Cardiovascular Emergencies

9

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Introduction

Cardiovascular emergencies comprise a major source of morbidity and mortality for the surgical intensive care unit patient. Many of the diagnoses discussed below represent some of the few truly emergent situations in modern medicine in which a delay of literally minutes can hasten an adverse outcome. Moreover, the trend toward surgical intervention on patients who in past years would have been considered too old or ill to undergo surgical intervention dictates that cardiovascular emergencies will remain a challenge for the surgical intensivist. We will discuss several scenarios, including acute myocardial infarction (AMI), pulmonary embolism (PE), cardiac tamponade, tension pneumothorax, aortic dissection, and mechanical complications of myocardial ischemia and infarction. The focus will be on diagnosis and early treatment of these life-threatening conditions.

Acute MI

Diagnosis: *EKG changes + biomarkers*

Therapy: *ASA, beta-blockers, heparin, nitroglycerin, second antiplatelet agent (*e.g., *clopidogrel), revascularization – time matters!*

Following surgery, cardiac complications are a major cause of morbidity and mortality [1]. Each year, it is estimated that at least 500,000 patients experience perioperative cardiac death, nonfatal acute MI (AMI), or nonfatal cardiac arrest [2]. Postoperative MI (PMI) rates have been estimated to be around 1 % for all noncardiac surgery patients and as high as 4–8 % for patients at risk for cardiac disease, with attendant PMI mortality rates in the 15–25 % range [1, 3–6].

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The etiologies for PMI vary and have been debated; traditionally, a major culprit is thought to be an increase in myocardial oxygen demand coupled with stenotic coronary artery disease [7]. This is further supported by the finding that only about a third of postsurgical patients suffering fatal PMI have an intracoronary thrombosis [8, 9]. Other authors have noted that over 50 % of PMI patients have evidence of plaque rupture [10].

Since the complications of PMI can be catastrophic, several risk assessment tools have been developed to stratify patients preoperatively. One such system is the Revised Cardiac Risk Index, which was derived from a population of 2,893 patients undergoing elective major noncardiac surgery and predicts the risk of major cardiac complications (cardiac death, acute MI, pulmonary edema, ventricular fibrillation or cardiac arrest, or complete heart block) [1]. Risk factors identified include performance of a high-risk procedure (vascular or open intraperitoneal/intrathoracic procedures), history of ischemic heart disease, history of heart failure, history of cerebrovascular disease, diabetes mellitus requiring insulin treatment, and a preoperative serum creatinine >2.0 mg/dL. A patient with no risk factors had a 0.4 % chance of a cardiac complication, (1) risk factor was associated with a 1.0 %, (2) risk factors with a 2.4 % risk, and (3) risk factors with a 5.4 % risk. High-risk patients may be referred for further cardiac testing including stress testing, echocardiography, or cardiology consultation.

Postoperatively, the diagnosis of PMI proceeds by the same established criteria as for other MI patients. The phrase "acute coronary syndrome" is used to denote any patient in which there is suspicion of myocardial ischemia and/or infarction. ACS encompasses three clinical entities: unstable angina (UA), ST-elevation MI (STEMI), and non-ST-elevation MI (NSTEMI). For the purposes of surgical patients, we will focus on the latter two categories. Unstable angina is a term used to refer to patients with clinical symptoms suggestive of myocardial ischemia, but who present without a rise in cardiac biomarkers or EKG changes suggestive of ischemia.

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Table 9.1 Diagnostic criteria for acute myocardial infarction

Rise and/or fall of cardiac biomarker (e.g., troponin I), with at least one sample above the 99th percentile upper limit of reference, with one of the following:
Symptoms of ischemia
New significant ST-segment/T-wave changes or new left bundle branch block
Emergence of pathological Q waves on electrocardiography
Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
Identification of an intracoronary thrombus by angiography
Or: Stent thrombosis associated with MI (as detected by coronary angiography), in the setting of myocardial ischemia, and associated with a rise and/or fall of cardiac biomarkers with at least one sample above the 99th percentile upper limit of reference

Adapted from Thygesen et al. [11]

The most recent universal definition of myocardial infarction is shown above (Table 9.1) [11]. In brief, an elevated cardiac biomarker coupled with at least one of the following makes the diagnosis of PMI: symptoms of ischemia, new EKG changes (pathological Q waves, left bundle branch block, ST-segment, or T-wave changes), imaging evidence of new loss of viable myocardium, or a new regional wall motion abnormality on echocardiography. Patients with ST-segment elevation are diagnosed with STEMI, while patients with one or more of the above criteria without ST-segment elevation are diagnosed with NSTEMI. Importantly, many of the classical symptoms associated with MI are absent in postoperative patients, thanks to the use of anesthetic and analgesic medications, and most PMIs tend to occur on the day of surgery or the day after [12]. Accordingly, cardiac enzymes should be trended every 6 h until downtrending in patients with a suspected cardiac complication. Troponin I has been shown to be sensitive (94 %) and specific (75 %) in the detection of major adverse cardiac events in postsurgical patients with at least one RCRI risk factor [13].

Management: STEMI

Following diagnosis, goals of STEMI management in the postsurgical patients involve securing the airway, stabilization of hemodynamics (including optimization of myocardial oxygen demand and afterload), pain relief, prevention of further thrombosis, and prompt revascularization. Many postsurgical patients have the added vulnerability of increased bleeding risk, which complicates decision-making in managing an MI.

After attention is paid to securing an appropriate airway, patients suffering from STEMI should be treated with beta-blockade (i.e., metoprolol) if blood pressure permits, statin therapy, narcotic pain medication, and acetylsalicylic acid and anticoagulant therapy if at all possible. Both aspirin and beta-blockers have been shown to durably reduce mortality following MI [14, 15]. Aspirin helps prevent thrombus propagation, while beta-blockers decrease myocardial oxygen demand. Statins, meanwhile, have a scientific rationale for use in acute MI based on their ability to improve endothelial function and reduce inflammation and thrombus formation [16]. Some authors have found that the use of statins in the early post-MI period is associated with a reduction in ischemic events and mortality [17–20], though subsequent meta-analyses have called these results into question [21, 22]. During this period, echocardiography is also indicated to assess the correlation between electrocardiographic and biochemical data and myocardial function.

