A primer for students regarding advanced topics in cardiothoracic surgery, part 1: Primer 6 of 7

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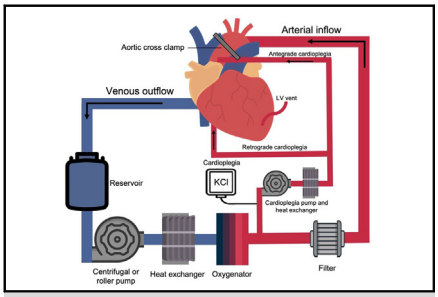

Simplified schematic diagram of cardiopulmonary bypass circuit.

CENTRAL MESSAGE

Cardiopulmonary bypass and extracorporeal membrane oxygenation are important modalities to support a patient's heart and/or lung function during cardiac surgery or cardiopulmonary failure.

CARDIOPULMONARY BYPASS

Cardiopulmonary bypass (CPB) was first successfully used by Dr John Gibbon Jr, in 1953 at the Jefferson Hospital in Philadelphia to repair an atrial septal defect.[1] As the name suggests, CPB was designed to substitute for the function of the heart and the lungs, thereby allowing a surgeon to operate on these structures in a relatively bloodless field. Specifically, CPB has 5 objectives:

- To oxygenate and remove carbon dioxide from the blood (replace pulmonary function);
- To provide sufficient systemic circulation (replace cardiac function);
- To provide a bloodless surgical field by draining the cardiopulmonary system;
- To thermoregulate the patient; and
- To protect the heart during the operation with thermoregulation and cardioplegia delivery.

A simplified schematic diagram of a CPB circuit is depicted in Figure 1. It is essential to understand the layout and the management of CPB to participate in the operative care of cardiac surgery patients.

The CPB circuit typically provides a constant flow of oxygenated blood. To do so, the venous side of the circulation needs to be drained from the patient using drainage (or venous) cannula(s), placed via the superior and inferior vena cava or their confluence at the right atrium. Once the cannulas are inserted, venous blood drains passively into the reservoir of the system located lower than the patient. In cases where passive drainage is insufficient, active suction can be used to increase venous drainage.

The blood is then oxygenated and ventilated with a membrane oxygenator, with carbon dioxide levels controlled using sweep flow (ventilating gas flow rate) before it is returned to the patient via an arterial cannula that can be positioned in the ascending aorta, axillary artery, femoral artery, or other arterial positions. Flow can be accomplished via a centrifugal pump or roller pump. Figure 2 depicts the relationship between afterload and flow rate for both pump types. Centrifugal pump is preload-dependent and afterload

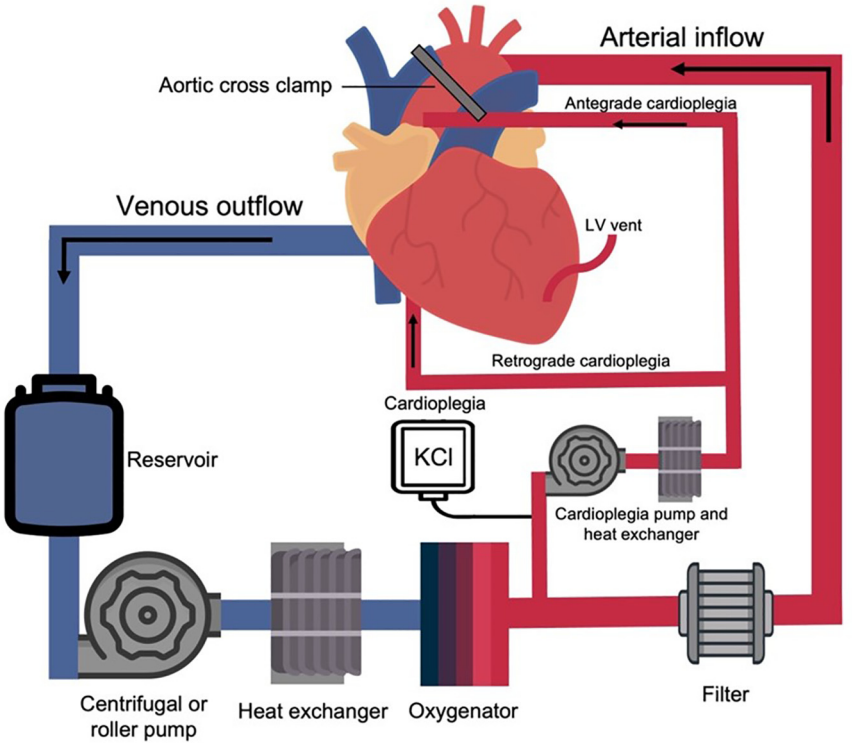

FIGURE 1. Simplified schematic diagram of a cardiopulmonary bypass circuit depicting the outflow, inflow (including cardioplegia administration routes), and major components. *LV*, Left ventricle; *KCl*, potassium chloride.

sensitive, whereas roller pump (further discussed in the Bloodless Field section) provides a relatively fixed flow rate as it works by positive fluid displacement.

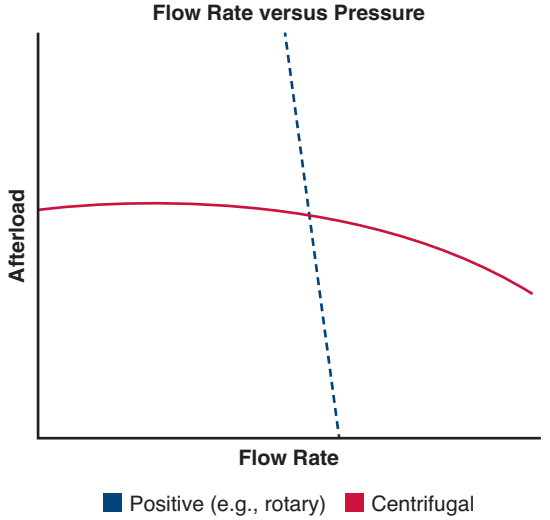

FIGURE 2. Simplified depiction of the relationship between pressure (measured as afterload) and flow rate of centrifugal and positive displacement (rotary or roller) pumps. The flow rate of centrifugal pumps decreases with increasing afterload, whereas positive displacement pumps have a relatively fixed flow rate. Figure modified from Ostadfar A. Biofluid flow in artificial, assistive and implantable devices. In: *Biofluid Mechanics*. Elsevier; 2016: 205-42. doi:10.1016/B978-0-12-802408-9.00005-3.

