

# ANESTHESIOLOGY™ 2014

OCTOBER 11-15 | NEW ORLEANS, LA

Session: L036  
Session: L061

## **Anesthetic Management of a Patient With Penetrating Chest Trauma**

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**Disclosures:** This presenter has no financial relationships with commercial interests

### **Stem Case and Key Questions Content**

A 26 year-old man presented himself to an outside hospital after being shot in the left chest. In the emergency department (ED), he became unresponsive and was noted to be in PEA (pulseless electrical activity). He had return of spontaneous circulation after brief CPR (30) and epinephrine. The trachea was intubated. A left chest tube was inserted which yielded 400 ml of blood. He received 5 units of blood and is transferred to your Level 1 trauma hospital. On arrival, he is intubated and sedated. BP 150/90. Pulse 130 RR 10. SpO<sub>2</sub> 99% on 100% O<sub>2</sub>. Examination reveals a single GSW to the left posterior axillary region. Chest x-ray from the outside hospital shows the bullet to be in the right chest. Breath sounds are diminished bilaterally and the cardiac exam is normal except for tachycardia. You are consulted to provide anesthesia for emergency operative intervention.

- 1) What aspects of the physical exam would be most relevant for anesthetic management?
- 2) What associated injuries would be expected given the mechanism of injury?
- 3) What landmarks would you use to determine the anatomic zone of injury?
- 4) Discuss the pros and cons of performing an ED thoracotomy?
- 5) What factors will determine if the patient requires further workup prior to going to the operating room?

A chest x-ray shows the left-sided chest tube to be in place, and evidence of a right hemothorax. Rapid CT angiogram of the chest reveals a large right hemothorax. No pericardial fluid is seen. The bullet is just next to the right atrium. A cardiac surgeon has been consulted. The patient has been scheduled for emergency right anterolateral thoracotomy by the trauma surgeon.

6. Evaluate your options for providing one-lung ventilation?
7. What monitors and IV access would you use?
8. Are there any additional labs or tests required?
9. What anesthetic agents would you use?
10. Discuss the advantages and disadvantages of permissive hypotension. What is your target blood pressure?

The tracheal tube is now exchanged for a double lumen tube. An arterial line and 9Fr internal jugular introducer have been inserted. Cross matched blood and plasma are being given to maintain systolic blood pressure of 80-100 mmHg. During thoracotomy, a massive amount of blood is evacuated from the right chest. However, there is no evidence of active bleeding. There

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is a hole through the mediastinal pleural just over the pericardium and obvious injury to the heart.

11. How will you evaluate the nature and extent of the heart injury?

Emergency transesophageal echocardiography (TEE) by cardiology shows normal left and right heart function, perforation of the posterior mitral valve leaflet (base of P2), severe mitral regurgitation, and traumatic atrial septal defect (inferior portion of the septum primum)

12. Review the impact of severe mitral regurgitation on anesthetic management?

The patient's right chest is packed and the patient is repositioned for sternotomy. Surgery consists of mitral valve repair and closure of the traumatic atrial septal defect (ASD).

13. What plans will you make for separation from cardiopulmonary bypass?

TEE by cardiology shows absence of intracavitary air, no residual mitral regurgitation, successful closure of the ASD, well preserved left ventricular function and moderately impaired right ventricular function. There are frequent PVCs and a new right bundle branch block.

14. What is the significance of the new arrhythmias? How will you manage right ventricular dysfunction?

Arrhythmias are now resolved and BP is stable. Heparin is reversed with protamine and the sternum is closed. The patient is noted to be bleeding from the right thoracotomy incision and the surgeon requests platelets.

15. How do you manage coagulopathy after bypass?

Coagulopathy is resolved. The right thoracotomy is no longer bleeding and the incision is closed.

