



Review

Dysrhythmias and heart failure complicating acute myocardial infarction: An emergency medicine review

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ABSTRACT

Introduction: Patients with acute myocardial infarction (AMI) may suffer several complications after the acute event, including dysrhythmias and heart failure (HF). These complications place patients at risk for morbidity and mortality.

Objective: This narrative review evaluates literature and guideline recommendations relevant to the acute emergency department (ED) management of AMI complicated by dysrhythmia or HF, with a focus on evidence-based considerations for ED interventions.

Discussion: Limited evidence exists for ED management of dysrhythmias in AMI due to relatively low prevalence and frequent exclusion of patients with active cardiac ischemia from clinical studies. Management decisions for bradycardia in the setting of AMI are determined by location of infarction, timing of the dysrhythmia, rhythm assessment, and hemodynamic status of the patient. Atrial fibrillation is common in the setting of AMI, and caution is warranted in acute rate control for rapid ventricular rate given the possibility of compensation for decreased ventricular function. Regular wide complex tachycardia in the setting of AMI should be managed as ventricular tachycardia with electrocardioversion in the majority of cases. Management directed towards HF from left ventricular dysfunction in AMI consists of noninvasive positive pressure ventilation, nitroglycerin therapy, and early cardiac catheterization. Norepinephrine is the first line vasopressor for patients with cardiogenic shock and hypoperfusion on clinical examination. Early involvement of a multi-disciplinary team is recommended when caring for patients in cardiogenic shock.

Conclusions: This review discusses considerations of ED management of dysrhythmias and HF associated with AMI.

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1. Introduction

Early complications associated with acute myocardial infarction (AMI) have decreased in the era of reperfusion therapy but remain a source of morbidity and mortality among emergency department (ED) patients. Among 10 million annual ED visits for chest pain, 625,000 patients are diagnosed with acute coronary syndrome (ACS) [1]. Approximately 30% of these patients have ST elevation myocardial infarction (STEMI). The prevalence of STEMI from 1998 to 2008 decreased from 133 to 50 cases per 100,000 patient admissions [2]. The risk of dysrhythmia from AMI varies by infarct location. In a multinational registry of patients with ACS, 4.6% developed cardiogenic shock [3]. Historically, ventricular dysrhythmias were the leading cause of death in AMI, but

cardiogenic shock is the leading cause of death among patients hospitalized for AMI in the modern era [4].

Patients with AMI complicated by dysrhythmia or heart failure represent a subset of patients at high risk for morbidity and mortality. Attentive supportive care addressing volume optimization, electrolyte replacement (especially for hypokalemia and hypomagnesemia), and avoidance of hypoxia and hyperoxia are important components of management for AMI complications [5]. Early cardiology consultation with urgent or emergent coronary angiography is indicated for the majority of these patients.

Application of evidence-based medicine is particularly challenging for the acute management of this patient population given the low prevalence of these conditions and frequent exclusion of patients with active coronary ischemia from clinical trials. The objective of this narrative review is to discuss the available evidence supporting treatment decisions for patients with AMI or recent MI presenting to the ED with dysrhythmias, heart failure (HF) with preserved blood pressure, or cardiogenic

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shock. Specifically, the review aims to discuss specific treatment considerations for these conditions when AMI is present.

2. Methods

This narrative review evaluates literature and guideline recommendations relevant to the ED management of AMI complicated by dysrhythmia or HF, with a focus on evidence-based considerations for ED interventions; however, this review will not cover cardiac arrest, general management of acute coronary syndrome (ACS), electrocardiogram (ECG) interpretation and rhythm recognition, and treatment considerations beyond acute stabilization in the ED. Authors searched PubMed and Google Scholar using a variety of keywords, including myocardial infarction, acute coronary syndrome, electrical complications, atrioventricular block, bradycardia, atrial fibrillation, ventricular tachycardia, cardiogenic shock, and acute heart failure for production of this narrative review. The literature search was restricted to studies published in English. Authors evaluated case reports and series, retrospective and prospective studies, systematic reviews and meta-analyses, and other narrative reviews. Authors also reviewed guidelines and supporting citations for emergency management of acute complications of AMI related to electrical complications, heart failure, or cardiogenic shock. Authors reviewed relevant articles and decided which studies to include for the review by consensus, with inclusion based on relevance to emergent care of electrical complications and heart failure in the setting of AMI. A total of 91 resources were selected for inclusion in this review. This is a narrative review, so no pooling of study data was performed.

