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REVIEW

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Arrhythmias After Acute Myocardial Infarction

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The incidence of arrhythmia after myocardial infarction has declined since the introduction of reperfusion techniques. Nevertheless, ischemic arrhythmias are often associated with increased morbidity and mortality particularly in the first 48 hours after hospital admission. This paper presents a comprehensive review of the epidemiology, characteristics, and management of ischemic tachy- and brady-arrhythmias focusing on the period shortly after myocardial infarction (MI) in patients with both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI).

INTRODUCTION

Arrhythmia after myocardial infarction (MI) is a common clinical problem requiring prompt recognition and treatment. The incidence of arrhythmia after MI has declined in the reperfusion era, however when present, may be associated with increased morbidity and mortality. In general, arrhythmias are more common in patients who do not undergo timely reperfusion, particularly those with resulting left ventricular ejection fraction (LVEF) depression. We present a comprehensive review of arrhythmia after MI, including both tachy- and brady-arrhythmias inclusive of recommended therapies and management. While prior ischemia and related myocardial scar can have a significant impact on arrhythmia, our review focuses specifically on the period shortly after MI in patients with both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI).

VENTRICULAR ARRHYTHMIAS

Accelerated Idioventricular Rhythm

Accelerated idioventricular rhythm (AIVR) is a common arrhythmia seen in the peri-infarct period and is generally considered a marker of reperfusion. Up to one third of patients presenting with STEMI will have AIVR

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Abbreviations: MI, myocardial infarction; LVEF, left ventricular ejection fraction; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; AIVR, accelerated idioventricular rhythm; PVCs, premature ventricular contractions; VA, Ventricular arrhythmias; VT, ventricular tachycardia; VF, ventricular fibrillation; NSVT, non-sustained ventricular tachycardia; ACS, acute coronary syndrome; AMI, acute myocardial infarction; NYHA, New York Heart Association; TIMI, thrombolysis in myocardial infarction; ACLS, Adult Advanced Cardiac Life Support; ICD, implantable cardioverter defibrillator; RCA, right coronary artery; DOAC, direct acting oral anticoagulant; AV, Atrioventricular; PCI, percutaneous coronary intervention; BBB, Bundle Branch Block; RBBB, right bundle branch block; LBBB, left bundle branch block; LAD, left anterior descending.

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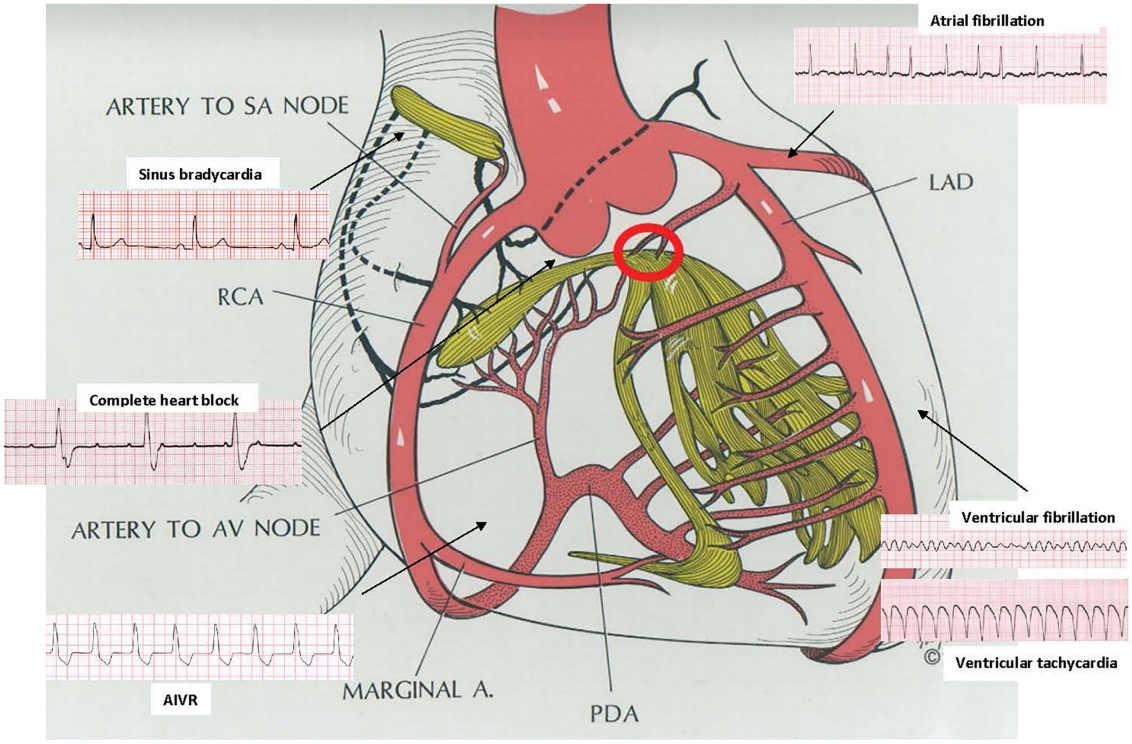


