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Extreme Measures: Procedures and Therapies for Life-threatening Situations

Introduction

The purpose of this article is to review procedures and therapies used in the difficult, life-threatening clinical circumstances. The authors review five procedures that are rarely used in extreme situations.

Emergency Department Thoracotomy

Emergency department thoracotomy (EDT) has become a well-accepted procedure in penetrating trauma within certain parameters. Its role in blunt trauma remains a matter of great controversy.[1]

Indications for ED Thoracotomy. The primary goals of EDT are to release pericardial tamponade, control hemorrhage, evacuate air emboli, and perform cardiac massage and internal defibrillation in patients sustaining blunt force and penetrating trauma who are too unstable for transport to the operating room. (*See Table 1.*) Indications to perform EDT are based on the physiological status of the patient, injury mechanism, and evidence of ongoing massive hemorrhage despite standard resuscitation measures.[2-7]

Blunt or Penetrating Trauma with Recent Loss of Signs of Life. Certain physiologic criteria must be present to warrant EDT. These include the presence of signs of life at the trauma scene, with loss of signs of life on arrival or in the ED. In penetrating trauma, the loss of signs of life can be extended up to 15 minutes prior to ED arrival, assuming appropriate initiation of CPR without a significant response to volume resuscitation, as evidenced by a systolic blood pressure (SBP) less than 70 mmHg.[2-7] The American College of Surgeons Committee on Trauma defines signs of life as pupillary response, spontaneous ventilation, measurable or palpable blood pressure, and/or cardiac electrical activity.[8] The literature expresses a general consensus that no signs of life in the field and on presentation to the ED is an independent contraindication to EDT; the risk to the health care provider and cost outweigh the benefit.[2,5-7]

Pericardial Tamponade. Regardless of the type of trauma, EDT should be considered in pulseless electrical activity (PEA), asystole, or inadequate cardiac output due to suspected pericardial tamponade.[2,7] This is independent of when the patient lost pulses, given that evacuation may result in return of spontaneous circulation despite greater than 15 minutes of CPR. Anoxic brain injury is likely in this scenario, but should not be the sole factor precluding EDT. Interestingly, according to the American College of Surgeons Committee on Trauma, asystole or PEA upon arrival to the ED is a contraindication to EDT, regardless of the suspicion of pericardial tamponade. However, the Western Trauma Association supports EDT in the setting of pericardial tamponade.[2,7] Prior to performing EDT, a tension pneumothorax should be excluded as the cause of decreased cardiac output. This is done with needle decompression of one or both pleural spaces. The highest survivability of pericardial tamponade

Executive Summary

- ED thoracotomy may be indicated for patients in arrest with suspected pericardial tamponade, massive intrathoracic hemorrhage, and control of hemorrhagic shock.
- Perimortem cesarean section is considered in the case of maternal cardiac arrest if the fetus is at least 24 weeks gestation.
- Fibrinolytic therapy is recommended for patients with massive pulmonary emboli who are hemodynamically compromised.
- ECMO is considered for patients in cardiac arrest from a reversible cause as a bridge to definitive treatment.

relieved with thoracotomy is seen in patients with penetrating injuries such as stab wounds or small lacerations of the heart.[6]

Massive Intrathoracic Hemorrhage. Conversion to open EDT is warranted in patients who undergo tube thoracostomy for evacuation of hemothorax with an initial output of greater than 1500 mL or ongoing output of 200 mL/hr in the first two hours.[9] Based on weight, this volume is defined as > 15 mL/kg initially or > 4 mL/kg/hr in the first two hours.[10] This is indicative of an injury to one or more of the great vessels of the thorax or penetrating injury to the heart. Each hemithorax can accumulate almost half of the patient's circulating blood volume in minutes.[7] EDT is required to visualize and control the source of bleeding. If the patient can survive transport to the operating room, open thoracotomy in the operating room is preferred.

Evacuation of Bronchovascular Air Embolism. In the setting of thoracic trauma, an alveolarvascular or bronchovascular communication can occur. After initiation of positive pressure ventilation, air emboli move down the pressure gradient to the pulmonary venous system. This can impede venous return to the left side of the heart. Propagation of air emboli to the coronary arteries causes obstruction with resultant ischemia and possible arrest. EDT with cross clamping of the pulmonary hilum prevents further propagation of air emboli. Furthermore, with the patient in the Trendelenburg position, air can be aspirated from the apex of the left ventricle.[7,11,12] Manual cardiac massage restores 55% of the normal cardiac output in the arrested heart and may also break up larger air pockets in the coronary arteries.[13,14]

Hemorrhagic Shock and/or Subdiaphragmatic Hemorrhage Control. Thoracic clamping of the descending aorta after EDT serves to redistribute blood flow to the coronary and cerebral arteries. Clamping of the thoracic aorta may reduce subdiaphragmatic blood loss from an abdominal injury. This provides a limited amount of time for surgical repair of the hemorrhage source. As soon as adequate blood volume has been restored and/or the site of hemorrhage has been controlled, the aortic clamp should be removed.[7,15,16]

Surgical Technique. The patient's left arm should be secured above the head to allow the largest area for surgical access. This is assuming that the source of injury is not isolated to the right hemithorax. The patient should be supine unless air embolism is suspected, in which case the patient is placed in the Trendelenburg position to minimize the risk of propagation. The thoracotomy incision starts just to the right of the sternum. If contralateral thoracotomy is required, this saves time in resection of the right chest. The excision should follow the natural curvature between the fourth and fifth ribs. The cut should extend through all the layers of the subcutaneous tissue and intercostal muscles in a single attempt. Once the pleural space is entered, the teeth of the rib retractors are inserted inside the pleural cavity. The long end of the handle should be directed posteriorly and caudal to the patient in case a right-sided thoracotomy is also warranted. Once the thoracic cavity is opened, rapid identification of the injury source should be made.[7,9]

The lung should be reflected superiorly to identify the pericardium. Alternatively, some have advocated intentional right mainstem intubation to deflate the left lung.[17] The phrenic nerve must be identified and avoided. The pericardium should be grasped with toothed forceps or hemostats with one hand, and the pericardium should be opened with blunt-tip scissors anterior and parallel to the phrenic nerve. Evacuation of the clot and isolation of the source of bleeding can be difficult to visualize in the beating heart. Therefore, a manual approach is often used. If injury to the ventricle is encountered, digital pressure can be used to attempt to control the bleeding. Several methods may be used to more definitely provide control. Sutures should be buttressed with pledgets to control cardiac surface bleeding when passing running or horizontal mattress sutures.[7,9] Staples can be used on the ventricle to rapidly control hemorrhage and minimize exposure risk.[9,18] A Foley catheter can be placed through the defect. Once in place, the Foley bulb can be inflated inside the ventricle and gentle traction can be applied to control bleeding.[9] Vascular clamps should be used on the more delicate tissue of the atrium or great vessels. Avoid suturing over the coronary arteries if possible. Of note, in the non-beating heart, cardiorrhaphy should be done prior to internal defibrillation or direct cardiac massage.

The indications for defibrillation are the same as those for non-traumatic cardiac arrest. Internal

defibrillation paddles are positioned on the surface of the heart anteriorly and posteriorly, with care to avoid the coronary arteries or phrenic nerve, if possible. Defibrillation begins at 10 joules and can be increased up to 50 joules in increments of 10 joules per defibrillation attempt.[19] Manual cardiac massage is done using a two-handed approach. This is done with both wrists touching, followed by a sequential clapping motion of the hands as they come together from the palms to the fingers. Intra-cardiac epinephrine may be warranted and is injected using a long, thin needle directly into the left ventricle, taking care to avoid the left anterior descending artery and its large diagonal branches.[7,13,14]

If the primary injury is to the descending aorta or one of the major intraabdominal branches, occlusion of the descending aorta is done to maximize coronary and cerebral perfusion. The inferior hilar ligament tethers the lung to the mediastinum and serves as a reflection point of the lung. Prior to isolation of the aorta, the mediastinal pleura must be incised carefully to avoid further aortic, hilar, or esophageal injury. This is best accomplished by palpation of the spine just superior to the diaphragm. The first structure anterior to the spine is the aorta. Blunt dissection is made through the pleura and around the aorta to release it from the spine posteriorly and esophagus anteriorly. This can be done with blunt hemostats or one's fingers. An orogastric tube can serve as a tactile reference to avoid clamping the esophagus. If possible, gentle pressure should be applied on the aorta during its isolation. While keeping one hand in the dissection plane to serve as a guide, the other hand inserts vascular clamps around the aorta. Once clamped, distal perfusion to the body is impaired and will convert to anaerobic metabolism, leading to accumulation of vasoactive agents and lactic acid, thus increasing morality with every minute of occlusion. Mortality following aortic cross clamping increases exponentially after approximately 30 minutes.[7,15,16]