Among the feared complications in providing systemic perfusion is entraining air within the circuit and causing embolic complications such as stroke. All circuits have fail-safe devices so flow is immediately stopped when air is detected in the system. De-airing the circuit is beyond the scope of this section. The patient must be sufficiently anticoagulated to be placed on CPB (further discussed in the Consequences of Cardiopulmonary Bypass section).

Additional Drainage

During CPB, venting or the removal of excess blood is important to prevent myocardial or vascular injury from the high pressures sometimes encountered during CPB. Multiple vents have been employed, but 2 are most common: left ventricular vent and aortic root vent (often termed root vent). The left ventricular vent is typically placed via the right superior pulmonary vein and passed through mitral valve into the left ventricular cavity. This vent drains the blood accumulated within left side of the heart from the bronchial and Thebesian veins. The aortic root vent allows suction to be applied to the aortic root, thereby indirectly emptying the left ventricle. Both vents have the dual role of evacuating air that has become trapped during surgery, thereby decreasing the risk of air embolism.

Bloodless Field

Among the benefits of CPB is that blood in the operative field (along with the blood in the patient's circulatory

system) can be returned to the CPB circuit and then back into the systemic circulation, thereby minimizing blood loss. By employing cardiotomy suckers, 1 of the roller pumps in the CPB circuit provides suction for the field. The sucker tips may be configured to be left in the dependent region in the operative field or be hand-held to enable directed aspiration of blood.

Additionally, blood can be salvaged using a cell-saver device that filters and processes the blood and packages it in blood bags that can be intravenously administered by the anesthesia team intraoperatively or postoperatively in an intensive care unit. This approach further reduces any net blood loss during the procedure by allowing blood loss at all stages to be returned to the patient.

Thermoregulation

The CPB circuit can heat and cool the oxygenated blood returning to the patient. Whereas the cardioplegia solution for myocardial protection is routinely cooled to 4 °C, the CPB circuit can cool and subsequently warm the patient in a systemic fashion. Systemic cooling can be of substantial benefit when performing surgery that is not safe metabolically at physiologic temperatures (discussed below). However, thermoregulation is associated with potential complications of which the surgeon and the perfusionist must be aware.

Myocardial Protection

Protection of the heart during surgery is vital. The CPB machine is able to cool and provide cardioplegia to the heart. Cardioplegia is designed to silence the electrical activity of the heart and reduce its metabolic rate. This process is accomplished through a cold (4 °C) solution with high potassium levels that reduces the electrochemical gradient leading to diastolic relaxation of the myocardium. Cardioplegia requires regular redosing, the timing of which depends on the type of cardioplegia being administered. The surgeon and perfusionist are in constant communication during the surgery regarding the timing, frequency, and volume of this redosing.

Avoiding ischemic insult to the myocardium during cardiac surgery is a primary goal of techniques of myocardial protection. However, myocardial injury may occur. The right ventricle tends to be susceptible to ischemic injury primarily for 2 reasons. First, depending on the procedure, cardioplegia may be administered and perfuse the myocardium in a retrograde fashion through the coronary sinus. Compared with the left ventricle, the right ventricle has more direct venous return via the Thebesian veins that drain into the cardiac chamber and not into the coronary sinus. Thus, retrograde cardioplegia will often favor left heart protection compared with that of the right heart. Second, the right ventricle has an anterior position, leading to greater exposure to room air and operating room lights, influencing optimal myocardial temperature and increasing risk of ischemic insult. Thus, topical hypothermia using iced saline solution/slush may be placed over the heart to enhance myocardial cooling.

An important approach to myocardial protection is infusion of antegrade cardioplegia directly into the coronary arteries. Antegrade cardioplegia is often delivered into the aortic root via 1 limb of the cardioplegia/root vent cannula (with the root vent clamped). This cannula is placed in the ascending aorta between the aortic crossclamp and a competent aortic valve. Delivery of the cardioplegia solution thus perfuses the myocardium via the coronary artery in an antegrade fashion. Alternatively, in patients undergoing aortic valve surgery or other surgery requiring an aortotomy, antegrade cardioplegia may be directly infused into the coronary ostia using handheld cannulas. Although beyond the scope of this primer, the atherosclerosis of the right and left coronary arteries can interfere with optimal antegrade cardioplegia administration.

CONSEQUENCES OF CPB

Volume Management

A key feature of the CPB circuit and one that distinguishes CPB from extracorporeal membrane oxygenation (ECMO) is that with the former the reservoir is open to the air. This system thus allows for cardiotomy suckers to be attached to the circuit, venous system to be passively drained, and potential for large volume infusions. A reservoir permits the surgeon and the perfusionist to regulate the amount of circulating volume in the patient. At the time of separating from CPB, the majority of the blood volume is generally returned to the patient to sustain hemodynamic stability.

Hemodilution

The CPB circuit is typically primed with crystalloid solution to ensure there is no air in the system prior to connecting to the patient. Upon initiating CPB, crystalloid will enter the patient's blood volume leading to hemodilution and potential ischemic complications. One method proposed to minimize hemodilution is retrograde autologous priming performed after the patient is attached to the CPB circuit but before initiation of bypass. In this fashion, a portion of the patient's blood is drawn into the circuit, displacing the crystalloid prime and reducing the hemodilution.

Anticoagulation

As noted, the CPB system includes a reservoir that is open to the air. This air-fluid interface, as well as the tubing for the circuit, results in a procoagulant and inflammatory response. To mitigate the risk of thrombus formation within the system, a patient is systemically anticoagulated with heparin before cannulation and initiating CPB. The degree of anticoagulation is monitored by the activated clotting time (ACT). As a general rule (recognizing variability

among surgeons), the target ACT should be >400 seconds. Of note, entrance of nonheparinized blood into the CPB circuit as well as other factors can influence the ACT; therefore, ACT is regularly monitored throughout the procedure.

Because of the dynamics of coagulation and anticoagulation, as well as inherent risks of CPB such as platelet dysfunction and effects of hypothermia, underlying coagulopathies or consumption of clotting factors can contribute to bleeding in the postoperative setting. Many have proposed the use of thromboelastogram, which aids in identifying the coagulation issues and the blood component to administer (eg, platelets vs fibrinogen vs clotting factors). An example of a thromboelastogram and its diagnostic utility is shown in Figure 3.

