retractor.

FIGURE 17-2

(**A, B**) The thoracotomy incision is performed through the fourth or fifth intercostal space; the incision should start to the right of the sternum, and begin curving into the axilla at the level of the left nipple. The Finochietto rib retractor is placed with the handle directed inferiorly toward the bed.



A



B

If the left anterolateral thoracotomy does not provide adequate exposure, several techniques may be employed. The costal cartilages can be readily divided with heavy scissors. Alternatively, the sternum can be transected for additional exposure with a Lebsche knife; care must be taken to hold the Lebsche knife firmly against the underside of the sternum when using the mallet to forcefully transect the sternum, or the tip of the instrument may deviate and result in an iatrogenic cardiac injury. If the sternum is divided transversely, the internal mammary vessels must be ligated when perfusion is restored; this may be performed using either a figure of eight suture with 3-0 silk or by clamping the vessel with a tonsil and individually ligating it with a 3-0 silk tie (Fig. 17-3). A concomitant right anterolateral thoracotomy produces a “clamshell” or “butterfly” incision and achieves wide exposure to both pleural cavities and anterior and posterior mediastinal structures (Fig. 17-4). Once the right pleural space is opened, the rib retractor should be moved to more of a midline position to enhance separation of the chest wall for maximal exposure. When visualization of penetrating wounds in the aortic arch or major branches is needed, the superior sternum is additionally split in the midline.

FIGURE 17-3

(**A, B**) Transverse division of the sternum requires individual ligation of the internal mammary arteries.

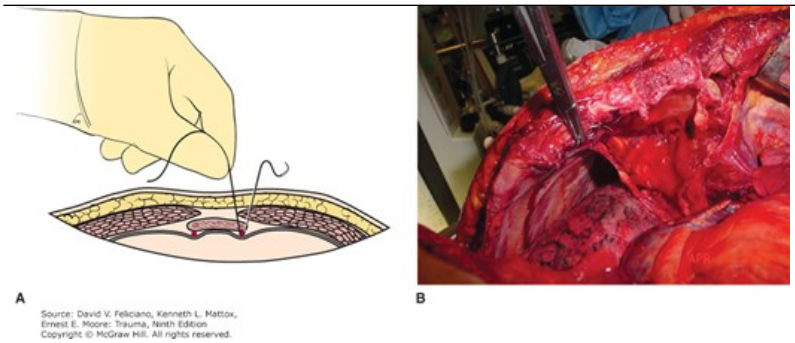
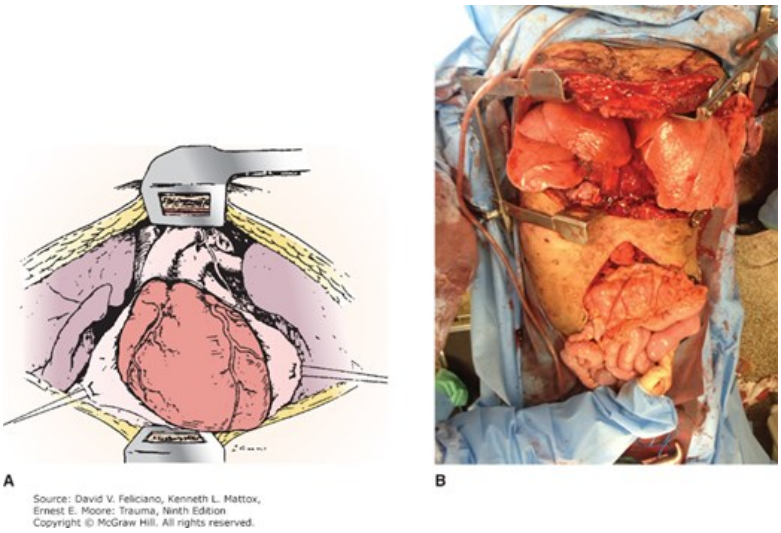


FIGURE 17-4

(**A, B**) A bilateral anterolateral ("clamshell") thoracotomy provides access to both thoracic cavities including the pulmonary hila, heart, and proximal great vessels.