Table 1: Indications for Emergent Thoracotomy in the Emergency Department‡

- Penetrating trauma with loss of signs of life in, on arrival to, or up to 15 minutes prior to arrival to the emergency department; blunt trauma with loss of signs of life in or on arrival to the emergency department*
- Hemothorax with initial chest tube output > 1500 mL or ongoing chest tube output > 200 mL in the first two hours^
- PEA or asystole in trauma patients with suspected pericardial tamponade not amenable to pericardiocentesis+
- Massive air embolism#
- Hemorrhagic shock with need for redistribution of blood flow or control of subdiaphragmatic hemorrhage

‡ All of these assuming +/- need for manual cardiac massage or internal defibrillation.
* WTA (2011): 18 institution meta-analysis: Contraindication to ED thoracotomy: > 15 minutes of CPR after loss of signs of life prior to ED arrival with penetrating injuries and > 10 minutes with blunt injuries.
Rhee et al meta-analysis: (2000) 24 studies 4,620 cases: ED thoracotomy is indicated in patients with signs of life in the field who do not respond to volume resuscitation as evidenced by SBP < 70 mmHg with loss of signs of life in the ED. Relative indication: penetrating abdominal injury or blunt thoracic injury with at least one sign of life in the field. Little benefit likely in all patients with loss of signs of life at scene. No absolute contraindication.
^ Ideally, these patients are transported to the operating room.
+ ACSCOT (2008): Contraindication to ED thoracotomy: pulseless blunt thoracic injuries, regardless of presence of electrical cardiac activity or suspected tamponade
# As evidenced by patients with penetrating chest injury who develop profound hypotension following intubation and positive pressure ventilation.

Outcomes. Survivability following ED thoracotomy is based on several factors, including mechanism of injury, location of major injury, and the physiologic status of the patient on arrival to the ED.[20] Rhee et al addressed the question of EDT survival via a meta-analysis involving 4,620 cases at 24 institutions. They found that overall survival following EDT was 7.4%, with most surviving patients reaching a full neurological recovery. Based on mechanism of injury, 8.8% of patients survived following penetrating injury, whereas 1.4% survived following blunt injury. Based on location, a survival rate of 10.7% was seen in those patients with thoracic injuries, and a rate of 4.5% was seen for abdominal injuries.[6] This number dropped to less than 1% for patients with multiple injuries. Finally, if signs of life were noted on arrival to the ED, survival rates following thoracotomy were 11.5% in contrast to 2.6% if signs of life were absent.[6] (*See Table 2.*) The decision to perform an EDT is complex. One EDT can cost on average $100,000 and substantial hospital resources.[6] Studies involving prehospital thoracotomies and thoracotomy in pediatric trauma, as well as continued observation of techniques and survival rates, are underway to further contribute to our understanding of this potentially life-saving but high risk procedure.[21-24]

Perimortem Cesarean Section

Perimortem cesarean section was described in literature dating as early as 800 BC.[25] Yet, emergent perimortem cesarean section was first described in the medical literature in the mid 1980s.[26] The literature on the indications, timing, and process of perimortem cesarean section during cardiac or traumatic

Table 2: Survivability of ED Thoracotomy

Overall	
• 7.4%	
Mechanism of Injury	
• Penetrating: 8.8%	
• Blunt: 1.4%	
Location of Injury	
• Thoracic: 10.7%	
• Abdominal: 4.5%	
Signs of Life on ED Arrival	
• Present: 11.5%	
• Not present: 2.6%	

maternal arrest comes largely from case reports and observational outcomes. In addition to trauma (blunt or penetrating), causes of cardiac arrest in the pregnant patient include venous thromboembolism, preeclampsia, eclampsia, anaphylactoid syndrome of pregnancy (previously known as amniotic fluid embolism), hemorrhage from placental abruption or previa, allergic reactions, hypermagnesemia, congenital or acquired structural heart disease, or infection.[27] Fortunately, it is still very rare.[28] If a gravid female presents in extremis and is not actively in labor, every effort should be made to transport the patient to the closest obstetrical unit following adequate resuscitation in the ED. Once pulses are lost and ACLS/ATLS protocols are initiated, an attempt to improve venous return to the heart is made by rolling the patient to the left lateral decubitus position with a wedge pillow. This reduces the gravitational compression of the inferior vena cava by the gravid uterus.

Rees et al studied the effective force of chest compressions when done at varying angles. They found that the minimum loss of compression force is at a 27° decubitus angle.[29] At this angulation, the maximal compressive force is 80% of the force generated in the supine position.[30] From this determination, the Cardiff resuscitation wedge was constructed. An alternative option is to use a caretaker's thighs as the person kneels against the patient's back to provide support during CPR. Also, the patient may be kept supine with someone manually displacing the uterus to the left.

If the gestational age is not known, rapid measurement of the fundal height can be assessed using the "finger breadth rule" in which the fundus is one finger-width above the umbilicus for every two weeks past 20 weeks gestation.[31] A fundus with two finger-widths above the umbilicus is consistent with a 24-week pregnancy.[31] The consensus opinion states that perimortem cesarean section should be considered only if the estimated gestational age is 24 weeks or greater, despite the fact that there are case reports of fetal survival at less than 24 weeks.[32] This is based on the likelihood of fetal survival and the fact that at 24 weeks, venocaval compression and anatomical changes often cause inadequate chest compressions and generation of pulses is rarely reported.[33] Perimortem cesarean section allows for more effective CPR and redistribution of up to 30% of the stroke volume away from the uterus and back to systemic circulation.[33,34] Based on the outcomes of several hundred maternal arrests, if perimortem section is to be performed, it should be initiated within four minutes of loss of pulses.[26,32] The goal is delivery of the fetus within five minutes.[25,27,35,36] An exponential decline in survival rates is seen at time intervals greater than five minutes. However, successful maternal resuscitation and viable fetuses have been delivered at longer time intervals.[25,26,34,37,38]

The following equipment should be available: sterile suction catheter tip on suction canister, scalpel, several hemostats, sterile towels and surgical sponges, retractors, blunt-tip scissors, basin for the placenta, large absorbable sutures, and suction bulbs. A classic long, vertical incision from the xiphoid to the symphysis pubis is performed. Care should be taken to avoid bowel or bladder injury, if possible. The bladder can be evacuated with a Foley catheter if time allows. Another midline vertical incision is made gently into the upper segment of the uterus and can be extended caudally with blunt scissors. Blunt-tip scissors are advisable for the chorionic membrane as well to avoid injury to the fetus. If the placenta is anterior, it must also be transected.

Once the fetus is delivered, it is held below the level of the mother until the cord is doubly clamped and cut. The placenta should then be evacuated gently with blunt manual dissection. Large, locking running sutures should be placed over the uterus to prevent bleeding once cardiac output is restored. This is followed by closure of the abdominal cavity in the same fashion. Some advocate for sterile abdominal packing if hemorrhage control is adequate to prevent abdominal compartment syndrome caused by bowel ileus and edema following ischemia.[35,36] CPR should be continued during this entire procedure.[35,36] Once delivered, a separate team, preferably the neonatal intensive care unit (NICU) team, will direct the resuscitation of the infant. The mother's vital signs need to be immediately reassessed as the resuscitation continues. Delivery can improve the maternal response to resuscitative efforts.[26,27]

Fibrinolytics for Pulmonary Embolus

The use of fibrinolytic agents for treatment of pulmonary embolism (PE) remains controversial. A massive PE is defined as acute PE with SBP < 90 for at least 15 minutes or requiring inotropic support not due to other causes of hypotension.[39] Cardiac arrest due to PE may occur via multiple mechanisms, including obstructive shock with right ventricular systolic dysfunction, arrhythmias induced by cardiac strain, release of mediators of pulmonary vasospasm, or respiratory failure. A submassive PE is defined as acute PE without hypotension but demonstrating evidence of either right ventricular dysfunction or myocardial necrosis.[39]

Fibrinolytic treatment appears

to improve frequency of return of spontaneous circulation (ROSC) in patients with cardiac arrest due to massive PE, although it is unclear whether there is a definite improvement in long-term survival.[40,41] Early bolus dosing of tenecteplase was associated with ROSC in one small pilot study of 35 patients.[40] A retrospective cohort study of 66 patients with massive PE-induced cardiac arrest showed more frequent ROSC among those treated with thrombolysis, but no statistically significant survival to discharge.[41] In a meta-analysis restricted to placebo-controlled, randomized trials of fibrinolysis for massive PE, 9.4% of patients treated with fibrinolytics had recurrent PE or death, compared to 19% for those treated with heparin alone (OR = 0.45, 95% CI 0.22-0.9). The number needed to treat was 10.[42] The number needed to harm has been estimated at 8 to 17.[42,43]

The 2010 American Heart Association ACLS Guidelines state that fibrinolytic therapy may be reasonable for cardiac arrest patients with presumed or known pulmonary embolism (class IIa, level of evidence B recommendation).[44,45] In the emergency department setting, bedside echocardiography may be useful for detecting a dilated, hypokinetic right ventricle suggestive of right heart strain and the presence of thrombus.