Figure 1. **Anatomy of tachy- and brady-arrhythmias**. (Modified with permission from: Levine, Herbert J. Clinical Cardiovascular Physiology. 1 April 1976.)

[1]. The mechanism is thought to be related to abnormal automaticity in the subendocardial Purkinje fibers and sympathetic stimulatory effects in infarcted tissue undergoing reperfusion (Figure 1) [2]. The rhythm is benign and self-limited. No treatment is recommended as the burden typically decreases in the days following MI.

Premature Ventricular Contractions

In the peri-infarct period, premature ventricular contractions (PVCs) may be a marker of cardiac electrical instability with the potential to degenerate into sustained ventricular arrhythmias (VA). PVCs have long been identified as a risk factor for sudden cardiac death (SCD) following MI [3]. Over thirty years ago, Mukharji and colleagues noted that frequent PVCs, defined as a frequency of greater than 10 PVCs per hour, were associated with significantly higher mortality in a 2-year follow up after MI, particularly in those with reduced ejection fraction [4]. Later, Maggioni and colleagues evaluated data from the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-2) study. These findings echoed prior studies: PVCs were an independent risk factor for total and sudden death in the first 6 months after acute myocardial infarction (AMI), particularly in those with greater than 10 PVCs per hour and those with complex premature ventricular beats [5]. Recognition of higher mortality in patients with PVCs after MI prompted the Cardiac Arrhythmia Suppression Trial (CAST) series [6,7]. Collectively, CAST concluded that use of antiarrhythmic drugs to suppress PVCs increased the risk of death, especially in patients with reduced ejection fraction. Finally, the Canadian Amiodarone Myocardial Infarction Arrhythmia Trial (CAMIAT) randomized patients with a mean of 10 or more PVCs per hour or at least one run of ventricular tachycardia (VT) after MI to treatment with amiodarone or placebo. A clinically important reduction in arrhythmic death and resuscitated ventricular fibrillation (VF) was seen among patients with frequent or repetitive PVCs taking amiodarone for up to 2 years after MI without an associated reduction in all-cause mortality. Taking these findings into consideration, amiodarone is a reasonable anti-arrhythmic choice for patients at significant arrhythmogenic risk after MI, when weighed against the risk of toxicity [8]. The mainstay of treatment of PVCs post-MI remains beta blockers, which blunt the increase in PVC complexity and frequency without the toxicities associated with amiodarone [9].

Non-sustained Ventricular Tachycardia

Non-sustained ventricular tachycardia (NSVT) is

typically defined as at least three consecutive tachycardic ventricular beats self-terminating in less than 30 seconds [10]. The clinical importance of post-MI NSVT remains controversial. Early studies suggesting that NSVT was associated with worse outcomes were generally performed in the pre-reperfusion era and focused on high risk patients including those with an LVEF of <40% [11,12]. Later analyses have not replicated this finding [5,13]. Indeed, Hohnloser and colleagues actually demonstrated a low predictive value of NSVT in the period shortly after MI for subsequent mortality [13].