When weaning or separating from CPB, reversal of heparin is required with protamine. During infusion of protamine, one main concern is the potential for protamine reactions, of which there are 3 types.

- Mild: A transient hypotension with administration
- Moderate: An anaphylactic-like response with bronchial constriction
- Severe: Profound pulmonary hypertension with systemic hypotension

Strategies to avoid adverse reactions include an initial intravenous testing dose by anesthesia and a slow infusion of protamine. Significant hemodynamic instability and hypotension requires vasopressor support and consideration of reheparinization and resuming CPB.

Inflammation

The air-fluid interface and tubing contact promotes an inflammatory environment. Additive effects include the mechanical shearing forces on the red blood cells and platelets from the CPB system and the use of cardiotomy suckers. These perturbations are more pronounced with roller pumps compared with centrifugal pumps, recognizing the tradeoffs of using the latter. The net result of CPB is reduced platelet function, decreased hematocrit level, and increased levels of free hemoglobin in the blood. One proposed mechanism is that nitric oxide reacts with free hemoglobin, reducing the levels of available nitric oxide and resulting in impaired vasodilation.[2] These events may lead to end-organ damage, in particular acute kidney injury.[3] Although the surgery itself may only take a matter of hours, CPB and the stress of surgery activate cellular cascades, which may be prolonged and require intensive postoperative management.

Failure to Separate from CPB

Successful separation from CPB involves the transition from pump-assisted circulation to spontaneous cardiopulmonary activity with adequate blood flow and tissue perfusion. This complex process requires the vigilance of the surgeon, anesthesiologist, and perfusionist. Fundamentally, it requires a stable cardiac rhythm, adequate hemostasis, and sufficient cardiac and respiratory function. Thus, the anesthesiologist utilizes transesophageal echocardiography to monitor for changes in contractility, air emboli, filling and/or distension of the cardiac chambers, and more. Meanwhile, the surgeon is visually inspecting the heart for contractility, stable rhythm, and hemostasis in the surgical field. In general, the venous drainage is gradually decreased (increasing the patient's blood volume) while reducing the arterial flow rate of the pump. This process allows the patient's own heart to take over the circulation. However, if the above-mentioned requirements are not met, the patient may not be able to be weaned from CPB. Older patients and those with preoperative left ventricular dysfunction, mitral regurgitation, coagulopathy, or longer ischemic time or duration of CPB are at increased risk of failure to wean.[4] The issue(s) precluding separation from bypass should be identified and addressed, if possible. If the patient remains dependent on CPB, temporary support options can

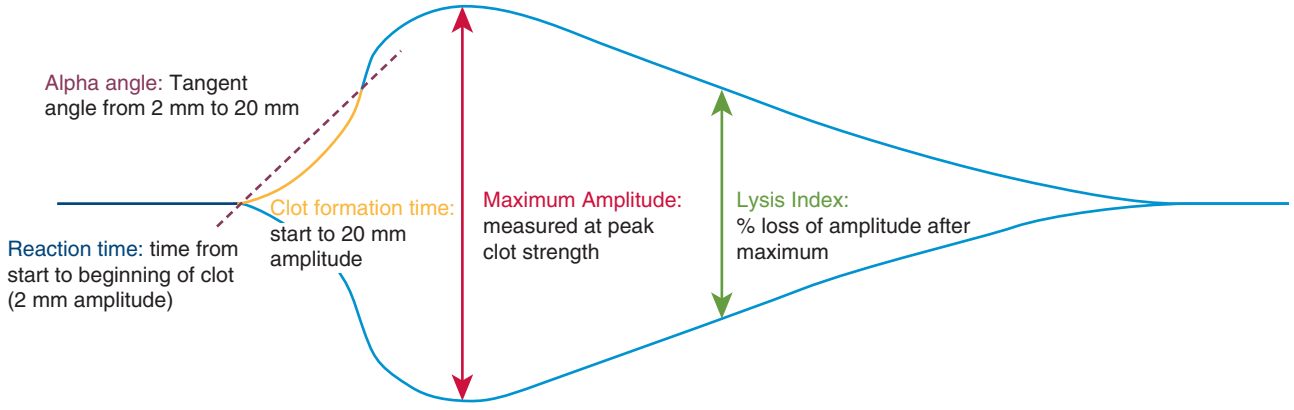

FIGURE 3. Sample thromboelastogram waveform, depicting reaction time, alpha angle, clot formation time, maximum amplitude, and lysis index. Aberrations in these values may provide insight into the nature of the coagulopathy.

be employed, including intra-aortic balloon pump, ECMO, or ventricular assist devices.

SPECIAL TOPICS RELATED TO CPB

Deep Hypothermic Circulatory Arrest

Operations where the patient is cooled significantly (20 °C or below) are termed deep hypothermic circulatory arrest (DHCA). The goal of DHCA is to decrease the metabolism of the entire body, most importantly the brain, to allow for periods of no systemic flow. This technique enables short durations of completely bloodless field, where conventional CPB is not sufficient. The main indications for DHCA are surgeries involving the aortic arch, such as ascending aortic dissection with an open distal anastomosis or arch aneurysm repair, and pulmonary thromboendarterectomy. A relationship between systemic temperature, estimated safe arrest time, and brain metabolism is depicted in Figure 4.

One issue with DHCA is knowing when the patient is sufficiently cooled based on their metabolic activity. The use of neuromonitoring (see below) helps to direct DHCA cooling. Monitoring brain activity allows the surgeon to know when the patient is sufficiently hypothermic (ie, electrocerebral inactivity). Electrocerebral inactivity indicates that the metabolic activity of the brain is low enough that it is safe to arrest or cease circulatory support. Typically, the duration of DHCA employed is <30 or 40 minutes, with an increased rate of deficits associated with longer periods. The use of DHCA also adds a substantial amount of time to the procedure because of the time required for cooling and rewarming.