For cardiac arrest without known PE, empiric fibrinolytic treatment has not been shown to be of clear benefit.[44] The use of tPA during CPR in pulseless electrical activity arrest does not improve survival to hospital discharge.[46] There was no difference in return of spontaneous circulation, survival, or improved neurologic outcome in one multicenter trial in which adult patients with witnessed out-of-hospital cardiac arrest were randomized to receive tenecteplase or placebo.[47] However, in one retrospective cohort analysis of out-of-hospital, non-traumatic cardiac arrest of all causes, recombinant tPA use during CPR was associated with increased ROSC (70.4% vs. 51% in controls) and improved 24-hour survival (48.1% vs. 32.9%).[48] A meta-analysis of thrombolysis in CPR did show improved ROSC, 24-hour survival, survival to discharge, and long-term neurological function, although patients treated with thrombolytics had increased risk of severe hemorrhage.[49]

In cases of acute PE without cardiac arrest, the value of fibrinolytics depends on whether the PE is massive, submassive, or low risk. Low-risk pulmonary emboli are those without hypotension, normal biomarkers (BNP, proBNP), and no evidence of right ventricular dysfunction. The benefit of thrombolysis does not outweigh the potential complications in patients with low risk PE.[39] Fibrinolytics appear to decrease death or recurrent PE in hemodynamically unstable patients or massive PE. Despite these benefits, fibrinolytics are used in only 30% of eligible patients.[50] This lack of thrombolytic use is greater in the elderly and patients with comorbidities.[51] Patients in these groups who were treated with thrombolytic therapy had a lower case fatality rate, regardless of age or comorbid conditions; therefore, the cautious approach may not be the safest approach.[51] Although fibrinolytic treatment is associated with more rapid recovery of right ventricular motion and restoration of pulmonary perfusion, these findings are not linked to decreased mortality in patients with submassive PE.[42,43,52,53] Whether this quicker recovery prevents long-term thromboembolic-induced pulmonary hypertension is unclear. Currently, there are two ongoing randomized, controlled trials to determine if there is a role for fibrinolytics in patients with submassive PE. At the present time, the decision to treat with thrombolytics must be made on a case-by-case basis.[54]

Currently, the U.S. Food and Drug Administration has approved three agents for treatment of PE: alteplase, streptokinase, and urokinase. Although not currently approved, clinical trials have been performed using tenecteplase and reteplase as well. Alteplase and tenecteplase have greater resistance to plasminogen-activating enzymes, and thereby have a longer duration of action.[39]

Absolute contraindications to fibrinolysis include CNS malignancy, prior intracranial hemorrhage, ischemic stroke within three months, active bleeding, suspected aortic dissection, bleeding diathesis, recent surgery near the spinal canal or brain, or head trauma. Other factors and relative contraindications include chronic anticoagulation, advanced age, pregnancy, and uncontrolled hypertension.[39] The use of thrombolytics and duration of CPR are not associated with a statistically significant increase in major bleeding complications with patients in extremis.[44,55,56]

Although intracranial hemorrhage is an absolute contraindication to fibrinolytics, there has been a case report of a patient with a recent intracerebral hemorrhage who received tPA with no rebleeding or new neurologic deficits.[57]

Percutaneous thromboembolectomy during active CPR has been described in several cases, with restoration of pulmonary circulation.[58] Emergency surgical embolectomy with cardiopulmonary bypass may be an option for patients for whom fibrinolysis is contraindicated, or who have refractory shock.[39,59-62]

Induced Hypothermia

Therapeutic hypothermia has been shown to improve neurological outcome in adult patients with witnessed out-of-hospital cardiac arrest due to ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) who remain comatose after restoration of spontaneous circulation. Hypothermia appears to limit reperfusion injury caused by systemic inflammation, formation of reactive oxygen species, and release of excitatory neurotransmitters after successful initial resuscitation.[63]

In one European multicenter, randomized trial, comatose survivors of VF cardiac arrest who were cooled

Table 3: Dosing of Fibrinolytics**

Medication	Dosing	FDA Approved for PE?
Alteplase	100 mg IV over 2 hours (15 mg bolus then 2-hour infusion of 85 mg)	Yes
Streptokinase	250,000 units IV bolus, then 100,000 units/hour infusion for 12-24 hours	Yes
Urokinase	4,400 units/kg bolus, then 4,400 units/kg per hour for 12-24 hours	Yes
Tenecteplase	Weight-based dosing over 5 seconds (30-50 mg with 5 mg adjustment every 10 kg from < 60 kg to > 90 kg)	No
Reteplase	Two 10-unit IV boluses 30 minutes apart	No
** Different dosing regimens have been used when treating cardiac arrest due to PE or presumed PE. Adapted from Jaff MR, et al. 2011.[39]		

were more likely to have a favorable neurologic outcome compared to the normothermia control group (55% vs. 39%, RR = 1.4; 95% CI: 1.08-1.81). Mortality at six months was also slightly lower among those in the hypothermia group (41% vs. 55%, RR = 0.74, 95% CI: 0.58-0.95).[64] An Australian trial of 77 out-of-hospital cardiac arrest patients randomized to hypothermia versus normothermia showed improved survival to discharge with sufficiently good neurologic function to return home or to a rehabilitation facility. Among those in the hypothermia group, 49% were discharged home or to rehab compared to 26% of those assigned to normothermia.[65]

The evidence of benefit for therapeutic hypothermia in patients presenting with non-shockable rhythms is less clear, mostly limited to observational studies with substantial risk of bias. Multiple studies with historical controls suggest that the beneficial effects of therapeutic hypothermia may be associated with any initial arrest rhythm.[65-71] Two non-randomized trials using concurrent controls also suggested that hypothermia may be of benefit for cardiac arrest with non-VF initial rhythms.[70,71] However, the neuroprotective effect of hypothermia in non-shockable rhythms is less clear. One recent meta-analysis of 12 non-randomized and two randomized trials with patients resuscitated from a non-shockable rhythm showed lower in-hospital mortality but no statistically significant difference in neurologic outcome.[72]

More research is needed to further characterize the benefits of inducing hypothermia in patients with initial rhythms other than VF or VT. A Cochrane Systematic Review in 2012 found that patients treated with hypothermia were more likely to have good cerebral recovery as well as survival to hospital discharge, compared to those with standard normothermic treatment.[73] The 2010 Guidelines for Advanced Cardiac Life Support recommend initiation of therapeutic hypothermia for all comatose adult post-cardiac arrest patients.[45]

It is unclear whether the timing of hypothermia induction results in better neurologic outcomes, although it may be ideal to initiate cooling as soon as possible after return of spontaneous circulation. Animal studies in mice, rats, and dogs suggest that early initiation of hypothermia within less than 20 minutes appeared more beneficial than when hypothermia was delayed.[74-76]

One case series of 986 patients showed no significant association between time to initiation of cooling and time to achieving target temperature within 3-6 hours, with improved neurologic outcome after discharge.[77] Another case study of 49 comatose out-of-hospital post-arrest patients showed that time to target temperature did not independently predict neurologic outcome.[78]

Induction of therapeutic hypothermia by EMS providers has not been shown to improve neurologically intact survival to discharge.[79,80]

Therapeutic hypothermia has been used with success in neonates with hypoxic ischemic encephalopathy, but there are few data regarding its use in cardiac arrest. A recent Cochrane Systematic Review showed no difference in survival or neurologic outcome. Therefore, the authors were unable to make specific recommendations for clinical practice.[81]

Therapeutic hypothermia may be considered for any patient who lacks meaningful response to verbal commands after return of spontaneous circulation. Potential contraindications include severe hemorrhage, intracranial bleeding, and hypotension refractory to multiple pressors.[63]

Induction may be performed via multiple methods, including intravascular heat exchange catheters and surface cooling devices. An initial IV bolus of 500 mL to 30 mL/kg ice-cold (4°C) normal saline or lactated Ringer's solution is a simple and effective method for lowering core temperature by up to 1.5°C.[82] Cold IV fluids can decrease core temperature more rapidly than endovascular catheters and may be administered in the pre-hospital setting.[83] External cooling may be more labor intensive, but may be achieved with cooling blankets, a cooling helmet, wet towels, fanning, or ice packs to the neck, axillae, and groin areas. External

cooling may also be complicated by the normal vasoconstriction of blood vessels in the skin at around 36.5°C, thereby reducing heat exchange.[84] Patients receiving therapeutic hypothermia should be cooled to a target temperature between 32°C and 34°C for 12-24 hours.[45,85]

Although central venous temperature is the gold standard, esophageal temperature may be the most accurate and feasible surrogate in the emergency department. Changes in rectal temperature may be delayed compared to acute core temperature fluctuations, and bladder temperature may be misleading if urine output is low.[45,86]

Shivering can slow down the cooling process and impede therapeutic hypothermia.[87-89] It can be prevented with neuromuscular blocking agents and sedation. However, paralytics may also mask seizure activity, and EEG may be indicated. In one of the landmark trials, pancuronium 0.1 mg/kg was given to prevent shivering.[64] Propofol may be used as well for sedation. Magnesium sulfate given intravenously can increase the shivering threshold.[63,84]

Low body temperature causes a mild coagulopathy, as clotting factors and platelets function less effectively. In one retrospective review of 69 cardiac arrest patients who underwent therapeutic hypothermia, 22% of patients had bleeding.[89] However, significant bleeding requiring transfusion occurred in only 4% of 986 patients studied in a Swedish case registry from 2004 to 2008, with higher risk among patients who had percutaneous interventions.[77]

Therapeutic hypothermia has become a class one recommendation by the American Heart Association for VF/VT arrests and is certainly not considered an extreme measure, although it may have been at one time.[90] The question now is what other clinical circumstances will benefit from therapeutic hypothermia. Will it be nonshockable rhythms (PEA or asystole), strokes, heart attacks without arrest, or some other condition?