Timing after MI appears to be the most important differentiator in determining the clinical importance of NSVT. Using data from the Metabolic Efficiency with Ranolazine for Less Ischemia in Non-ST Elevation Acute Coronary Syndrome – Thrombolysis in Myocardial Infarction 36 (MERLIN-TIMI 36) trial, including over 6,500 patients hospitalized with non-ST elevation acute coronary syndrome (ACS), Scirica and colleagues performed arrhythmia analyses for a total of 6,345 patients with the primary outcome of SCD [14]. NSVT occurring within 48 hours of admission for non-ST elevation ACS was not associated with an increase in SCD. However, NSVT episodes lasting 4 to 7 (18.5% vs 2.9%, adjusted HR 2.3, 95% CI 1.5 to 3.7, p<0.001) or greater than 8 beats (6.8% vs 4.3%, adjusted HR 2.8, 95% CI 1.5 to 5.1, p=0.001) in the subsequent year was associated with a significantly greater risk of SCD compared to those without VT. This effect was independent of baseline characteristics and LVEF [14]. These findings likely reflect mechanistic differences between early and late VAs. NSVT during acute ischemia is more likely due to transient automaticity or triggered activity in the region of ischemia or infarction, compared with late NSVT which is more likely due to scar-mediated ventricular re-entry. The mainstay of therapy for NSVT in the period shortly after MI is similar to that for PVCs and includes beta blockers and, when appropriate, the addition of amiodarone or other antiarrhythmic drugs.

Ventricular Tachycardia and Ventricular Fibrillation

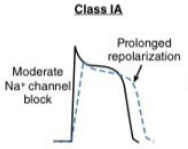
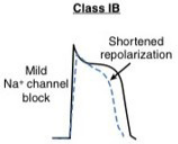
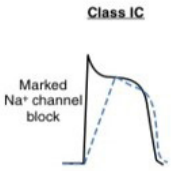
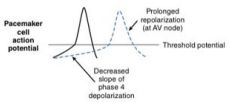
There are multiple contributing and interrelated processes induced by ischemia contributing to ventricular arrhythmogenesis in the peri-infarct period including, but not limited to, enhanced automaticity, cell death and injury generating substrate for VA, and changes in autonomic tone. In addition, some ischemia-related changes persist even in apparently healed myocardium which may increase the risk of future VAs if ischemia recurs (Figure 1) [15].

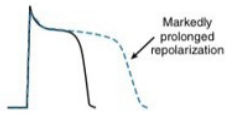
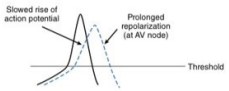
Approximately 6% of patients hospitalized with acute MI will develop VT or VF within 48-hours of admission [16]. The incidence of ischemic ventricular tachyarrhythmias has declined over time. In a review of hospital incidence and case-fatality rates, the incidence of sustained VAs decreased from 14.3% to 10.5% for VT and from 8.2% to 1.7% for VF between 1986 and 2009 [17], though both VT and VF after acute MI are associated with increased short-term mortality [5,17-19]. Data on long-term mortality has varied based on the type of ACS (ie, NSTEMI versus STEMI), type of VA, time to onset after acute hospitalization, and clinical interventions. Early reperfusion techniques and use of medical therapy including statins, ACE-inhibitors, and beta-blockers have decreased the incidence of ACS-related complications such as heart failure and LV dysfunction, complications thought to increase the risk of late VAs [20-27]. Despite these interventions, ischemic VAs, specifically when present at the time of hospital admission, have been associated with a 43-45% mortality at 2.5 years [16].

Numerous studies have identified features associated with an increased risk of VAs after MI. In a study of patients following their first AMI, intercurrent heart failure (RR 7.11; P<0.001), recurrent ischemic events (RR 4.25; P<0.001), diabetes (RR 2.45; P=0.01), chronic kidney disease (HR 2.53; P=0.01), and defibrillator implantation (HR 11.62; P<0.001) were all associated with an increased risk of VAs and sudden cardiac arrest [28]. In an analysis of pooled patient data from five multicenter randomized controlled prospective survival trials, demographic and clinical risk factors for 1-year mortality secondary to arrhythmia after AMI were identified in high risk patients defined as those with LVEF ≤40% or those with VAs. Factors conferring increased 1-year mortality included advanced age, male sex, prior MI or angina, history of hypertension, low baseline systolic blood pressure, Q waves, increased heart rate at the time of presentation, and worse NYHA functional class. All were associated with increased risk of arrhythmic, cardiac, and all-cause mortality. Good TIMI flow post-PCI was associated with lower mortality [29].

