Conduction abnormalities after myocardial infarction

AUTHORS: Peter J Zimetbaum, MD, Joseph E Marine, MD, FACC, FHRS
SECTION EDITORS: Bradley P Knight, MD, FACC, Bernard J Gersh, MB, ChB, DPhil, FRCP, MACC, James Hoekstra, MD
DEPUTY EDITOR: Susan B Yeon, MD, JD

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Jan 2024.**
This topic last updated: **Jan 02, 2024.**

INTRODUCTION

Electrical conduction abnormalities are well-recognized complications of acute myocardial infarction (MI). They may be caused by either autonomic imbalance or ischemia/infarction involving the conduction system. The most common arrhythmic consequence is bradycardia, which may or may not be symptomatic. Complete heart block with a slow escape rhythm is a potentially life-threatening event in this setting if not detected and treated promptly. In addition, it is important to recognize which bradyarrhythmias are transient and which are likely to progress to irreversible and symptomatic high-degree atrioventricular (AV) block.

The major conduction abnormalities associated with acute MI will be reviewed here. Supraventricular arrhythmias, including sinus bradycardia, are discussed separately. (See "Supraventricular arrhythmias after myocardial infarction" and "Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features".)

ANATOMY AND ELECTROPHYSIOLOGY

Anatomy — The sinoatrial (SA) node is located high (superiorly) in the right atrium at the junction of the crista terminalis (a thick band of atrial muscle at the border of the atrial appendage) and the superior vena cava. The SA node is located beneath the epicardial surface of the crista terminalis; there is a layer of atrial muscle between the SA node and the

endocardium so that it does not occupy the entire thickness of the atrial myocardium. Human histologic studies have demonstrated that the SA node has a crescent-like shape with an average length of 13.5 mm [1].

Following atrial activation, the impulse reaches the AV node. This structure generates a slow calcium-mediated action potential (figure 1). Thus, there is a delay in impulse transmission through this structure. After leaving the AV node, the bundle of His divides at the juncture of the fibrous and muscular boundaries of the interventricular septum into the right and left bundle branches (figure 2).

- The right bundle branch courses down the right side of interventricular septum near the endocardium in its upper third, deeper in the muscular portion of the septum in the middle third, and then again near the endocardium in its lower third. The right bundle does not branch throughout most of its course, but it begins to ramify as it approaches the base of the right anterior papillary muscle, with fascicles going to the septal and free wall of the right ventricle (RV). The apical free wall at the base of the right anterior papillary muscle is the earliest site of RV activation.

- The left bundle branch penetrates the membranous portion of the interventricular septum under the aortic ring. Shortly thereafter, it divides into several discrete branches [2-5]:

  - An anterior fascicle that crosses the left ventricular (LV) outflow tract and terminates in the Purkinje system of the anterolateral wall of the LV.

  - A posterior fascicle that fans out extensively inferiorly and posteriorly.

  - In about 65 percent of hearts, a separate fascicle to the interventricular septum.

  - In all patients there is early septal activation, either by a discrete septal branch or septal extensions of the posterior fascicle.

  - It should also be noted that evidence suggests that certain fibers in the His bundle are predestined to form the left bundle branch.

Blood supply — In order to fully understand the relationship between MI and dysrhythmia, it is helpful to review the vascular supply of the different components of the conduction system (figure 3) [6]:

- SA node – Supplied by the right coronary artery (RCA) in 60 percent of patients; by the left circumflex artery (LCX) in 40 percent.

- AV node – Supplied by the RCA in 90 percent (AV nodal branch); by the LCX in 10 percent of patients.
- His bundle – Supplied by the RCA (AV nodal branch) with a minor contribution from the septal perforators of the left anterior descending artery (LAD).
- Main or proximal left bundle branch – The LAD provides most of the blood supply for the left bundle branch, particularly for the initial portion. There may be some collateral flow from the RCA and LCX systems. To the extent that the His bundle has predestined fibers to the left bundle, His bundle blood supply is also important.
- Left posterior fascicle – The proximal portion of the left posterior fascicle is supplied by the AV nodal artery and, at times, by septal branches from the LAD. The distal portion has a dual blood supply from both anterior and posterior septal perforating arteries.
- Left anterior fascicle – The left anterior and mid-septal fascicles are supplied by septal perforators of the LAD and, in about one-half of subjects, by the AV nodal artery.
- Right bundle branch – The right bundle branch receives most of its blood supply from septal perforators from the LAD coronary artery, particularly in its initial course. It also receives some collateral supply from either the RCA or LCX coronary systems, depending upon the dominance of the coronary system.

Electrophysiology — While it may appear that the electrical signals from the SA node to the atrial periphery can exit randomly, there appear to be preferential pathways of conduction from the sinus pacemaker cells to the atrium [7]. Whether these are functional or anatomical exit paths remains unclear. The conduction velocity within the SA node is very slow compared with non-nodal atrial tissue. This is a result of poor electrical coupling arising from the relative paucity of gap junctions in the center of the SA node compared with the periphery [8].