3. Discussion

3.1. Perfusion of the cardiac conduction system

An understanding of coronary perfusion and the cardiac conduction system can assist in predicting the occurrence and prognosis of different dysrhythmias based on infarct location. Table 1 displays the most common sources of perfusion for the cardiac conduction system in terms of the right coronary artery (RCA), posterior descending artery (PDA), left circumflex artery (LCX), and left anterior descending artery (LAD) [6,7]. Fig. 1 depicts the cardiac conduction system. The RCA is the most common culprit artery in inferior AMI (80% of cases), followed by LCX lesions [6]. The LAD is the most common culprit artery in anterior MI, and the proximal LCX is the most common in lateral MI [6,8].

3.2. Bradydysrhythmias

Bradydysrhythmias occur in 25–30% of patients with AMI. Sinus bradycardia (SB) is the most common bradydysrhythmia, but the presence of SB in AMI is not associated with increased mortality [9,10]. Hemodynamic compromise due to bradycardia from AMI is rare, occurring in approximately 1 per 10,000 ED visits. AMI is an infrequent cause of hemodynamically compromising bradycardia requiring ED care, estimated at 14% of cases in one European ED registry [11]. Third-degree atrioventricular block (AVB) is the most common rhythm among patients with unstable bradycardia associated with AMI [9].

Two mechanisms are responsible for the majority of bradydysrhythmias in the setting of AMI. First, physiologic changes associated with AMI result in a state of autonomic instability, and increased parasympathetic input to the cardiac conduction system can trigger bradydysrhythmias. Second, AMI may cause ischemia of the cardiac conduction system [9]. Many bradydysrhythmias are due to a combination of these two mechanisms. The type of bradydysrhythmia, timing of onset after AMI, and location of coronary infarct can assist in determining the most probable mechanism for an individual patient.

3.2.1. Atrioventricular block (AVB)

AVB associated with AMI can be divided into early-onset and late-onset AVB. Early-onset AVB develops <6 h of AMI onset and is frequently due to increased parasympathetic input from autonomic imbalance in AMI [12]. Myocardial reperfusion may play a role in triggering this reflex [13]. Early-onset AVB usually presents with very slow ventricular rates which are responsive to atropine [12]. Late-onset AVB presents >6 h after AMI onset and is frequently due to ischemia of the cardiac conduction system. Late-onset AVB tends to present with wide QRS complexes and higher ventricular rates than early-onset AVB. Compared to all comers with AVB, patients with AVB in the setting of AMI tend to be younger and present with chest pain and hypotension [14].

Although first-degree AVB is typically a benign finding, weak evidence suggests that first-degree AVB carries an increased risk of progression to high-grade AVB (HAVB) in the setting of AMI. Among 14 patients identified with HAVB >6 h after AMI, 12 patients had a hemodynamically stable period with first-degree AVB prior to developing HAVB [12]. Cardiac monitoring for patients with first-degree AVB in the setting of AMI is recommended to evaluate for progression to HAVB [15].

AVB was more common in posterior MI (7.3%) than anterior MI (3.0%) in one study of elderly patients with AMI [16]. HAVB consists of type II second-degree AVB and third-degree AVB. The vast majority of HAVB in AMI is third-degree AVB. Among patients with STEMI in a recent prospective registry, 2.2% of patients had HAVB at the time of admission [17]. This represents a decrease from historical estimates of 2.7–14% prevalence of HAVB among patients admitted with STEMI [17]. HAVB is slightly less common when including all patients with ACS, with 1.3% prevalence on presentation [18]. Risk factors for developing HAVB among STEMI patients include RCA culprit lesion lesions, age > 65 years, hypertension, diabetes, and female gender [19]. In one study of patients with STEMI, in-hospital mortality was greater in patients with HAVB (29% versus 18% in patients without HAVB), but HAVB was not independently associated with mortality on multivariable analysis [17].

Mortality and risk of hemodynamically instability are greatly affected by the location of MI, with inferior MI having a more favorable prognosis than anterior MI. HAVB in the setting of inferior MI is usually due to ischemia proximal to the bundle of His [20]. The escape rhythm tends to have a narrow QRS, and many patients tolerate this rhythm without hemodynamic compromise [6]. HAVB from anterior MI is usually due to a larger infarct causing ischemia to the trifascicular conduction system distal to the AV node [21]. The escape rhythm tends to be slow with a wide QRS and has a greater risk of hemodynamic compromise than the escape rhythm in inferior MI [6,9].

3.2.2. Management

Atropine increases automaticity of the SA and AV nodes by inhibiting parasympathetic input. Indications for atropine include sinus bradycardia with hypoperfusion, symptomatic second-degree AVB, or third-degree AVB associated with a narrow QRS complex (Class IIa, LOE B) [9,22]. In a retrospective review of patients with hemodynamically unstable bradycardia (n = 86) or AVB (n = 35) who received atropine in the prehospital setting, 47.3% of patients demonstrated improvement in heart rate and blood pressure following atropine administration. Approximately half of these responses were transient, while the remainder

Table 1
Perfusion of the cardiac conduction system.

