Chapter 23
Complications of Myocardial Infarction and Cardiovascular Emergencies

Nai-Lun Chang and Adam S. Budzikowski

Complications of Myocardial Infarction

1. Mortality rates due to complications of acute myocardial infarctions (AMIs) have been trending downward in recent years. With early reperfusion strategies and optimized medical therapy, early recognition and intervention are crucial [1, 2].
2. Complications can be separated into three main categories—arrhythmic, inflammatory, and mechanical.
3. Arrhythmic complications:
   (a) Common complications after acute MI.
   (b) Extensive MI with left ventricular (LV) failure.
      - Often improves with afterload reduction and treatment of pulmonary vascular congestion
   (c) Supraventricular tachyarrhythmias:
      - Triggered by excessive sympathetic activation.
      - Persistent sinus tachycardia.
   (d) Atrial fibrillation/atrial flutter:
      - The incidence rate of atrial fibrillation is 10–15% among patients who have AMIs.
      - Higher risk of heart failure (HF), stroke, shock, and mortality.

N.-L. Chang (✉) · A. S. Budzikowski
Division of Cardiovascular Medicine-EP Section, SUNY Downstate Medical Center, Brooklyn, NY, USA
e-mail: Nai-Lun.Chang@downstate.edu; abudzikowski@downstate.edu

H. Z. Mieszczanska, A. S. Budzikowski (eds.), *Cardiology Consult Manual*,
https://doi.org/10.1007/978-3-319-89725-7_23

- Rate versus rhythm control:
  - For patients with hemodynamic instability, immediate synchronized electrical cardioversion of 200 Joules is indicated. If AF does not respond to cardioversion, IV amiodarone can be used.
  - For patients who do not have hypotension, beta-blockers or calcium channel blockers can be used if no other contraindications.
  - Digoxin as an adjunct for rate control in patients with reduced LV systolic function or HF.
  - Indications/contraindications of anticoagulation should be considered.

(e) Bradyarrhythmias and intraventricular conduction defects:
- Transient.
- May occur immediately post reperfusion.
- High-degree atrioventricular (AV) block and persistent bundle branch block are strong predictors of cardiac death in the setting of MI [3].
- Extensive anterior and inferior MI may cause bundle branch block and AV blocks.
- Idioventricular rhythm is suggestive of reperfusion, albeit not sensitive or specific.
- Temporary transvenous pacemaker (TVP) needed, if:
  - Symptomatic bradycardia unresponsive in medical therapy (Class I).
  - High-grade AV blocks (second or third AVB), whether patient symptomatic or not.
  - New bundle branch (persistent or alternating) or bifascicular block for anterior or lateral MI [4].
  - Alternating BBB.
- Indications for permanent pacemaker:
  - Persistent high-degree AV block, with or without bundle branch block (Class I; LOE B, C) [4], even if patient is asymptomatic.

(f) Ventricular arrhythmias—ventricular fibrillation (VF) and ventricular tachycardia (VT) (see also Fig. 23.1):
- Most commonly occur during the first 48 h post-STEMI.
  - Polymorphic VT is associated with recurrent myocardial ischemia [5].
- VTs that occur post initial 48 h or in context of cardiogenic shock have poor prognosis:
  - Associated with depressed LV function and myocardial scar.
  - Need to rule out recurrent ischemia.
- Non-sustained VT:
  - Start beta-blocker, if no contraindications.
- Sustained VT is defined as three or more consecutive premature ventricular contractions (PVCs) at a rate > 100 bpm and lasting >30 s or PVCs causing hemodynamic compromise.
- Ventricular fibrillation:
  - Emergent defibrillation.