Both early, occurring within 48 hours of hospital admission, and late VAs, occurring greater than 48 hours post-MI, are associated with increased in-hospital and long-term mortality [19,30]. Studies performed after the introduction of fibrinolytics echo these findings [18,31,32]; early VF [33] as well as late VT and VF [29] are associated with increased mortality. Data extracted from the original Assessment of Pexelizumab in Acute Myocardial Infarction (APEX-AMI) data showed that among patients experiencing ischemic VA, 64% did so prior to primary PCI and 90% within 48-hours of STEMI symptoms. Ninety-day mortality was worse in patients with VA than in those without (23.2% vs 3.6%; adjusted HR 3.63; 95% CI 2.59-5.09). Moreover, late VT or VF was associated with worse mortality when compared with early VT or VF with a 5 and 2-fold increase in 90-day

Table 1. Anti-arrhythmics for Ventricular Arrhythmias after AMI

Drug	Vaughan Williams Classification Mechanism		ECG intervals [36]			Recommendations	COR [35]
			PR	QRS	QT		
Disopyramide Procainamide Quinidine	**Class Ia** Slow cardiac depolarization (phase 0) via fast-Na channel blockade. Prolong the cardiac action potential and refractory period leading to slowed repolarization via K-channel blockade (phase 3).		-	↑	↑	1. IV procainamide can be useful to terminate VT 2. Prophylactic use of procainamide after AMI associated with increased mortality. Not recommended [37]	IIa -
Lidocaine Mexieitine	**Class Ib** Slow cardiac depolarization (phase 0) via fast-Na channel blockade. Shorten repolarization via increasing the resting potential of phase 4.		-	-	-/↓	1. IV therapy for recurrent, sustained VTA post-AMI [38] 2. Lidocaine prophylaxis after AMI associated with increased mortality. Not recommended [37]	IIa III
Ecainide Flecainide Propafenone	**Class Ic** Slow cardiac depolarization (phase 0) via most potent blockade of fast-Na channels. Significantly decreases cardiac action potential upstroke and conduction velocity.		↑	↑	-/↑	Class IC antiarrhythmics not recommended in patients with prior MI 1. Slowed conduction with Flecainide and Propafenone may exacerbate ventricular arrhythmias post-AMI. Not recommended. 2. Ecainide and flecainide increase the risk of mortality post-AMI. Not recommended [6]	III
Beta-blockers	**Class II** B-adrenergic receptor inhibition. Decreases sympathetic drive, mitigating upslope of phase 4 depolarization, decreases SA node firing.		-/↑	-	-	1. First line. Excellent safety profile and effective in reducing VAs and SCD [37,39] 2. IV beta blockers may reduce mortality in polymorphic VT after-MI [40] 3. IV beta-blockers may be effective in patients with recent AMI with recurrent VT/VF despite DCCV and use of other antiarrhythmic medications	- IIa IIa

Amiodarone Dofetilide Dronaderone Ibutilide	**Class III** Block K channels responsible for phase 3 repolarization, prolonging the cardiac action potential.		-/↑	-/↑	↑	All prolong the QT. Pose a theoretical risk for polymorphic VT. Risk is likely lower for amiodarone [41]	
						1. Amiodarone not recommended for routine prophylaxis of VAs	III
						a. High dose amiodarone associated with higher mortality	
						b. Moderate dose amiodarone not superior to placebo [42]	I
						2. Amiodarone recommended for use in cardiac arrest due to VF or polymorphic VT unresponsive to CPR, defibrillation or vasopressor therapy	
						3. No data regarding use of dronaderone, ibutilide, or dofetilide	
Sotalol						1. IV amiodarone or sotalol be considered for termination of hemodynamically stable VT	IIb
						2. Typically avoided in patients with advanced LV dysfunction (LVEF <20%) due to negative inotropic effects and risk of torsades de points [43]	-
Verapamil Diltiazem	**Class IV** Selective L-type calcium channel blockade decreases phase 0 depolarization rate, decreases conduction velocity, and increases the AV node refractory period.		↑	-	-	1. No role in the treatment of VTA	III
						2. IV verapamil in the setting of sustained VT, particularly in patients with prior MI, has been associated with hemodynamic collapse	
						3. Should be particularly avoided in patients with VT in the setting of HFrEF	