Conduction location	Right coronary artery	Left circumflex artery	Left anterior descending artery
Sinoatrial node	60%	40%	0%
Atrioventricular node	90%	10%	0%
Right bundle branch block ^a	33%	13%	52%
Left bundle branch block ^a	17%	0%	43%

^a The location of culprit lesions in patients in GUSTO I and TAMI trials with new onset right and left bundle branch block in AMI [7].

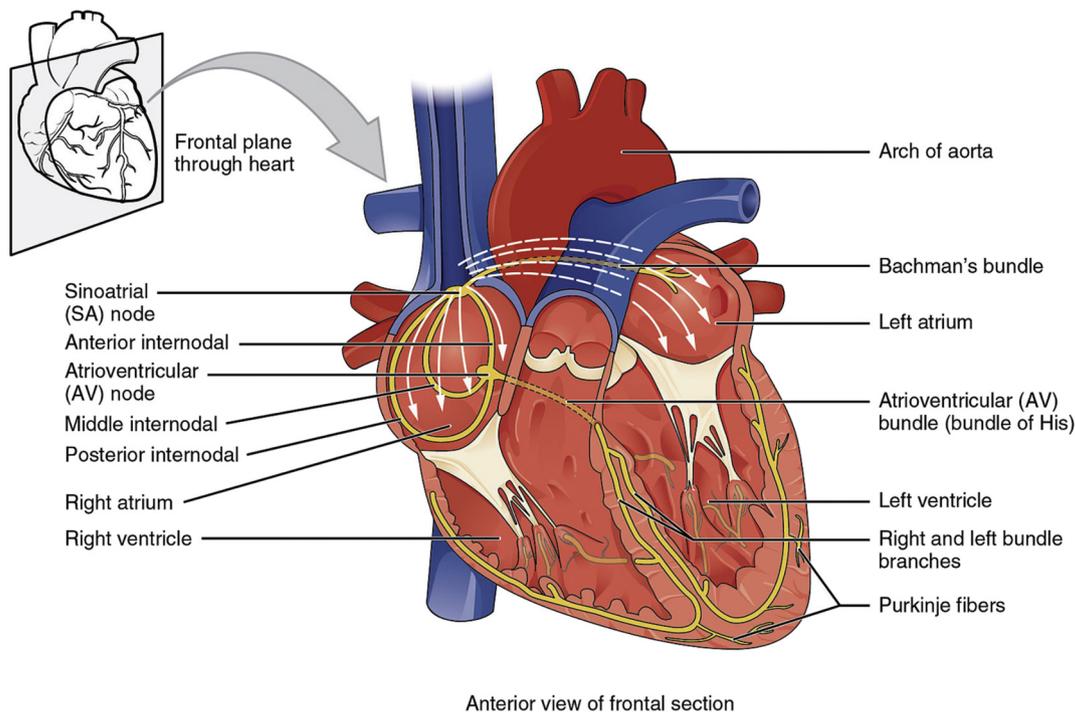


Fig. 1. Cardiac conduction system.

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were sustained throughout the prehospital phase of care. On subgroup analysis, the 34.4% of patients with AMI had a similar response to atropine compared to patients without AMI [23]. Potential adverse effects of atropine include worsening of HAVB, increased risk of dysrhythmia, and progression of infarction. In the same prehospital study, 4 patients (3.1%) developed ventricular dysrhythmias [23]. This risk of dysrhythmia was lower than a prior study of 56 intensive care unit (ICU) patients receiving atropine for SB in which 5% of patients had increased premature ventricular contractions and 5% had ventricular dysrhythmias [24]. Atropine is typically not effective in patients with HAVB, warranting consideration of other measures.

Epinephrine and dopamine are recommended alternative agents if atropine is ineffective (Class IIa, LOE B) [25]. A strategy of epinephrine administration in place of atropine is somewhat supported by an anesthesia algorithm for bradycardia management in the operating room, but no primary literature support is offered for this recommendation [26]. Mortality and adverse events were similar in a feasibility study of 82 patients randomized to dopamine or transcutaneous pacing (TCP) after failure of atropine for unstable bradycardia in the prehospital setting. The use of epinephrine prior to atropine for symptomatic bradycardia in AMI is a deviation from ACLS recommendations with minimal literature supporting this strategy, though it should be considered as a potential first-line therapy in patients with HAVB.