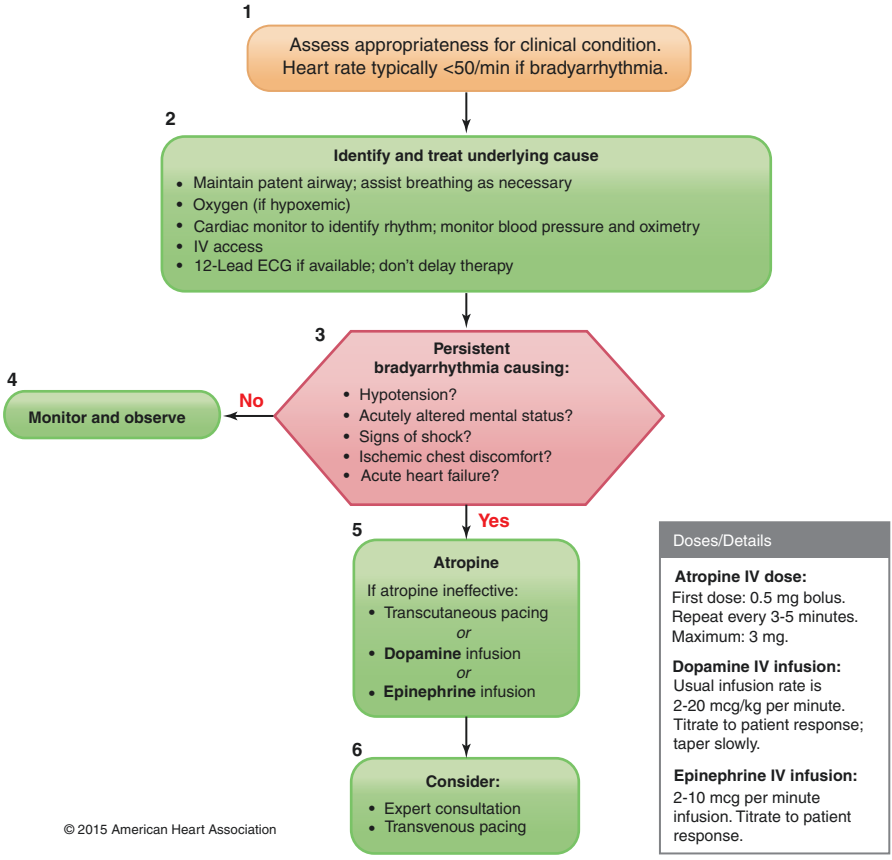


Fig. 23.1 Adult tachycardia (with pulse). Reprinted with permission from [34]

- Prevention of VF or VT is focused on electrolyte abnormality correction and revascularization for recurrent ischemia.
- Acute treatment in the setting of hemodynamic instability:
  - DCCV + IV amiodarone or lidocaine.
  - Revascularization if due to recurrent ischemia (recurrent angina ± new ECG changes).
  - Replete electrolytes (Keep K > 4 mEq/L and Mg > 2 mg/dL).
- Prophylactic suppression of premature ventricular contraction with lidocaine is not recommended, as it elevates risk of excess mortality by fatal bradycardia [4, 6, 7].
- Implantable cardioverter-defibrillator (ICD):
  - Not recommended in the setting of acute MI.
  - Current guidelines recommend deferring ICD implantation for at least 40 days following MI [8].

  - Overall mortality was not improved with early, prophylactic ICD therapy [9].
  - However, ICD therapy is indicated before discharge in patients who develop sustained VT/VF more than 48 h after STEMI, provided the arrhythmia is not due to transient or reversible ischemia, reinfarction, or metabolic abnormalities.
- LifeVest® (ZOLL, Pittsburgh, PA, USA) [10–13]:
  - Wearable external defibrillator that provides continuous protection for patients with elevated risk for sudden cardiac death
  - Indications:
    Serves as a bridge to cardiac transplantation or ICD placement.
    Early post-MI patients with markedly reduced LV function.
    Infected ICDs awaiting for device reimplantation.
    New-onset nonischemic cardiomyopathy with severe LV dysfunction during a trial of optimization of medical therapy.

4. Inflammatory:
   (a) Early pericarditis (2–4 days post MI):
   - Usually with extensive or anterior MI.
   - Pericardial rubs on physical exam.
   - EKG showing diffuse concave ST elevations with PR depressions.
   - Small pericardial effusion may be present on transthoracic echocardiogram (TTE).

   (b) Late pericarditis/Dressler's syndrome (weeks post MI):
   - Autoimmune reaction.

   (c) Treatment [4, 14]:
   - High-dose aspirin (Class I).
   - Adjunct colchicine, acetaminophen, or opioids may be useful (Class IIb).
   - Avoid NSAID and steroids (Class III).

   (d) Mechanical complications:
   - With early reperfusion strategy, the incidence of mechanical complications after acute myocardial infarction has declined. However, rapid deterioration of a patient's clinical status with new physiological exam findings should raise suspicion of mechanical complications and call for urgent diagnosis and surgical intervention [15].
   - Urgent echocardiography with color-flow Doppler is used as the initial diagnostic modality in the diagnosis and differentiation of the conditions.
   - Preoperative optimization, with an intra-aortic balloon pump and vasodilators, may help to reduce the afterload on the compromised ventricle following AMI. It may improve cardiac output in the short term but should not delay expedient surgical intervention in the setting of acute mitral regurgitation (MR).
   - Surgical intervention remains the mainstay of treatment in patients with mechanical complications of AMI, with dismal outcomes for patients treated medically.

(e) Rupture of LV free wall:
- A serious complication of ST-elevation myocardial infarction with very high mortality rate [16]. The SHOCK registry showed that mortality with free wall rupture was over 60% [17].
- It occurs in 0.5% of patients post MI.
- Occurs most often in anterior or transmural MI, elderly, and female patients.
- Signs and symptoms:
  - Recurrent chest pain with sudden hemodynamic collapse.
  - Patients may show signs of cardiac tamponade.
- Physical findings: diminished heart sounds, new murmurs, pulsus paradoxus, and jugular venous distention may be found.
- Diagnostics:
  - Urgent bedside echocardiography.
  - Right heart catheterization may show equalization of chamber pressures (right atrial, right ventricular diastolic, pulmonary wedge, and pulmonary arterial pressures) consistent with cardiac tamponade.
- Management: aggressive supportive care while waiting for cardiac surgery for emergent repair.