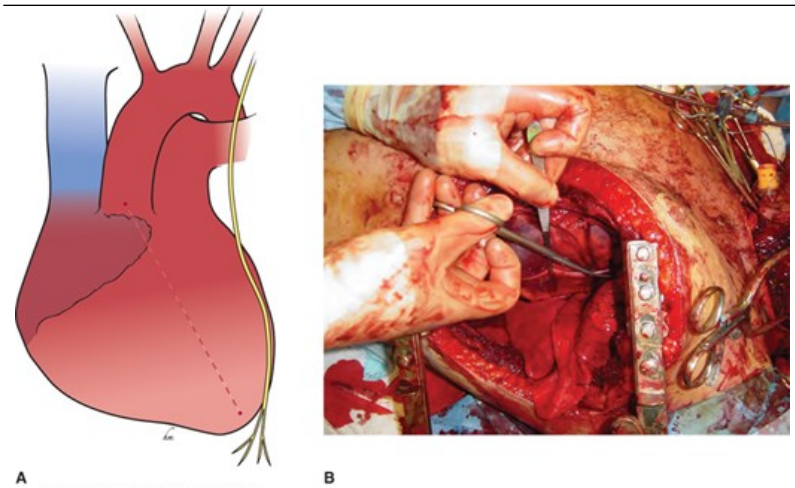


Pericardiotomy and Cardiac Hemorrhage Control

The pericardium is incised widely, starting at the cardiac apex and extending toward the sternal notch, anterior and parallel to the phrenic nerve (Fig. 17-5). If the pericardium is not tense with blood, it may be picked up at the apex with toothed forceps and sharply opened with scissors. If tense pericardial tamponade exists, a knife or the sharp point of a scissors is often required to initiate the pericardiotomy incision. Blood and blood clots should be completely evacuated from the pericardium. The heart should be delivered from the pericardium by placing the right hand through the opening in the pericardium posterior to the heart, encircling the right side of the heart and pulling it into the left chest. This effectively places the left side of the pericardium behind the heart allowing access to the cardiac chambers for repair of cardiac wounds and access for effective open cardiac massage.

FIGURE 17-5

(**A, B**) Pericardiotomy is done with toothed pickups and curved Mayo scissors; the incision begins at the cardiac apex, anterior to the phrenic nerve, and extends on the anterior surface of the heart toward the great vessels.



Prompt hemorrhage control is paramount for a cardiac injury. In the beating heart, cardiac bleeding sites should be controlled immediately with digital pressure on the surface of the ventricle and partially occluding vascular clamps on the atrium or great vessels. Efforts at definitive cardiorrhaphy may be delayed until initial resuscitative measures have been completed. In the nonbeating heart, cardiac repair is done prior to defibrillation and cardiac massage. Cardiac wounds in the thin-walled right ventricle are best repaired with 3-0 nonabsorbable running or horizontal mattress sutures. Buttressing the suture repair with Teflon pledgets is ideal for the thinner right ventricle. When suturing a ventricular laceration, care must be taken not to incorporate a coronary vessel into the repair. In these instances, vertical mattress sutures should be used to exclude the coronary and prevent cardiac ischemia. In the more muscular left ventricle, control of bleeding can occasionally be achieved with a skin-stapling device if the wound is a linear stab wound whose edges coapt in diastole. Low-pressure venous, atrial, and atrial appendage lacerations can be repaired with simple running or purse string sutures of 3-0 Prolene. Posterior cardiac wounds may be particularly treacherous when they require elevation of the heart for their exposure; closure of these wounds is best accomplished in the OR with optimal lighting and equipment. For a destructive wound of the ventricle or for inaccessible posterior wounds, temporary inflow occlusion of the superior and inferior vena cava may be employed to facilitate repair (see Chapter 30). BioGlue may be used as a hemostatic agent in such cases. Use of a Foley catheter for temporary occlusion of cardiac injuries has been suggested; in our experience, this may inadvertently extend the injury due to traction forces.