Pacer pads should be placed prophylactically in patients presenting with bradycardia in the setting of AMI. TCP should be initiated as a bridge to transvenous pacing or definitive management in hemodynamically compromising bradycardia unresponsive to atropine or not meeting atropine indications. A recent prospective, observational study in the ED found increased systolic blood pressure and HR with the use of TCP in patients with bradycardia unresponsive to atropine [27]. Although data are limited by small sample size, 2 randomized controlled trials (RCTs) examining TCP for symptomatic bradycardia in the prehospital setting found lower mortality among patients with a pulse on EMS arrival to the scene who received TCP versus those who did not receive TCP [28,29].

The decision to start transvenous pacing in the ED is multifactorial based on the presenting rhythm, hemodynamic status, hospital setting,

and consultant support. While placing TCP pads can be accomplished easily, transvenous pacing is more technically challenging. In a study of 277 ED patients with bradycardia and hemodynamic compromise, 20% of patients required transvenous pacing. The risk of requiring transvenous pacing was doubled among the 40 patients with bradycardia secondary to AMI [11]. The risk of progression to HAVB and development of hemodynamic compromise is partially determined by the location of infarction. Anterior MI with AVB is commonly associated with infranodal ischemia with a greater risk than inferior infarctions for progression to higher grade AVB and hemodynamic compromise. Patients with first-degree or type I second-degree AVB should have cardiac monitoring to evaluate for progression to HAVB. Patients with second-degree type 2 AVB are at significant risk of progression to third-degree AVB. The presence of a new bundle branch block increases the risk of requiring transvenous pacing. Patients with third-degree AVB, particularly in the setting of anterior infarction, are likely to develop hemodynamic instability requiring transvenous pacing [30]. In summary, patients with AMI and hemodynamically compromising bradycardia not responsive to atropine should be managed with TCP and/or epinephrine, with consideration of placing a transvenous pacemaker.

3.3. Atrial fibrillation

The reported prevalence of atrial fibrillation (AF) complicating AMI has varied between 2% and 21% over the last 30 years [31]. A recent large retrospective study of patients with ACS reported a 7.6% prevalence of new AF during hospitalization for AMI [32]. Multiple factors likely play a role in the development of AF during AMI [16]. A commonly proposed mechanism is left ventricular dysfunction leading to increased atrial pressures [33]. Other possible contributing factors include ischemia of the atria, metabolic abnormalities, or autonomic instability associated with AMI [16].

Risk factors for developing AF in the setting of AMI include advanced age, male gender, and tachycardia or heart failure on presentation [31,32]. Cardiogenic shock on presentation has the strongest association of any risk factor with an odds ratio (OR) of 1.58 [31]. AF in the setting of

ACS is associated with increased risk of mortality, repeat MI, and ischemic stroke [32].

3.3.1. Management

Before initiating interventions for AF in the setting of AMI, an assessment is needed as to whether AF is a primary cause or significant contributor to the patient's hemodynamic condition [34]. Tachycardia may be a compensatory mechanism to AMI, and adverse events are common following rate control interventions. Among 15 patients receiving rate control for AF with rapid ventricular rate (RVR) in the setting of ACS, 27% of patients developed significant hypotension in one study [35].

Synchronized cardioversion is indicated to treat AF in the setting of AMI if any of the following conditions are present: 1) hemodynamic instability, 2) uncontrolled RVR despite pharmacotherapy, or 3) evidence of ongoing cardiac ischemia despite pharmacotherapy [36]. A recent expert opinion manuscript described stability as a continuum, and the presence of AMI should decrease the threshold to treat a patient with AF as hemodynamically unstable [37].

In cases where cardioversion is unsuccessful or not performed, pharmacotherapy recommendations for acute rate control of AF depend on clinical evidence of heart failure or hemodynamic instability. Amiodarone is recommended in patients presenting with reduced ejection fraction or hemodynamic instability (Class IIb, LOE B) [36]. A retrospective study of 38 ICU patients with refractory atrial dysrhythmias found decreased heart rate and increased blood pressure in patients receiving amiodarone. The majority of patients receiving diltiazem, esmolol, or digoxin had decreases in systolic blood pressure (SBP) without slowing the HR [38]. The choice of rate control agent in hemodynamically stable patients is less clear. The American Heart Association/American College of Cardiology (AHA/ACC) and European Society of Cardiology (ESC) guidelines recommend intravenous beta blockers (Class I, LOE C) over non-dihydropyridine calcium channel blockers (Class IIb, LOE C) for acute rate control of hemodynamically stable patients in the setting of ACS [33,36]. This recommendation is largely based on expert opinion, as studies for AF with RVR usually do not include patients with AMI [39–41]. The same guidelines recommend avoiding non-dihydropyridine calcium channel blockers if any evidence of heart failure or hemodynamic instability is present (Class IIb, LOE C) [36].