(f) Ventricular septum rupture (VSR):
- 1–2% of patients following AMI and usually between 3 and 5 days after AMI [15].
- Often in elderly, female, and hypertensive patients with poor collaterals to the infarcted area and in patients with delayed or lack of perfusion therapy.
- Sign, symptoms, and physical findings:
  - Sudden onset of heart failure or shock with new loud harsh holosystolic murmur, which is heard loudest at the left sternal edge, associated with a parasternal thrill.
  - The differential is acute mitral regurgitation secondary to papillary muscle rupture.
- Diagnostics:
  - ECG may show intra- or infranodal conduction abnormalities.
  - Transthoracic echocardiogram (TTE) to assess defect size and magnitude of shunt.
- Management:
  - Pressors, inotropes, and vasodilator agents.
  - Intra-aortic balloon pump (IABP) may be needed as a bridge to surgical repair [4].
  - Emergent surgical repair is necessary, even if patient is hemodynamically stable (Class I).

(g) Acute mitral regurgitation (MR) post MI:
- A life-threatening complication with a poor prognosis.
- High index of suspicion is needed if patient becomes hemodynamically unstable post MI.

- Incidence rate ranges from 3% [18] up to 39% [17].
- Occurs either from papillary muscle rupture or ischemic LV remodeling (functional ischemic MR) that restricts the posterior leaflet.
- Papillary muscle rupture resulting in MR occurs within 2–7 days of MI and occurs in 0.25% of patients following MI.
- The posteromedial muscle is affected more often than the anterolateral papillary muscle.
- Signs and symptoms:
  - Variable
  - May present as sudden pulmonary edema, with or without shock
- Physical findings: new systolic murmur. However, due to rapid equalization of pressure between the left atrium (LA) and left ventricle (LV), murmur may not be audible.
- Diagnostics:
  - CXR showing pulmonary edema.
  - TTE showing severe MR with flail leaflet.
- Management:
  - Medical therapy ± IABP support, to reduce afterload and increase forward volume and output, while waiting for urgent surgical repair.
  - Valve replacement, rather than repair, is necessary and required.

(h) Left ventricular aneurysm (LVA):

- Defined as a localized area of myocardium with abnormal outward bulging and deformation during both systole and diastole.
- The rate of LVA after AMI is approximately 3–15%.
- Signs and symptoms: severe LV dysfunction, leading to heart failure and cardiogenic shock if aneurysm is large.
- Diagnostics:
  - ECG showing persistent ST elevation after AMI that appears in the same leads as those showing the acute infarct.
  - TTE showing wide neck aneurysmal and dyskinetic segments, "smoke," and possible mural thrombus.
- Management:
  - IV vasodilators ± IABP for acute left ventricular failure.
  - Optimized medical therapy for chronic heart failure.
  - Surgical correction may be considered if heart failure is refractory to medical therapy or there is development of ventricular arrhythmia.

(i) LV pseudoaneurysm:

- Contained myocardial rupture or perforation.
- High mortality rate [19].
- Usually with inferior MI—proximal RCA occlusion with impaired flow to RV marginal branch.
- Signs and symptoms:
  - May be asymptomatic.
  - Variable (e.g., chest discomfort, dyspnea, etc.).

- Diagnostics:
  - ECG showing persistent ST elevations.
  - TTE showing a narrow neck pouching.
  - Ventriculogram on the left heart.
- Management:
  - Surgical correction recommended, despite symptomatology or size of the pseudoaneurysm, to prevent spontaneous rupture.

(j) Left ventricular mural thrombus (LVMT):
- Develops after anterior infarcts of the LV wall.
- Incidence ranges from 20 to 40% [20].
- Anticoagulation with warfarin for mural thrombus and embolization (Class I):
  - Target INR 2–3.
  - In addition to dual antiplatelet therapy (DAPT).

(k) Cardiogenic shock (CS):
- Cardiogenic shock remains a leading cause of mortality in the setting of an acute myocardial infarction, due to end organ failure [21].
  - Criteria:
    SBP <80–90 mmHg
    Pulmonary congestion
    Signs of low peripheral perfusion in the setting of severely depressed cardiac index <2.2 L/min (m^2) with support or <1.8 L/min (m^2) without support
    Elevated left ventricular filling pressures (pulmonary capillary wedge pressure >15–18 mmHg) [22, 23]
  - Risk factors:
    Elderly (>70 years of age)
    Female
    Multivessel coronary artery disease
    Extensive LV infarct, especially anterior MI
    ST elevation MI with new LBBB
    Prior history of CAD and HF
  - Causes [17]:
    Left (79%) or right ventricular failure (3%)
    Mechanical complications of acute MI (~12%)
    Iatrogenic (e.g., medication overdose)
    Ventricular outflow tract obstruction
    Cardiac tamponade
    Arrhythmia induced
  - Signs and symptoms:
    Variable (hypotension, altered mental status, dyspnea, oliguria, etc.)
    Right ventricle (RV)-related shock: high jugular venous pulse (JVP) and clear lungs
    Left ventricle-related shock: high JVP with pulmonary edema

