

Cardiac arrest

Acute coronary syndrome

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Introduction

Cardiac arrest is a rare but serious cause of morbidity and mortality in the perioperative setting. Ischemic heart disease is the leading cause of death in the world.¹ Ambulatory surgery centers should be prepared to treat acute coronary syndrome (ACS) and have a system in place to stabilize and transfer the patient to a higher acuity level of care facility, and if possible one that has a cardiac catheterization lab. The principles in this case apply to any operating room (hospital-based and outpatient), off-site anesthetizing location, or clinic.

The three cases presented here for learning purposes are patients with preexisting coronary artery disease presenting for elective ambulatory surgery. Theses cases will offer an opportunity to discuss key points in early detection, communication, treatment, and management.

Educational objectives

- 1. Take appropriate steps to manage a patient with known or possible coronary artery disease
- 2. Detect early signs of myocardial ischemia or cardiac arrest
- 3. Emergently treat cardiac arrest

Scenario 1: STEMI in operating room

A 76-year-old male, 82 kg patient is in the operating room for a shoulder arthroscopy. The patient has a history of hypertension, hyperlipidemia, coronary artery disease, and diabetes mellitus type 2, all of which are moderately well controlled with medications. General anesthesia induction was uneventful. During surgical incision, tachycardia and ST segment elevation in leads II and V5 were detected.

Expected actions:

- 1. Assess clinical status and check all vital signs
- 2. Assure ventilation and oxygenation
- 3. Consider early treatment prior to confirming diagnosis

Scenario continued:

Acute hypotension quickly ensues with decrease in end-tidal CO_2 .

Expected actions:

- 1. Call for help and ask for crash cart
- 2. Consider placing invasive arterial line
- 3. Check blood pressure and other vital signs
- 4. Differential diagnosis of acute myocardial infarction, pulmonary embolus, anaphylaxis, hypovolemia
- 5. Consider placement of trans-esophageal echo probe (TEE) for diagnosis (if available)

Scenario continued:

TEE reveals anteroseptal and inferior wall hypokinesis. (If no TEE available, downsloping ST-segments are seen on anterior and inferior leads of 12-lead ECG.)

Expected actions:

- 1. Consider acute myocardial ischemia causes: coronary artery spasm, plaque rupture, demand ischemia
- 2. Begin vasodilator and inotrope infusion as needed to maintain blood pressure (nitroglycerin and epinephrine)

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Scenario continued:

Ventricular ectopy, deteriorates into ventricular fibrillation

Expected actions:

- 1. Begin basic life support (BLS). This is chest compressions at ratio of 30 compressions to two breaths, rate at least 100, depth at least 2" (5 cm), allow for recoil
- 2. Get defibrillator and attach pads
- 3. Simultaneously secure airway depending on expertise of available staff (mask ventilate, supraglottic airway, or intubation)
- 4. Defibrillate at 200 joules, repeat after 2 min at 360 if unsuccessful

Scenario continued:

Rhythm converts to sinus rhythm with occasional PVCs, blood pressure 98/74, HR 88

Expected actions:

- 1. Continue treatment with fluids and vasoactive medications
- 2. Continue to monitor vital signs and echo imaging
- 3. Arrange disposition to higher level of care facility with cardiac catheterization lab capabilities

The drill ends with resuscitation that continues in transport to hospital with cardiac catheterization lab.

Scenario 2: Chest pain in recovery

A 77-year-old female 67 kg patient is in the post anesthesia care unit (PACU) recovering from a carpal tunnel release under local anesthesia with monitored anesthesia care. She has a history of chronic kidney disease (CKD) requiring dialysis, hypertension, diabetes mellitus type 2, and coronary artery disease. She had suffered a heart attack 12 months previously, requiring two stents in her proximal LAD and one in the mid RCA. The MI was complicated by heart failure with an EF of 24% requiring an aortic balloon pump, but she has regained some functional capacity walking two blocks slowly. The patient begins to complain of severe crushing substernal chest pain upon arrival in PACU and calls out to her nurse.