Emergency Cardiopulmonary Bypass: Extracorporeal Membrane Oxygenation

Extracorporeal membrane oxygenation (ECMO) provides temporary cardiorespiratory support for patients in respiratory or cardiac failure. Veno-venous (VV) circuit ECMO provides gas exchange only, and is used to support patients with acute lung injury such as ARDS. Cardiac ECMO utilizes a veno-arterial (VA) circuit that propels blood through the body to maintain circulation in addition to gas exchange.[91]

Deoxygenated blood is transported by a pump through a membrane oxygenator where gas exchange takes place. A heat exchanger in the device maintains normal blood temperature, which may potentially be used for therapeutic hypothermia in the setting of cardiac arrest. Vascular access can be established peripherally with percutaneous cannulation of the femoral vessels by a Seldinger technique or cutdown procedure.[91,92]

Complications of ECMO include bleeding, especially at cannulation sites, which may be exacerbated by thrombocytopenia and the need for systemic anticoagulation with heparin to prevent thrombus formation. Massive gas embolism from air in the circuit and tubing rupture may be catastrophic. Decreased blood flow through the circuit is the most common complication, and is usually due to hypovolemia.[91] With femorofemoral ECMO in the setting of cardiac arrest, oxygenated blood travels in a retrograde fashion up the aorta. In the absence of effective left ventricular output, this may result in elevated left ventricular pressures and pulmonary edema or hemorrhage.[92,93]

Extracorporeal cardiopulmonary resuscitation (E-CPR) is an emerging application of ECMO as an adjunct to conventional ACLS for refractory cardiac arrest patients. Most studies to date on E-CPR have reported survival benefit among patients who have in-hospital cardiac arrests. In 1986, one hospital in San Diego created a program for rapid mobile initiation of ECMO for in-hospital cardiac arrests. Case-series data collected over 20 years showed 46% overall survival and a 30-day survival rate of 27.7%.[94] One retrospective analysis of adults suffering witnessed cardiac arrest in the hospital showed that patients treated with ECMO had improved survival with minimal neurologic impairment at discharge and at six months.[95] A prospective, observational study of 975 in-hospital cardiac arrest patients who had CPR for longer than 10 minutes showed that ECMO was associated with improved survival to discharge (28% vs. 12.3%) and one-year survival (18.6% vs. 9.7%).[96]

Duration of ECMO does not appear to be associated with survival. Even with prolonged CPR times in one study, with a mean of 47.6 minutes (+/- 13.4 minutes), 66.7% of patients eventually were able to be weaned off ECMO. Survival discharge was seen in 31.6%. Among survivors, only 5.6% had severe neurologic deficit.[97] Duration of CPR prior to initiation of ECMO, however, is associated with survival. In one study of 135 adult in-hospital cardiac arrest patients, probability of survival for CPR duration of 30, 60, or 90 minutes was 50%, 30%, or 10%, respectively. Favorable neurologic outcomes were reported in 89% of survivors.[98]

Among pediatric patients with in-hospital cardiac arrest treated with ECMO, one study of 27 patients showed a 41% survival rate. Nonsurvivors had higher lactate levels, longer duration of CPR, higher incidence of renal failure, and longer time to ECMO initiation.[99]

The role of ECMO in treating patients with out-of-hospital cardiac arrest is not well established. Neurologic outcomes were generally poor. In the 1990s, ED initiation of ECMO was reported in a case series of 10 patients. Although vascular access and circulatory support was achieved, the mean survival was two days, with no long-term survivors.[100] A French study of 51 consecutive patients with witnessed out-of-hospital refractory cardiac arrest who

received ECMO immediately upon arrival showed only 4% survival with favorable neurologic outcome at day 28.[101] In Japan, a retrospective study of adult patients with witnessed out-of-hospital cardiac arrest found three-month survival of 22.7% among patients treated with cardiopulmonary bypass, versus 9.9% with standard resuscitation. There was no statistically significant difference in neurologic outcome.[102]

Another study of 162 adult patients with witnessed cardiac arrest who received CPR for more than 20 minutes showed 29.2% survival in the ECMO group compared to 8.3% of those receiving standard treatment. Pupil diameter greater than 6 mm was associated with poor neurological survival, but duration of CPR was not a significant factor.[103]

More recently, there have been encouraging results in several reports with small numbers of patients. Shinar and colleagues reported a case of successful ECMO initiation in the ED for an adult patient with refractory ventricular fibrillation secondary to acute myocardial infarction. This patient subsequently underwent cardiac catheterization, and a left anterior descending artery occlusion was visualized and stented. He was discharged on hospital day 9 with no neurologic sequelae.[92] A subsequent study from this institution reported eight patients who had successful E-CPR in the ED, with five surviving to hospital discharge neurologically intact.[93] Preliminary data from the SAVE-J multicenter, nonrandomized prospective cohort study based in Japan compared 180 patients treated with E-CPR with 134 patients who received standard treatment. These were mostly witnessed, out-of-hospital arrests, with half of the patients receiving lay-rescuer CPR at the scene. Average time to arrival at the ED was approximately a half hour. Survival with favorable neurologic outcome was 12.4% in the E-CPR group, compared to 1.6% among the controls.[104]

Other future applications of ECMO for cardiac arrest may include field resuscitation. This has been attempted in France and Germany, where the EMS systems emphasize direct provision of medical care by physicians. The first case of field initiation of ECMO was reported in France in an adult athlete who had refractory cardiac arrest during a road race. ECMO was started in the ambulance within one hour of the arrest. The patient was found to have a right coronary artery occlusion, which was treated with angioplasty. The patient was successfully weaned off ECMO within 48 hours.[105] One recent case report of a 9-year-old pediatric drowning patient in cardiac arrest described the field application of a new portable mini-ECMO system in Germany. Although effective circulation and gas exchange was established, the patient did not survive.[106]

The decision to initiate ECMO in the ED depends on identification of the appropriate patient. More research is needed to define predictors of favorable outcomes and specific indications for treatment in order to identify good candidates and to prevent futile treatment. Cost is also a consideration in the decision to start ECMO. In 2005, a cost-utility analysis of pediatric patients at Emory University requiring cardiac ECMO for a mean duration of 5 days revealed a median cost of $156,324 per patient.[107] However, as the use of ECMO in cardiac arrest care is still not widely used, the true costs have not been well studied. The 2010 ACLS guidelines state that ECMO may be considered for patients with a potentially reversible cause of cardiac arrest, such as a drug overdose, and if the time period without blood flow is brief.[108] Survival among patients on ECMO was seen mostly among patients with a condition amenable to definitive intervention, such as pulmonary embolism or a respiratory process.[109,110] In such cases, ECMO is an important bridge to survival rather than a definitive treatment itself. Future studies will clarify the indications and optimal use of ECMO. Its use is clearly not a solitary endeavor and will require a multidisciplinary team with appropriate administrative and ancillary support.[111]

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CME Questions

1. The optimal time to perform a perimortem cesarean section is within what time interval from maternal cardiac arrest?
   A. 10 minutes
   B. 16 minutes
   C. 4 minutes
   D. never indicated

2. The most likely patient in cardiac arrest to be resuscitated successfully with an emergency department thoracotomy is a patient with:
   A. a shotgun wound to the chest
   B. a stab wound to the chest
   C. a gunshot wound to the abdomen
   D. blunt trauma

3. Which patient with a pulmonary embolism would benefit from treatment with a fibrinolytic?
   A. a pregnant patient with an oxygen saturation of 95% and severe chest pain
   B. a patient with a normal EKG and normal troponin
   C. a patient with a mild elevation of troponin and a blood pressure of 100/60
   D. a patient whose blood pressure remains 80/60 for more than 15 minutes

4. Currently, the role of thrombolytics in the treatment of submassive pulmonary embolus is best described as:
   A. contraindicated
   B. potentially useful
   C. the harm clearly outweighs the benefit
   D. the benefit clearly outweighs the harm

5. All of the following are complications of ECMO *except*:
   A. delayed cooling
   B. excessive bleeding
   C. air embolus
   D. pulmonary edema