3.4. Accelerated idioventricular rhythm

Accelerated idioventricular rhythm (AIVR) is characterized by a ventricular rhythm with a heart rate of 60–120 beats per minute [42]. A rate >120 beats per minute suggests ventricular tachycardia (VT). AIVR is seen most commonly following reperfusion with PCI or thrombolytic therapy, but it may rarely be seen in the setting of AMI. The presence of AIVR does not affect prognosis. AIVR is a stable escape rhythm, and antidysrhythmic treatment is not recommended [43].

3.5. Ventricular tachycardia

VT is a ventricular rhythm with a QRS duration >120 milliseconds, a rate of at least 120 beats per minute, and loss of association between atrial and ventricular depolarization [44]. Sustained VT is estimated to occur following 1% of AMI [45]. This decrease from historical estimates of 3–5% is presumably secondary to improved cardiac care to reduce infarct size in AMI [45]. VT usually occurs remotely from the MI event as a result of reentry pathways at the site of myocardial scar [45]. In the setting of AMI, VT is usually due to abnormal automaticity causing focal activity at the border of the infarction. In early ischemia, mild hyperkalemia increases cardiac myocyte excitability. As ischemia progresses, worsening hyperkalemia results in slowed conduction and decreased excitability. This mismatch in cells bordering the infarct may

result in depolarization-induced automaticity, producing focal activity which can promote VT [46].

Risk factors for VT include large infarct size and reduced ejection fraction [45]. Multiple algorithms exist for distinguishing ventricular tachycardia from other causes of regular wide complex tachycardia on ECG [47]. The occurrence of regular wide complex tachycardia during or shortly after AMI strongly suggests VT [48].

3.5.1. Management

Immediate cardioversion is recommended for VT with a pulse in the setting of ischemic chest pain or hemodynamic instability [49,50]. Stable monomorphic VT is typically defined as the prevalence of VT in the setting of minimal symptoms. The threshold to categorize a patient with AMI as unstable VT should be low, with consideration of immediate cardioversion [44]. For patients with a pulse and persistent VT refractory to cardioversion, intravenous amiodarone is recommended (Class I, LOE A) along with further attempts at cardioversion [25]. Among 18 patients (8 with AMI) receiving amiodarone in the setting of unstable VT, 78% had termination of VT with a protocol involving repetitive shocks and amiodarone boluses [51].

For hemodynamically stable patients with regular wide complex tachycardia, the 2017 AHA guidelines for ventricular dysrhythmias include procainamide (Class IIa, LOE A), amiodarone (Class IIb, LOE B), and sotalol (Class IIb, LOE B) [50]. The PROCAMIO trial found improved termination of VT (67% vs 38%) and fewer major adverse events (9% vs 41%) for procainamide versus amiodarone, respectively [52]. Notably, this trial did not include patients with AMI or severe anginal symptoms [52]. A randomized parallel study comparing procainamide with lidocaine in patients with spontaneous monomorphic VT found procainamide terminated VT in 12 of 15 patients, compared with 3 of 14 patients receiving lidocaine [53]. A retrospective cohort of 97 infusions of amiodarone or procainamide for stable sustained VT in the ED included 9 patients with STEMI or NSTEMI. Among patients receiving procainamide, 19% experienced hypotension, requiring discontinuation of the infusion [54]. In summary, procainamide has recently been shown to terminate stable VT more frequently than amiodarone, but there is little evidence to support procainamide use in the setting of AMI. Most patients with VT in the setting of AMI should undergo cardioversion with administration of repeated shocks and intravenous amiodarone if VT persists. Table 2 summarizes dysrhythmia management in AMI.

3.6. Acute heart failure associated with AMI

Myocardial ischemia was identified as the precipitating factor in 15% of heart failure admissions in the OPTIMIZE-HF trial [55]. In-hospital mortality was independently higher (adjusted OR 1.2) for patients with ACS as the precipitating factor compared to all HF admissions [55]. The degree of HF on admission is commonly characterized by physical examination using the Killip classification [56]. Patients with Killip class I have no evidence of HF on physical examination. Patients with Killip class II and III have preserved SBP of at least 90 mmHg with pulmonary edema involving less than a third (class II) or greater than a third (class III) of the lung fields on physical examination. Patients with Killip class IV have cardiogenic shock, with evidence of pulmonary edema and SBP <90 mmHg [56]. Increasing Killip classification was associated with greater mortality in a study of non-STE ACS patients, which found 30 day mortality of 2.8% in Killip class I, 8.8% in Killip class II, and 14.4% in Killip class III or IV [56]. Risk factors for HF complicating AMI include advanced age, hypertension, diabetes, and prior MI [56,57].

3.6.1. Heart failure with normal or elevated blood pressure

Management directed towards HF in the setting of LV dysfunction from AMI is similar to the ED management of HF not associated with

Table 2
Management considerations for dysrhythmias complicating acute myocardial infarction.