- = no or little effect. Vaughan Williams' classification of antiarrhythmic drugs based on their effect on cardiac action potentials. 0, = 0mV. The dotted curves indicate the effects of the drugs; Lilly L. Pathophysiology of heart disease, 2003 [36].

mortality, respectively [29]. Composite data from the Acute Coronary Syndrome Israeli Survey (ACSIS) Registry [20] found a 2.1% incidence of early and 1.7% incidence of late VA, with a statistically significant increase in overall mortality (p<0.001) in patients in both groups, with a specific increase in the risk of in-hospital mortality (HR = 3.84, CI 1.77-6.78, p<0.001 in early VTA, HR 8.23, CI 4.84-13.98, p<0.001 in late). In this analysis, long-term mortality was significantly associated with late VA, particularly sustained VT (HR 5.17, CI 1.54-17.27, p=0.007). Overall, in-hospital mortality is generally increased for those with early VT and VF, while late VA may be associated with increased mortality in the longer term.

Treatment of VA depends on clinical stability. Pulseless VT and VF should be treated according to the Adult Advanced Cardiac Life Support (ACLS) guidelines [34,35], specifically, immediate electrical defibrillation and cardiopulmonary resuscitation. Antiarrhythmic drugs like amiodarone may be indicated when defibrillation attempts are unsuccessful or in the setting of recurrent VA. In the acute setting, intravenous antiarrhythmic drugs like amiodarone and lidocaine can suppress unstable ischemic VAs. Anti-arrhythmic therapies for VA and supporting evidence are detailed in Table 1 [36-43].

Aggressive treatment of underlying disorders including ischemia and electrolyte abnormalities, specifically hypokalemia and hypomagnesemia, are critically important. When VT is clinically stable, antiarrhythmic intervention alone may be adequate [35]. Catheter ablation of VAs in the peri-infarct period is generally not appropriate. Techniques to suppress autonomic drive as adjunctive treatment for peri-infarct VAs are emerging, including stellate ganglion blockade [44,45].

In addition to medical therapies, the implantable cardioverter defibrillator (ICD) plays an important role in the treatment of post-MI VA. Landmark randomized controlled trials of the 1990s and early 2000s demonstrated benefit of the ICD over medical therapy including antiarrhythmic therapy in high-risk populations with prior MI and reduced LVEF largely due to prevention of SCD [46-48]. The Multicenter Automatic Defibrillator Implantation Trial (MADIT) investigators [47] and Multicenter Unsustained Tachycardia (MUSTT) trial [49] studied patients with ischemic cardiomyopathy with reduced left ventricular function, ≤35% and ≤40%, respectively, with the additional criteria of observed NSVT and inducible VT at invasive electrophysiological study in the latter. Both studies demonstrated a significant survival benefit in the group receiving an ICD (MADIT: HR for overall mortality 0.46; CI 0.26 to 0.82; p=0.009; MUSTT: RR 0.24 95% CI 0.13 to 0.45; p<0.001). Subsequent data from the Multicenter Automatic Defibrillator Implantation Trial II (MADIT II) [46] and Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) [48] further demonstrated a survival benefit in ischemic cardiomyopathy patients with advanced LV dysfunction (LVEF <30% and <35%, respectively) even in the absence of VA or inducible VT (MADIT II: HR 0.69, 95% CI, 0.51 to 0.93; P=0.016). In the ischemic cohort enrolled in SCD-HeFT, ICD placement was associated with a 21% decreased risk of death (HR 0.79, 97.5% CI 0.60 to 1.04, p=0.05). Despite clear overall benefit, the benefits of preventing arrhythmic sudden death do not outweigh the risks of device implant until 30-40 days post revascularization in part due to the competing risks of death from mechanical causes after MI [50,51].