Advanced Cardiac Life Support Interventions Including Cardiac Massage

The restoration of organ and tissue perfusion may be facilitated by a number of interventions.[61] First, a perfusing cardiac rhythm must be established. Early defibrillation for ventricular fibrillation or pulseless ventricular tachycardia has proven benefit, and evidence supports the use of amiodarone (with lidocaine as an alternative) following epinephrine in patients refractory to defibrillation. Magnesium may be beneficial for torsades de pointes; other dysrhythmias should be treated according to current guidelines.[61] Familiarity with the internal cardiac paddles and appropriate charging dosages in joules are required (Fig. 17-6). In the event of cardiac arrest, bimanual internal massage of the heart should be instituted promptly (Fig. 17-7). We prefer to do this with a hinged clapping motion of the hands, with the wrists apposed, sequentially closing from palms to fingers. The ventricular compression should proceed from the cardiac apex to the base of the heart. The two-handed technique is strongly recommended, as the one-handed massage technique poses the risk of myocardial perforation with the thumb.

FIGURE 17-6

(**A, B**) Internal paddles for defibrillation are positioned on the anterior and posterior aspects of the heart.

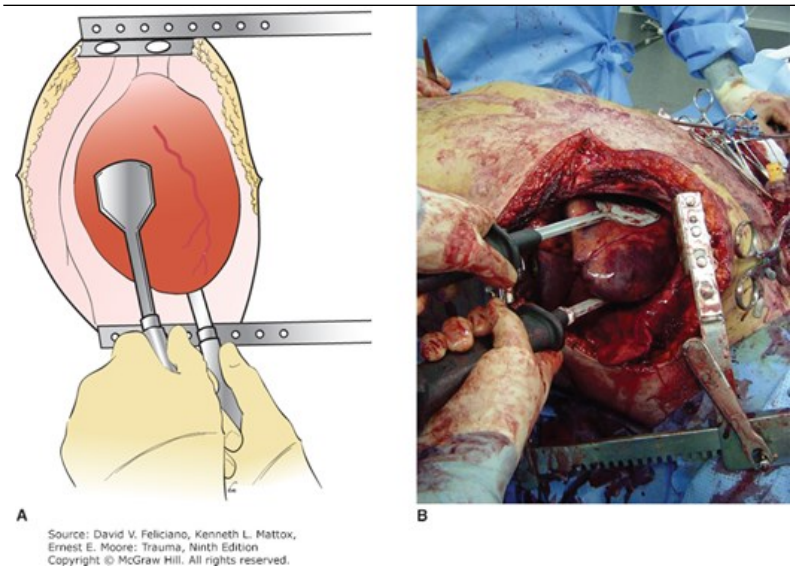
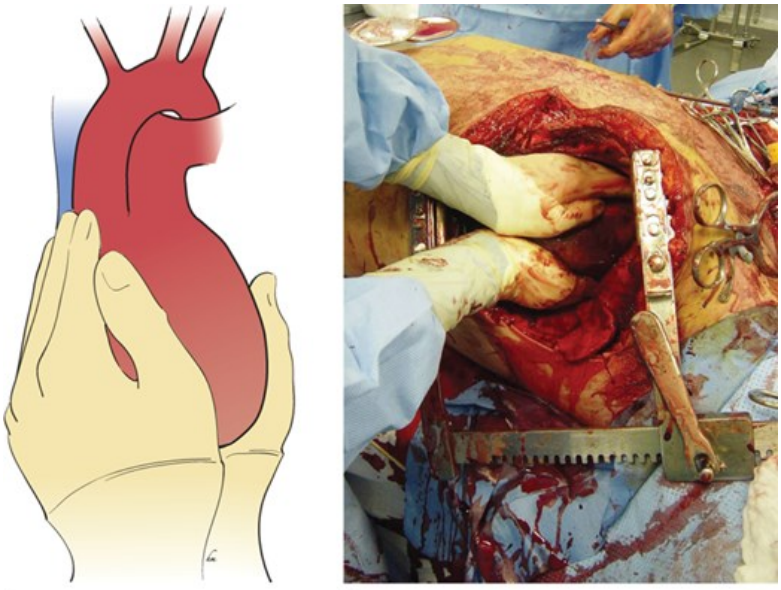


FIGURE 17-7

(**A, B**) Open cardiac massage is performed with a two-handed hinged technique; the clapping motion sequentially closes the hands from palms to fingers.