6. Which of the following treatments has been shown to increase the shivering threshold?
   A. normal saline
   B. phenobarbital
   C. magnesium
   D. morphine

7. Therapeutic hypothermia is a class I recommendation by the American Heart Association for which type of patient?
   A. asystolic arrest
   B. ventricular fibrillation
   C. sepsis
   D. head trauma

8. The biggest advantage of induced hypothermia is:
   A. decreased arrhythmias
   B. improved neurologic outcome
   C. decreased coagulopathy
   D. less hyperglycemia

9. What amount of initial drainage from a hemothorax is an indication for a thoracotomy?
   A. 200 mL
   B. 500 mL
   C. 750 mL
   D. 1500 mL

10. Beyond what gestational age should perimortem cesarean section be considered in the setting of a maternal arrest?
    A. 10 weeks
    B. 34 weeks
    C. 24 weeks
    D. 16 weeks

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Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

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Emergency Medicine Reports
Practical, Evidence-Based Reviews in Emergency Care

Extreme Measures: Procedures and Therapies for Life-threatening Situations

Indications for Emergent Thoracotomy in the Emergency Department‡

- Penetrating trauma with loss of signs of life in, on arrival to, or up to 15 minutes prior to arrival to the emergency department; blunt trauma with loss of signs of life in or on arrival to the emergency department*
- Hemothorax with initial chest tube output > 1500 mL or ongoing chest tube output > 200 mL in the first two hours^
- PEA or asystole in trauma patients with suspected pericardial tamponade not amenable to pericardiocentesis+
- Massive air embolism#
- Hemorrhagic shock with need for redistribution of blood flow or control of subdiaphragmatic hemorrhage

‡ All of these assuming +/- need for manual cardiac massage or internal defibrillation.
* WTA (2011): 18 institution meta-analysis: Contraindication to ED thoracotomy: > 15 minutes of CPR after loss of signs of life prior to ED arrival with penetrating injuries and > 10 minutes with blunt injuries.
Rhee et al meta-analysis: (2000) 24 studies 4,620 cases: ED thoracotomy is indicated in patients with signs of life in the field who do not respond to volume resuscitation as evidenced by SBP < 70 mmHg with loss of signs of life in the ED. Relative indication: penetrating abdominal injury or blunt thoracic injury with at least one sign of life in the field. Little benefit likely in all patients with loss of signs of life at scene. No absolute contraindication.
^ Ideally, these patients are transported to the operating room.
+ ACSCOT (2008): Contraindication to ED thoracotomy: pulseless blunt thoracic injuries, regardless of presence of electrical cardiac activity or suspected tamponade
# As evidenced by patients with penetrating chest injury who develop profound hypotension following intubation and positive pressure ventilation.

Dosing of Fibrinolytics**

Medication	Dosing	FDA Approved for PE?
Alteplase	100 mg IV over 2 hours (15 mg bolus then 2-hour infusion of 85 mg)	Yes
Streptokinase	250,000 units IV bolus, then 100,000 units/hour infusion for 12-24 hours	Yes
Urokinase	4,400 units/kg bolus, then 4,400 units/kg per hour for 12-24 hours	Yes
Tenecteplase	Weight-based dosing over 5 seconds (30-50 mg with 5 mg adjustment every 10 kg from < 60 kg to > 90 kg)	No
Reteplase	Two 10-unit IV boluses 30 minutes apart	No

** Different dosing regimens have been used when treating cardiac arrest due to PE or presumed PE.
Adapted from Jaff MR, et al. 2011.[39]

Survivability of ED Thoracotomy

Overall
- 7.4%

Mechanism of Injury
- Penetrating: 8.8%
- Blunt: 1.4%

Location of Injury
- Thoracic: 10.7%
- Abdominal: 4.5%

Signs of Life on ED Arrival
- Present: 11.5%
- Not present: 2.6%

Supplement to *Emergency Medicine Reports*, September 8, 2013: "Extreme Measures: Procedures and Therapies for Life-threatening Situations." *Authors:* **Dennis Hanlon , MD, FAAEM**, Vice Chairman, Operations, Associate Professor of Emergency Medicine, Allegheny General Hospital, Pittsburgh, PA. **Michael Yeh, MD**, Senior Resident, Combined EM/IM Program, Allegheny General Hospital, Pittsburgh, PA; and **Jennifer Nelson, MD**, Senior Resident, Emergency Medicine Residency, Allegheny General Hospital, Pittsburgh, PA.

Trauma Reports

PRACTICAL, EVIDENCE-BASED REVIEWS IN TRAUMA CARE

Volume 14, Number 5 **Sept/Oct 2013**

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Civilian Blast Injury

Blast injuries are commonly thought of as incidents that occur in other countries, not here in the United States. The majority of clinicians are not prepared to deal with the devastation of a civilian blast incident and the resulting injury patterns. The author reviews expected injury patterns, triage decisions, and current therapies.

— Ann M. Dietrich, MD, Editor

Introduction

With more than 54 local wars and armed conflicts in the first decade of this century[1] and notable terrorist activity in Afghanistan, Great Britain, India, Iraq, Pakistan, and Spain, it is easy to see why many American clinicians view terrorist blast injuries as an overseas issue. However, in the two decades from 1983 to 2002, more than 36,110 criminal bombing incidents occurred in the United States. During the decade between 1992 and 2002, more individuals were injured or killed by bombs on U.S. soil than all of the U.S. citizens killed during this same period in terrorist events overseas.[2]

High-profile terrorist bombings leading to mass casualties have occurred on American soil. In the past two decades, the 1995 bombing of the Murrah Federal Building in Oklahoma resulted in 759 injuries and 168 deaths;[3] the 2001 World Trade Center bombing in New York led to nearly 4,000 casualties and 3,000 deaths;[4,5] and most recently, in 2013, three people were killed and 264 injured when two improvised explosive devices (IEDs) were detonated at the Boston Marathon.[6]

Conventional explosive devices, either traditional or improvised, remain the terrorists' weapon of primary choice,[7] and blast injury is the common result. Despite these facts, we are relatively unprepared as emergency providers and systems to treat mass casualties as a result of blast injuries.[2,3,7,8] This review provides a primer on the physics, common injury patterns, and triage for blast injury.

Physics

Terrorist explosive devices are often weapons of convenience. These devices are categorized as high-energy or low-energy.[9] Examples of high-energy explosives include trinitrotoluene (TNT), plastic explosives such as C-4, and fertilizer-based explosives. The Oklahoma City bombing was the result of a high-energy explosive: the combination of nitrate fertilizer and fuel oil configured for maximum explosive effect as fuel-air explosive.[10]

Low-energy explosives include black powder and petroleum products. The World Trade Center explosions were the result of fuel-filled commercial aircraft. The Boston Marathon bombers probably used a combination of black powder, nails, and ball bearings packed into pressure cookers detonated with standard egg timers.[11]

High-energy explosives create a blast effect as the result of transient overpressurization. A brief period of high pressure is followed by a transient low pressure of longer duration, which can suck debris into the scene of injury. The

Executive Summary

- Secondary to the multiple simultaneous mechanisms of injury, the blast patient is often more seriously injured than his multiple trauma cohort, and there is a "multidimensionality of injury" in these patients, largely because primary, secondary, tertiary, and quaternary blast effects may impact the victim simultaneously.
- Delayed injury, although uncommon, is really a delay of injury presentation. These injuries are typically primary blast injuries to the hollow organs, which do not manifest on initial roentgenogram, but should be anticipated based on clinical presentation and mitigated by careful observation and reassessment.
- The mid-facial skeleton contains large air-filled cavities and is susceptible to the spalling effects of the blast wave and implosion, resulting in "crushed egg shell" fractures of the sinus walls.
- Tympanic membrane (TM) rupture is common because of the relatively low pressure needed to perforate an eardrum.
- Research has shown that patients with skull fracture, burns greater than 10% of the body surface, and penetrating injuries to the head or torso were more likely to suffer a blast lung injury, and require early critical intervention at a level 1 trauma center.

Table 1. Classification of Blast Injuries[13,50]

Classification	Type	Mechanism	Typical Injuries
Primary	Blast wave		
	Implosion	Air-filled structures rupture from over-pressurization	Tympanic membrane rupture, blast lung, GI rupture
	Spalling	Explosive energy transfer in tissue interfaces of differing density	Lung, liver, brain contusion
	Inertia	Acceleration and deceleration forces lead to shearing injury	Mesenteric tears, axonal injury
Secondary	Blast wind	Bomb fragments, displaced foreign bodies	Penetrating or blunt multi-system injury
Tertiary	Blast wave and wind	Individual or structure thrown or crushed	Blunt or penetrating multi-system injury
Quaternary	By-products of explosion	Fireball and toxic agents	Burns and inhalation injuries

Classification of blast injury based on the mechanism of blast effect, based on the Zukerman classification developed during WWII[13] and modified from Plurad[50]

result is a shock wave that travels at supersonic speeds and a blast wind. The leading edge of this shock wave can injure tissue in its path (primary blast injury) by implosion, spalling, and inertia. The blast wind can move objects in its path, resulting in secondary blast injuries from flying debris and projectiles, or tertiary blast injuries from victims or objects that are hurled or structures that collapse. Quaternary blast injury results from the by-products of combustion such as burns and inhalation injuries.[8,9,12] (*See Table 1.*)

In the open air, blast energy rapidly dissipates with distance in inverse relation to the cube of the distance from the blast. For this reason, the distance from the blast is important in predicting injury and subsequent survival.[8,13] The Department of Homeland Security published a Bomb Stand-off Chart, which provides estimated safe distances from ground zero for a given TNT equivalent. (*See Table 2.*) The blast effect is magnified in water by an estimate of three times, and because water is less compressible than air, the wave travels for a greater distance.[8] Table 3 provides an estimate of the effect of blast over-pressurization.