  - Physical exam:
    Cold, mottled skin
    Faint and rapid peripheral pulses
    Jugular venous distension
    Rales
    Distant heart sounds
    Additional heart sounds (S3 or S4)
  - Diagnostics:
    Urgent echo to evaluate the cause of CS and to rule out tamponade and other mechanical complications of MI
    Pulmonary artery catheter (Swan-Ganz): diagnose and guide the shock therapy (monitor the pulmonary capillary pressure and the cardiac output)
- Management:
  - Early revascularization has survival benefit in patients with cardiogenic shock [24].
  - 2013 ACCF/AHA guidelines [4]:
    Emergent revascularization with either PCI or CABG is preferred (Class I).
    If patient is not a candidate for PCI or CABG, fibrinolytic therapy should be used if not contraindicated (Class I).
    Medical support with pressors and inotropes should be guided by hemodynamic monitoring and individualized as per clinical scenario.
    The use of mechanical hemodynamic support, such as intra-aortic balloon pump (IABP), may be helpful in patients who are unstable despite medical therapy (Class IIa). Alternative ventricular devices may also be considered (Class IIb).
  - 2016 ESC guidelines [25]:
    In contrast, the use of IABP as a routine management is not recommended (Class III).
    Volume support with bolus of normal saline or lactated Ringer's is first-line treatment, if patient has no signs of fluid overload (Class I).
    Norepinephrine is the preferred vasopressor over dopamine, in the setting of persistent hypotension (Class IIb).
    Dobutamine to augment cardiac output (Class IIb).
  - Medical therapy:
    Vasodilators (e.g., nitroprusside and nitroglycerin) have limited roles in cardiogenic shock due to their hypotensive effect.
    Vasopressors to maintain arterial pressure (mean arterial pressure 60–65 mmHg) for adequate end organ perfusion:
    Norepinephrine (0.2–3 μg/kg/min)
      First-line agent [26]
      Potent alpha and mild beta 1 agonist

Dopamine (3–20 mcg/kg/min)
- At low dose, acts on dopaminergic receptors producing renal vasodilation
- At higher dose, works on beta 1, dopaminergic, and alpha receptors
- High life ~2 min

Epinephrine (0.05–1 mcg/kg/min)
- Nonspecific adrenergic agonist
- Potent inotrope and chronotrope
- Associated with a higher rate of lactic acidosis, tachycardia, and arrhythmia [27]

Phenylephrine (0.5–15 mcg/kg/min)
- Alpha-adrenergic agonist
- Generally avoided in cardiogenic shock, since it increases afterload without augmenting cardiac contractility [27, 28]

Inotropes for severe LV dysfunction (low cardiac output):

Dobutamine (2.5–40 mcg/kg/min)
- Strong beta 1 and weaker beta 2 and alpha 1 agonist
- Has vasodilator and inotropic effect
- Half-life ~2 min

Milrinone (50 mcg/kg bolus followed by 0.375–0.75 mcg/kg/min infusion)
- Phosphodiesterase inhibitor
- Direct vasodilator and positive inotrope (weak chronotrope)
- Marked hypotensive effect
- Long half-life ~2.5 h
- Dose reduction by 50% in patients with renal failure
- Avoid when systolic pressure <90 mmHg

– Mechanical circulatory support (MCS) [23, 29]:

Early intervention with MCS may be considered in refractory shock.

Percutaneous circulatory assist devices, such as the Impella (Abiomed) and TandemHeart, are superior to medical therapy alone.

Consider venoarterial extracorporeal membrane oxygenation (ECMO) as preferred temporary MCS, for patients with defective gas exchange who will not rapidly improve with another MCS device.

Used as a bridge for heart transplant [30].

– The 2017 CULPRIT-SHOCK trial [31] showed that in patients with acute MI and cardiogenic shock showing multivessel disease on a cardiac angiogram, percutaneous coronary intervention to culprit lesion alone was superior to multivessel intervention.

This is in contrast to the current guideline [32], which recommends multivessel revascularization, if there is no contraindication (Class IIb).

In an older study from 2004, multivessel PCI in the setting of acute MI results in higher complication rate [33].

Bradycardia (Fig. 23.2)

1. Defined as heart rate < 60 bpm with symptoms (e.g., dyspnea, altered mental status, chest discomfort, etc.)
2. Evaluate for potential causes:

   (a) Iatrogenic
   - Medications (e.g., overdose of AV nodal block agents, digoxin toxicity, opioid overdose, etc.)
     - Digoxin/digitalis (cardiac glycoside).
     - AV node conduction suppression.
     - Increase of vagal tone.
     - Metabolized by the liver.
     - Half-life ~1–5 days.
     - Not dialyzable.
     - Toxicity associated with level > 2 ng/mL; however symptoms may occur at lower levels.