Expected actions:

- 1. Call for help and ask for crash cart
- 2. Obtain 12-lead ECG

- 3. Administer oxygen via nasal cannula, increase as needed to maintain oxygen saturation greater than 90%
- 4. Administer chewable aspirin 162–325 mg non enteric coated
- 5. Give sublingual nitroglycerin as needed for chest pain
- 6. Administer morphine intravenously as needed for chest pain
- 7. Consider differential diagnosis including aortic dissection, pericarditis, pulmonary embolus, pneumonia, pneumothorax, pleuritis, gastrointestinal reflux disease, esophageal spasm, costochondritis, etc.

Scenario continued:

ST elevation is noted in the anterior leads V1–V4, ventricular ectopy begins, and the cardiac rhythm rapidly deteriorates to ventricular fibrillation

Expected actions:

- 1. Begin basic life support
- 2. Get defibrillator and attach pads
- 3. Simultaneously secure airway depending on expertise of available staff (mask ventilate, supraglottic airway or intubation)
- 4. Defibrillate at 200 joules, repeat after 2 min at 360 if unsuccessful

Scenario continued:

Defibrillation success, patient is stable, blood pressure is 94/62, HR 85.

Expected actions:

- 1. If freestanding center, call EMS for transfer using pre-established institutional management protocols of STEMI
- 2. If tertiary care center, institute single call system for mobilization to cardiac catheter lab with cardiology consult for angiography
- 3. Establish arterial line and send labs for multimarker evaluation with cardiac troponin and creatine kinase MB (CK-MB)
- 4. Support circulation with inotropes and antiarrhythmic as necessary

Scenario continued:

The drill ends with implementation of protocol to transfer patient to facility with cath lab alerted to possible acute stent thrombosis.

Scenario 3: Bradycardic arrest

A 59-year-old male is in the operating room receiving a retrobulbar block by the ophthalmologist for a planned vitrectomy. He has a past medical history of diabetes mellitus type 2, HTN, sleep apnea, and hyperlipidemia. After injection of 3 ml of lidocaine 2% mixed with bupivacaine 0.75% the patient becomes severely bradycardic to a rate of <20. The patient is given glycopyrrolate 0.2 mg IV with no response and subsequently is given atropine 0.4 mg IV. The patient then responds with sinus tachycardia at a rate of 142 and begins to complain of chest pain.

Expected actions:

- 1. Treat tachycardia with beta-blocker
- 2. Administer O_2 via nasal cannula to keep saturation greater than 90%
- 3. Obtain 12-lead ECG
- 4. Obtain serial cardiac troponin levels (at presentation and at 3 and 6 hours)
- 5. Review medical history and reassess risk

Scenario continued:

A 12-lead ECG reveals down-sloping ST depression and T-wave inversion anteriorly. Patient continues complaining of symptoms of chest tightness and inability to breathe. Patient becomes diaphoretic. Heart rate decreases to 90 but chest pain continues.

Expected actions:

- Administer sublingual nitroglycerin 0.4 mg every 5 min up to three doses (contraindicated if patient on phosphodiesterase inhibitor)
- 2. Give intravenous morphine for pain
- 3. Administer chewable aspirin 162–325 mg non enteric coated
- 4. Call for help for cardiac evaluation if available
- 5. Consider use of oral beta-blockers, calcium channel blockers, and statins

Scenario continued:

Drill ends with making urgent arrangements to transfer patient to cardiac care unit or another facility because of ongoing chest pain.

Debriefing

Briefings happen before a learning experience, and debriefings after. Debriefing is a form of feedback, and allows the participants to reflect on their knowledge and behavioral and technical skills. It is important to have the participants feel safe to question and not take the feedback as a personal attack, but to welcome the opportunity to find their own knowledge gaps and or discover themselves what they needed to do better. That is the art of debriefing. The participants should feel empowered to recognize themselves their own strengths and weaknesses. It is thought that this active learning enhances memory.