As much of initial treatment is aimed at halting intracoronary thrombotic processes prior to reperfusion, postoperative patients require specialized decision-making to balance the competing risks of losing myocardium versus inducing life-threatening bleeding. Aspirin, or clopidogrel for aspirin-intolerant patients, should be administered early following PMI diagnosis if the patient is not actively bleeding. In postoperative patients, anticoagulation with unfractionated heparin is preferred because it is quickly reversible. Thrombolytic agents are traditionally considered contraindicated due to bleeding risk; moreover, early postoperative patients have been historically excluded from major thrombolytic trials. Similarly, glycoprotein IIb/IIIA inhibitors are not typically used in postoperative patients due to their high associated bleeding risk.

Unfortunately, precise data on the risk of surgical bleeding induced by treatment of PMI are scarce. In and of itself, major bleeding has been identified as a risk factor for myocardial infarction, which creates difficulties in investigating this relationship [23–25]. Significant surgical site bleeding associated with PMI treatment appears to be relatively uncommon, however. In one study of 120 patients with postoperative ACS (87 % of whom were subsequently fully heparinized), 9.2 % of treated patients experienced clinically significant bleeds, but of these, only three were related to the surgical site, and five were gastrointestinal bleeds [26]. In another series of 48 patients referred for percutaneous coronary intervention (PCI) after experiencing PMI within 7 days of surgery, nine patients (18.8 %) required red blood cell transfusion, but only one (2.1 %) developed bleeding related to the surgical site.

Though bleeding data in surgical populations suffering PMI is rare, several studies have highlighted the risk of continuing antiplatelet therapy in the early perioperative period in all patients. The POISE-2 trial examined continued aspirin use preoperatively and during the early perioperative period and found that this practice had no effect on the composite rate of death or myocardial infarction, but did slightly increase the risk of major bleeding (4.6 % vs. 3.8 % in non-aspirin-treated

controls, $p=0.04$) [27]. These risks were most significantly increased in aspirin-treated patients on postoperative days 0 through 7, with the risks becoming comparable by postoperative day 8. Similarly, another trial of combined clopidogrel and aspirin treatment given within 5 and 2 days prior to coronary artery bypass grafting (CABG), respectively, found this strategy actually increased the risk of both PMI and postoperative bleeding [28].

Reperfusion itself is the most important and lifesaving aspect of MI therapy [29]. As fibrinolytic therapy is typically too risky for the PMI patient given bleeding risks, the first step in this process is percutaneous coronary intervention (PCI), which should be considered in consultation with cardiology for all PMI patients. Though specific data on PMI patients are scarce, data in emergency department populations suggest that each 30-min delay from symptom onset to PCI increases the relative risk of 1-year mortality by 8 % [30]. At the same time, PCI virtually mandates the use of dual antiplatelet therapy, as aspirin and clopidogrel substantially reduce the risks of stent thrombosis within 30 days as well as death, MI, or repeat revascularization within a year [31, 32].

Finally, though primary surgical revascularization is not usually performed due to logistical constraints, STEMI patients may be referred for coronary artery bypass grafting for several indications. These include persistent or recurrent ischemia following PCI, high-risk anatomy such as left main or triple vessel disease, or a mechanical complication of AMI (discussed below). Additionally, patients who can be stabilized and revascularized percutaneously following their MI but who still have significant stenoses may be referred for CABG as well.

Management: NSTEMI

Treatment principles of NSTEMI largely parallel those for STEMI, including early optimization of myocardial oxygen demand, administration of antiplatelet and anticoagulant medications, and cardiology consultation to pursue possible revascularization. However, as opposed to STEMI, both conservative and invasive strategies have been proposed and debated for the management of NSTEMI [33]. The former calls for medical therapy, consisting of aspirin, clopidogrel, and heparin, with angiography only if the patient exhibits evidence of recurrent ischemia. Stabilized patients may undergo noninvasive stress testing (e.g., treadmill, echocardiography, or nuclear) at a later point to assess the need for angiography. Several recent studies suggest that low-risk female patients may benefit from an initially conservative strategy [34–36].

In contrast, the invasive strategy calls for routine angiography early after the diagnosis of PMI. This approach is favored for patients with recurrent angina or ischemia, elevated biomarkers, worsening heart failure, hemodynamic instability, arrhythmia, or other high-risk features [33]. One study of NSTEMI patients found early pretreatment with aspirin, heparin, clopidogrel, and tirofiban plus angiography within 6 h of diagnosis was associated with improved survival as compared to a strategy of pretreatment for 3–5 days prior to MI [37].

Acute Pulmonary Embolism

Diagnosis: *Clinical suspicion, ABG, bedside echocardiography (RV strain), and/or CT-PA*

Therapy: *Heparin (consider empiric treatment for renal insufficiency or clinical urgency); if in extremis, consider thrombolysis or thrombectomy.*

One of the most common complications experienced in surgery is deep venous thrombosis (DVT) and its feared counterpart, pulmonary embolism (PE). Without any form of prophylaxis, DVTs can occur in 10–40 % of medical and general surgical patients and up to 40–60 % of trauma and orthopedic surgery patients [38]. A recent large series of trauma and orthopedic surgery patients found that though only 0.47 % of patients developed PE as diagnosed by computed tomography pulmonary angiogram (CT-PA) scanning, their attendant mortality rate was 15.3 % [39]. One autopsy series of over a thousand patients who died following surgical procedures found that 32 % of these patients suffered a PE; in 29 % of the entire series, PE was determined to be the cause of death [40].

The clinical presentation of PE can vary widely and is suggested by symptoms including dyspnea, pleuritic pain, and cough, particularly in the presence of DVT symptoms (e.g., calf pain, unilateral extremity edema). Hypoxia and hypotension can also be presenting signs of PE, though it is relatively unusual for patients to present in frank shock. Additionally, many surgical patients already have one or more risk factors for pulmonary embolism, such as advanced age, cardiac or respiratory failure, prolonged immobility, the use of central venous lines, and prior DVT [41, 42]. A number of risk scoring systems have been devised to organize such risk factors into a pretest probability. One such clinical decision rule is the Canadian Pulmonary Embolism Score, also known as Wells' Criteria (Table 9.2). As originally studied, patients with a low clinical probability of PE based on a low Wells' score (0–1) and a negative D-dimer test had no further testing, and the diagnosis of PE was considered excluded. All other patients underwent ventilation-perfusion scanning in the original study, with bilateral deep venous ultrasonography performed if the scan was nondiagnostic. This algorithm has been shown to have a negative predictive value of 99.5 % in an emergency department patient population [43, 44].