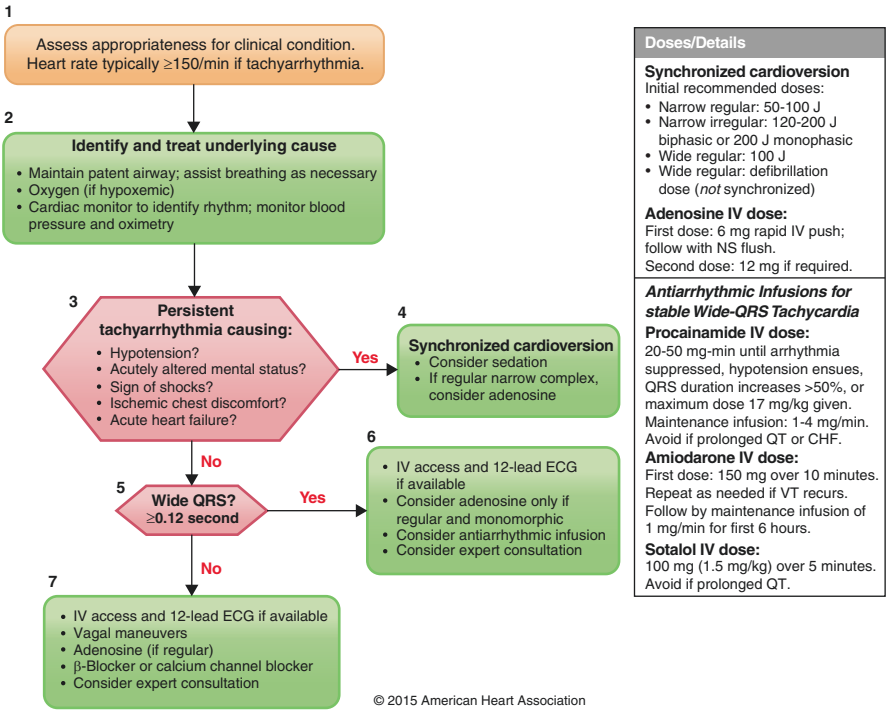


Fig. 23.2 ACLS treatment of bradycardia. Reprinted with permission from [34]

  - Variable symptoms: nausea, blurry vision, palpitations, altered mental status, GI distress, syncope.
  - EKG can show sagging ST depression and T wave (digoxin effect). It just indicates that patient is on digoxin and is not a marker of digoxin toxicity.
  - Multitude of ECG changes, such as bradyarrhythmias, AV blocks (first, second, or third), premature ventricular contractions, ventricular tachycardia (bidirectional), slow atrial fibrillation/flutter, paroxysmal atrial tachycardia with AV block, bidirectional ventricular tachycardia, etc.
- Management of digoxin toxicity:
  - Discontinuation of digoxin, correction of electrolyte imbalance.
  - Digoxin digoxin-specific antibody fragments are used to treat significant dysrhythmia from digitalis toxicity. The decision to use digoxin-specific antibody fragments is not dependent on the serum digoxin concentration.

(b) Electrolyte abnormalities:
- Hyperkalemia:
  - Hyperkalemia is defined as a potassium level >5.5 mEq/L.
  - Moderate hyperkalemia is a serum potassium >6.0 mEq/L.
  - Severe hyperkalemia is a serum potassium >7.0 mEq/L.
  - EKG changes: peaked T waves, widening and flattening of P wave, PR prolongation, QRS prolongation. With higher potassium level (>8.0 mEq/L), the progressively widened QRS eventually merges with the T wave, forming a sine wave pattern. Ventricular fibrillation or asystole follows.
- Treatment:
  - IV calcium.
  - IV insulin with glucose.
  - Consider beta-adrenergic agonist therapy (e.g., nebulized albuterol).
  - Therapy to remove excess potassium (diuretics, kayexalate, dialysis).

(c) Heart blocks:
- First-degree AV block = prolonged PR interval (>0.2 s)
  - Usually asymptomatic and does not cause hemodynamic instability
  - If isolated, does not require any specific treatment
- Second-degree AV block:
  - Mobitz I (Wenckebach) = progressive PR interval prolongation followed by non-conducted P wave
    Rarely causes hemodynamic instability.
    Low risk of progressing to complete heart block (provided QRS is narrow).

If asymptomatic, no specific treatment required. Decrease or avoid use of AV node-blocking drugs.

If symptomatic, there is usually improvement after addressing the underlying issue, and pacemaker may be indicated as well.

- Mobitz II = non-conducted P wave without progressive prolongation of PR interval

More likely to cause symptoms (most often syncope) and hemodynamic instability, which may occur spontaneously.

High risk of progression to complete heart block or sudden cardiac death.

Emergent need for pacing may be indicated if symptomatic and/or hemodynamically unstable.

Avoid drugs that can cause bradycardia, keep electrolyte levels within normal, and evaluate/treat any underlying disorder.

- Third-degree AV block = AV dissociation
  - Often, not always, accompanied by hemodynamic instability and/or symptoms, including syncope, altered mental status, hypotension, and sudden cardiac death.
  - Usually requires temporary pacing (particularly in patients with wide complex escape) (until permanent pacemaker can be placed or underlying condition is addressed).
  - Isoproterenol may be attempted to accelerate a ventricular escape rhythm, however with a low probability for efficacy.
  - In certain situations, a dopamine infusion may be a temporary alternative to improve the heart rate.
  - Evaluation and treatment of any underlying disorder are crucial (e.g., dialysis for hyperkalemia, antibiotics for Lyme disease, coronary reperfusion for MI).