Bradydysrhythmias	<ul style="list-style-type: none"> - Place transcutaneous pacer pads. - Atropine is more likely to be successful in AVB occurring <6 h of AMI onset. - Minimal literature supports the use of epinephrine prior to atropine, except in cases of HAVB or those patients unresponsive to atropine. - Anterior MI with AVB has greater risk of progressing to high grade AVB. - Hemodynamically compromising bradycardia unresponsive to atropine should be treated with cardiac pacing and/or epinephrine. - Transvenous pacing may be required.
Atrial fibrillation	<ul style="list-style-type: none"> - Increased risk of hypotension if acute pharmacologic rate control is required. - Determine whether the rhythm is the primary cause of patient decompensation or a compensatory mechanism. - Synchronized electrocardioversion is recommended for patients with hemodynamic instability and uncontrolled RVR or ischemia despite pharmacotherapy. - Amiodarone may be used in patients with reduced EF or hemodynamic instability if electrocardioversion is unsuccessful or not performed. - Avoid calcium channel blockers if heart failure is present.
Accelerated idioventricular rhythm	<ul style="list-style-type: none"> - Antidysrhythmic medication is not indicated.
Ventricular tachycardia	<ul style="list-style-type: none"> - Assume VT for regular, wide complex tachycardia in the setting of AMI. - Early electrocardioversion with repeat shocks and amiodarone if VT persists is the strategy with greatest literature support.

Abbreviations: AVB - atrioventricular block, AMI - acute myocardial infarction, VT - ventricular tachycardia, RVR - rapid ventricular rate, EF - ejection fraction.

AMI [58]. Initial resuscitation to address pulmonary edema includes noninvasive positive pressure ventilation and nitroglycerin as tolerated by blood pressure [34]. Oxygen saturation should be maintained above 90% [59]. One RCT found fewer intubations and AMI with high dose nitrate and low dose furosemide therapy, compared to high dose furosemide and low dose nitrate therapy [60].

The presence of AMI is not a contraindication to diuretic use in acute HF management. Diuretic therapy is recommended by recent ESC guidelines (Class I, LOE C) for patients with STEMI and evidence of volume overload on clinical examination [5]. Early use of diuretics in HF has recently received a great deal of attention. An observational study showing an association between early furosemide treatment and decreased in-hospital mortality excluded patients with ACS requiring urgent catheterization [61]. As ESC guidelines recommend urgent catheterization for all patients with decompensated HF in the setting of ACS, this study may not apply to the HF subgroup with AMI [59]. In summary, management directed towards HF in AMI should focus on nitroglycerin therapy and noninvasive positive pressure ventilation to address pulmonary edema. An intravenous dose of diuretic equal to the patient's home maintenance dose can be given if clinical assessment suggests volume overload [62].

3.6.2. Cardiogenic shock

Cardiogenic shock (CS) occurs in 4–9% of patients with AMI [63]. In a study of multiple registries, prevalence of CS in AMI in 2005 was 5.7%, decreased from 6.9% in 1995 [64]. Risk factors for CS include advanced age, female gender, prior MI, and diabetes [4,65,66]. The majority of CS is precipitated by STEMI and typically occurs hours to days after initial presentation [63].

The most common etiology of CS in the SHOCK registry was left ventricular failure (78%) [67]. Isolated right ventricular failure and mechanical complications precipitated CS in 3% and 12% of patients, respectively [67]. Anterior MI (55%) and posterior MI (46%) were the most common infarct locations [67].

Patients with AMI complicated by CS can rapidly decompensate due to a positive feedback loop: 1) myocardial ischemia leads to decreased ejection fraction, 2) decreased diastolic blood pressure worsens coronary perfusion pressure, and 3) decreased coronary perfusion worsens coronary ischemia. This loop causes significant challenges in management.

3.6.3. Management

Patients with CS in the setting of AMI require immediate resuscitation and cardiology consultation. Echocardiography is needed to evaluate for mechanical complications [33]. This review will not specifically cover mechanical complications of AMI. Emergent cardiac catheterization is indicated if any of the following conditions persist despite medical management: ST segment deviation on ECG, anginal symptoms, or end organ hypoperfusion [33].

The decision to initiate vasopressors rests on a balance of benefit and risk. Vasopressors can improve coronary perfusion by increasing diastolic blood pressure. Adverse effects of vasopressors include increased myocardial oxygen consumption and increased risk of dysrhythmias and infarct progression [68].

The choice of vasopressor agent depends on the degree of hypotension, the patient's perfusion status on clinical assessment, emergency provider comfort with use of different agents, and preference of consultants assisting with patient care. A reasonable target blood pressure is a mean arterial pressure (MAP) of at least 65 mmHg [69,70].