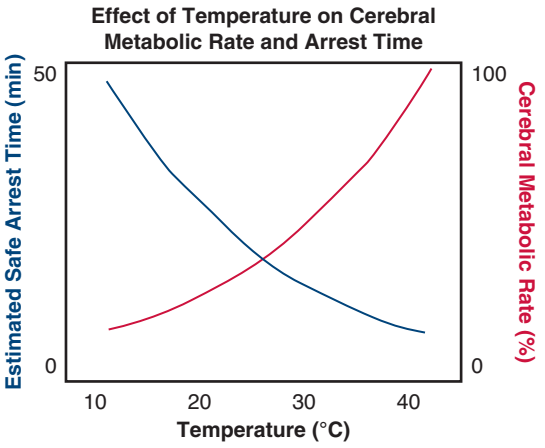

FIGURE 4. Relationship between temperature, cerebral metabolic rate (*red line*, percent of baseline levels), and the estimated time of safe circulatory arrest (*blue line*, minutes). As the temperature is lowered, the relative cerebral metabolic rate decreases and safe circulatory arrest time increases. Figure modified from Tian DH, Yan TD. Current status of open surgery in aortic arch surgery. In: *New Approaches to Aortic Diseases from Valve to Abdominal Bifurcation*. Elsevier; 2018: 307-13. doi:10.1016/B978-0-12-809979-7.00027-4.

The rewarming gradient (how cold the patient is compared to the temperature of the blood from the CPB circuit) is held around 10 °C or less and is adjusted so that warming is less than 0.5 °C per minute. Rapid rewarming can lead to damaging the brain and vasculature and protein denaturation.

One strategy to limit cerebral ischemia is to selectively perfuse the brain during hypothermic circulatory arrest using antegrade cerebral perfusion (ACP) or retrograde cerebral perfusion. These techniques, where ACP may be employed with moderate hypothermic circulatory arrest, continue to be evaluated and often are dependent of the extent of surgery (ie, anticipated circulatory arrest time) and surgeon preference. In recent meta-analyses, the postoperative incidence of stroke, early mortality, and permanent neurological dysfunction was similar between ACP and retrograde cerebral perfusion.[5,6] However, a trend toward decreased temporary neurological dysfunction was found in ACP.[5,6] Given these findings, the ideal cerebral protection strategy should be individualized based on patient characteristics, surgeon preference, and hospital resources.[7]

Neuromonitoring

The goal of neuromonitoring intraoperatively is to detect signs of central nervous system ischemia. The neuromonitoring techniques facilitate operative decision making to reduce neurological deficits after cardiac surgery. Neuromonitoring is employed in cases DHCA and aortic surgery (thoracic or thoracoabdominal). There are 3 complementary approaches of monitoring: electroencephalogram (EEG), somatosensory evoked potential (SSEP), and motor evoked potential (MEP).

EEG assesses electrical silence during DHCA and potential ischemia during the operation. A key abnormal finding is asymmetrical changes in the EEG waveforms. SSEPs are used to determine spinal cord ischemia, particularly involving the posterior spinal arteries and dorsal column. SSEPs are assessed by stimulating sensory nerves at the extremities and seeing the propagation of that signal back to the brain. Finally, MEPs stimulate the motor cortex and observe the propagation through the descending motor tracts to the extremities. MEPs are sensitive to ischemia involving the anterior spinal arteries and, by extension, the radicular arteries of the aorta.

These measurements help determine whether there is a risk of spinal cord ischemia and subsequent deficit; the findings allow the surgeon to intervene. For example, SSEPs are typically normal if perfusion is 20 mL/100 g/hour. Spinal cord damage occurs at rates of 10 mL/100 g/hour or lower. In between these 2 values, the SSEPs will be abnormal, allowing for interventions such as permissive hypertension. For MEPs, ischemia of the radicular arteries, and thus the anterior spinal artery, triggers abnormal signals. This

modality is useful for procedures such as thoracic endovascular aortic repair for descending thoracic aortic pathology or thoracoabdominal aneurysm repair, during which flow to the radicular arteries may be compromised. If the MEPs are abnormal, a surgeon may decide to reimplant the intercostal arteries (for open surgical procedures), increase the blood pressure, or decrease the intracranial/spinal cord pressure.

Left Heart Bypass

Another type of CPB is left heart bypass. As implied, this technique bypasses only the left side of the heart, maintaining normal flow through the right side and the native pulmonary circulation. Indications for this approach are open surgical procedures involving the descending thoracic or thoracoabdominal aorta. Left heart bypass allows for perfusion of the branches of the aorta that are not directly excluded during the surgery (ie, by the crossclamp) and requires close monitoring. Depending on the cannulation setup, the bypass circuit will not drain all the flow into the left atrium. Instead, there is a balance of how much blood to allowed into the left ventricle, and thus antegrade aortic flow, and how much blood will be diverted to the rest of the systemic circulation via distal aortic or arterial cannulation. Of note, in extensive descending and thoracoabdominal aortic surgery, left heart bypass may not be feasible, and conventional CPB and DHCA may be necessary.

ECMO

ECMO is a form of temporary circulatory support that has been used over the last decade following the H1N1 influenza pandemic. Since its clinical success in 1970,[8] ECMO has become an accepted support modality for patients with respiratory and/or cardiac failure refractory to all other therapies. Although conceptually similar to CPB, modifications to the ECMO circuit allow for a longer duration of cardiopulmonary support than afforded by traditional CPB. ECMO is a closed circuit with no open reservoir where blood contacts air and does not require the same degree of anticoagulation as CPB.

Components and Configurations of ECMO Circuit

ECMO drains venous blood from the patient, exchanges gases, then returns the blood back to the patient. The main components of the ECMO circuit are the inflow and outflow cannulas (defined in relation to the pump, not the patient's body) for draining and returning blood, respectively, a pump, and an oxygenator. Other components include pressure and flow sensors, a heat exchanger for heating or cooling blood, and arterial or venous ports for drawing blood.

Important ECMO concepts in a clinical setting include flow rate, speed, fraction of inspired oxygen, and sweep. Flow rate describes the volume of blood per minute being delivered. Depending on the patient's native cardiac output and the setting of the ECMO circuit, ECMO flow may account for a variable percentage of the patient's total cardiac output. Flow is modulated using the pump speed (in rotations/minute). Sweep gas is how carbon dioxide is removed from the patient's blood and is accomplished by flowing a gas (typically 100% oxygen) through the oxygenator. Sweep gas flows through the oxygenator, which is a microporous tube, while the patient's blood flows around it. This interface between the sweep gas and the blood is where gas exchange occurs. The pressure gradient between the patient's blood flow and the sweep gas flow determines the level of oxygenation and ventilation. Increased sweep gas flow (measured in liters per minute) leads to increased decarboxylation of the blood. Thus, increasing the sweep can take over pulmonary function when a patient is very ill, and titrating the sweep down can be done to assess a patient's ability to ventilate before decannulation. Finally, fraction of inspired oxygen, as in ventilators, is the percentage of oxygen in the gas being flowed through the oxygenator. This determines the partial pressure gradient between the patient's blood gas and is thus important for both oxygenation and ventilation.