Table 9.2 Wells' criteria (Canadian Pulmonary Embolism Score)

Risk factor	*Points assigned*
Clinical signs and symptoms of DVT	3
PE is #1 diagnosis or equally likely as another	3
Heart rate >100	1.5
Immobilization for ≥3 days or surgery within the prior 4 weeks	1.5
Previous objectively diagnosed PE or DVT	1.5
Hemoptysis	1
Malignancy within last 6 months	1
Clinical probability of pulmonary embolism	*Score total*
Low (1.3 % chance of PE)	0–1
Intermediate (16.2 % chance of PE)	2–6
High (40.6 % chance of PE)	>6

Adapted from Wells et al. [43]

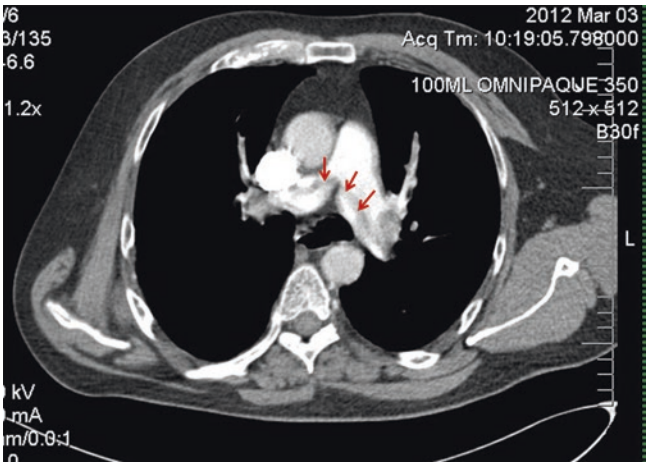

Fig. 9.1 Saddle pulmonary embolus (*arrows*) on computed tomography scanning with intravenous contrast. (Figure courtesy of T. Metkus, M.D.)

For patients who may be safely imaged, the definitive diagnostic test in the modern era is computed tomographic pulmonary angiography (CT-PA) or, less commonly, ventilation-perfusion scanning. CT-PA has been shown to be extremely sensitive and specific with regard to the diagnosis of PE, particularly populations at moderate or high risk of PE (Fig. 9.1). One large series found that CT-PA coupled with venous phase imaging had a sensitivity of 90 % and a specificity of 95 % for the diagnosis of PE, though this population was not exclusively postsurgical [45].

In patients with multiple risk factors who experience a sudden, unexplained change in hemodynamic status – for example, the critically ill bed-bound patient – prompt institution of therapy prior to definitive diagnostic testing may be lifesaving. Empiric anticoagulation and/or thrombolytic therapy is indicated for patients with a high likelihood of having PE in whom definitive testing is dangerous. Ancillary studies such as bedside echocardiography showing right ventricular strain may be helpful in these scenarios.

The standard treatment of PE, after providing respiratory and hemodynamic support as appropriate, is anticoagulation with low molecular weight heparin (LMWH) or unfractionated heparin (UFH). In surgical patients who are typically deemed at increased risk of bleeding, UFH is usually chosen because it is the shortest-acting agent and can be reversed with protamine sulfate. Additionally, since renal insufficiency can affect the pharmacokinetics of anticoagulation therapy, UFH is preferred in patients with underlying renal disease due to its ease of monitoring. A typical UFH protocol is weight based, with a bolus dose of 80 units/kg given followed by an infusion at 15–20 units/h. The activated partial thromboplastin time (aPTT) is monitored at the beginning of therapy and every 4–6 h thereafter to target a goal range of aPTTs. In our institution, this range is typically 65–80 s for patients not deemed at excessive bleeding risk; the 50–65 s range represents a second choice available to clinicians. Of note, prompt institution of therapy is essential: the risk of recurrent PE may be as high as 25 % when the aPTT is not therapeutic within the first 24 h after heparinization [46].

For patients presenting with massive PE as indicated by persistent hypotension (usually defined as a systolic blood pressure less than 90 mmHg or a decrease of greater than 40 mmHg in systolic pressure from baseline), often with right ventricular dysfunction, thrombolytic therapy may be indicated [47]. Thrombolytic alteplase (Genentech, San Francisco, CA) at a dose of 100 mg infused over 2 h has FDA approval for the treatment of massive PE [47]. Though a mortality benefit to the administration of thrombolytic therapy was seen in a recent meta-analysis (OR 0.53), this came at the cost of a dramatically increased incidence of major bleeding events (OR 2.73) [48]. The same study noted the incidence of major bleeding events to be 9.2 % in patients receiving thrombolysis versus 3.4 % in patients treated with anticoagulation therapy alone, and a 1.5 % risk of intracranial hemorrhage as opposed to 0.2 % in the anticoagulation-only group. Moreover, recent surgery is often considered an absolute contraindication to thrombolytic therapy. A promising option for these patients is catheter-directed thrombolysis, which may offer some of the advantages of thrombolytic therapy without the same systemic exposure. Catheter-directed thrombolysis (CDT) is typically performed using low-profile (<10 French) catheters and may involve mechanical fragmentation or aspiration of emboli, as well as intraclot thrombolytic injection. CDT is reported to have a clinical success rate of 86.5 % (defined as stabilization of hemodynamics, resolution of hypoxia, and overall survival from PE), with a major complication rate of 2.4 % [49], though it has not been well studied in surgical populations.

Finally, pulmonary embolectomy is usually reserved for patients with massive PE and right ventricular strain on echocardiography, with or sometimes without impending hemodynamic collapse [50, 51]. Though the mortality rate

for this procedure has declined over the last few decades, it remains near 20 % [52]. One recent series of 20 patients operated on emergently reported a survival-to-discharge rate of 95 % [53], while another reported that 94 % of emergent patients survived to hospital discharge, with 83 % alive at 3 years [51].