Cardiac Arrest (Figs. 23.3, 23.4, and 23.5) [34, 35]

Ventricular fibrillation/pulseless ventricular tachycardia

- Sudden loss of mechanical activity in the heart
- May be multifactorial in etiology (cardiovascular, neurological, inflammatory, trauma, metabolic, etc.)
- Basic life support (BLS) and advanced cardiopulmonary life support (ACLS) algorithms

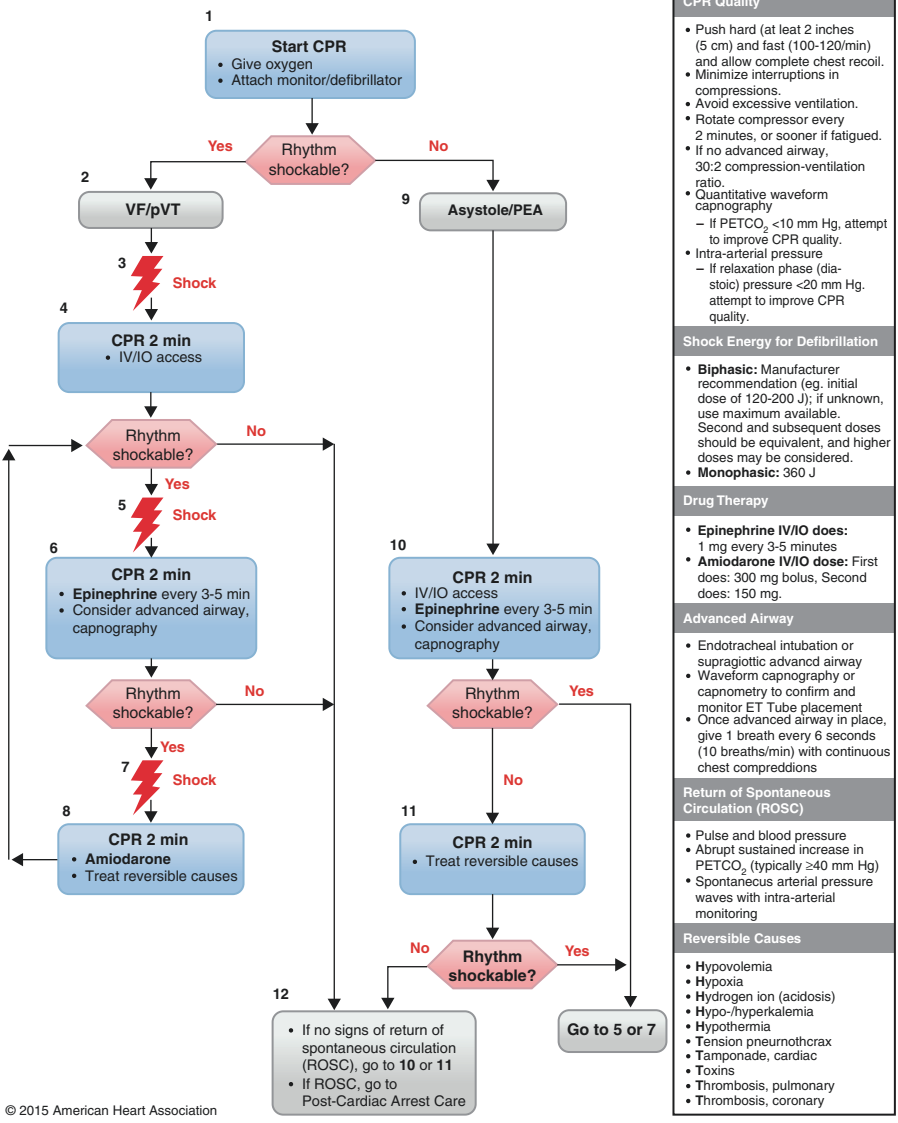
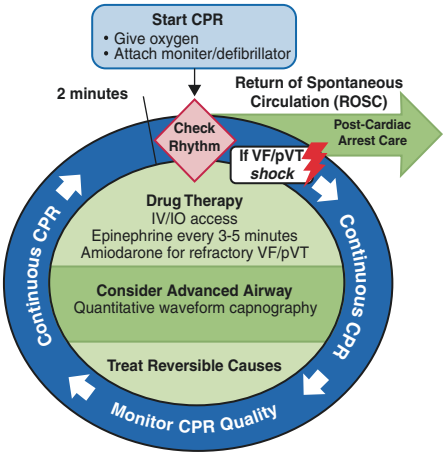


Fig. 23.3 Adult cardiac arrest algorithm—2015 update. Reprinted with permission from [34]



CPR Quality

- Push hard (at least 2 inches (5 cm) and fast (100-120min) and allow complete chest receil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sconer if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If $PETCO_2$ <10 mm Hg. attempt to improve CPR quality
- Intra-artenal pressure.
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality.

Shock Energy for Defibrillation

- **Biphasicc**: Manufacturer recommendation (eg. initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent dose should be equivalent, and higher doses may be considered.
- **Monophasic**: 360 J

Drug Therapy

- **Epinephrine IV/IO dose**: 1 mg every 3-5 minutes
- **Amiodarene IV/IO dose**: First dose: 300 mg bolus, Second dose 150 mg.

Advanced Airway

- Endotracheal intubation or supragiottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt austained increase in $PETCO_2$ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneurnothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Fig. 23.4 Adult cardiac arrest circular algorithm—2015 update. Reprinted with permission from [34]

Pulseless electrical activity

- Organized heart rhythm observed on ECG or telemonitor but without a detectable pulse
  - Check for underlying causes.
  - Mnemonic of 5 Hs and 5 Ts (hypo-/hyper-kalemia, hypothermia, hypoxemia, hypovolemia, hydrogen ion (acidosis) and toxins, tamponade, tension pneumothorax, thrombosis (coronary or pulmonary embolism (PE))).
- See AHA BLS/ACLS algorithms, (Figs. 23.3, 23.4, and 23.5) [34, 35].