- 1. Identify three things the team felt went well during the scenario.
- 2. Identify three things the team felt should or could be improved.
- 3. What equipment was difficult to locate or use?
- 4. Was crash cart or TEE probe difficult to obtain?
- 5. Was help easily accessible?
- 6. What was not available that would have been helpful?
- 7. What would have been done differently?
- 8. Discuss when you thought transfer to hospital with or without cardiac catheterization lab would be necessary.
- 9. Discuss the steps of ACLS resuscitation and algorithm.
- 10. Identify up to three improvements that the facility will incorporate after the drill to prepare them for a similar case.

Discussion

The American College of Cardiology (ACC) and American Heart Association (AHA) provided updated guidelines for risk assessment of non-cardiac surgery and need for perioperative workup and management.² The ACC & AHA have also provided updated recommendations for management of acute coronary syndrome.^{3,4} The underlying principle for their recommendations is to use appropriate key clinical factors such as type of surgery and patient comorbidities to assess risk, which guides the degree of preoperative cardiac workup for non-cardiac surgery. For patients experiencing acute coronary syndrome, efficient teamwork is essential in stabilizing patient transfer to a hospital with cardiac catheterization lab capabilities.

Initial medical therapy should consist of immediate antiplatelet anticoagulant therapy. Despite the proliferation of newer antiplatelet and antithrombotic agents, aspirin continues to be the mainstay of initial management and should not be forgotten. Oxygen,

nitrates, beta-blockers, calcium channel blockers, and statins are Class I therapy (benefit is greater than risk, procedure/treatment should be performed/administered). Morphine sulfate is reasonable for treatment of continued pain despite maximal medical therapy, even though it is classified as a type IIb agent (benefit greater or equal to risk, additional studies needed. Procedure treatment may be considered).³

Nitrates in the setting of phosphodiesterase therapy, NSAIDS, IV beta-blockers when shock is present, and immediate-release nifedipine in the absence of a beta-blocker are all potentially harmful interventions in acute coronary syndrome.³

The International Liaison Committee on Resuscitation (ILCOR) includes representatives from the AHA, the European Resuscitation Council (ERC), the Heart and Stroke Foundation of Canada (HSFC), the Australian and New Zealand Committee on Resuscitation (ANZCOR), and the Resuscitation Council of Asia (RCA). They have established an updated ILCOR Universal Cardiac Arrest Algorithm,¹ which has only two pathways, shockable or non-shockable cardiac rhythm. These documents are available on the ILCOR website (see www.ilcor.org). A simple algorithm to follow has the benefit of allowing the staff to focus on the quality of the CPR efforts and early defibrillation, which has been shown to improve outcomes.

Early recognition of myocardial ischemia is the key to management, along with having existing protocols and systems in place for handling emergencies. In an effort to do no harm, medical professionals may have a tendency toward inaction rather than action, and avoid starting CPR out of fear of breaking a patient's ribs, or even damaging their professional reputation if wrong. This is called omission bias⁵ and must be avoided.

Once the emergency is recognized and the team is mobilized, effective outcomes are the results of deliberate practice. It is not only important to educate the ambulatory center staff about the protocols, but retraining the staff at intervals of six months may be necessary to improve skills and improve retention.⁶

There are several key points which have been shown in recent years to correlate with improved outcomes and should be emphasized in training¹:

- 1. Early defibrillation is lifesaving and AED use should not be restricted to trained medical personnel
- 2. Defibrillation should not be delayed. One rescuer can begin chest compressions while the AED or defibrillator is obtained