Tamponade

Diagnosis: *Clinical suspicion (Beck's triad, equalization of right & left heart pressures), echocardiography*

Therapy: *Volume, administration, drainage*

Acute cardiac tamponade occurs when fluid under pressure accumulates inside of the pericardial sac. The elasticity of the pericardium is limited to accommodating the physiologic amounts of fluid which normally surround the heart. As excess fluid accumulates, the pericardium stiffens (i.e., compliance decreases) and compression of the heart itself occurs, which impairs cardiac filling. Worsening tamponade is associated with progressively declining preload and a corresponding drop in cardiac output and blood pressure. "Beck's triad" refers to the distended neck veins, muffled heart sounds, and low arterial blood pressure which can be seen in cases of acute tamponade. Additionally, patients with a pulmonary artery catheter in place may exhibit equalization of pressures between right and left sides of the heart.

Tamponade can be seen in a wide range of clinical situations. Etiologies can be subdivided into pericardial effusions, which tend to be medical in nature, and hemorrhage into the pericardium, more often seen in surgical populations. Within this subset, hemorrhage into the pericardium has three major causes: trauma to the myocardium itself, either blunt or penetrating, free ventricular wall rupture following myocardial infarction, or hemorrhage as a result of an aortic dissection.

Tamponade following trauma is a grave event, but it will lead to earlier arrest and better preserved blood volume than injuries that result in hemorrhage and arrest from hypovolemia. The overall survival rate for penetrating cardiac trauma is generally poor, with typical survival figures reported as 10.8 % [54], 14 % [55], or 19.3 % [56] in some series; gunshot wound patients fare less well than those with isolated stab wounds, especially those limited to the right ventricle. One series of 212 patients with penetrating cardiac trauma found that only 96 were even transported to the trauma center (45.3 %). Of those 96, 48 presented with tamponade (22.6 %), and of those, 27 survived (12.7 %) [56]. Though some series have not found that tamponade at presentation is predictive of survival or mortality [57], other series have suggested improved survival among patients presenting with tamponade alone as opposed to those in frank hypovolemic shock [54], highlighting the urgency of rapid intervention. Data indicate that tamponade following blunt trauma is equally serious. Victims of blunt cardiac rupture are unlikely to survival to the hospital, and overall mortality even within those initial survivors lies in the 60–90 % range [58, 59].

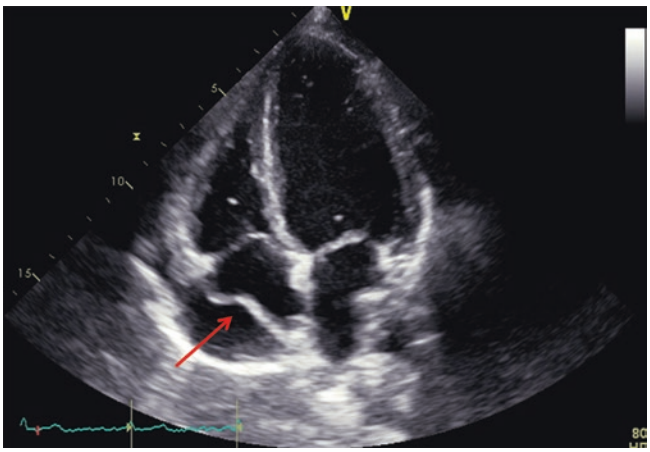

Fig. 9.2 Pericardial effusion with right atrial collapse (*arrow*). (Figure courtesy of T. Metkus, M.D.)

Management of traumatic cardiac tamponade patients follows Advanced Trauma Life Support (ATLS) protocols. In stable patients, hemopericardium may be diagnosed with Focused Assessment with Sonography for Trauma (FAST) ultrasound scanning. Aggressive volume resuscitation is critical to maintaining intracardiac filling pressures but needs to be coupled with rapid definitive hemorrhage control (Fig. 9.2).

In contrast to medical cases of cardiac tamponade arising from pericardial effusions, pericardiocentesis may not always be appropriate for surgical patients because it fails to address the underlying traumatic defect in the myocardium. Pericardiocentesis may be useful as a bridge to definitive surgical therapy, however, and is still taught as part of the ATLS curriculum. A recent review article noted that most studies of pericardiocentesis are biased toward survivors and that the procedure is used as a sole intervention in trauma patients in only 2.1 % of patients [60]. Hemodynamically stable patients presenting with hemopericardium after penetrating chest trauma may be candidates for a subxiphoid pericardial window performed in the operating room; evidence suggests this approach may shorten ICU and hospital stays without any decrement in survival [61]. It is important to note that inducing anesthesia in a patient with significant hemopericardium may worsen hemodynamic compromise. Unstable trauma patients may be taken emergently to the operating room or may undergo emergency department resuscitative thoracotomy should they meet ATLS criteria.

Tamponade can also occur secondary to two primary cardiac events, namely, acute myocardial infarction (MI) or acute aortic dissection. Following MI, weakened myocardium

can rupture, allowing the free passage of blood into the pericardial space. Free wall or left ventricular aneurysm rupture requires emergent operative repair and will be discussed further below. Cardiac tamponade can also complicate acute aortic dissection, occurring in 8.4 % [62] to 18.7 % [63] of all dissection patients in recent series. Tamponade typically complicates an ascending or type A dissection when rupture of the aorta into the pericardium near the aortic root results in hemopericardium under essentially arterial pressures. The presence of cardiac tamponade in acute aortic dissection is independently associated with a higher mortality risk [64, 65], with one series of 674 type A dissection patients reporting mortality of 24.6 % overall and 54.0 % in patients presenting with tamponade [63]. Tamponade as a result of MI or aortic dissection generally requires emergent operative intervention, discussed below. Pericardiocentesis has been suggested to be harmful in cases of acute aortic dissection [66], as it fails to address the underlying disease process.

Finally, no textbook of surgical intensive care would be complete without noting that cardiac tamponade should always be suspected in cardiothoracic surgery patients with declining arterial blood pressure and rising CVP, even in the presence of apparently functioning mediastinal drainage tubes. The intensivist must always be attuned to this possibility, particularly if chest tube output has dropped suddenly. The inadequate placement or failure of these tubes due to clot can lead to inadequate drainage of ongoing bleeding and hemodynamic compromise. Though some advocate "stripping" or "milking" chest tubes to prevent this, a Cochrane Library meta-analysis of chest tube clearance methods found insufficient evidence to support or refute the need for such maneuvers [67]. These patients may require reopening of the chest in the ICU and/or reexploration in the operating room.