16. The surgeon requests change over to a single lumen tracheal tube. Discuss your options. How do you proceed?

## **Model Discussion Content**

### ***Anatomy***

The thorax contains vital organs and vasculature which are protected by the bone structure of the vertebral column, sternum and ribs. Inferiorly the thorax is demarcated by the diaphragm while the structures of the neck and lung apices are found superiorly. The primary organs within the thorax are the heart, the lungs, the great vessels and the esophagus. The surface of the heart exposed to the anterior chest wall is formed 55 percent from the right ventricular wall, 20 percent from the left ventricular wall, 10 percent from the right atrial wall, 10 percent from the ascending aorta and pulmonary artery, and 5 percent from the vena cavae. The right atrium (RA) is superior and the right ventricle (RV) lies inferiorly. Together the RA and RV form a crescent which wraps around the ellipsoidal left ventricle (LV). The left anterior descending coronary artery (LAD) courses over the ventricular septum which separates the LV and RV. A portion of the LV inferiorly is at risk for trauma originating anteriorly. The great vessels consist of the aorta, pulmonary arteries, pulmonary veins, vena cava and their major intrathoracic branches. The great vessels occupy a central mediastinal location and connect with the base of the heart. The apex of the heart lies in the left thoracic cavity. The heart is contained within the pericardium, a tough fibrous sack which limits cardiac motion. Rapid accumulation of fluid, particularly following penetrating cardiac trauma or aortic dissection, in the pericardial space can result in cardiac tamponade and hypotension (1).

### ***Mechanism of Injury***

The incidence of traumatic injury to the heart is difficult to establish. Many patients with cardiac

injury succumb at the scene of the injury. Aggressive resuscitation and rapid transport by trained personnel enable more patients with potentially lethal cardiac trauma to reach the hospital alive (2). The survival of these patients is directly related to rapidity of diagnosis, which requires a high index of suspicion and treatment of their injury.

Trauma to the heart and great vessels, can be divided into blunt and penetrating trauma. Blunt trauma is produced by deceleration, direct energy transfer, and compression. Rapid deceleration results in shear forces on the heart and great vessels (3). Depending on the force vector and body positioning during deceleration, various injuries result including blunt cardiac injury and aortic transection at the attachment of the ligamentum arteriosum. Chest compressions during cardiopulmonary resuscitation (CPR) are an iatrogenic cause of blunt cardiac trauma which may produce injury. Penetrating cardiac trauma is further subdivided into low velocity and high velocity injuries. Stab wounds are the prototypical low velocity wound whereas many gunshot wounds (GSW) are high velocity wounds. High velocity wounds transfer significant energy to surrounding tissue often resulting in more tissue destruction than that produced by the projectile's specific pathway.

### ***Penetrating Cardiac Trauma***

Victims of GSWs have more severe physiologic impairment than those with stab wounds. GSWs cause larger defects in the myocardium and pericardium, through-and-through wounds, and a larger number of injuries in other vital organs leading to hemorrhage and exsanguination (1). Stab wounds are more likely to produce small injuries that seal off during systole, leading to cardiac tamponade. Cardiac tamponade and exsanguination are immediately life-threatening complications of penetrating cardiac trauma. Prompt recognition and treatment is required to avoid poor outcomes. These wounds may produce a variety of lesions affecting the pericardium, cardiac wall, interventricular septum, cardiac valves, chordae tendineae, papillary muscles, and coronary vessels (1). In a study of 711 penetrating cardiac trauma patients, there was a 47% mortality rate (stab wounds 54%, GSW 42%). Right heart wounds were noted in 64% of penetrating cardiac trauma victims (24% RA, 40% RV) while LV injury was noted in 40%. Approximately 5% of patients suffered injury to a coronary artery with a predominance for LAD. Additional defects reported were the creation of a ventricular septal defect (VSD) and damage to the mitral valve. Occasionally, penetrating cardiac trauma can occur from a blunt injury. If sufficient blunt force to the thorax results in fracture of the rib or sternum, the sharp bone fragments can be displaced into the heart with a resultant penetrating wound. Mortality from penetrating heart trauma is high. In a study of 117 patients, mortality from stab wounds was ~15% whereas GSW resulted in an 81% mortality. Cardiac tamponade was noted in 53% of the patients with stab wounds. However mortality was only 8% in this subpopulation. The authors concluded that cardiac tamponade was associated with improved survival in patients with stab wounds of the heart. Exsanguination was an important contributing factor in the fatalities. Penetrating injury to the great vessels may also occur and the clinical presentation varies greatly depending on the site and extent of the wound. Penetrating trauma of the proximal pulmonary arteries, terminal pulmonary veins or vena cava has a very high mortality rate (~75%). Fractures of the sternum or rib(s) can result in avulsion of the internal mammary or intercostal arteries, respectively. It is important to recognize that these vessels can be lacerated iatrogenically (during needle decompression of a tension pneumothorax or during chest tube placement).