The Defibrillator in Acute Myocardial Infarction Trial (DINAMIT) trial randomized 674 patients 6-40 days after MI with a reduced LVEF to ICD versus no ICD. During follow up, up to 50% of the sudden deaths were due to mechanical causes including left ventricular rupture or acute mitral regurgitation. No mortality difference was demonstrated between the two groups (HR 1.08; 95% CI 0.76-1.55, p=0.66) [50]. Similar findings were made in the Immediate Risk Stratification Improves Survival (IRIS) trial in which 898 patients at high risk for sudden death after MI (LVEF <40 and a heart rate of ≥90 bpm on first available EKG and/or NSVT) were randomized to early ICD versus medical therapy. There was no difference in mortality between groups (HR 1.04, 95% CI 0.81-1.35 p=0.78) [51]. Even without device implantation, as in the case of the wearable defibrillator, no mortality benefit was seen in a modern high risk population treated early, although problems with device adherence may have diminished the benefits of this device administered early in the post-infarct period [52].

SUPRAVENTRICULAR ARRHYTHMIAS

Sinus Bradycardia

Sinus bradycardia may be caused by ischemic injury to the sinus node or by a vagally-mediated reflex. Sinus bradycardia can occur in 15 to 25% of patients after AMI, especially involving the inferior wall, since the right coronary artery (RCA) supplies the SA node in about 55% of people, with the remaining 45% supplied by the left circumflex artery. Generally, sinus bradycardia associated with AMI resolves within 24 hours and is associated with worse outcomes [53]. Medical treatment with intravenous atropine or temporary pacing is indicated when there is associated hemodynamic compromise. When sinus bradycardia persists beyond the immediate post-infarct period and is associated with hemodynamic compromise or symptoms, permanent pacing is indicated [54,55]. Although guidelines do not recommend a specific timeline for pacemaker implant post-infarct, a waiting period of at least 72 hours is generally accepted. Also,

when a pacemaker is indicated post-infarct and a patient would otherwise meet criteria for an ICD but is inside the 30-40 day waiting period, implantation of an ICD is reasonable to reduce procedural exposure [56].

Sinus Tachycardia

Sinus tachycardia occurs in up to 40% of patients presenting with AMI [57,58]. It is a nonspecific finding and may reflect catecholaminergic surge, pain, hemodynamic compensation, medication effects, or other factors. Data from the era prior to reperfusion suggest that persistent sinus tachycardia can reflect a large anterior MI with depressed LVEF in which case the rhythm is associated with increased morbidity, high early and increased 30-day mortality [57]. Importantly, because sinus tachycardia may reflect a compensatory process, caution should be used in initiating treatment, including use of beta blockers.

Atrial Fibrillation/Atrial Flutter

Atrial fibrillation occurs in 6-21% of patients with MI [59-61]. Atrial fibrillation occurring early after MI is typically the result of atrial ischemia, while elevated filling pressures and atrial stretch contribute to later onset (Figure 1) [62]. Risk factors associated with developing atrial fibrillation include advanced age, increased extent of myocardial damage, heart failure symptoms, depressed left ventricular function, and multi-vessel coronary disease [59,60].

Atrial fibrillation in the setting of AMI is associated with increased mortality [59-61]. A review by Schmitt and colleagues demonstrated that onset after AMI was an independent predictor of both in-hospital and long-term mortality [59]. Another study showed that new onset atrial fibrillation after primary PCI for acute STEMI was a powerful independent predictor of major bleeding (HR 1.74, 95% CI 1.30 to 2.34 p=0.0002) and major adverse cardiac events (HR 1.73, 95% CI 1.27 to 2.36) at 3 years compared with those without atrial fibrillation [63]. This increase in mortality may reflect atrial fibrillation as a surrogate marker of heart failure, elevated filling pressures, and volume overload after MI [59]. Alternatively, atrial fibrillation may give rise to VAs due to ischemia, varying R-R intervals or activation of the sympathetic nervous system [61].