Tension Pneumothorax

Diagnosis: *Clinical suspicion (tracheal deviation, decreased breath sounds, jugular venous distension, hypotension), radiography*

Therapy: *Acute decompression*

A tension pneumothorax occurs when air accumulates in the pleural space under pressure. This occurs as a result of a pneumothorax coupled with an impediment to air extravasation from the pleural space – the so-called "one-way-valve" effect. In this manner, air can enter the pleural space, but cannot leave. As air accumulates under pressure exceeding atmospheric pressure, the heart and great vessels are compressed, leading to a decrease in cardiac preload and a drop in cardiac output. Typically, a tension pneumothorax results from a lung laceration (e.g., from a fractured rib or stab wound), though it is theoretically possible to have tension physiology with a chest wall laceration alone as well. Positive-pressure ventilation can create (e.g., the rupture of a lung bleb) or exacerbate situations leading to tension physiology.

Clinically, the classical signs of a tension pneumothorax are decreased breath sounds on the affected side, shift of the trachea away from the affected side (where the tension is building), mediastinal shift away from the affected side, and depression of the affected side's hemidiaphragm. Tension pneumothorax is one of the most common causes of death in battlefield combat injuries and is one of the most common civilian traumatic injuries as well, with a reported incidence of 20 % in patients admitted to trauma centers [68, 69]. Equally important for the intensivist is the fact that tension pneumothoraces may occur insidiously in the intensive care unit patient. The prevalence of positive pressure ventilation as well as invasive procedures such as central line placement can all be complicated by pneumothorax. The classic example is a mechanically ventilated patient who undergoes subclavian central line placement, develops an iatrogenic pneumothorax, and then develops tension physiology due to ongoing positive pressure ventilation coupled with a parenchymal lung injury.

A chest radiograph may be obtained for definitive diagnosis of a pneumothorax; the clinical signs and symptoms mentioned above are useful for determining if tension physiology is occurring. More recently, the increased use of computed tomography (CT) scans in trauma patients has revealed a high incidence of "occult" pneumothoraces which are not appreciated on chest radiology alone. In one series of 230 trauma center patients who were discharged with a diagnosis of pneumothorax, over half (54.8 %) had pneumothoraces missed by presentation clinical examination and chest radiography which were only appreciated following CT imaging [70]; such pneumothoraces are termed occult pneumothoraces.

The treatment for a tension pneumothorax is aimed at relieving the built-up intrathoracic pressure which impairs cardiac preload and therefore cardiac output. Traditionally, tension pneumothorax has been treated by tube thoracostomy, typically performed in the fourth or fifth rib interspaces on the anterior axillary line of the affected side. The tube is directed apically. This allows the escape of pressurized air from the pleural space and insertion of a suitable tube to provide negative pressure suction and therefore reexpand the collapsed lung. For the occult pneumothorax patient – for example, an intensive care unit patient undergoing imaging for another indication – it has been recommended that all patients requiring positive pressure ventilation undergo tube thoracostomy patient to preclude the development of tension physiology. One small randomized trial found that in occult pneumothorax patients requiring positive pressure ventilation, 8 of 21 observed patients progressed to require tube thoracostomy, with three of these developing tension physiology [71]. Another randomized

trial found that 20 % of observed occult pneumothorax patients progressed to require tube thoracostomy, though those who underwent initial tube thoracostomy did not have a survival differential versus those who were observed [72].

Aortic Dissection

Diagnosis: *Clinical suspicion (part of any chest pain differential), asymmetric pulse exam, CT aortography or transesophageal echocardiography. *MUST distinguish type A from type B.**

Therapy: *Negative ionotropy followed by afterload reduction; if type A, immediate surgery. If type B, medical management unless malperfusion, unremitting chest pain, hemorrhagic (left) pleural effusion, continued fall in hemoglobin, uncontrollable hypertension, and rising creatinine (normotensive acute kidney injury).*

Aortic dissection occurs as a result of a tear in the aortic intima, either primary in nature or as a result of an underlying medial hemorrhage. Disruption of the intima allows blood under arterial pressure in the aortic lumen to force its way through the media and thereby separate the intima from the media and/or adventitia, creating a dissection flap and a "false lumen." As blood continues to separate the arterial wall layers, the dissection can spread. Proximally, this may affect the aortic valve and extend into the pericardial interior, resulting in hemopericardium and potentially cardiac tamponade. Distally, dissections of the aorta can involve any of the great vessels to the upper circulation, as well as the visceral vessels. The subsequent potential compromise of blood flow to end organs and resulting ischemia is referred to as malperfusion. Dissections are classified according to their involvement of either the ascending aorta (Stanford type A) or the descending aorta (i.e., distal to the left subclavian artery: Stanford type B) [73]. Alternatively, Debakey's classification describes three types: Type 1, dissections starting in the ascending aorta and extending at least into the aortic arch; type 2, dissections limited to the ascending aorta alone; and type 3, dissections starting in the descending aorta and extending proximally or distally [74].

Aortic dissection is a relatively rare disease, with an incidence of about 3 per 100,000 persons per year, about two thirds of them male [62, 75, 76]. Patients typically are older males, though younger patients are more likely to have a connective tissue disorder (e.g., Marfan syndrome or Loeys-Dietz syndrome), have a bicuspid aortic valve, or have a history of prior cardiac surgery [77]. Cystic medial necrosis, a disorder of large arteries characterized by loss of elastic and muscle fibers in the media, is often present in connective tissue disorder patients presenting with aortic dissection. Presenting individuals may report substernal chest pain or "tearing" or sharp pain in the posterior chest, sometimes radiating to the back. Some patients may experience syncopal episodes as part of their presentation, and a history of hypertension may be present in 72 % of patients [62]. Symptoms of aortic insufficiency may be present if the dissection has propagated proximally to involve the aortic valve or root. If the aortic arch vessels are involved, patients may present with a pulse or blood pressure variation between the right and left arms. Other clues to diagnosis include recent procedural history: in a recent study of 464 aortic dissection patients, 17.9 % were noted to have had prior cardiac surgery, and 2.2 % experienced their aortic dissection secondary to a cardiac catheterization procedure [62].