There are numerous factors one must consider before initiating ECMO for a patient. These include the severity of the cardiac and pulmonary failure, whether or not it is responsive to more conservative treatment modalities, the overall prognosis of the patient, and the current resource utilization of the hospital system. The only absolute contraindication to ECMO is a preexisting condition that is incompatible with recovery, such as advanced malignancy or severe brain injury. Relative contraindications include very poor prognosis, advanced age, and severe coagulopathy.

ECMO configurations can be generally classified into 2 different types (Figure 5).

- Veno-venous (VV) ECMO: In VV ECMO, the circuit both drains the blood from and returns the blood to the patient's venous system. Because the blood is returned to the venous system prior to entering the pulmonary circulation, VV ECMO relies on the patient's native cardiac output to circulate the newly oxygenated blood throughout the body. The most common configuration of VV ECMO consists of the inflow cannula placed in a femoral vein with drainage holes extending all the way up the inferior vena cava, with an outflow cannula placed in the right internal jugular vein. However, other options exist, such as femoral-femoral cannulation or a double lumen catheter inserted into the right internal jugular vein. Notably, the ECMO circuit is established in series with the patient's existing cardiopulmonary system and therefore does not augment cardiac function. VV ECMO is primarily indicated for patients with isolated respiratory failure. According to the Extracorporeal Life Support Organization, VV ECMO is indicated for

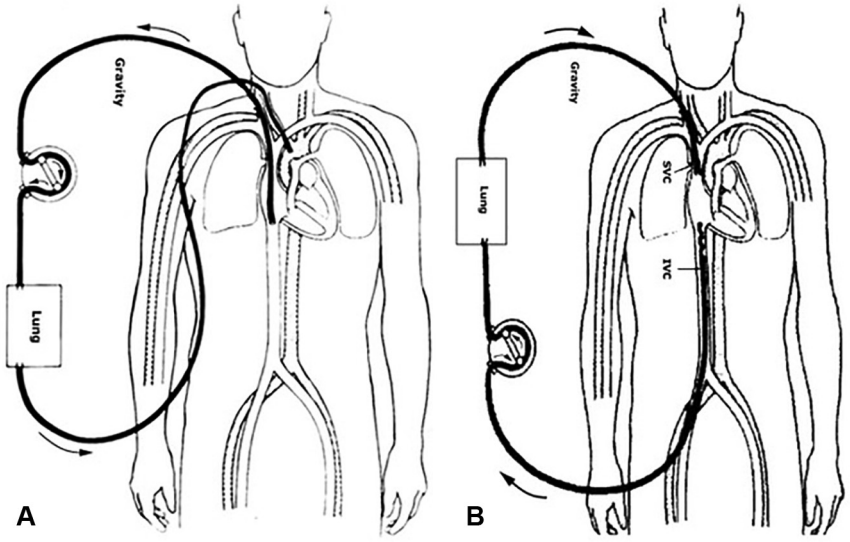

FIGURE 5. Diagrams of common extracorporeal membrane oxygenation (ECMO) configurations A, A venoarterial ECMO circuit with right internal jugular vein drainage and right carotid artery infusion. B, A venovenous ECMO circuit with right common femoral vein drainage and right internal jugular vein infusion. *SVC*, Superior vena cava; *IVC*, inferior vena cava. Figure adapted from public domain image. Van Meurs K, Lally K, Peek G, Zwischenberger. Right internal jugular vein drainage and right carotid artery infusion; Extracorporeal Life Support Organization, Ann Arbor 2005.

patients in: hypoxemic respiratory failure (Pao_2/fraction of inspired oxygen ratio <80 mm Hg) after optimal medical management or hypercapnic respiratory failure (pH <7.25), despite optimal conventional mechanical ventilation.[9] Specifically, bacterial pneumonia, viral pneumonia, and trauma-related acute respiratory distress syndrome are the most frequent indications.

- Veno-arterial (VA) ECMO: In VA ECMO, the inflow cannula still drains blood from the venous system, but the outflow cannula is placed into the patient's arterial system, thus creating a parallel circuit that can effectively augment the patient's cardiac output (partial bypass support). VA ECMO can be placed peripherally, with the inflow cannula placed in a femoral or internal jugular vein and an outflow cannula placed in the femoral artery. This peripheral configuration results in retrograde flow in the aorta, which mixes with the native cardiac output, and has important implications for potential left ventricular strain, root stasis, pulmonary edema, and north-south syndrome. Other peripheral cannulation sites include the axillary or subclavian arteries. Central cannulation, on the other hand, involves drainage from the right atrium and outflow into the aorta, which provides antegrade flow in conjunction with the native cardiac output. This method may facilitate the transition from CPB to ECMO if the patient fails to wean from CPB intraoperatively. VA ECMO is indicated for patients suffering from cardiac failure with or without respiratory failure. It is most commonly used to support patients in cardiogenic shock (systolic blood pressure <90 mm Hg), including acute coronary syndrome, myocarditis, pulmonary embolism, or failure to wean from CPB. Depending on the clinical context, VA ECMO can be utilized as a bridge to recovery or as a bridge to definitive therapies, such as ventricular assist device implantation or cardiac transplantation.
- Although VV and VA ECMO comprise the large majority of ECMO circuits used, there other circuits that utilize hybrid or parallel configurations that may be seen. These strategies may be useful if support on standard circuits is insufficient.[10] Veno-arteriovenous ECMO may be used if a patient supported by VV ECMO develops worsening cardiac function, necessitating placement of an arterial inflow cannula. Moreover, a patient supported by VA ECMO may lack adequate venous return, requiring placement of a second venous outflow cannula.

ECMO Complications

Several factors contribute to ECMO-related complications, including the physiologic condition of the patient, the nature of the materials used in the circuit, the amount of anticoagulation required to prevent thrombosis within the system, and the challenges associated with managing the circuit over time. To simplify, the following are technical and nontechnical complications. Technical complications include vascular injury during cannulation, cannula malposition, and mechanical failure of the circuit due to air emboli or thrombus formation. Although individual events are relatively rare, when combined, nearly one-third of ECMO runs require replacement of the system due to technical issues. Common nontechnical

complications include hemorrhage, neurologic injury, infection/sepsis, and thromboembolism. In general, VA ECMO is associated with higher rates of complications compared to VV ECMO, partially due to the cannulation of the arterial system. In particular, lower extremity ischemia can occur secondary to the return cannula placed in the femoral artery, which can be mitigated with a distal perfusion catheter. A summary of common complications associated with VA and VV ECMO is shown in Table 1.