Norepinephrine is recommended as the first line vasopressor for CS in the setting of AMI with SBP <70 mmHg [68]. An RCT comparing dopamine and norepinephrine in shock found that dysrhythmias were more common with the use of dopamine (24%) versus norepinephrine (12%) [71]. A subgroup analysis of patients with CS found decreased mortality in patients randomized to norepinephrine compared to dopamine [71]. In a pilot study of 30 patients with CS not precipitated by AMI, 20% of patients treated with epinephrine had dysrhythmias compared to 0% in the norepinephrine-dobutamine arm [72].

Patients with CS and SBP 70–100 mmHg require careful consideration in selecting the appropriate vasopressor or inotrope. Norepinephrine is recommended if signs of shock are present on clinical assessment [68]. This is a change from previous guidelines, which recommended dopamine as the first line agent for this patient population [30]. Norepinephrine is recommended over epinephrine, as epinephrine is associated with increased oxygen consumption, lactate levels, and mortality rates [72,73]. Dobutamine is recommended if the patient has adequate perfusion and lack of severe end organ dysfunction by clinical assessment [68].

Dobutamine primarily acts as a beta 1 and beta 2 agonist. The net effect at low doses is increased myocardial contractility and vascular smooth muscle relaxation. This vasodilation may cause a decrease in blood pressure. Adverse effects of dobutamine include increased myocardial oxygen consumption and increased risk of dysrhythmia. Non-sustained VT occurred in 13% of patients during the first 24 h of dobutamine infusion in 1 study [74]. Milrinone is a phosphodiesterase inhibitor, which acts as an inotrope and vasodilator. Parenteral milrinone has a half-life of 2–4 h, so initiation of this medication in the ED can have prolonged clinical effects and is generally avoided [68]. Inotropes have not been shown to improve mortality in cardiogenic shock, but these agents can improve coronary perfusion in the acute shock state until other treatments, such as PCI or mechanical support, are performed [68,75,76].

In cases of CS due to AMI, myocardial revascularization is the only evidenced-based therapy with proven survival benefit [70]. However, mechanical circulatory support is an essential part of the management of CS and is commonly utilized. Non-pharmacologic measures to improve organ perfusion include the use of an intra-aortic balloon pump

(IABP), Impella placement, and veno-arterial extracorporeal membrane oxygenation (ECMO) [77–81]. By decreasing afterload, IABP improves cardiac output, systemic pressures, coronary perfusion pressure, and end-organ perfusion. Impella and TandemHeart devices are percutaneous left ventricular assist devices that provide mechanical circulatory support but do not alter afterload [70]. Hemodynamic optimization should not delay definitive treatment via percutaneous closure device or primary revascularization [82–84].

3.6.4. Right ventricular failure

A minority of patients have CS due primarily to right ventricular (RV) failure, as seen in 2.8% of CS patients in the SHOCK registry [67]. These patients tend to be younger and present with inferior and posterior infarctions [85]. Causes of RV failure during an AMI can generally be divided into 3 main categories: insufficient myocardial contractility, excessive preload, and excessive afterload [86]. Insufficient myocardial contractility is primarily due to local myocardial ischemia or overstretching of the RV free wall. Excessive preload can occur from acute left-to-right shunt in conditions such as a ventricular septal rupture. Excessive afterload is typically due to acute left heart failure [86].

3.6.5. Management

Proper fluid management is critical for successful management of RV failure. The goal is to optimize RV preload and perfusion while avoiding fluid overload. If low intravascular volume is suspected, fluid resuscitation should be instituted as quickly as possible. In the setting of clear intravascular depletion, clinicians should use low-volume boluses, such as 250 mL of a crystalloid solution. Frequent serial assessments of end-organ perfusion, including blood pressure, urine output, capillary refill, and serum lactate, are needed to reduce the risk of iatrogenic volume overload [87]. Care is needed to avoid volume overloading the RV. High RV pressures can impair left ventricular function due to bowing of the interventricular septum into the left ventricle [85]. Additionally, greater RV dilation from volume overload can increase RV ischemia and free wall tension. Diuresis can improve cardiac output in the settings of RV volume overload, but treatments to reduce preload should be instituted with caution, as preload reduction beyond optimal levels will decrease cardiac output [86].