Patients who develop atrial fibrillation after MI are at higher risk of stroke both in-hospital and at follow-up [59,60], even when sinus rhythm is restored prior to discharge. Siu and colleagues studied 431 patients with AMI, preserved LVEF, and transient in-hospital atrial fibrillation with spontaneous conversion to sinus rhythm prior to hospital discharge. These patients were discharged on antiplatelet therapy alone. Risk of subsequent atrial fibrillation was significantly higher in the group with transient in-hospital atrial fibrillation (22 vs 1.3%, p<0.01), with a significant difference in ischemic stroke risk (10.2 vs 1.8%, p<0.01) [64].

The risk of developing atrial fibrillation appears to be highest in the peri-infarct period. In a sub-study of Cardiac Arrhythmias & Risk Stratification After Acute Myocardial Infarction (CARISMA), Jons and colleagues followed implantable cardiac monitors in a population of 271 patients post-MI with LVEF<40% without prior atrial fibrillation. Over a 2-year period, atrial fibrillation was diagnosed in 39.3% of patients (95% CI 33.7%-45.5) and was most likely in the first 2 months after AMI (16%). By year 2 of monitoring, the risk of developing atrial fibrillation approached the risk in the general population. Compared with patients without atrial fibrillation, investigators additionally found an increased risk of major adverse cardiac events in those with atrial fibrillation (HR 2.73 (95% CI 1.35 to 5.50, p=0.005)). Importantly, more than 90% of patients with recorded atrial fibrillation were asymptomatic [65].

Management depends on multiple clinical factors. Atrial fibrillation causing hemodynamic compromise from either rapid or slow ventricular response requires standard ACLS including emergent cardioversion or pacing and infusion of antiarrhythmic or rate control medications such as amiodarone [66]. Otherwise, the mainstay of therapy follows standard management strategies, in combination with stroke risk management.

A detailed discussion of anticoagulation for stroke prevention in atrial fibrillation in the setting of standard dual antiplatelet therapy for treatment of ACS is beyond the scope of this review. In general, more recent evidence has shown promising results with a short course of triple therapy (ranging from 1 week to 1 month) followed by use of single anti-platelet therapy with a direct acting oral anticoagulant (DOAC) or vitamin K antagonist for patients in whom therapeutic anticoagulation is indicated for stroke prevention, especially in the setting of pre-existing atrial fibrillation [67-72].

High-Grade Atrioventricular Block

High-grade atrioventricular (AV) block (inclusive of second-degree type 2 AV block and complete heart block) is a rare complication of AMI occurring in less than 5% of patients in the reperfusion era [73-77]. Reperfusion has led to a dramatic decrease in the occurrence of high-grade AV block complicating STEMI with an incidence of 2% in 2005 compared with 5.1% in 1975 [74,78,79]. Patients who develop high-grade AV block after MI tend to be older, have higher Global Registry of Acute Coronary Events (GRACE) risk scores, and experience more in-hospital complications including myocardial re-infarction, heart failure, cardiogenic

shock, and VA [77]. While rare, high-grade AV block is associated with high morbidity and mortality [74,79-81]. In an analysis of 2003-2012 National Inpatient Sample database, Harikrishnan and colleagues found that STEMI patients with complete heart block had higher in-hospital mortality than those without (20.4% vs 8.7%; adjusted OR: 2.47; 95% CI: 2.41-2.53 [81]. Gang and colleagues analyzing hospital register data and the Danish National Patient Register similarly found a significantly increased adjusted mortality rate among patients with high-grade AV block compared to those without (HR 3.14 95% CI 2.04-4.84, p<0.001).

The mechanism of high-grade AV block varies according to anatomy. The AV node is supplied by the distal branches of the right coronary artery in 90% of patients and from the distal portions of the left circumflex artery in 10%. In general, conduction disturbances associated with inferoposterior infarctions are primarily related to ischemia or due to enhanced vagal activity at the level of the AV node (Figure 1). As such, these conduction disturbances tend to be transient and responsive to atropine. Conversely, conduction disturbances associated with anterior infarcts usually occur below the AV node, imply extensive septal necrosis, are less likely to recover [74,75], and may be exacerbated by atropine. As such, those with high-grade AV block from an anterior MI when the left anterior descending artery is the culprit are more likely to require permanent pacing and have a higher mortality than those with AV block resulting from an inferior MI with a right coronary artery or left circumflex artery culprit [76,77,79]. There is one notable exception to this general rule: when AV block due to inferior MI is accompanied by RV failure and cardiogenic shock, mortality remains high [75].