- 3. Incomplete chest recoil is common and full release after each compression should be emphasized
- 4. "No flow" fraction should be minimized, i.e. continuous CPR maintained
- 5. Unscheduled mock codes improve mock code performance in hospital personnel
- 6. Rescuer should be alternated every 2 min to prevent rescuer fatigue which results in deterioration of chest compression quality, specifically depth of compression (>38 mm)
- 7. It is reasonable to use cognitive aids (checklists) during resuscitation. Caution is advised so that unintentional delay in initiation of the correct protocol does not occur. It is difficult for the event leader to simultaneously lead and read, and thus the role of a reader has been suggested.⁷

In the three drill cases presented, the first patient had a spontaneous non-STEMI related to a primary coronary artery process such as an atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombosis. In this case it would be important to make the decision early to transfer the patient to a facility capable of PCI or an in-house transfer to the cardiac catheterization laboratory. The goal of a 90 minute door-to-device time is quite feasible in most cases, since the initial symptoms have been witnessed. However, if this is not practical, then the decision to start fibrinolytic therapy should be considered.

The second patient had STEMI related to stent thrombosis. This presents itself as a sudden dramatic event, and the urgency is even greater to get the patient to a catheterization lab. Stent thrombosis often presents as devastating ventricular arrhythmias and cardiac collapse.

It is important to understand that an estimated 600,000 coronary stents⁸ are placed annually in the United States, and that the cumulative incidence of non-cardiac surgery following coronary artery stenting is more than 10% at one year and over 20% at two years.⁹ Despite evidence-based guidelines on antiplatelet therapy, there is still much controversy on managing perioperative antiplatelet medication. Even though patients are told by medical staff to stay on their aspirin perioperatively, they often will still discontinue it on their own. The hypercoagulable state induced by the stress of surgery, along with a rebound effect from stopping aspirin, can increase the chances of a late stent thrombosis.¹⁰

The third case was unstable angina related to ischemic imbalance; the oxygen demand was higher than the supply because of the tachycardia and underlying

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coronary artery disease and it was unclear whether it would evolve to an MI or not. This patient needs follow-up in a cardiac care unit, and may become unstable at any point.

As anesthesiologists and anesthetists, we are used to taking care of the patients ourselves. It is indeed a paradigm shift to rely on a system to help us take care of these patients, and the key element to saving lives is having systems in place that ensure smooth transfers to the appropriate facilities.

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Chapter

Reversible causes of cardiac arrest

Steven Butz

Introduction

Cardiac arrest can occur for reasons other than ischemic heart disease. The American Heart Association classifies them into the reversible causes of cardiac arrest, the H's and T's. The mnemonic stands for, in this case, hypoxemia, hypovolemia, hydrogen ion (acidosis), hypo-/hyperkalemia, hypothermia, thrombosis (pulmonary and cardiac), toxins, tamponade (cardiac), and tension pneumothorax. A study of European in-hospital cardiac arrest attributed 42% of 258 cardiac arrests to these reversible causes.¹

This chapter will direct you through scenarios that may be seen in an ambulatory setting that are derived from these etiologies.

Educational objectives

- 1. Encourage review of situations to identify potentially reversible causes of cardiac arrest.
- 2. Be able to enumerate causes of reversible arrest.
- 3. Evaluate clinical setting for presence of diagnostic and therapeutic items to differentiate causes of cardiac arrest.

Scenario 1: Pneumothorax

A 38-year-old male with aggressive testicular cancer presents for central line insertion for chemotherapy. He undergoes an uneventful general anesthetic with propofol, remifentanil, ketorolac, and ondansetron. A laryngeal mask airway is used to control his ventilation.

In the recovery room, the patient gets agitated and is fighting the nursing staff trying to calm him and keep him restrained. The patient screams that he has trouble breathing.

Expected actions:

1. Evaluate monitors for signs of hypoxia and arrhythmias.

- 2. Apply oxygen.
- 3. Assess vital signs.

Scenario continued:

The patient is hypotensive with blood pressure 54/28 with heart rate of 138 and oxygen saturation of 82% with $4 \log p$ by facemask.