One additional complication is harlequin syndrome (aka, north-south syndrome).[11] In this case, as the cardiac function starts to recover, ejecting some blood through the left ventricular outflow tract, the VA ECMO circuit is still providing the majority of systemic perfusion. If there is pulmonary dysfunction and the blood ejected from the left ventricle is not sufficiently oxygenated, the first organs this deoxygenated blood will perfuse are the heart, via the coronary circulation, and the brain. This complication is best monitored by right upper extremity or head saturations. Otherwise, the pulse oxygenation reading may appear fine–due to the VA ECMO oxygenation—while the heart and/or brain experience ischemic insults.

ADULT CARDIAC AND GENERAL THORACIC EMERGENCIES

Cardiac Emergencies

Blunt cardiac injury. Blunt cardiac injury, such as contusion, is often the result of sudden decelerations during motor vehicle collisions but can also occur from falls and crush injuries. The mechanism of injury includes shearing from rapid deceleration, pressure fluctuations in the chest and abdomen, and compression of the heart between the sternum and spine.[12] Signs and symptoms include shortness of breath, midsternal chest pain, persistent tachycardia, new-onset arrhythmia, and conduction abnormalities on electrocardiogram (EKG).[13] Progression to cardiogenic shock is possible and can involve rupture of valves, septum, or ventricular or atrial walls. Commotio cordis is a rare type of BCI in which low-impact chest trauma (eg, by a projectile during sports) can cause sudden cardiac arrest. Initial evaluation should follow Advanced Trauma Life Support protocol. A focused assessment with sonography for trauma exam may be used to look for hemothorax or pericardial effusion.

Hemorrhage, tamponade, and tension pneumothorax should be considered as causes of hypotension. EKG, echocardiography, and serial cardiac biomarkers (eg, troponin) are warranted. Computed tomography (CT) of the chest may identify cardiac injury and/or sternal fracture. Management of valve, septal, or ventricular wall injury requires immediate surgical consultation. Signs of myocardial infarction warrants cardiac catheterization with possible percutaneous coronary intervention, coronary artery bypass grafting, or thrombolytic therapy. Cardiac tamponade may require pericardiocentesis or surgical evacuation. Cardiac dysfunction (eg, hypotension, bradycardia, arrhythmia, heart failure, or hemodynamic instability) warrants continuous cardiac monitoring in the intensive care unit and cardiology consultation.

Penetrating cardiac injury. The mechanism of injury most commonly includes stab wounds from a sharp object (knife or broken rib) or firearm injury.[14] The right ventricle is commonly involved due to its anterior anatomical location, but more than one cardiac chamber may be involved in up to 30% of cases. Hemorrhage from penetrating cardiac injury can result in cardiac tamponade. Management of hemodynamically unstable patients requires fluid resuscitation and immediate transfer to the operating room. A focused assessment with sonography for trauma exam to locate source of bleeding may be performed in the emergency department or operating room before intervention. Evaluation of hemodynamically stable patients includes CT of the chest and/or abdomen with CT angiography to assess the thoracic aorta.[15] Management includes efforts to stop the bleeding and prevent contamination of the chest with abdominal contents in the case of concomitant abdominal trauma. If blood is seen in a subxiphoid pericardial window, sternotomy and open exploration of the chest may be required, particularly in the setting of hemodynamic instability.[16] If the patient is hemodynamically stable and there is no active bleeding, a drain may be placed with subsequent patient observation.[17]

When the chest must be explored for further evaluation of cardiac injury and/or persistent bleeding, various approaches may be used. Left anterolateral thoracotomy via the fourth or fifth intercostal space is the incision of choice for patients in extremis (ie, pulseless). It allows access to the apical anterior left and right ventricles. It does not provide access to the superior right ventricle or right atrium. Other

TABLE 1. Common complications associated with venoarterial (VA) and venovenous (VV) extracorporeal membrane oxygenation (ECMO)

Complication*	VA ECMO	VV ECMO
Technical		
Cannulation site vascular injury (dissection, hematoma, bleeding)	++	++
Cannula malposition	+	+
Air embolism	+	+
Oxygenator failure	++	++
Nontechnical		
Bleeding	+++	++
Thromboembolism	++	+
Coronary hypoxia	+	–
Neurologic injury	+++	++
Infection/sepsis	++	++
Pulmonary hemorrhage	+	–
Acute kidney injury	++	+

*Technical and nontechnical complications are listed separately. The number of plus signs (+, ++, and +++) indicates the relative occurrence rate of each during VA or VV ECMO.

indications for resuscitative thoracotomies include decompression of cardiac tamponade, direct control of blunt thoracic injuries, and clamping of the proximal aorta to prevent massive hemorrhage. After initial chest assessment, the inferior pulmonary ligament is incised, and the lung is mobilized to allow inspection of the mediastinum, pericardium, and phrenic nerve. A pericardial incision anterior and parallel to the phrenic nerve allows for evacuation of fluid from the pericardial sac. For patients in extremis with suspected injury to the upper right ventricle or atria, a clamshell thoracotomy extending the left anterolateral thoracotomy across the sternum to the contralateral intercostal space may be performed. The sternum is divided horizontally using a sternal saw and the bilateral internal mammary arteries are divided and ligated. A median sternotomy is the incision of choice for anterior (precordial) injuries. This method is useful in a trauma setting because the incision can be extended into the abdomen for an exploratory laparotomy or into the neck. CPB may have a role when performing emergency surgery for cardiac injury in a hemodynamically unstable patient, although the outcome is often poor in this setting.[18]

Definitive repair of cardiac injuries is performed by repairing cardiac tissue using nonabsorbable suture. For ventricular injuries, interrupted horizontal mattress sutures are used, often with pledgets. Larger injuries may require patch repair. Atrial injuries are repaired using polypropylene suture with or without pledgets. When repairing cardiac tissue close to a coronary artery, a mattress suture is placed deep to the artery to avoid compromising the artery; the patient should undergo immediate coronary angiography after the procedure to ensure artery patency. Coronary artery injury due to trauma warrants bypass grafting and may be performed without CPB unless ventricular function is compromised with hemodynamic instability.[19] After repair of the heart, the rest of the mediastinum should be examined, including the pleural spaces, posterior pericardium, and internal thoracic arteries. Intraoperative echocardiography is recommended to assess valvular and chamber function.