Table 2. Bomb Threat Stand-off Chart

Threat Description Improvised Explosive Device (IED)	Explosives Capacity[1] (TNT Equivalent)	Building Evacuation Distance[2]	Outdoor Evacuation Distance[3]
Pipe bomb	5 lbs	70 ft	1200 ft
Suicide bomber	20 lbs	110 ft	1700 ft
Briefcase/suitcase	50 lbs	150 ft	1850 ft
Car	500 lbs	320 ft	1500 ft
SUV/van	1000 lbs	400 ft	2400 ft
Small moving van/ delivery truck	4000 lbs	640 ft	3800 ft
Moving van/water truck	10,000 lbs	860 ft	5100 ft
Semi-trailer	60,000 lbs	1570 ft	9300 ft

1. These capacities are based on the maximum weight of explosive material that could reasonably fit in a container of similar size.
2. Personnel in buildings are provided a high degree of protection from death or serious injury; however, glass breakage and building debris cause some injuries. Unstrengthened buildings can be expected to sustain damage that approximates five percent of their replacement cost.
3. If personnel cannot enter a building to seek shelter, they must evacuate to the minimum distance recommended by Outdoor Evacuation Distance. This distance is governed by the greater hazard of fragmentation distance, glass breakage, or threshold for ear drum rupture.
Source: Department of Homeland Security
Department of Homeland Security Bomb Threat Stand-off Chart. This chart provides an estimate of safe distance from the blast epicenter for a given charge of TNT. Note that 5 pounds of TNT is dangerous at up to a quarter mile. Further detail is available from dhs.gov.

Closed spaces significantly modify and amplify the blast effect. Walls and other hard surfaces reflect the wave and extend its duration, leading to a greater transfer of energy to susceptible organ systems.[13,14] Low-energy explosives can also have a primary blast effect, which is quickly mitigated by distance, and injuries are usually due to secondary and tertiary blast effects. In the Centennial Olympic Park bombing in 1996, advance warning and a low-energy explosive allowed for an orderly evacuation, which minimized casualties.[15]

Initial Assessment

The initial assessment and management of patients with blast injuries does not differ from the management of any multiple-injury trauma victim, and should follow standard Advanced Trauma Life Support (ATLS) principles.[16] There are, however, some differences in injury patterns and potential pitfalls specific to the organ systems involved, which will be discussed below. (*See Table 4.*) An understanding of the mechanism of injury is especially critical to understanding and managing the patient who has sustained a blast injury. The explosive agent used, the medium of wave propagation (air vs. water), the presence of flying debris and shrapnel, distance from the blast, open vs. closed environment, building collapse, and fire all provide different wounding mechanisms and morbidity and mortality rates.[8,12-14]

Additionally, because of the multiple simultaneous mechanisms of injury, the blast patient is often more seriously injured than his or her multiple trauma cohort, and there is a "multidimensionality of injury" in these patients, largely because primary, secondary, tertiary, and quaternary blast effects may impact the victim simultaneously.[8] This means time is of the essence and the opportunity for missed injury is magnified. Delayed injury, although uncommon, is really a delay of injury presentation. These injuries are typically primary blast injuries to the hollow organs, which do not manifest on initial roentgenogram, but should be anticipated based on clinical presentation and mitigated by careful observation and reassessment.[12] (*See Tables 4 and 5.*)

Maxillofacial Skeleton

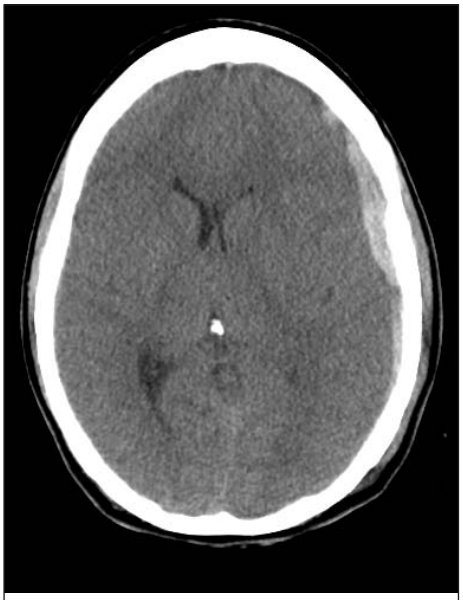
The most common injuries are blunt and penetrating trauma as a result of secondary and tertiary blast effects. However, the blast wave can cause differential acceleration/

Table 3. Blast Pressure Effects

Pressure (kPa)	Effect
30	Shatters glass
100	50% chance of tympanic membrane rupture
200	100% tympanic membrane rupture, minimum pressure for lung injury
500	50% chance of lung injury
900	50% chance of death
2000	Lethal

Adapted from Boffard and MacFarlane.[31] The blast effects are governed by the size and type of charge, distance from the blast, and the medium of propagation. For example, 25 kg of TNT produces 1500 kPa (150 psi) of over pressure for 2 milliseconds at the epicenter and travels at up to 8,000 meters/second.[51] The resultant blast wind can be of hurricane proportions.[31]

Figure 1. Subdural Hematoma



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deceleration forces (inertia), which can lead to transverse shearing fractures of the mandible.[1] The mid-facial skeleton contains large air-filled cavities and is susceptible to the spalling effects of the blast wave and implosion. This may result in "crushed egg shell" fractures of the sinus walls.[1] Isolated maxillofacial injury rarely leads to death, and should be managed as appropriate within the context of the victim's other injuries.

Ear

Injury to the external ear is usually the result of secondary, tertiary, or quaternary blast injury.[17] The blunt and penetrating injuries require appropriate wound care, debridement, and repair. Injuries to the middle and inner ear are often the result of primary blast injury. Hearing loss, tinnitus, and ear pain are common and often temporary. Vertigo is unusual and should suggest the possibility of concomitant head injury.[17,18] Tympanic membrane (TM) rupture is common because of the relatively low pressure needed to perforate an eardrum. (*See Table 3.*) Ossicular injury is uncommon and suggests significant trauma.[17] The physical examination should include a hearing evaluation.[17] Most TM ruptures will heal spontaneously; however, referral to an otolaryngologist is appropriate.[17,18]

The common wisdom has been that a TM injury is a harbinger of potential occult primary blast injury. Recent evidence, however, suggests that while TM injury is common, its presence or absence does not include or exclude other injuries.[9,17,19,20] In the survivors of the 2005 London bombings, TM rupture as a biomarker of concealed primary blast injuries had a sensitivity and specificity of 50%, and a low positive predictive value. External evidence of injury may be a more appropriate triage tool.[21]

Eye

Primary blast injury to the eye can lead to globe disruption, retinal injury, and hyphema.[14] However, penetrating injury from flying debris and shrapnel as the result of secondary blast injury is the more common cause of eye injury.[22,23] One major receiving center from the 2004 Madrid bombing reported an incidence of ocular injury in 16% of their patients with minor injuries and 15% of their patients with critical injuries.[24] Ocular injury was the second most common injury (26%) in the injured survivors of the 2001 World Trade Center bombing.[4] Symptoms include loss of visual acuity, eye pain, and foreign body sensation. Appropriate wound care should be provided for external injuries. Emergency management for injury to the globe (evaluation, irrigation, topical antibiotics, and patching) should be followed with specialty evaluation and management for complex injuries.