Asystole

- Absence of any myocardial electrical activity.
- See AHA BLS/ACLS algorithms, (Figs. 23.3, 23.4, and 23.5) [34, 35].

Cardiac tamponade—please refer to Chap. 12 for more in-depth discussion.

Hypertensive emergency—please refer to Chap. 15 for more in-depth discussion.

Symptomatic tachyarrhythmia—please refer to Chaps. 17–19 and 21 for more in-depth discussion.

Aortic dissection—please refer to Chap. 22 for more in-depth discussion.

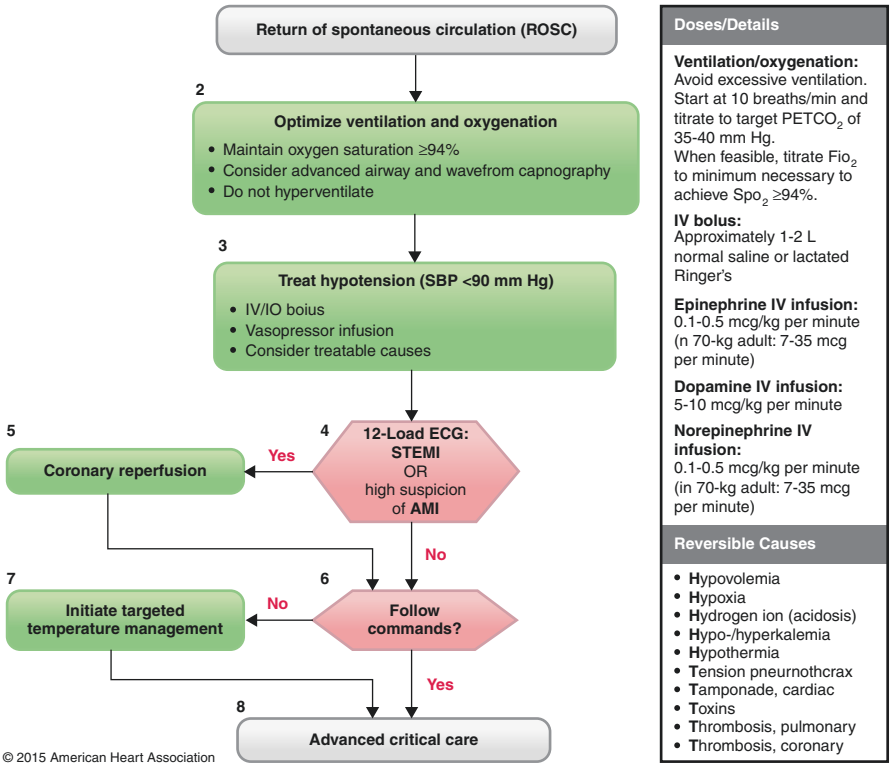


Fig. 23.5 Adult Immediate post-cardiac arrest care algorithm—2015 update. Reprinted with permission from [35]

References

1. French JK, Hellkamp AS, Armstrong PW, Cohen E, Kleiman NS, O'Connor CM, Holmes DR, Hochman JS, Granger CB, Mahaffey KW. Mechanical complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from APEX-AMI). Am J Cardiol. 2010;105:59–63.
2. Anderson JL, Morrow DA. Acute myocardial infarction. N Engl J Med. 2017;376:2053–64.
3. Bloch Thomsen PE, Jons C, Raatikainen MJP, et al. Long-term recording of cardiac arrhythmias with an implantable cardiac monitor in patients with reduced ejection fraction after acute myocardial infarction: the cardiac arrhythmias and risk stratification after acute myocardial infarction (CARISMA) study. Circulation. 2010;122:1258–64.
4. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice guidelines: developed in collaboration with the American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions. Catheter Cardiovasc Interv. 2013;82:E1–27.
5. Wolfe CL, Nibley C, Bhandari A, Chatterjee K, Scheinman M. Polymorphous ventricular tachycardia associated with acute myocardial infarction. Circulation. 1991;84:1543–51.