Cardiac tamponade. Cardiac tamponade is the accumulation of fluid in the pericardial sac either acutely or subacutely. Compression of all cardiac chambers from fluid accumulation reduces chamber diastolic compliance and filling. Venous return normally peaks during ventricular systole and early diastole. As fluid in the pericardium accumulates, venous return is seen primarily during ventricular systole. Eventually, severe tamponade completely inhibits venous return, which is evident because of decreased cardiac output and low systemic blood pressures.[20]

Acute cardiac tamponade can occur from trauma and rupture of the heart or aorta (intrapericardial component) or as a complication of invasive procedures and requires urgent reduction in pericardial pressure. Subacute cardiac tamponade results from the accumulation of fluid over days to weeks and can be associated with neoplastic, uremic, or idiopathic pericarditis. In acute cardiac tamponade, patients may have signs of hypotension and decreased cardiac output (eg, cool extremities, peripheral cyanosis, and decreased urine output) as well as jugular venous distention with possible muffled heart sounds on auscultation. In subacute cardiac tamponade, the fluid accumulates slowly, and the patient may not initially experience any symptoms. Eventually, compression of cardiac chambers can result in symptoms of dyspnea, chest fullness or discomfort, peripheral edema, and fatigue. Sinus tachycardia is seen in almost all patients with cardiac tamponade.

Initial evaluation of a patient with suspected cardiac tamponade includes echocardiography, chest radiograph, and EKG. Urgent echocardiography is the test of choice in a hemodynamically unstable patient with suspected tamponade. EKG may show low-voltage QRS complexes and electrical alternans. Chest radiograph will show an enlarged cardiac silhouette. On echocardiogram, collapse of the right atrium during end-diastole (atrial relaxation) and collapse of the right ventricle during early diastole may be seen. During inspiration, the atrial and ventricular septa may move leftward. Inferior vena cava plethora marked by less than 50% reduction in inferior vena cava diameter with inspiration reflects elevated central venous pressure may be seen in tamponade. Diastolic pressures may equalize in all 4 cardiac chambers. When evaluating a patient for cardiac tamponade, other potential diagnoses include myocardial infarction (particularly with right ventricular involvement), pulmonary embolism, and aortic dissection. Diagnostically, EKG can identify infarction; jugular venous distention is not present in aortic dissection (unless associated with cardiac tamponade); and none of these listed should causes pulsus paradoxus.

Aortic dissection. Aortic dissection often results from an intimal tear, leading to blood flow into the medial layer of the aortic wall. Risk factors for aortic dissection include hypertension, genetic disorder (eg, Marfan syndrome, Ehlers-Danlos syndrome, or Turner syndrome), degenerative aortic disease, inflammatory conditions (eg, Takayasu arteritis or giant cell arteritis), and aortic instrumentation, trauma, or surgery.[21] Stanford type A aortic dissection involves the ascending aorta, whereas Stanford type B dissection does not involve the ascending aorta; the importance of this distinction in the acute setting is that urgent surgery is necessary for type A dissection. The aortic dissection is considered acute if symptoms occurred within 14 days, subacute if between 15 and 90 days, and chronic as more than 90 days.[22] Acute type A dissection is considered a surgical emergency because of potential life-threatening complications, including acute aortic regurgitation, cardiac tamponade, stroke, myocardial infarction, and aortic rupture. Acute type B aortic dissection typically involves the descending thoracic or thoracoabdominal aorta and

occasionally the aortic arch; if uncomplicated, it can be managed with aggressive medical management.

Patients presenting with acute aortic dissection may complain of sharp stabbing or tearing chest pain that radiates to the back. Other notable signs include a new murmur, signs of malperfusion (eg, stroke, myocardial infarction, paraplegia, and mesenteric or peripheral ischemia), Horner syndrome, or hemodynamic instability. Initial evaluation should include EKG (may show signs of acute coronary syndrome), d-dimer, electrolytes, complete blood count, lactate dehydrogenase, cardiac biomarkers, coagulation parameters, and chest radiograph (showing widened mediastinum or pleural effusion). For hemodynamically stable patients in the emergency room, a thoracic CT angiography should be obtained. For patients who cannot undergo CT angiography, transesophageal echocardiography is warranted.

Management of patients with uncomplicated acute type B dissection includes placement of 2 large bore intravenous lines and continuous monitoring of heart rate (target <60 bpm) and blood pressure (target systolic blood pressure <100-120 mm Hg). Esmolol or labetalol drip is used. If the patient cannot tolerate beta blockers, verapamil or diltiazem can be used. If the systolic blood pressure remains high, a nicardipine or nitroprusside infusion can be started once the heart rate is consistently below 60 bpm. Pain should be controlled using intravenous opioids, and a Foley catheter is placed to assess urine output and kidney perfusion. Complicated acute type B dissection includes those with malperfusion and is treated surgically or with endovascular procedures such as aortic stent-grafting and/or fenestration.

Acute type A dissection requires emergency surgery directed at replacement of ascending aorta and open distal anastomosis (as in hemiarch repair). The surgical steps include excision of the intimal tear, interposition graft replacement, and, if necessary, resuspension or replacement of the aortic valve. There is limited evidence of endovascular stent-grafting in the management of ascending aortic dissection; however, novel devices are undergoing clinical investigation. The frozen elephant trunk procedure involves repairing the ascending aorta using an open approach while using a stent graft to manage the descending aorta. Hybrid frozen elephant trunk repair has demonstrated similar or improved outcomes compared to conventional aortic arch repair, with favorable remodeling of the descending thoracic aorta.[23,24]

GENERAL THORACIC EMERGENCIES

Esophageal Perforation

Esophageal perforation may occur from increased intraluminal pressure at sites of anatomic narrowing and sites that are narrowed due to malignancy, foreign body, or physiological dysfunction. Endoscopic instrumentation of the esophagus is a common etiology of iatrogenic perforation.[25] Other causes include Boerhaave syndrome (from forceful emesis), trauma, and intraoperative injury. Presenting signs and symptoms include chest and abdominal pain, fever, subcutaneous emphysema in the neck, and Hamman's sign (crunching sound on chest auscultation). For patients who are clinically stable, flexible esophagoscopy is warranted to assess the location of perforation. Alternatives to endoscopy include water-soluble esophagography swallow study with Gastrografin (generic name, diatrizoate; Bracco Diagnostics, Liebel-Flarsheim Company). For patients who are clinically unstable, flexible esophagoscopy should be performed in the operating room or intensive care unit.