Brain

Brain injury is a common cause of death in blast injury. One hundred sixty-seven people died as a result of the Oklahoma City bombing in 1995. Head injury was the second most common cause of death (14%), with multiple trauma the leading cause (73%). Fifty-two percent of critically injured patients treated at the closest hospital during the 2004 Madrid bombings sustained head injuries.[24] Blunt and penetrating injury can result from primary, secondary, and tertiary blast effects.[23] Blunt injury can range from concussion to diffuse axonal injury;

Table 4. Overview of Explosion-related Injuries

System	Injury or Condition
Auditory system	Tympanic membrane rupture, ossicular disruption, cochlear damage, foreign body
Cardiovascular	Cardiac contusion, myocardial infarction from air embolism, shock, vasovagal hypotension, peripheral vascular injury, air embolism-induced injury
Extremity injuries	Traumatic amputation, fractures, crush injuries, compartment syndrome, burns, cuts, lacerations, acute arterial occlusion, air embolism-induced injury
Gastrointestinal	Bowel perforation, hemorrhage, ruptured liver or spleen, sepsis, mesenteric ischemia from air embolism
Neurologic system	Concussion, closed and open brain injury, stroke, spinal cord injury, air embolism-induced injury
Ocular injury	Perforated globe, foreign body, air embolism, fracture
Renal injury	Renal contusion, laceration, acute renal failure due to rhabdomyolysis, hypotension, and hypovolemia
Respiratory system	Blast lung, hemothorax, pneumothorax, pulmonary contusion and hemorrhage, A-V fistulas (source of air embolism), airway epithelial damage, aspiration pneumonitis, sepsis

Adapted from Centers for Disease Control and Prevention. Explosions and blast injuries: A primer for clinicians. Available at http://www.bt.cdc.gov/masscasualties/explosions.asp.

however, subarachnoid hemorrhage and subdural hemorrhages occur most frequently in fatalities.[23] (*See Figure 1.*)

Recent studies using diffusion tensor imaging suggest there is a component of axonal injury in military personnel with blast-related mild traumatic brain injury.[25] However, it is not clear if isolated primary blast injury at a distance leads to mild traumatic brain injury in the absence of a direct blow.[25-27]

Evaluation and management of head injury should follow the basics of emergency management as outlined in ATLS.[16] Because patients sustaining injury as a result of a blast are more critically injured than their multisystem trauma cohort,[8] avoidance of hypoxia and hypotension are essential, and the early involvement of neurosurgical specialists in the initial management is appropriate.

Chest

Blast lung injury (BLI) implies proximity to the blast and is a common cause of mortality at the scene of bomb blasts.[9] (*See Figure 2.*) It is also a frequent cause of morbidity for survivors.[28,29] Avidan et al analyzed a two-decade experience with BLI and found a 71% incidence of BLI in blast victims admitted to the intensive care unit (ICU) of their Israeli trauma center.[28]

The blast wave causes a combination of implosion, inertia, and spalling, which can lead to bronchoalveolar disruption, pulmonary contusion, and arterial air emboli.[9,21,28,29] Survivors generally present with hypoxemia and respiratory distress.[29] Bloody sputum and evidence of barotrauma (pneumothorax) are not uncommon, even though radiologic evidence may lag by 12 to 24 hours.[12] Secondary injury may also lag presentation by several hours. Eckert et al advocate observation for at least 18 hours and selected bronchoscopy to evaluate quaternary airway injury based on their experience at a combat support hospital in Iraq.[30]

Penetrating injuries as a result of flying debris and projectiles, blunt injuries as a result of falls and crush injuries, and inhalation injury and burns should be managed emergently with ATLS principles.[16] Definitive management should be provided with advanced pulmonary and surgical critical care.

Abdomen

Abdominal blast injury can be both blunt and penetrating. In the 2004 Madrid bombings, 12 of the 243 patients (5%) and 10 of 27 (37%) critically injured patients treated at the nearest hospital sustained abdominal injuries.[24] The

Table 5. Clinical Signs and Symptoms of Explosion-related Injuries

System	Injury or Condition
Auditory system	• Blood oozing from the mouth, nose, or ears* • Eardrum hyperemia, hemorrhage, or rupture* • Deafness* • Tinnitus* • Earache*
Cardiovascular	• Tachycardia • Fall of mean arterial blood pressure
Gastrointestinal	• Nausea* • Abdominal tenderness* • Abdominal rigidity*
Neurologic system	• Vertigo • Retrograde amnesia
Ocular injury	• Eye irritation** • Hyphema** • Distorted pupil** • Decreased vision** • Blindness** • Funduscopic findings of retinal artery air embolism**
Respiratory system	• Cyanosis* • Ecchymosis or petechiae in hypopharynx* • Cough (often dry)* • Tachypnea (often preceded by a short period of apnea)* • Dyspnea* • Hemoptysis* • Rales or moist crepitation in lung fields* • Chest pain*

* Most common findings
** Common findings

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experience in the Oklahoma City bombing in 1995 was quite different. Four of a total of 759 patients sustained life-threatening abdominal injuries: one bowel transection, two splenic lacerations, one kidney laceration, and one liver laceration.[10] (*See Figures 3 and 4.*) Both of these bombings involved high-energy explosives, but the mechanisms were quite different. In Madrid, suicide bombers used military explosives and shrapnel to detonate in a closed space at close proximity.[24] As expected, the injuries included a combination of blunt and penetrating trauma, and were relatively common in seriously injured survivors. In Oklahoma City, the explosive was an improvised fuel-oil bomb,[10] and the abdominal injuries were blunt and uncommon.

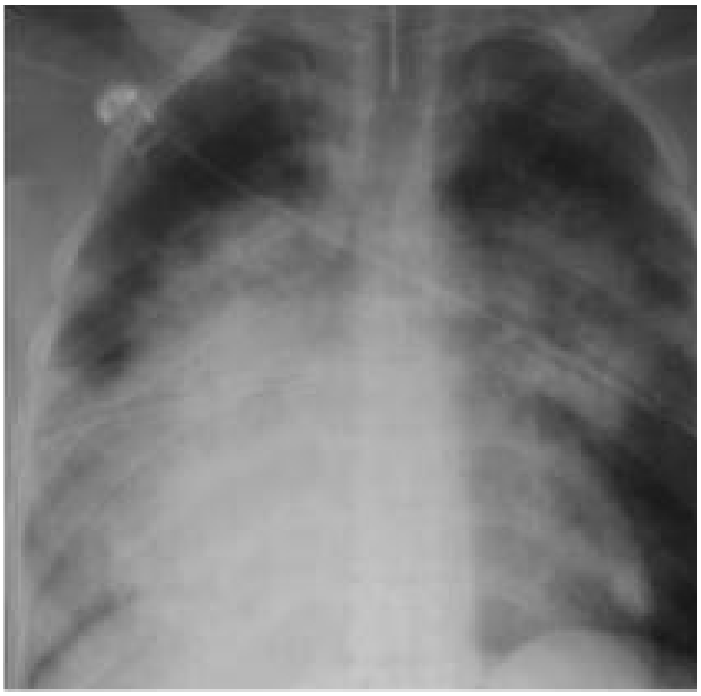
Primary blast injury is relatively uncommon in survivors. Implosion can rupture air-filled bowel wall, spalling forces can disrupt viscera at tissue interfaces (i.e., lung/liver, or spleen), and shearing forces associated with inertia typically affect mesentery.[8,12,31] These effects are significantly increased under water.[32] The emergency management of these patients does not change from the management of the multiple-injury patient, except as mentioned above.

Extremities

Soft-tissue injury accounts for the majority of trauma in civilian blast events.[33] In the Oklahoma City bombing, soft-tissue trauma was the most common injury, followed by fractures and dislocations.[3] Primary blast injury has been shown to cause fractures and amputations;[34] however, the most common cause of extremity injury is related to secondary and tertiary blast effects. Injuries can range from simple soft-tissue trauma to amputations. In the military experience in Afghanistan and Iraq, extremity fractures accounted for 82% of combat injuries, and the majority of the fractures were open.[35] In the civilian bombings, the incidence of extremity injury is much lower. In the Madrid bombings of 2004, only 17% of survivors sustained extremity fractures, and only one patient sustained a traumatic lower extremity amputation.[24] Civilian traumatic amputations as a result of blast injury, however, are often lethal because the blast energy required to amputate a limb is usually lethal to other organ systems.[12,36,37] This was illustrated in the 2005 London bombing in which six of seven patients with traumatic amputations of the upper extremity died at the scene.[36] Almogy et al reported a three-year experience with 15 suicide bombings in Israel.[21] In their series, 63 of 74 (85%) patients with traumatic amputations died at the scene. The Boston bombings of 2013 resulted in several amputations, but few fatalities. These blasts were low-energy and in the open air.

Evaluation should proceed by standard ATLS protocols.[16] Special consideration should be given to

Figure 2. Blast Lung



Reprinted with permission from: Wolf YG. Vascular trauma in high-velocity gunshot wounds and shrapnel-blast injuries in Israel. *Surg Clin North Am* 2002;82:237-244.