Pharmacologic adjuncts to increase coronary and cerebral perfusion pressure may be needed. The first agent in resuscitation at this juncture is intracardiac epinephrine. Epinephrine should be administered using a specialized syringe, which resembles a spinal needle, directly into the left ventricle. Typically, the heart is lifted up slightly to expose the more posterior left ventricle, and care is taken to avoid the circumflex coronary during injection. Although epinephrine continues to be advocated during resuscitation, there is a growing body of data suggesting that vasopressin may be superior to epinephrine in augmenting cerebral perfusion and other vital organ blood flow.[62] Administration of calcium, while theoretically deleterious during reperfusion injury, increases cardiac contractility and may be helpful in the setting of hypocalcemia produced by blood component transfusion (see Chapter 16). Although metabolic acidosis is common following RT and resuscitation,[63] the mainstay of therapy is provision of adequate alveolar ventilation and restoration of tissue perfusion. Sodium bicarbonate therapy has not been proven beneficial in facilitating defibrillation, restoring spontaneous circulation, or improving survival. However, it may be warranted following protracted arrest or resuscitation

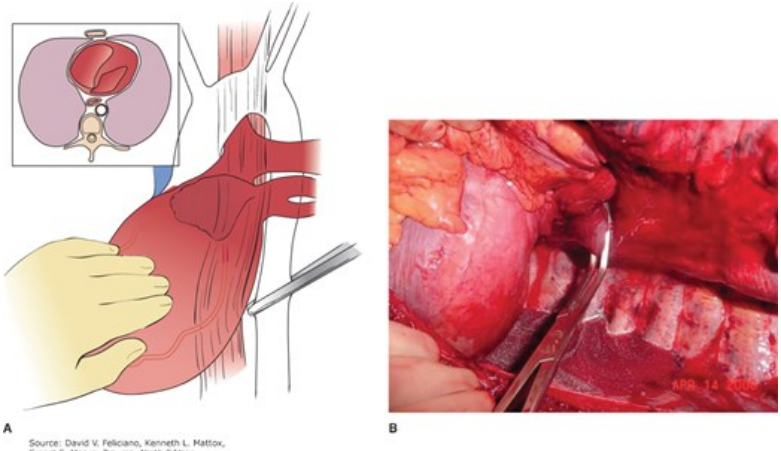
because catecholamine receptors may be desensitized with advanced metabolic acidosis.

Thoracic Aortic Occlusion and Pulmonary Hilar Control

Following thoracotomy and pericardiotomy with evaluation of the heart, the descending thoracic aorta should be occluded to maximize coronary perfusion if hypotension (SBP <70 mm Hg) persists. We prefer to cross-clamp the thoracic aorta inferior to the left pulmonary hilum (Fig. 17-8). Exposure of this area is best provided by elevating the left lung anteriorly and superiorly. Although some advocate taking down the inferior pulmonary ligament to better mobilize the lung, this is unnecessary and risks injury to the inferior pulmonary vein. Dissection of the thoracic aorta is optimally performed under direct vision by incising the mediastinal pleura and bluntly separating the aorta from the esophagus anteriorly and from the prevertebral fascia posteriorly. Care should be taken in dissecting the aorta, and completely encircling it may avulse thoracic and other small vascular branches. Alternatively, if excessive hemorrhage limits direct visualization, which is the more realistic clinical scenario, blunt dissection with one's thumb and fingertips can be done to isolate the descending aorta. Once identified and isolated, the thoracic aorta is occluded with a large Satinsky or DeBakey vascular clamp. If the aorta cannot be easily isolated from the surrounding tissue, digitally occlude the aorta against the spine to effect aortic occlusion. Although occlusion of the thoracic aorta is typically performed after pericardiotomy, this may be the first maneuver upon entry into the chest in patients sustaining extrathoracic injury and associated major blood loss.