Expected actions:

- 1. Assess for jugular venous distension and bilateral breath sounds.
- 2. Assess EKG waveform for signs of hyperkalemia.
- 3. Prepare for intubation.
- 4. Consider bolus of IV fluid.
- 5. Consider chest x-ray.

Scenario continued:

Suddenly, the patient's eyes roll back into his head and he slumps to the side. ECG rhythm is still sinus tachycardia. There is no pulse.

Expected actions:

- 1. Call for help.
- 2. Begin ACLS for pulseless electrical activity (PEA).
- 3. Assess for cardiac tamponade versus tension pneumothorax during secondary assessment.
- 4. CPR is to continue to provide perfusion.

Scenario continued:

There are no breath sounds on the left chest, the side with the new central line.

Expected actions:

1. Needle aspiration of air is performed in left chest at second intercostal space anteriorly at the mid-clavicular line.

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- 2. Consider placement of chest tube if available.
- 3. Ultrasound may be used as a guide for evaluation of pneumothorax.

Scenario continued:

Pulse returns with relief of tension pneumothorax. Plans should be made for transfer to hospital.

Scenario 2: Acidosis and hypovolemia

A 51-year-old, 56 kg female patient with type 1 diabetes has undergone a screening colonoscopy. She successfully completed her prep and had documented her blood sugars at home. Her A-1-c is 7.1, but was running in the 200s in the previous days and not feeling well. After her prep, her glucose was 300–400 mg/dl despite treating with subcutaneous insulin at home. On arrival that morning, her sugar was 275 mg/dl. Your anesthesia colleague was consulted by the gastroenterologist to help get the case completed since the patient wanted it done and had completed her bowel preparation. The anesthesiologist agreed to do the case since the 275 mg/dl reading was improved from the previous day. Propofol sedation was given and a bolus of 1 l of lactated Ringer's solution was given prior to starting.

Your colleague left due to a family emergency and you are called to the recovery room 30 min after the patient arrived. She has not woken up.

Expected actions:

- 1. Assess the patient and review history.
- 2. Check blood glucose.
- 3. Check ketones if available. May require placing urinary catheter to check urine dipstick.
- 4. Assess for hypovolemia and give bolus of normal saline.

Scenario continued:

Glucose is "high" on glucometer. Before patient can be aroused, the EKG shows peaked T-waves that evolve into pulseless ventricular tachycardia.

Expected actions:

- 1. Call for help.
- 2. Begin CPR and ACLS protocol for pulseless VT/VF. CPR needs to be continued throughout administration of medications until pulse is restored.
- 3. Advanced airway management. Intubation is indicated for patients undergoing CPR; *alternative*

methods of oxygenation may be used if personnel with intubation skills are unavailable.

4. Perform secondary assessment after initial treatment with focus on treatment of acidosis from diabetic ketoacidosis (DKA) and hypovolemia.

Scenario continued:

Shocks have been unsuccessful. A colleague recommends placing a Foley catheter and gets dark urine with positive ketones on a dipstick test. No triggering agents were used. Your colleague also recommends hanging hetastarch since she is obviously dry and to give a 15 unit bolus of IV insulin.

Expected actions:

- 1. Continue CPR and ACLS.
- Begin treatment for hyperkalemia.
 a. calcium
 - b. regular insulin IV
- 3. Give fluid bolus of 1 l of normal saline.

Scenario continued:

Next round of shocks restore normal sinus rhythm. Patient is still ventilated. There has been no urine production.

Expected actions:

- 1. Make arrangements for transfer.
- 2. Blood gas may be drawn if possible to process.
- 3. Continue volume resuscitation with normal saline.
- 4. May begin insulin treatment with either normal saline drip at 0.05 units/kg/h or ultra rapid insulin 0.5 units /kg subcutaneous injection.

Scenario continued:

The drill would end when ambulance arrives. Facility personnel likely to go along to provide report and ventilation.