Common mechanisms of death after penetrating cardiac trauma are from cardiac tamponade and exsanguination (1). History of chest trauma, physical examination findings of Beck's triad

(distended neck veins, hypotension and muffled heart tones) or pulsus paradoxus supports the diagnosis of cardiac tamponade. The neck veins may not be distended when tamponade is accompanied by severe hypovolemia. ECG may show electrical alternans. If the pericardial defect caused by penetrating trauma is large and remains open, the blood drains into the pleural space and produces hemothorax and hemorrhagic shock.

### ***Diagnostic Strategies***

Owing to the absence and/or lack of specificity of some of the clinical manifestations of acute cardiac tamponade, its diagnosis may be overlooked unless a high index of suspicion is maintained. Chest radiography is nonspecific, but it can identify hemothorax or pneumothorax and demonstrate an enlarged cardiac silhouette suggesting pericardial fluid. Other possibly indicated examinations include ultrasonography, central venous measurements, subxiphoid pericardial window, thoracoscopy, and pericardiocentesis.

### ***Ultrasonography***

Focused assessment for the sonographic examination of the trauma victim (FAST) evaluates four anatomical windows for the presence of intra-abdominal or pericardial fluid. FAST examination. Echocardiographic features of tamponade include collapse of the thin, flexible right atrial free wall for greater than 1/3 of systole, diastolic collapse of the right ventricle, inferior vena cava plethora, and respiratory variations of left and right ventricular diastolic filling. Transthoracic or transesophageal echocardiography may be useful in identifying and characterizing valvular abnormalities and septal defects.

### ***Subxiphoid Pericardial Window***

Subxiphoid pericardial window has been performed both in the emergency department and in the operating room with the patient under either local or general anesthesia (1). Via subxiphoid vertical incision, a small hole is made in the pericardium to determine the presence of blood. The disadvantage of a subxiphoid pericardial window is that it is an invasive procedure, and if a major injury is found, a second thoracic incision is required for definitive repair. Ultrasonographic evaluation has almost eliminated the role of subxiphoid pericardial window in the evaluation of cardiac trauma.

### ***Pericardiocentesis***

In the setting of trauma, cardiac tamponade is acute and caused by hemorrhage. Clot forms quickly and is not amenable to needle drainage. Currently, many trauma surgeons discourage pericardiocentesis for acute trauma. Recurrence of tamponade and subsequent increase in mortality, as well as a significant incidence of false negative results and potential for iatrogenic injury, makes pericardiocentesis a less than optimal diagnostic tool.

### ***CVP***

In the absence of immediate availability of ultrasonography, the determination of CVP is the best test for reinforcing the suspected diagnosis of acute traumatic cardiac tamponade. The combination of shock and an elevated CVP in a patient with cardiac trauma should immediately suggest cardiac tamponade. Other differential considerations of these signs include tension pneumothorax, RV failure, myocardial contusion, superior vena cava obstruction, ruptured tricuspid valve, or preexisting severe pulmonary disease.

GSWs of the heart have several unique considerations with respect to anesthetic management.

The potential exists for transmediastinal injury including the great vessels and the esophagus. Traumatic esophageal perforation may be worsened with TEE. Placement of a TEE probe may therefore be contraindicated. Missile embolus can occur with GSW of the heart. This occurs when the bullet or shrapnel fragment penetrates a vascular structure and then is carried by blood flow until it lodges in the arterial tree at a remote site where it can produce end-organ ischemia. The trauma care team can be distracted by the penetrating cardiac trauma and neglect to search for missile embolus preoperatively. Appropriate evaluation for missile embolus should occur prior to leaving the operating room to avoid prompt return for embolectomy.

### ***Blunt Cardiac Trauma***

Myocardial contusion (also called cardiac contusion) is a common diagnosis in patients with a history of blunt chest trauma. Considerable controversy exists about the diagnostic criteria and clinical significance of myocardial contusion. A diagnosis of blunt cardiac trauma consistent with myocardial contusion is typically made from a history of chest trauma along with abnormalities in the ECG, cardiac enzymes and/or echocardiography. Serum cardiac troponins, troponin I and troponin T, are highly specific for myocardial injury. The majority of patients diagnosed with cardiac contusions have unremarkable recovery from a cardiac standpoint. At the cellular level, blunt cardiac trauma can result in myocardial hemorrhage, myocardial edema, myocardial inflammation and myocyte necrosis. During histopathological examination cellular disruption is noted and correlates linearly with force of blunt trauma. Damage at the tissue and cellular level is heterogeneously distributed, with patchy areas of trauma interspersed with normal appearing myocardium. This results in abnormal electrical conduction and contractility of the damaged myocardium. Cardiac contractile dysfunction resulting from myocardial contusion typically improves over time.