FIGURE 17-8

(**A, B**) Aortic cross-clamp is applied with the left lung retracted superiorly, below the inferior pulmonary ligament, just above the diaphragm. The flaccid aorta is identified as the first structure encountered on top of the spine when approached from the left chest.



Control of the pulmonary hilum has two indications. First, if coronary or systemic air embolism is present, further embolism is prevented by placing a vascular clamp across the pulmonary hilum (Fig. 17-9). Associated maneuvers such as vigorous cardiac massage to move air through the coronary arteries and needle aspiration of air from the left ventricular apex and the aortic root are also performed (Fig. 17-10). Second, if the patient has a pulmonary hilar injury or marked hemorrhage from the lung parenchyma, control of the hilum may prevent exsanguination. Hilar control is best performed with a large Satinsky clamp or temporarily with digital control (see Chapter 30).

FIGURE 17-9

(**A, B**) A Satinsky clamp is used to clamp the pulmonary hilum for hemorrhage control or to prevent further bronchovenous air embolism.

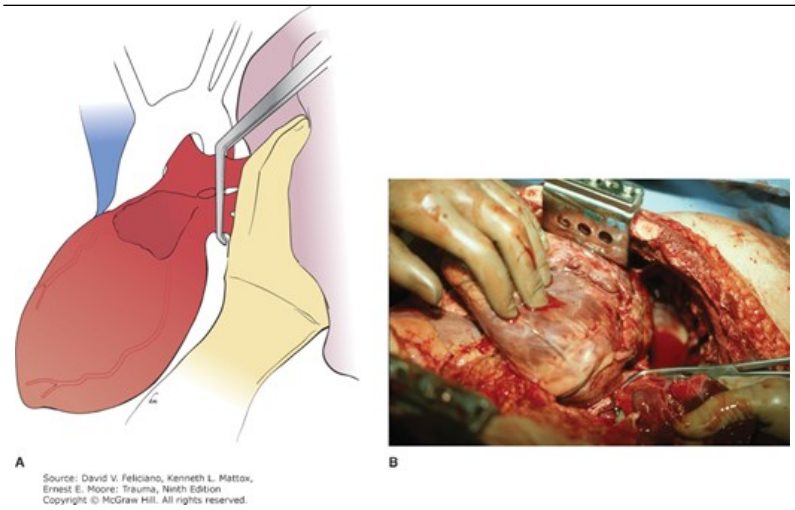
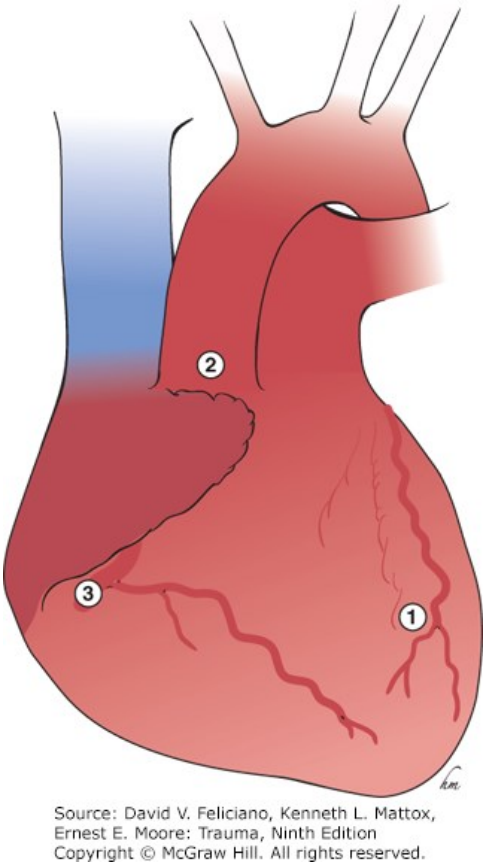


FIGURE 17-10

In cases of bronchovenous air embolism, sequential sites of aspiration include the left ventricle (1), the aortic root (2), and the right coronary artery (3).