Scenario 3: Anaphylaxis

A 17-year-old female is undergoing a left ACL repair. She has a known egg allergy and a history of asthma and psoriasis. A femoral nerve catheter was placed preoperatively and the procedure is being performed under general anesthesia. The anesthetic used is a total IV anesthetic (TIVA) due to history of severe PONV with previous surgery. She is being maintained on propofol and ketamine infusions with nitrous oxide, ketorolac, dexamethasone, and ondansetron. A dose of Chapter 2: Reversible causes of cardiac arrest

cefazolin was administered preoperatively. The airway is managed with a supraglottic airway.

Approximately 15 min into the procedure, the oxygen saturation has dropped to 91% and tidal volumes are decreased to 200 ml.

Expected actions:

- 1. Examine the patient by auscultating the lungs and looking at the skin for rashes.
- 2. Look at the end-tidal CO₂ tracing for evidence of bronchospasm.
- 3. Hand-ventilate and administer albuterol for bronchospasm.

Scenario continued:

Blood pressure cycles and is now 48/23. Heart rate is 118 bpm.

Expected actions:

- 1. Epinephrine 100–200 mcg IV administered.
- 2. Volume expansion with crystalloid.
- 3. Review of possible allergic triggers: propofol (egg allergy), ketorolac (asthma-related aspirin allergy), antibiotic, latex.
- 4. Work to remove possible trigger.

Scenario continued:

Patient's cardiac rhythm deteriorates into ventricular fibrillation. Patient's face is noticeably edematous.

Expected actions:

- 1. Begin ACLS protocol for VT/VF.
- 2. Proceed with cardiac compression and intubation.
- 3. Administer high-dose epinephrine.
- 4. Surgery should be closed/aborted.

Scenario continued:

Sinus rhythm returns after 2 cycles of epinephrine and shocks. Patient remains hypotensive.

Expected actions:

- 1. Epinephrine drip should be started.
- 2. Patient should be dosed with higher-dose dexamethasone. Consider adding diphenhydramine and H₂ blocker.

Scenario continued:

The drill ends with arrangements for transfer and completion of secondary survey.

Debriefing

- 1. Identify up to three or more things the team felt went well with the scenario.
- 2. Identify up to three or more things the team felt they should have done differently.
- 3. What equipment was difficult to locate or use?
- 4. Were the H's and T's addressed early?
- 5. Were suggestions from staff taken and considered?
- 6. What aspects of patient history or case scenario were there to lead to correct diagnosis?
- 7. What processes worked well and what needs to be improved?

Discussion

Although the protocols for resuscitation for ventricular fibrillation/tachycardia and pulseless electrical activity continue regardless of cause, identifying the etiology may lead to a successful resolution more quickly. The cause of cardiac arrest in hospital is most often primary cardiac disease, but the H's and T's account for 42% of arrests.¹ The numbers are likely to be different in the outpatient setting and actually may be higher for non-cardiac causes as patients with severe cardiac disease are selected away from an ambulatory setting. Aspects of the H's and T's relevant to the scenarios presented will be described below.

Scenario 1: Pneumothorax

Hypoxemia by itself may lead to cardiac arrest. The etiology probably combines other components of the H's and T's. As a person is deprived of oxygen, anaerobic metabolism will begin. This contributes to acidosis. The high oxygen consumption of the heart makes it particularly vulnerable to low oxygen levels. If the cause is combined with poor ventilation, the hypercarbia will further increase acidosis making the myocardium more vulnerable to arrhythmias. Shifts in pH will also affect potassium levels, causing more irritability. Toxins can cause hypoxemia as in the case of cyanide or carbon monoxide.

Treatment of hypoxia is reversing the primary cause. For ambulatory surgery, the most likely successful maneuver will be re-establishing a patent airway. This may be more difficult in a patient with a history of obstructive sleep apnea as they are at higher risk for desaturation in the recovery room than the healthy patient. Toxins each have an individual treatment, and identification of the toxin will go a long way to determine the next steps in treatment.