Clinically, the RV is at greatest risk for blunt cardiac trauma as this is the most anterior chamber of the heart. RV contusion can result in RV contractile dysfunction which in turn leads to systemic hypotension from decreased left ventricular filling (2). RV contusion is frequently associated with pulmonary contusion which can synergistically contribute to right heart failure. Pulmonary contusion results in increased interstitial pulmonary edema and hemorrhage, diffusion abnormalities and hypoxia which all contribute to increased pulmonary artery resistance. The contribution made by positive pressure ventilation further augments the pulmonary arterial pressures. Mechanical ventilation, decreased pulmonary compliance (with increased peak and plateau airway pressures) and utilization of positive end-expiratory pressure (PEEP) all serve to increase mean airway pressure which translates into increased pulmonary arterial pressure. Concomitant pneumothorax and hemothorax can add to the increased intrathoracic pressure. Pulmonary contusion can thus cause acute pulmonary hypertension concurrent with attenuated RV function to manifest as right heart failure.

Cardiac dysrhythmia is a typical sign of myocardial contusion. Sinus tachycardia is the most common abnormality noted but can only be attributed to myocardial contusion when other causes of tachycardia have been ruled out, notably hypovolemia and pain. All dysrhythmias have been associated with myocardial contusion including supraventricular arrhythmias, conduction delays and ventricular dysrhythmias. Fatal ventricular arrhythmias can result from a reentry mechanism in myocardial contusion, although this is rare. Definitive diagnosis of myocardial contusion is difficult. A clinical history of chest trauma, even a minor low-speed impact, is the only constant feature of the diagnosis. Physical examination may reveal signs suggestive of trauma. Fractures of the ribs, sternum or clavicle are particularly correlated with

myocardial contusion. Chest radiographs may also provide clues to the diagnosis. Radiographic findings of sternal or rib fractures, pulmonary contusion, pneumothorax, hemothorax or widened mediastinum should raise suspicion of myocardial contusion. The primary diagnostic modalities for myocardial contusion are ECG, troponin I and echocardiography. TEE has superior diagnostic capabilities compared to transthoracic echocardiography and is feasible in a high percentage of the trauma population.

Myocardial contusion can impact anesthetic management. Specifically these patients are at increased risk for hypotension and dysrhythmia. Risk factors associated with perioperative mortality in myocardial contusion patients include atrial fibrillation, aortic rupture and advanced age. Patients who display any dysrhythmia during a procedure or have hypotensive episodes attributed to myocardial contusion should have increased postoperative observation and monitoring (4).

### ***Initial Management Issues***

On initial presentation to the emergency center, airway, breathing, and circulation (ABCs) under Advanced Trauma Life Support (ATLS) protocol are evaluated and established. Two large-bore intravenous catheters are inserted, and blood is typed and cross-matched. The patient is examined for signs of symptoms of cardiac tamponade. The presence of a pneumothorax or hemothorax, which is often associated with penetrating cardiac trauma, must be treated expeditiously with tube thoracostomy (3). Bedside echocardiography should be performed as quickly as possible to establish the diagnosis of pericardial effusion with tamponade physiology, which then mandates urgent surgical repair. Patients with penetrating cardiac injury invariably require surgical repair. The location (operating room versus emergency department) and timing (immediate versus urgent) depends on the patient's clinical status. Emergency department thoracotomy is a drastic, dramatic, and potentially lifesaving procedure (5-7). With thoracotomy, the physician seeks to (1) to relieve any cardiac tamponade, (2) to support cardiac function with direct cardiac compression and/or cross-clamping of the aorta to improve coronary perfusion, and (3) internal defibrillation when indicated. The physician should have a systematic plan prior to opening the chest. The decision should be based on a realistic judgment that the patient has a chance of survival, but will not tolerate any delay in operative intervention. It is also important to consider not performing thoracotomy for cases in which there is virtually no chance of salvaging a neurologically uncompromised patient. Important information in formulating a decision to perform emergency department thoracotomy includes time of injury, transport times to the emergency department, the time of vital signs or cardiac electrical activity or both ceased. Patients with penetrating trauma with signs of life in the field, even if only electrical activity on cardiac monitor or agonal respirations, are candidates for emergency department thoracotomy if transport times are less than 10 minutes. For patients in cardiac arrest, tracheal intubation and short duration of CPR correlate with survival following thoracotomy.