COMPLICATIONS AND CONSEQUENCES OF RESUSCITATIVE THORACOTOMY

Procedural Complications

Technical complications of RT involve virtually every intrathoracic structure. The list of such misadventures includes lacerations of the heart, coronary arteries, aorta, phrenic nerves, esophagus, and lungs, as well as avulsion of aortic branches to components of the mediastinum. Previous thoracotomy

virtually assures technical problems from the presence of dense pleural adhesions and is therefore a relative contraindication to RT. Additional postoperative morbidity among ultimate survivors of RT includes recurrent chest bleeding; infection of the pericardium, pleural spaces, sternum, and chest wall; and postpericardiotomy syndrome.

Importantly, there is a finite risk to the health care providers and trauma team performing an RT.[64] The use of RT by necessity involves the rapid use of sharp surgical instruments and exposure to the patient's blood. Even during elective procedures in the OR, the contact rate of patient's blood with the surgeon's skin can be as high as 50%, and the contact rate of patients' blood with health care workers' blood is as high as 60%. The overall seroprevalence rate of HIV among patients admitted to the ED for trauma is around 4%, but is much higher among the subgroup of patients most likely to require an RT, for example, 14% of penetrating trauma victims and nearly 30% of intravenous drug abusers. Caplan et al[65] found that 26% of acutely injured patients had evidence of exposure to HIV (4%), hepatitis B (20%), or hepatitis C virus (14%); there was no difference in the incidence comparing blunt to penetrating trauma. Thus, the likelihood of a health care worker sustaining exposure to HIV or hepatitis in the ED is substantial. The risk of contagion from exposures to HIV and other bloodborne pathogens can be minimized by the use of appropriate barrier precautions, minimizing the use of needles and scalpels, and selective use of RT.

Hemodynamic and Metabolic Consequences of Aortic Cross-Clamping

Aortic cross-clamping may be lifesaving during acute resuscitation, but there is a finite cost to the patient. Occlusion of the aorta results in an increase in blood pressure, but there is an associated 90% reduction in femoral artery SBP; in addition, abdominal visceral blood flow decreases to 2% to 8% of baseline values.[32,33] Therefore, cross-clamping magnifies the metabolic cost of shock by reducing local blood flow to abdominal viscera even further. This results in tissue acidosis and increased oxygen debt, and may ultimately contribute to postischemic multiple organ failure.[33] Additionally, return of aortic flow may not result in normalization of flow to vital organs; in animal models, blood flow to the kidneys remained at 50% of baseline despite a normal cardiac output. The metabolic penalty of aortic cross-clamping becomes exponential when the normothermic occlusion time exceeds 30 minutes, both in trauma and in elective thoracic aortic procedures.[66,67] Hypoxia of distal organs, white blood cells, and endothelium induces the elaboration, expression, and activation of inflammatory cell adhesion molecules and inflammatory mediators; this systemic inflammatory response syndrome has been linked to impaired pulmonary function and multiple organ failure[68] (see Chapter 63). Consequently, the aortic clamp should be removed as soon as effective cardiac function and adequate systemic arterial pressure have been achieved.

Removal of aortic occlusion may result in further hemodynamic sequelae.[69] Besides the abrupt reperfusion of the ischemic distal torso and washout of metabolic products and inflammatory mediators associated with aortic declamping, there are direct effects on the cardiopulmonary system. The return of large volumes of blood from the ischemic extremities, with its lower pH, elevated lactate, and other mediators, may exert a cardiodepressant activity on myocardial contractility.[70] Overzealous volume loading during aortic occlusion may also result in ventricular strain, acute atrial and ventricular dilatation, and consequently, precipitous cardiac failure.[34] Following release of aortic occlusion, there is impaired left ventricular function, systemic oxygen utilization, and coronary perfusion pressure in the postresuscitation period.[27,68] The transient fall in coronary perfusion may not be clinically relevant in patients with efficient coronary autoregulation; however, in patients with coronary disease or underlying myocardial hypertrophy, this increase in cardiac work may result in clinically critical ischemia.[27]