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In our case, the hypoxemia was most likely due to either a tension pneumothorax or even a pericardial effusion. Either can present as an acute or chronic problem, each with different treatments. For this patient, the onset was acute and needed emergent treatment. On physical exam, tracheal deviation away from the affected side is seen. Jugular venous distension and hyperresonance to percussion are late findings.² The diagnosis can be difficult in an ambulatory setting. A chest film can be performed during a code to diagnose a pneumothorax. However, a skilled ultrasonographer may also be able to diagnose it with an ultrasound machine.³ An article comparing AP chest films with non-radiologist sonographers demonstrated equivalent sensitivity and specificity. A tension pneumothorax may be diagnosed by ultrasound when there is an absence of lung sliding and B lines are present. B lines are seen in an anteriomedial view in the mid-clavicular line. These are vertical lines that arise from the pleural line and extend to the lower edge of the screen.4

Treatment of a tension pneumothorax is with a 14or 16-gauge needle inserted into the second intercostal space in the mid-clavicular line. This will release the pressure with a rush.² After relief, there needs to be careful monitoring to ensure that there is no recurrence of the flap-valve mechanism causing the initial pneumothorax, requiring a chest tube placement.

A cardiac tamponade would be possible if there were proximal rupture of the vasculature or atrium with the wire used to place the central line. Hemodynamically significant tamponade may show the signs of jugular venous distension (JVD) and pulsus paradoxus. The JVD reflects the higher right atrial filling pressures. Pulsus paradoxus is an exaggeration of the decrease in blood pressure that occurs with respiration. Typically, blood pressure drops 10 mmHg when increased negative intra-thoracic pressure decreases the left atrial filling. With tamponade, filling is further restricted and the blood pressure drop is more pronounced.⁵ Other signs of tamponade are dyspnea, tachycardia, and decreased heart tones. Diagnosis with ultrasound is made when effusion is identified and there is collapse of the right chamber size in the cardiac cycle. These findings are very accurate for the diagnosis.³

Treatment for cardiac tamponade should include airway management and CPR as needed. The patient should be placed with chest elevated 45°. The perixyphoid area should be prepped and draped. Ideally, a 16- or 18-gauge cardiac needle about 15 cm long should be attached via a stopcock to a 50 ml syringe.

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Figure 2.2 Approach used to aspirate fluid causing cardiac tamponade

Using ultrasound or careful EKG monitoring, the needle is advanced from just inferior-lateral to the left of the xyphoid at a 45° angle toward the left mid-clavicle. Once the pericardium is entered (it may be felt by the person advancing the needle), blood is withdrawn. Symptoms may reverse quickly even after a small amount of blood is withdrawn. A clamp may be used to stabilize the needle at the skin or a catheter may be threaded in to withdraw the blood. The EKG should be viewed looking for evidence of entry into the ventricle or atrium. Ventricular penetration will cause ST elevations and atrial penetration may cause PR elevation.²

Scenario 2: Acidosis and hypovolemia

The issues woven into this scenario include hypovolemia, acidosis, diabetes, and coverage issues. The resulting controversies are the care for ketoacidosis and fluid resuscitation. Recognition of hyperkalemia would be critical to success, as would the correct treatment.

Hypovolemia in an ambulatory surgery center may be present for many different reasons. Fasting patients are already dry, but a healthy patient should be able to compensate. Patients on diuretics or having bowel preparations will be further depleted. Relative hypovolemia may occur in cases of vasodilation caused by sympathetic blockade or anaphylaxis and shock. Life-threatening hypovolemia in the surgical setting can be from extreme or unnoticed blood loss. Bleeding from a laparoscopic procedure may continue until the patient decompensates.

Volume replacement is the first treatment until the cause of hypovolemia is addressed. The fluid of choice is controversial when considering crystalloid versus colloid. Previous thought was that only one third of administered crystalloids stayed intravascular. Newer