### ***Definitive Treatment***

Definitive treatment involves surgical exposure through a thoracotomy or median sternotomy (1). Thoracotomy usually necessitates one-lung ventilation. Significant blood loss should be anticipated. Coagulopathy is common and multifactorial. Adequate intravenous access should be verified. Arterial line and CVP monitoring is routine. Blood products should be immediately available during the operative course. Thermal conservation measures should be employed early in the operative course to limit the adverse effect of hypothermia on hemostasis. A rapid infusor should be used to deliver warmed fluids and blood. Obtaining a coagulation profile via

thromboelastography will guide rational blood component therapy administration. A massive transfusion protocol may be necessary. Chest tube placement should be considered prior to or coincident with anesthetic induction because of the risk of pneumothorax. The choice of induction agents is less important than the selection of an appropriate dose. Stab wounds of the heart can usually be repaired without the use of cardiopulmonary bypass (CPB). Temporary asystole can be induced with adenosine which allows the surgeon time to accurately place the required number of sutures in a semibloodless and motionless field to adequately control hemorrhage (8). Rapid bolus of adenosine, 6-12 mg, IV results in acute inhibition of sinus node and atrioventricular node function, sinus bradycardia, transient atrioventricular block, and asystole. During the period of asystole, the heart is rendered completely flaccid and more amenable to manipulation, especially when dealing with the lateral wall. Owing to adenosine's ultrashort half-life, asystole lasts for approximately 15-20 seconds with prompt restoration of sinus rhythm afterwards. ECG monitoring, pacing backup, corrective measures for untoward hemodynamic effects (e.g., ephedrine, low dose epinephrine, phenylephrine), and defibrillation capabilities are required. Certain injuries will require CPB such as valvular disruption, pulmonary artery lacerations and ventricular septal defects. CPB permits extensive rotation, retraction, and displacement of the heart. It allows cardiac repair to be carried out without any heart motion and is necessary for repair of inaccessible wounds, large left atrial, coronary sinus, or high posterior LV wounds and coronary artery repair or bypass. The coronary arteries must be avoided in all cases of cardiac suture (9). Treatment of RV and LV dysfunction and arrhythmias are often required. Pacing wires are placed for treatment of heart block or bradycardia. Patients who have suffered shock or cardiac arrest secondary to tamponade or bleeding often have received massive amounts of volume and inotropes. Hypothermia, acidosis and coagulopathy will require ongoing treatment in the intensive care unit and postoperative mechanical ventilation is routine in these situations. Residual lesions and delayed sequelae of penetrating cardiac trauma include residual shunts and fistulae, valvular dysfunction, and ventricular pseudoaneurysms.

### ***One-Lung Ventilation***

Options for one-lung ventilation and lung isolation include double lumen tubes (DLT), Univent, and bronchial blocker (10). Left sided DLTs are preferred at the authors' institution because they provide excellent lung isolation, are quickest to place successfully, permit bronchoscopy and suction to the isolated lung, and allow addition of continuous positive airway pressure (CPAP). The main disadvantage is non-optimal postoperative two-lung ventilation. Bronchial blockers are used for patients with known difficult airway anatomy and in patients whose tracheas are already intubated. Tube exchange from single lumen to DLT is also an option. Fiberoptic bronchoscopy is vital for positioning DLTs and bronchial blockers. If the patient requires postoperative mechanical ventilation, the previously placed DLT may be withdrawn so that the bronchial cuff is in the mid-tracheal position. The bronchial tube is then used as a single lumen tube and the tracheal tube is clamped. This may be necessary when the risk of re-intubation with a single lumen tube is judged to be unacceptably high. Otherwise, tube exchange is generally done using an airway exchange catheter and profound neuromuscular block. Profound neuromuscular blockade prevents any deleterious movement during tube exchange such as coughing/bucking, significantly increasing the success rate. There is always a risk of losing the airway during a tube exchange. A back-up plan, including a surgical airway, is mandatory. With a bronchial blocker technique, tube exchange is not necessary.

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