Initial management of esophageal perforation or rupture includes discontinuing oral intake, placing a large bore intravenous line and providing fluid resuscitation, and administering intravenous broad-spectrum antibiotics, such as ampicillin/sulbactam, piperacillin/tazobactam, or a carbapenem, to cover for aerobes and anaerobes. Clindamycin plus a fluoroquinolone or ciprofloxacin is also acceptable, depending on local resistance. In cases of complicated esophageal perforation, antifungal coverage is likely necessary. The patient should also be prepared for surgical intervention, including obtaining a complete blood count, coagulation studies, type and screen, and chest radiograph, which may show widened mediastinum, pneumomediastinum, and/or pleural effusion. Any pleural effusion should be managed with chest tube drainage.

The overall goals of treatment are to clean out any debris or infection and prevent ongoing contamination. Depending on the size and location of the perforation, treatment includes conservative management, endoscopic or endoluminal therapy (eg, stenting or clipping), and primary surgical repair.

Traditionally, primary surgical repair has been employed, which includes debriding the devitalized tissue from the perforation site, incising the muscular layer longitudinally along the muscle fibers to expose the perforation, and closing the mucosa with absorbable interrupted sutures and the muscular layer with interrupted nonabsorbable sutures. A vascularized pedicle flap (commonly, the intercostal muscle flap) may be necessary to enhance the repair. Exceptions to performing a primary repair include cases of diffuse mediastinal necrosis, large perforation that cannot be reapproximated, esophageal malignancy or preexisting end-stage benign esophageal disease (such as achalasia), or if the patient is clinically unstable, in which case an esophagectomy with immediate or delayed reconstruction may be necessary.[26,27]

Postoperatively, the patient should remain taking nothing by mouth for 7 days and will require nutritional support through a jejunal feeding tube or parenteral nutrition via a central line. The patient should remain on intravenous broad-spectrum antibiotics for 7 to 10 days. A contrast esophagogram should be obtained on postoperative day 7 if the patient is clinically stable. Provided that there is no evidence of a leak or postoperative ileus, oral feedings can recommence. Drains should remain in place until the patient can tolerate oral feeding without clinical evidence of leak.

Tension Pneumothorax

A pneumothorax is the accumulation of air in the pleural space between the visceral and parietal pleura. Primary spontaneous pneumothorax occurs in the absence of clinical lung disease, whereas secondary spontaneous pneumothorax occurs as a complication of underlying lung disease such as chronic obstructive pulmonary disease, cystic fibrosis, or malignancy. Patients presenting with primary spontaneous pneumothorax typically have a benign course and can often be managed conservatively. However, there is a risk of recurrence that is managed with pleurodesis via video-assisted thoracic surgery or thoracotomy.[28] Patients with secondary spontaneous pneumothorax often have recurrent pneumothoraces and require early intervention with plans for preventing recurrence.

Patients presenting with pneumothorax may display dyspnea, chest pain, hypoxia, absence of breath sounds to auscultation, hyperresonance to percussion, and in extreme cases, hemodynamic instability. Patients who present with pneumothorax should be assessed for clinical stability, which is defined as having a normal blood pressure, a heart rate between 60 and 120 bpm, a respiratory rate less than 24 breaths per minute, an oxygen saturation above 90% on room air, and the ability to speak in whole sentences.[29] A tension pneumothorax causes hemodynamic instability when the accumulation of air within the hemithorax leads to collapse of lung tissue, possible compression of the heart, and shift of thoracic structures to the contralateral side. There should be a high index of suspicion for tension pneumothorax in patients with worsening dyspnea, hypotension, diminished breath sounds on the affected side, distended neck veins, and tracheal deviation from the affected side.

Unstable patients should undergo tube thoracostomy or needle decompression within the second intercostal space at the midclavicular line if chest tube decompression is delayed. Immediate needle decompression is a lifesaving and temporizing measure to reduce intrapleural pressure and allow for improvement in cardiac output. A thoracostomy tube should be placed after the patient has stabilized. Stable patients may be treated with small-bore catheters ($\leq$14Fr) or chest tubes ($\leq$22Fr). However, for patients with larger air leaks, concomitant empyema or hemothorax, tension pneumothorax, and barotrauma from mechanical ventilation, a large bore chest tube (24Fr-28Fr) is recommended.

Hemothorax

Some injuries that can result in a hemothorax include aortic rupture, myocardial rupture, injuries to hilar structures, and injuries to lung parenchyma and intercostal or mammary blood vessels. To diagnose a hemothorax on chest radiograph, a minimum of 300 mL blood accumulation is typically needed; ultrasound can aid in diagnosis. Hemothorax can be treated with tube thoracostomy using a size 28Fr to 32Fr chest tube. Immediate drainage $\geq$20 mL/kg (approximately 1500 mL) blood, shock, and persistent (>3 mL/kg/hour) bleeding requires surgical thoracotomy.[30]

Flail Chest

A flail chest occurs most commonly because of blunt thoracic trauma. A flail chest results when rib fractures in $\geq$3 ribs in $\geq$2 locations on each rib creates a floating segment that moves paradoxically to the rest of the chest wall. During inspiration, the flail segment is pulled inward and, during expiration, pushed outward (paradoxical movement). Patients with suspected flail chest should also be assessed for sternal fractures and pulmonary contusion given the nature of the injury. The abnormal motion of the flail chest segment compromises normal respiratory function and can cause increased work of breathing and necessitate endotracheal intubation and ventilatory support in some cases.[31] These severe injuries are associated with high rates of morbidity and mortality in elderly patients and warrant prompt management.[32]

Patients with a flail chest should be closely monitored clinically for respiratory compromise and given supplemental oxygen as an initial first step. Further, the respiratory status should be monitored using pulse oximetry as well as continuous waveform capnography. Noninvasive ventilatory support to deliver positive pressure is often necessary, and endotracheal intubation may be required with worsening respiratory status. Additionally, surgical fixation with rib plating has demonstrated benefit in patients with flail chest, decreasing ventilatory and analgesic requirements.[33]

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