6. Echt DS, Liebson PR, Mitchell LB, Peters RW, Obias-Manno D, Barker AH, Arensberg D, Baker A, Friedman L, Greene HL. Mortality and morbidity in patients receiving encainide, flecainide, or placebo. The cardiac arrhythmia suppression trial. N Engl J Med. 1991;324:781–8.
7. Pratt CM, Moye LA. The cardiac arrhythmia suppression trial: casting suppression in a different light. Circulation. 1995;91:245–7.
8. Hohnloser SH, Kuck KH, Dorian P, Roberts RS, Hampton JR, Hatala R, Fain E, Gent M, Connolly SJ, Investigators DINAMIT. Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. N Engl J Med. 2004;351:2481–8.
9. Steinbeck G, Andresen D, Seidl K, et al. Defibrillator implantation early after myocardial infarction. N Engl J Med. 2009;361:1427–36.
10. Feldman AM, Klein H, Tchou P, et al. Use of a wearable defibrillator in terminating tachyarrhythmias in patients at high risk for sudden death: results of the WEARIT/BIROAD. Pacing Clin Electrophysiol. 2004;27:4–9.
11. Epstein AE, Abraham WT, Bianco NR, Kern KB, Mirro M, Rao SV, Rhee EK, Solomon SD, Szymkiewicz SJ. Wearable cardioverter-defibrillator use in patients perceived to be at high risk early post-myocardial infarction. J Am Coll Cardiol. 2013;62:2000–7.
12. Piccini JP, Al-Khatib SM, Hellkamp AS, Anstrom KJ, Poole JE, Mark DB, Lee KL, Bardy GH. Mortality benefits from implantable cardioverter-defibrillator therapy are not restricted to patients with remote myocardial infarction: an analysis from the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT). Heart Rhythm. 2011;8:393–400.
13. Klein HU, Goldenberg I, Moss AJ. Risk stratification for implantable cardioverter defibrillator therapy: the role of the wearable cardioverter-defibrillator. Eur Heart J. 2013;34:2230–42.
14. Berman J, Haffajee CI, Alpert JS. Therapy of symptomatic pericarditis after myocardial infarction: retrospective and prospective studies of aspirin, indomethacin, prednisone, and spontaneous resolution. Am Heart J. 1981;101:750–3.
15. Kutty RS, Jones N, Moorjani N. Mechanical complications of acute myocardial infarction. Cardiol Clin. 2013;31:519–31, vii–viii.
16. Birnbaum Y, Chamoun AJ, Anzuini A, Lick SD, Ahmad M, Uretsky BF. Ventricular free wall rupture following acute myocardial infarction. Coron Artery Dis. 2003;14:463–70.
17. Hochman JS, Buller CE, Sleeper LA, Boland J, Dzavik V, Sanborn TA, Godfrey E, White HD, Lim J, LeJemtel T. Cardiogenic shock complicating acute myocardial infarction—etiologies, management and outcome: a report from the SHOCK Trial Registry. Should we emergently revascularize occluded coronaries for cardiogenic shocK? J Am Coll Cardiol. 2000;36:1063–70.
18. Lavie CJ, Gersh BJ. Mechanical and electrical complications of acute myocardial infarction. Mayo Clin Proc. 1990;65:709–30.
19. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. J Am Coll Cardiol. 1998;32:557–61.
20. Nihoyannopoulos P, Smith GC, Maseri A, Foale RA. The natural history of left ventricular thrombus in myocardial infarction: a rationale in support of masterly inactivity. J Am Coll Cardiol. 1989;14:903–11.
21. Werdan K, Gielen S, Ebelt H, Hochman JS. Mechanical circulatory support in cardiogenic shock. Eur Heart J. 2014;35:156–67.
22. Reynolds HR, Hochman JS. Cardiogenic shock: current concepts and improving outcomes. Circulation. 2008;117:686–97.
23. van Diepen S, Katz JN, Albert NM, et al. Contemporary management of cardiogenic shock: a scientific statement from the American Heart Association. Circulation. 2017;136:e232–68.
24. Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. SHOCK investigators. Should we emergently revascularize occluded coronaries for cardiogenic shock. N Engl J Med. 1999;341:625–34.
25. Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure: the task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) developed with

the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2016;37:2129–200.
26. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. J Am Coll Cardiol. 2004;44:E1–E211.
27. Levy B, Perez P, Perny J, Thivilier C, Gerard A. Comparison of norepinephrine-dobutamine to epinephrine for hemodynamics, lactate metabolism, and organ function variables in cardiogenic shock. A prospective, randomized pilot study. Crit Care Med. 2011;39:450–5.
28. Klein T, Ramani GV. Assessment and management of cardiogenic shock in the emergency department. Cardiol Clin. 2012;30:651–64.
29. Rihal CS, Naidu SS, Givertz MM, et al. 2015 SCAI/ACC/HFSA/STS clinical expert consensus statement on the use of percutaneous mechanical circulatory support devices in cardiovascular care: endorsed by the American Heart Association, the Cardiological Society of India, and Sociedad Latino Americana de Cardiologia Intervencion; Affirmation of Value by the Canadian Association of Interventional Cardiology-Association Canadienne de Cardiologie d'intervention. J Am Coll Cardiol. 2015;65:e7–e26.
30. Hoefer D, Ruttmann E, Poelzl G, Kilo J, Hoermann C, Margreiter R, Laufer G, Antretter H. Outcome evaluation of the bridge to bridge concept in patients with cardiogenic shock. Ann Thorac Surg. 2006;82:28–33.
31. Thiele H, Akin I, Sandri M, et al. PCI strategies in patients with acute myocardial infarction and cardiogenic shock. N Engl J Med. 2017. https://doi.org/10.1056/NEJMoa1710261.
32. Levine GN, Bates ER, Blankenship JC, et al. 2015 ACC/AHA/SCAI focused update on primary percutaneous coronary intervention for patients with st-elevation myocardial infarction: an update of the 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention and the 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction. J Am Coll Cardiol. 2016;67:1235–50.
33. Corpus RA, House JA, Marso SP, et al. Multivessel percutaneous coronary intervention in patients with multivessel disease and acute myocardial infarction. Am Heart J. 2004;148:493–500.
34. American Heart Association. Web-based Integrated Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care—Part 7: Adult Advanced Cardiovascular Life Support. ECCguidelines.heart.org
35. American Heart Association. Web-based Integrated Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care—Part 8: Post-Cardiac Arrest Care. ECCguidelines.heart.org.