Much of the existing data regarding high-grade AV block complicating MI is from cohorts of patients presenting with STEMI. However, in a review by Misumida and colleagues of National Inpatient Sample data of STEMI and NSTEMI patients, high-grade AV block was more common than sinus node dysfunction in STEMI. The opposite was true of NSTEMI. The study also noted that patients with NSTEMI and high-grade AV block were more likely to require permanent pacemaker placement than patients with STEMI, suggesting lower rates of spontaneous resolution in the NSTEMI population. Despite these differences, high-grade AV block in both NSTEMI and STEMI patients was associated with high mortality rates. [73]. This finding was replicated in a global registry of ACS in which high-grade AV block was associated with in-hospital death in all forms of ACS: the odds ratio of death in the presence versus absence of high-grade AV block was 3.0 for STEMI, 6.4 for non-STEMI, and 8.2 for unstable angina (p<0.001) [77].

The primary treatment for patients presenting with high-grade AV block complicating AMI is reperfusion therapy [82]. For patients with symptomatic or hemodynamically significant AV block in the setting of MI that is likely due to block at the AV node, atropine is a reasonable intervention [54]. Dopamine and epinephrine may be considered in the unstable patient. Aminophylline has shown promise in a small case series, but rigorous data are lacking [47-54,75-83]. Temporary pacing is indicated for patients presenting with AMI with significant bradycardia or AV block that is medically refractory or associated with ongoing symptoms [54].

As noted, AV conduction often recovers following reperfusion and resolution of high vagal tone. When it does not, permanent pacing is indicated. When permanent pacing is planned, defibrillator therapy can be considered in a patient who would otherwise be subject to a reperfusion waiting period in order to reduce procedures and optimize resource utilization [56].

BUNDLE BRANCH BLOCK (BBB)

New BBB complicating AMI is uncommon in the reperfusion era [84]. Notably, unifasicular block, particularly left anterior fascicular block has a relatively benign prognosis while complete right (RBB) or left bundle branch (LBB) block are associated with higher in-hospital and long term mortality [84]. The RBB and the distal portion of the anterior LBB are supplied by septal perforators from the LAD. The LBB receives dual supply from distal branches of the right coronary and proximal circumflex vessels making it more resistant to permanent injury. Generally, no treatment is indicated for isolated BBB developed in the setting of AMI aside from reperfusion. Left-right dyssynchrony can develop in the setting of LBBB and can be associated with progressive LV dysfunction and heart failure. In this setting, cardiac resynchronization therapy with or without defibrillator therapy is considered.

CONCLUSION

Arrhythmias after MI are common clinical problems. The anti-arrhythmic properties of reperfusion have changed the incidence and overall outcomes for both ventricular and supraventricular arrhythmias in the setting of STEMI and NSTEMI. Increased electrical stability of infarcted myocardium with reperfusion has been effective in reducing ventricular events [85,86], supraventricular arrhythmias such as atrial fibrillation [87,88], and in improving survival in patients with high-grade AV block complicating ACS. These improvements are all likely related to the attenuation of the ischemic insult to the conduction system, reduction in the risk of re-infraction and a reduction in the overall infarct size [77].

There are many potential arrhythmic complications in the early post MI period. Appropriate treatment is often dependent on patient factors including timing of presentation, left ventricular function, clinical status, and co-morbidities. Some peri-infarct arrhythmias including VAs, atrial fibrillation, and persistent high-grade AV block, require treatment. Other arrhythmias such as sinus bradycardia and sinus tachycardia, as well transient high-grade AV block may require acute treatment, but often resolve with reperfusion and time. In all cases, the ability to recognize these rhythms, understand their likelihood, and appreciate the associated risks improves preparedness and hopefully outcomes for these vulnerable patients.

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