Systemic hypotension should be avoided. Cardiac output in this patient population is difficult to augment as increasing RV afterload worsens right heart hemodynamics. Correction of hypercapnia, acidemia, and alveolar hypoxia can reduce RV afterload [86]. Vasopressors can improve coronary perfusion pressure and reduce right heart ischemia [87]. Norepinephrine maintains coronary perfusion while slightly augmenting inotropy, making it an ideal first line agent [87,88]. Vasopressin may decrease pulmonary vascular resistance [89], whereas phenylephrine generally increases pulmonary vascular resistance [90]. Dobutamine increases risk of tachycardia and decreases systemic vascular resistance. Both of these effects are detrimental in RV failure, so dobutamine should be avoided in this patient population [88,91].

Electrocardioversion of new-onset dysrhythmias should be strongly considered in RV failure [87,91]. Adequate filling time and atrial contractions are needed to maintain cardiac output, so dysrhythmias are not well tolerated [87]. Cardiodepressants such as beta blockers and calcium channel blockers impair RV function and should usually be avoided.

Close monitoring for hemodynamic decompensation is needed if nitrates or mechanical ventilation are used. Increased intrathoracic pressure from positive pressure ventilation can reduce RV preload and increase RV afterload. Patients with RV failure requiring intubation are at high risk for peri-intubation hemodynamic collapse due to impaired venous return and decreased systemic vascular resistance from administration of sedatives. Emergency clinicians should be prepared to address profound hypotension if the decision is made to intubate a patient with RV failure [87]. Low tidal volumes and plateau pressures

are preferable mechanical ventilation settings, and positive end expiratory pressure should be minimized. Permissive hypercarbia and hypoxia should be avoided, as they may contribute to pulmonary vasoconstriction.

Patients may require mechanical circulatory support in the setting of refractory RV failure. Currently available devices include an in situ centrifugal pump (CentriMag), an axial catheter-based pump (Impella RP), and a catheter with an extracorporeal centrifugal pump (PROTEK Duo) [70]. Venoarterial ECMO remains another option as a bridge to myocardial recovery, durable mechanical circulatory support, heart transplant, or decision for palliative therapy [70]. Table 3 summarizes management of heart failure complicating AMI.

4. Conclusions

Acute complications in the post-AMI period include dysrhythmias and heart failure. Dysrhythmias include sinus bradycardia, AVB, AF, VT, and several others. The benefits of atropine outweigh the potential adverse effects for the majority of patients with AMI presenting with symptomatic bradycardia. The decision to initiate or prepare for transvenous cardiac pacing should incorporate the presenting rhythm, location of infarction, and hemodynamic status of the patient. The risk of significant hypotension is increased for acute rate control of AF with RVR if AMI is present. Regular wide complex tachycardias in the setting of AMI should be managed as VT, with a low threshold to treat by cardioversion. Patients presenting with HF associated with AMI and no right ventricular involvement should be managed with early noninvasive positive pressure ventilation and nitroglycerin as tolerated by blood pressure. Norepinephrine is the first line vasopressor for patients with cardiogenic shock and end organ hypoperfusion. Early involvement of a multi-disciplinary team is recommended for patients with

Table 3

Management considerations for heart failure complicating acute myocardial infarction.

Heart failure from LV dysfunction	<ul style="list-style-type: none"> - Management of pulmonary edema in AMI is similar to patients without AMI with early non-invasive positive pressure ventilation and nitroglycerin therapy if there is no evidence of right ventricular involvement. - Diuretic therapy is indicated in AMI if volume overload is suspected after volume redistribution.
Cardiogenic shock	<ul style="list-style-type: none"> - Early involvement of multidisciplinary team is needed including cardiology for echocardiography and possible cardiac catheterization, as well as cardiothoracic surgery. - Target a MAP of 65 mmHg in combination with clinical signs of perfusion. - Norepinephrine is the recommended first line vasopressor if SBP is <70 mmHg or there is clinical evidence of hypoperfusion. - Consider inotropic therapy with SBP 70–100 mmHg and no hypoperfusion. Milrinone has longer clinical effects than dobutamine. - Shock associated with AMI requires revascularization. - Patients with refractory shock require mechanical circulatory support.
RV failure	<ul style="list-style-type: none"> - Cardiac output is preload dependent and often improves with IV fluids, but volume overload can also reduce cardiac output. - Avoid hypotension; vasopressors may be required with IV fluids. - Utilize electrocardioversion for hemodynamically unstable dysrhythmias and RV failure. - Patients are at high risk of peri-intubation hemodynamic collapse due to effects of sedatives and positive pressure ventilation. - Mechanical circulatory support may be required for refractory RV failure.

Abbreviations: SBP – systolic blood pressure, mm – millimeters, Hg – Mercury, AMI – acute myocardial infarction, MAP – mean arterial pressure, IV – intravenous, LV – left ventricular, RV – right ventricular, AMI – acute myocardial infarction.

cardiogenic shock. An understanding of these complications is vital in optimizing care of patients in the post-AMI period.

Conflicts of interest

None.

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