CT aortography remains the predominant means of diagnosing of aortic dissection, as it is rapid and readily available. CT images can be helpful in assessing not only the anatomy and extent of dissection but also sequelae including intraluminal thrombus and hemopericardium. Sensitivity and specificity of CT are both excellent and have been reported in the range of 98 % and 100 %, respectively [78, 79]. Though slower, MRI is also considered to be highly accurate in the diagnosis of aortic dissection and is better than CT at identifying the dissection's point of origination [78]. Transesophageal echocardiography requires esophageal intubation and the hemodynamic risks of risk of procedure sedation. TEE is quite sensitive but somewhat less specific than CT or MRI (in the range of 77–85 %) [78, 80]. However, advantages to TEE include that it can be performed at the bedside without moving an unstable patient, and it allows the added benefit of assessing any component of aortic regurgitation which may be present in an ascending dissection.

The management of an aortic dissection depends on its anatomic location. Ascending or type A dissections (Debakey classes 1 and 2) are true surgical emergencies and should involve prompt cardiothoracic surgical consultation for operative repair. In contrast, descending or type B dissections (Debakey class 3) are managed nonoperatively unless the patient has evidence of ongoing malperfusion or hemorrhage. Acutely, prior to the consideration of operative intervention, all patients should be admitted to a monitored setting and undergo proper airway management, including intubation in unstable patients and adequate opioid analgesia as needed. Both blood pressure and heart rate must then be controlled in a systematic fashion. In order to minimize the force of left ventricular ejection (i.e., the change in pressure over change in time or "dP/dT"), a beta-blocker such as esmolol or labetalol should be given to lower the blood pressure to a systolic goal of 100–120 mmHg with a heart rate of around 60 [77]. Calcium channel blockers such as diltiazem and verapamil are an acceptable alternative in the rare patient who cannot tolerate beta-blockers. For additional blood pressure control, vasodilating agents are then added, such as sodium nitroprusside.

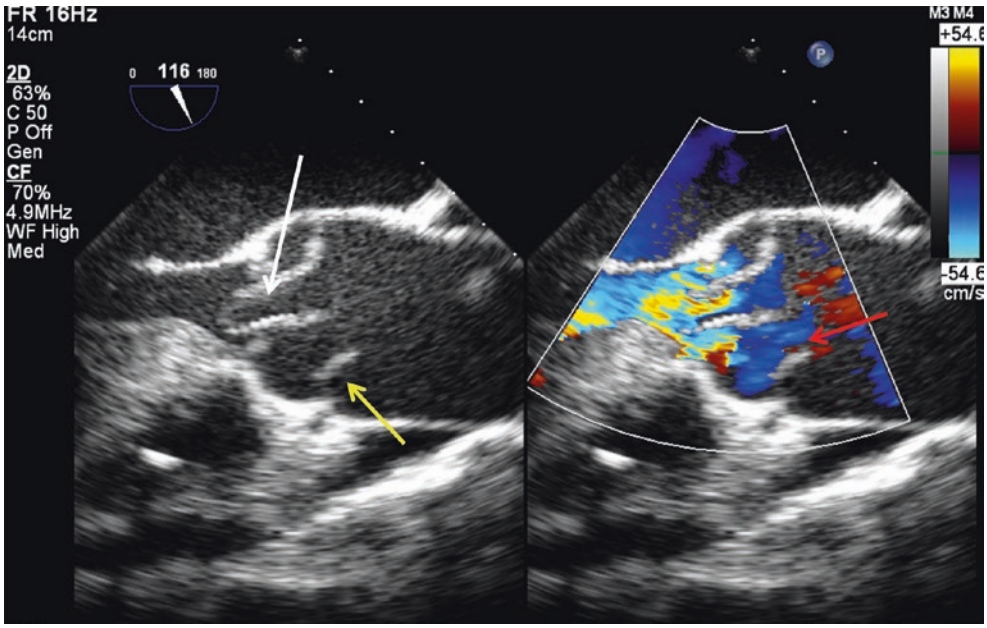

Fig. 9.3 Aortic dissection flap with aortic insufficiency. *White arrow* shows aortic valve leaflets. *Yellow arrow* shows dissection flap. *Red arrow* shows aortic insufficiency arising from flap. (Image courtesy of T. Metkus, M.D.)

Patients who present *in extremis* are exceptionally challenging to manage. These patients may already be in hypovolemic shock from blood loss or extracardiac obstructive shock from cardiac tamponade if the ascending dissection has resulted in hemopericardium. These patients will require emergent intubation and volume resuscitation with blood products. Pericardiocentesis is to be avoided in patients with signs of tamponade, as the patient's increased intrapericardial pressure may be the only factor preventing further bleeding and sudden hemodynamic collapse [66].

Operatively, goals of surgery as originally articulated by Debakey et al., and later by Bahnson and colleagues, involve excision of the intimal tear, removal or obliteration of the point of entry into the false lumen, and aortic reconstruction with a synthetic graft [74, 81, 82]. Cardiopulmonary bypass is used, as well as hypothermic circulatory arrest if circulation to the head vessels must be compromised during repair of the aortic arch. Additionally, if the aortic dissection involves the aortic valve and aortic insufficiency is present, valve replacement is required (Fig. 9.3).

In the series of Hagan et al., 72 % of type A dissections were managed surgically (with some patients not offered surgery due to advanced age or other comorbidities), while only 20 % of type B aneurysms were operated upon [62]. Surgically treated acute type A dissection patients experienced a 26 % in-hospital mortality rate (versus 58 % of type A patients treated medically), while medically treated type B patients had a 10.7 % mortality rate. However, mortality was highest in type B patients who required operation, at 31.4 %. Overall operative mortality for the repair of ascending aortic dissections remains in the 15–35 % range at experienced centers [62, 83, 84].