OPTIMIZING OXYGEN TRANSPORT FOLLOWING RT

Following RT, patients are frequently in a tenuous physiologic state. The combination of direct cardiac injury, ischemic myocardial insult, myocardial depressants, and pulmonary hypertension adversely impact postinjury cardiac function (see Chapter 58). Additionally, aortic occlusion induces profound anaerobic metabolism, secondary lactic academia, and release of other reperfusion-induced mediators. Consequently, once vital signs return, the resuscitation priorities shift to optimizing cardiac function and maximizing oxygen delivery to the tissues. The ultimate goal of resuscitation is adequate tissue oxygen delivery and cellular oxygen consumption (see Chapter 57). Circulating blood volume status is maintained at the optimal level of cardiac filling in order to optimize cardiac contractility, and the oxygen-carrying capacity of the blood is maximized by keeping the hemoglobin above 7 to 10 g/dL. If these measures fail to meet resuscitative goals (eg, resolution of base deficit or clearance of serum lactate), inotropic agents are added to enhance myocardial function.

FUTURE CONSIDERATIONS

Defining Nonsalvageability

As clinicians faced with increasing scrutiny over appropriation of resources, it is critical to identify the patient who has permanent neurologic disability or death. Although there may be a widespread belief that survivors of RT have long-term functional or neurologic impairment, a recent analysis reports the majority have no long-term sequelae.[71] Resuscitative efforts should not be abandoned prematurely in the potentially salvageable patient, but field assessment of salvageability is unreliable.[43,72-76] Our clinical pathway attempts to optimize resource utilization, but outcomes must continue to be evaluated, searching for more definitive predictors of neurologic outcome. For example, markers of brain metabolic activity such as increased serum neuron-specific enolase activity appear to have prognostic significance for irreversible brain damage.[77] Although the optimal outcome of RT is undoubtedly the neurologically intact survivor, some have recognized the impact of RT on the potential salvageability of organs for donation.[78] The use of more advanced monitoring devices in the ED, together with further elucidation of the characteristics of irreversible shock, may permit a more physiologic prediction of outcome for these critically injured patients in the future.

Temporary Physiologic Hibernation

A potential adjunct in the care of traumatic arrest is the timely application of hypothermia. Recent randomized studies suggest the use of hypothermia for central nervous system protection after nontraumatic cardiac arrest.[79,80] In these studies, patients randomized to a period of mild-to-moderate hypothermia (32–34°C) after cardiac arrest had improved neurologic outcomes compared to those kept normothermic. This favorable effect was presumably due to a decrease in cerebral metabolic demand during hypothermia. In addition, hypothermia may reduce oxygen radical generation and inflammatory mediator production. By extension, then, if an injured patient in transport could be cooled to a minimal metabolic rate (ie, suspended animation), one can posit that transfer to definitive care might be possible.[81,82] Application of this principle to the multiply injured or bleeding patient, however, is problematic. Rapid cooling is not currently practical in the field, and there are legitimate concerns about the adverse effects of hypothermia on immune function and effective clot formation. Overall, although some[83] preclinical work supports the application of hypothermia after resuscitation from hemorrhage, other investigators have reached the opposite conclusion.[84]

Temporary Mechanical Cardiac Support

The concept of temporary mechanical cardiac support for the failing heart following injury is intuitively attractive. Unfortunately, experience with the intra-aortic balloon pump in this scenario has been unrewarding. The advent of centrifugal pumps (Bio-Medicus Pump; Bio-Medicus, Inc., Minneapolis, MN), which allow partial cardiac bypass without systemic anticoagulation, offers another potential means for increasing salvage of the moribund patient. Centrifugal pumps have become the standard approach for open repair of a torn descending thoracic aorta (see Chapter 30).[85,86] The adjunctive use of extracorporeal membrane oxygenation may also play a critical role in supporting the patient with massive injuries or early multiple organ failure.[87] Finally, hypothermia circulatory arrest may find utility in a broad spectrum of patients with injuries that are considered "irreparable."[88,89] Although a political issue, restoring perfusion in a patient with unsurvivable injuries is a potential source of organs for donation,[90] particularly with kidneys that can tolerate prolonged periods of warm ischemia.

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