Figure 3. Liver Laceration

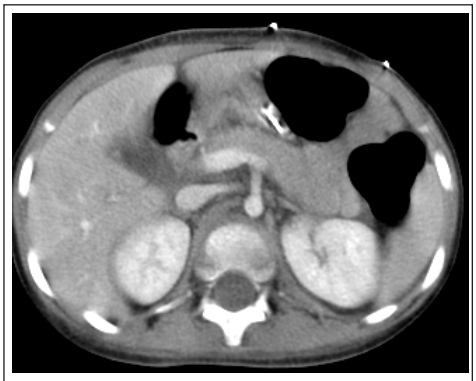


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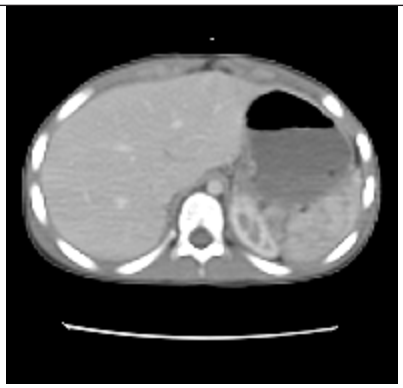
Figure 4. Spleen Fracture



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the surrounding environment and the potential for contamination and secondary infection.[23] One unique aspect of terrorist bombing is the potential for biologic foreign bodies and the risk of blood-borne disease these biologic fragments may carry.[38] Tourniquets are often discouraged in civilian practice, but can be life-saving in traumatic amputations. Surgical completion of the amputation is often a difficult decision, but should be made based on the potential viability and projected functionality of the injured extremity.[35,36]

Pregnancy

Blast injury in pregnancy is uncommon. Mallonee et al reported three of the 167 deaths (1.8%) in the Oklahoma City bombing were pregnant women.[10] Marti et al reported one maternal fetal death from massive hemoperitoneum in 36 patients (3%) treated at their institution following the 2004 Madrid bombings.[39] The fetus may be cushioned by amniotic fluid; however, the placenta would be subject to implosion and shearing forces,[14,40] and the mother would be subject to all of the other mechanisms and injuries described above. Initial assessment and management should follow standard ATLS protocols.[16] Postmortem cesarean section for blast injury is rare, but should be considered for the viable fetus in the case of sudden maternal death.[41]

Burns

The primary blast is associated with a brief fireball at detonation.[23] These burns are often lethal[14] because they herald a close proximity to the blast[12] and are associated with the other primary blast injuries discussed above. Burns and inhalation injuries make up the majority of quaternary blast injuries.[14] Among the 790 injured survivors in the World Trade Center attack in 2001, 386 (49%) were treated for smoke inhalation.[4] The combination of burns and inhalation injuries with the constellation of other injuries sustained by these patients can make them difficult to manage.[12]

Triage

Mass casualty events as the result of a blast are dramatic events and muster an immediate and often less than coordinated response. Despite this, the vast majority of survivors are walking wounded (*see Table 6*), and the over-triage rate is typically 50-90%.[43] Additionally, the receiving

Table 6. Civilian Blast Event Severity of Injury

Event and Year (Reference)	Casualties	Dead	Treated		Explosive
			Hospitalized	Released	
Oklahoma City 1995[10]	759	168 (22%)	83 (14%)	508 (86%)	Fertilizer and fuel oil
Atlanta Olympics 1996[15]	111	1 (<1%)	24 (22%)	87 (78%)	Pipe bomb and shrapnel
World Trade Center 2001[4,5]	3922	2819 (72%)	181 (17%)	810 (73%)	Jet fuel
Madrid 2004[24]	2000	191 (10%)	91 (29%)	221 (71%)	Military explosives and shrapnel
London 2005[42]	775	56 (7%)	27 (14%)	167 (86%)	Military explosives and shrapnel
Boston Marathon 2013[6]	267	3 (1%)	20 (8%)	244 (92%)	Black powder and shrapnel

Casualties for the World Trade Center attack and the Madrid bombings are estimates based on the available literature. Disposition of casualties at the hospital for Madrid and London are based on single institutional experience. The differences in mortality are related to the explosive agent used (high-energy vs. low-energy), open vs. closed space, and high-rise vs. ground level.

facility for the largest influx of patients is often the nearest facility and not necessarily the facility most capable of handling the injuries.[42] Triage at the scene should be performed by experienced personnel, and patient distribution allocated based on available resources and patient need.[42] Triage at the receiving facility should also be done by experienced clinicians and with the understanding that most of the patients seen will not be critically injured.[42,43]

Although injury to the tympanic membrane is the most common blast injury and has been heralded as a harbinger of more serious blast injury, the correlation doesn't hold. It is a poor diagnostic tool for triage. Serious injury is usually obvious. The mechanism is a more important predictor for occult injury, and history becomes an important indicator of blast exposure. The combination of mechanism and evidence of external injury can often help to identify those patients in need of critical resources. (*See Table 7.*) Almogy et al described a retrospective analysis of 15 suicide bomb attacks treated over a three-year period (1994-1997) in Israeli hospitals.[21] These authors found that patients with skull fracture, burns greater than 10% of the body surface, and penetrating injuries to the head or torso were more likely to suffer a blast lung injury, and would require early critical intervention at a level 1 trauma center.

The initial management of these patients should follow damage-control principles to allow for the greatest good to the largest number of victims.[13,42,43] Resource allocation for definitive management will typically mirror those resources used in trauma; however, the seriously injured are a magnitude of several times more severely injured than their typical multi-system counterparts. Experience from military conflicts can help to guide their management.[44] In the Oklahoma City bombing, general surgery, ophthalmology, orthopedics, neurosurgery, and vascular surgery were utilized (in decreasing order) for patients needing operative intervention.[3]

Although it is counter-intuitive, blood usage is not out of proportion to the injury and does not exceed local resources.[42,44] Predictive models from military[44] and civilian experience[13,39,42,43,45-49] may help with disaster planning and allocation of resources.

There are a number of excellent courses to help emergency providers better understand the basics of mass casualty and its management. These include: The National Disaster Life Support™ (NDLS™) course from the National Disaster Life Support Foundation (formerly a collaboration with the AMA); Collaborative Disaster Planning Processes from the American College of Emergency Physicians (ACEP) and the Federal Emergency Management Agency (FEMA); and Disaster Management and Emergency Preparedness (DMEP) from the American College of Surgeons Committee on Trauma Disaster and Mass Casualty Management Committee.

Table 7. Considerations for Injury Severity

Blast Force

- Explosive energy (high-energy vs. low-energy)
- Distance from ground zero
- Energy dissipates by the cube of the distance from the blast

Environment

- Building collapse (high-rise vs. low-rise)
- Confined space vs. open air explosions
- Urban vs. rural (less population dense) settings

Projectiles

- Environmental debris vs. intentional shrapnel

By-products of explosion

- Fire
- Smoke
- Toxins

Anatomic Markers of Severe Injury

- Traumatic amputation
- Blast lung injury
- Severe head injury
- Torso trauma
- Multidimensional injury

Adapted from Ciraulo DL, Frykberg ER. The surgeon and acts of civilian terrorism: Blast injuries. *J Am Coll Surg* 2006;203:942-950.

Conclusion

Blast injury provides a unique challenge in management. There are often multiple survivors, and many of them have minor injuries. This can lead to a dramatic surge in patient inflow to the facility closest to the event. The seriously injured patient can be subjected to a multitude of mechanisms, including primary, secondary, tertiary, and quaternary blast. They are often more severely injured and more complex in their presentation than their multiple trauma counterparts. The emergency provider should understand and anticipate the basics of blast injury to provide optimum care. Continuing education in mass casualty and disaster management is strongly encouraged.

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CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

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CME/CNE Questions

1. Blast-injured patients are more seriously injured than their multiple-trauma counterparts because of:

   A. primary blast injury
   B. secondary blast injury
   C. tertiary blast injury
   D. quaternary blast injury
   E. all of the above

2. Tympanic membrane rupture is an accurate predictor of occult primary blast injury.

   A. true
   B. false

3. Which of the following is (are) the best predictor(s) of occult primary blast injury?

   A. tympanic membrane rupture
   B. external injury
   C. mechanism of injury
   D. B and C
   E. none of the above

4. The most common cause of blast injury is:

   A. primary blast injury
   B. secondary blast injury
   C. tertiary blast injury
   D. quaternary blast injury
   E. quintenary blast injury

5. Explosions are dramatic events, but over-triage is uncommon.
   A. true
   B. false

6. Blast-injured patients at the scene of a mass casualty event should be routinely transferred to a trauma center.
   A. true
   B. false

7. In a blast injury mass casualty situation, management should include:
   A. scene transfer to a trauma center
   B. damage-control principles
   C. hospitalization and observation of all victims evaluated in the ED to exclude occult blast injury
   D. all of the above
   E. none of the above

8. Traumatic amputation is often:
   A. a lethal injury in civilian experience
   B. survivable in the recent military experience
   C. associated with other severe blast injuries
   D. best managed with damage-control principles
   E. all of the above

9. Which of the following is true?
   A. Quaternary blast injury is uncommon in survivors.
   B. Infectious risk does not differ from other multiple-injury patients.
   C. Burn patients are managed identically to non-blast counterparts.
   D. All of the above are true.
   E. None of the above are true.

10. Which of the following is true of primary blast lung injury?
   A. It is usually evidenced on admission with clinical findings.
   B. It uniformly occurs with blast over pressures of 300 psi.
   C. It may be associated with quaternary blast effect on the airways.
   D. A and C are true.
   E. None of the above are true.

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