In recent years, endovascular repair of aortic dissection has been attempted successfully, with or without fenestration of the stent graft. These techniques have been most widely employed for complicated type B dissections (i.e., dissections with the presence of malperfusion or evidence of impending rupture), with some investigators reporting lower rates of paraplegia and mortality as compared to open surgical repair [85]. The VIRTUE trial of endovascular stent grafting for aortic dissection reported 3-year survival of 82 % among patients with an acute type B dissection requiring intervention [86]. Another group reported an 84 % survival rate over a median of 53 months of follow-up [87]. With these results, many surgeons now believe that the endovascular approach is the preferred means of treating complicated type B aortic dissections [88, 89].

Traumatic Aortic Injury

Diagnosis: *Mechanism of injury, CT aortography*

Treatment: *Endovascular or open repair*

No discussion of aortic dissection is complete without mention of the devastating consequences of traumatic aortic injury. Though patients with penetrating aortic injuries typically rapidly suffer exsanguination and death, blunt aortic injury (BAI) may be seen in trauma patients who survive to hospital presentation [90]. Shear forces sustained during rapid deceleration events (e.g., high-speed motor vehicle collisions, airplane crashes, falls from height) are typically implicated in BAI; for example, 73 % of one major commercial airline crash's victims suffered aortic injuries [91]. The most common sites of injury within the aorta are the isthmus,

ascending aortic and arch, and the distal thoracic aorta [92, 93]. As with aortic dissection, diagnosis is most commonly established by CT, and the same initial medical management principles apply, including the use of beta-blockade and aggressive antihypertensive infusions. However, management principles of BAI differ in that other life-threatening injuries are usually stabilized prior to surgical repair of the aortic injury. In a large prospective trial, delayed repair (>24 h after injury) of BAI was associated with improved survival regardless of the presence or absence of major associated injuries [94]. Stent grafts have been employed frequently in the traumatic setting as well and have been associated with relatively favorable outcomes [95, 96].

Mechanical Complications of MI: Ventricular Septal Defect and Free Wall Rupture

Diagnosis: *Physical exam (harsh systolic murmur), echocardiography*

Therapy: *Decrease afterload (consider IABP); hypoxia or shock mandate emergent surgery*

Ventricular septal defects (VSD) have been reported to complicate about 0.2 % of acute MI cases in the modern era and are associated with 30-day mortality rates in the 75 % range [97, 98]. Typically, these occur when an infarct is of sufficient size to result in a large transmural infarction in the septal myocardium. Ruptures may be "simple," in which a straight path is created between the right and left ventricles, or "complex," in which the path of rupture and dissection of blood travels serpiginously through the septum and may result in defects far apart from each other in each respective ventricle. One autopsy series found that simple VSDs tend to be associated with anterior infarcts, while complex VSDs are associated with inferior infarcts [99]. Subsequent left-to-right shunting of blood may impose a marked hemodynamic strain on a struggling heart, depending on the severity of the infarct and resultant VSD.

Traditionally, the classic time period for VSDs and/or free wall rupture to occur is around 5–6 days following acute MI, roughly the time taken for infarcted myocardium to weaken sufficiently [100]. Interestingly, in the modern era of aggressive intervention and revascularization, the median time to VSD occurrence is less than 24 h [101].

Clinical clues to the diagnosis of VSD include increased chest pain, new ST elevations, a pansystolic murmur, or frank cardiogenic shock. VSD can be a sudden event, and acute changes in an AMI patient's condition may alert the clinical to the possibility of VSD. Echocardiography may show the frank septal rupture, left-to-right flow on color Doppler modes, or right ventricular dysfunction in the case of hemodynamically significant VSDs (Fig. 9.4).

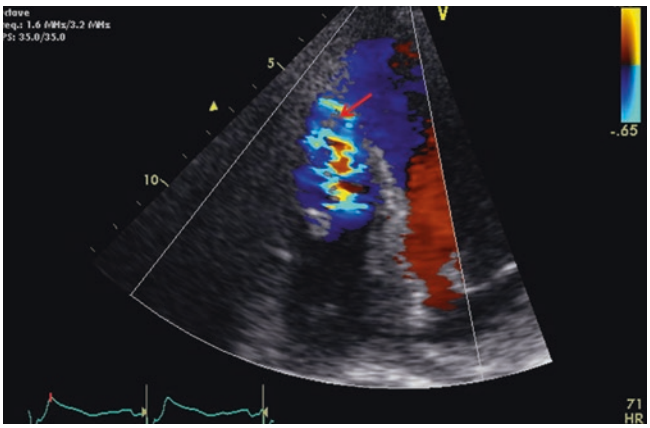

Fig. 9.4 VSD with left-to-right shunting on color Doppler (*red arrow*). (Image courtesy of T. Metkus, M.D.)

Management of the patient with VSD represents a marked challenge, as the few medical therapies available to the clinician are usually already in place at the time of diagnosis. As with the therapy of AMI in general, goals include optimization of coronary and end-organ perfusion, minimization of myocardial oxygen demand, and the reduction of SVR to minimize left-to-right shunting through the VSD. Operative repair remains a mainstay of therapy. Historically, repairs were delayed for as long as 1 month out of concern for the VSD patient's poor hemodynamic condition, as well as the inability of necrotic myocardium to hold sutures. However, since the majority of VSD patients are in cardiogenic shock, survival rates with medical management alone were extremely poor – overall survival in one recent registry found 19 % survival in an operative management group, but only 4 % survival in patients treated medically [101]. Accordingly, with the exception of hemodynamically stable VSD patients whose defects are sufficiently small to allow operative delay, most patients are considered for emergent operations.

Operative repair takes place on cardiopulmonary bypass. A left ventriculotomy is usually performed to gain access to the septum [102]. The surgeon must find myocardium of sufficient strength to hold sutures which will anchor a pericardial patch; this may require not only debridement of necrotic tissue around the defect but also enlargement of the defect itself. The patch must be of sufficient size to minimize tension and preclude the recurrence of a defect. A more recent method of repair, infarct exclusion, involves suturing the pericardial patch to healthy myocardium far from the defect in order to entirely exclude the defect and surrounding tissue from the left ventricular cavity [103]. For example, an anterior VSD would be excluded by suturing the patch to the septum and lateral wall. This method has the advantage of not only closing the defect but also preventing further resection of potentially viable myocardium and preserving left ventricular geometry.

Mechanical Complications of MI: Left Ventricular Aneurysm

Diagnosis: *Echocardiography*

Therapy: *Anti-remodeling therapy, anticoagulation if thrombus present; aneurysmectomy for systemic embolization or refractory symptoms; emergent surgery for rupture*

Left ventricular aneurysms (LVA) result from post-MI healing and scarring and are usually defined as well-delineated, thin segments of the ventricular wall which contain no viable muscle. These aneurysms typically balloon outward paradoxically during systole and are hence termed dyskinetic (or sometimes akinetic). As with VSDs, the incidence of LVA has declined in the modern era of early reperfusion; current figures suggest around 10 % of all AMI patients will develop an LVA. Interestingly, one study found only 7.2 % of patients who underwent revascularization developed LVAs, as opposed to 18.8 % who could not have their infarct-related artery reopened [104]. About three quarters of LVAs occur in the anterior or apical LV walls. Infarct expansion occurs rapidly after AMI via neutrophil-mediated proteolysis [105, 106]. Like VSDs, these lesions are prone to rupture in the early post-MI time period. As ventricular remodeling occurs and scar tissue replaces the infarcted myocardium, the LVAs remain unable to contract and expand appropriately with systole and diastole. These changes, coupled with the compensatory hypertrophy and ventricular dilation which occurs following MI, may further increase myocardial oxygen demand and lead to heart failure.

In addition to worsening heart failure, LVA patients may also present with angina or arrhythmias related to the scar tissue. Mural thrombus has been reported to be present in up to half of patients who undergo surgical correction and seems to be associated with increasing aneurysm size in older reports [107–109]; accordingly, some patients may suffer systemic embolization resulting in cerebrovascular accidents or peripheral arterial occlusion.

Medical therapy for LVA consists of treatment to ameliorate LV remodeling, typically with beta-blockers and angiotensin-converting enzyme inhibitors, and anticoagulation were required for the presence of intraventricular thrombus. Indications for aneurysmectomy include persistent arrhythmias or heart failure refractory to medical therapy, refractory angina, and systemic embolization in patients with contraindications to oral anticoagulation. Typically, revascularization, when indicated, is performed concomitant with aneurysmectomy, since this approach appears to improve survival [110]. Additionally, patients presenting with LVA and/or free wall rupture require emergent surgery.

Mechanical Complications of MI: Papillary Muscle Rupture and Acute Mitral Regurgitation

Diagnosis: *Physical exam (harsh systolic murmur), echocardiography*

Therapy: *Decrease afterload (consider IABP); hypoxia or shock mandate emergent surgery*

Just as infarcted myocardium weakens, resulting in VSD or LVA, so too can the papillary muscles suffer damage during AMI. As these structures control the mitral valve, acute mitral regurgitation can result. The valve leaflets and chordae tendineae are not directly affected by ischemia. However, the posteromedial papillary muscle is usually only supplied by a single artery – the right coronary artery or the circumflex artery – and is therefore at highest risk of an ischemic insult. Meanwhile, the first circumflex marginal and first diagonal arteries both supply the anterolateral papillary muscle, giving it a degree of protection during AMI as compared to its counterpart [111].

Acute mitral regurgitation occurs via two mechanisms. In the first, papillary muscle rupture as a result of infarction and subsequent weakening causes flail mitral valve leaflets. Though an infarction causing total rupture of the papillary muscle common trunk may precipitate prompt hemodynamic collapse, a partial rupture of the trunk or only one head of the muscle may be less severe [112]. Acute mitral regurgitation may also result from poor coordination of the mitral apparatus. Not only may papillary muscle shortening be impaired by infarction but also dysfunction of the LV wall can impede proper valve leaflet coaptation. For example, if the ventricular wall adjacent to a leaflet infarcts, it will dilate and can cause a central leak as the ipsilateral leaflet is pulled slightly away from its proper position.

In the SHOCK trial, moderate or greater mitral regurgitation following myocardial infarction was present in about 40 % of AMI patients who underwent echocardiography, and these patients had poorer survival than AMI patients with mild or no mitral regurgitation [113]. Another study of AMI patients found that about 10 % of AMI patients presenting in cardiogenic shock had clinically significant acute MR [114]. The incidence of papillary muscle rupture is harder to pinpoint, but is thought to account for up to 5 % of all AMI deaths and is usually fatal should a complete rupture occur [115].

Medical management of moderate or severe acute MR follows the same principles as cardiogenic shock following AMI. IABP placement in this setting has been shown, in a calf model, to increase cardiac output while decreasing the degree of MR [114]. Surgical therapy is the only viable corrective therapy for papillary muscle rupture; it carries

high-operative mortality rates (around 20–30 %), but lower mortality rates as compared to medically managed patients [112, 116, 117]. Valve replacement (as opposed to repair) is required in the presence of papillary muscle necrosis. Though survival may be similar between matched patients undergoing repair versus replacement, patients undergoing repair for severe MR following AMI have higher reoperative rates due to mitral valve failure [118, 119]. Notably, in one large series, no survival differences were seen between repair and replacement among high-risk patients [118].

Future Horizons: The Emerging Role of Extracorporeal Life Support in Cardiovascular Emergencies

Though cardiopulmonary bypass is hardly new, the ever-expanding use of extracorporeal life support technologies like extracorporeal membrane oxygenation (ECMO) to maintain patients whose own pulmonary and/or circulatory systems are failing represents a new frontier in medicine. Currently accepted indications for ECMO include potentially reversible causes of cardiopulmonary failure refractory to traditional management, such as hypoxic and hypercapnic respiratory failure, refractory cardiogenic shock, cardiac arrest, failure to wean from cardiopulmonary bypass after cardiac surgery, and as a bridge to heart and/or lung transplantation.

Previously reserved only for highly specialized indications, ECMO utilization has increased dramatically even since the mid-2000s, with a decline in overall mortality rates from above 40 to 33 % in one large series [120]. ECMO has now been shown to be associated with reasonable survival rates in a variety of settings, including acute respiratory distress syndrome, in patients who would otherwise assuredly have nearly 100 % mortality rates [121]. Additionally, new modalities, such as low-flow ECMO for CO_2 removal (extracorporeal carbon dioxide removal or ECCO$_2$R), represent promising new therapeutic options for selected patients.

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