With atrial activation, the electrical impulse reaches the AV node. This structure generates a slow calcium-mediated action potential (figure 1). Thus, there is a delay in impulse transmission through this structure.

Once the action potential traverses the AV node, it activates the proximal portion of the bundle of His, a specialized conducting tissue that generates a fast action potential. The bundle of His divides into the main left and right bundle branches, which consist of bundles of Purkinje cells covered by a dense sheath of connective tissue. Purkinje cells are specialized to conduct rapidly at 1 to 3 m/second, as phase 0 is dependent on the rapid inward sodium current (figure 4),

resulting in nearly synchronous depolarization of the terminal His Purkinje system and the adjacent ventricular myocardium.

TYPES OF CONDUCTION ABNORMALITIES

Conduction abnormalities are manifest on the surface electrocardiogram (ECG) and are broadly categorized as follows:

- Left bundle branch block (LBBB). (See "Left bundle branch block", section on 'ECG findings and diagnosis' and "Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block or a paced rhythm".)
- Left anterior, left posterior, or left septal (median) fascicular block. (See "Left anterior fascicular block" and "Left posterior fascicular block" and "Left septal fascicular block".)
- Right bundle branch block (RBBB). (See "Right bundle branch block", section on 'ECG findings and diagnosis'.)
- First-degree AV block. (See "First-degree atrioventricular block", section on 'Definition'.)
- Second-degree AV block. (See "Second-degree atrioventricular block: Mobitz type I (Wenckebach block)", section on 'ECG findings and diagnostic maneuvers' and "Second-degree atrioventricular block: Mobitz type II", section on 'ECG findings'.)
- High-grade (ie, two or more consecutive blocked P waves) AV block. (See "Third-degree (complete) atrioventricular block" and "Second-degree atrioventricular block: Mobitz type II".)
- Third-degree (complete) AV block. (See "Third-degree (complete) atrioventricular block", section on 'Electrocardiographic findings'.)

INCIDENCE

In the era of primary percutaneous coronary intervention (PCI), the rates of post-MI conduction abnormalities are decreasing, with rates between 1 and 3 percent reported in most studies. Additionally, new BBB complicating acute MI is uncommon in the reperfusion era (0.73 and 0.15 percent of patients developed RBBB and LBBB, respectively, in the first 60 minutes after presentation in one large study) [9].

Determining the incidence and prognostic significance of new conduction abnormalities associated with acute MI is difficult for several reasons:

- Data are most commonly generated from retrospective reviews, sub-analyses of clinical trial data, or administrative databases.
- The incidence of conduction system disease depends on the cohort studied. For example, older patients are more likely to have underlying conduction system disease at baseline. Additionally, patients treated with prompt reperfusion with PCI have a lower incidence of conduction abnormalities compared with those who received thrombolytic reperfusion therapy, who in turn have lower rates of conduction abnormalities when compared with older studies in the era prior to reperfusion.
- It is frequently difficult to distinguish between ECG conduction abnormalities that are chronic (pre-existing) and those that are new abnormalities associated with the infarction.

The National Registry of Myocardial Infarction 2 (NRMI-2) in the United States evaluated the incidence of **bundle branch block** in 297,832 patients admitted to a hospital with an acute MI between 1994 and 1997: 6.7 percent of patients had an LBBB and 6.2 percent had an RBBB on the initial ECG [10]. A similar rate of LBBB (9 percent) was noted in a prospective analysis of over 88,000 patients in Sweden [11]. Because both of these series only assessed the presence of BBB on the initial ECG, these data provide no information on the incidence of new conduction disease in acute MI.

The largest experience with **high-degree AV block** in the fibrinolytic era comes from a review of almost 76,000 patients with ST-elevation MI (STEMI) enrolled in four large randomized trials, in which the overall incidence was 6.9 percent (9.8 percent with inferior MI and 3.2 percent with anterior MI) [12]. In the primary PCI era, most studies have shown continued decreases in the rate of high-degree AV block, ranging from 1 to 3 percent of patients, with higher rates seen in patients with STEMI compared with non-ST elevation MI (NSTEMI) [13-19]. (See 'Conduction disturbances based on infarct location' below.)

- In a study of 418,396 United States patients with acute MI (32.5 percent STEMI with 77 percent receiving PCI, 67.5 percent NSTEMI with 37 percent receiving PCI) between 2010 and 2014 who were enrolled in the National Inpatient Sample database, high-degree AV block was seen in 2.4 percent of patients with STEMI compared with 0.6 percent of patients with NSTEMI [19]. Of the 1745 patients who developed high-degree AV block, a higher proportion of patients with NSTEMI (30 percent) underwent permanent pacemaker implantation than those with STEMI (10.6 percent). High-degree AV block was associated

with elevated risk of in-hospital mortality in both NSTEMI (17 versus 3.8 percent, p<0.001) and STEMI (18 versus 8.2 percent) patients.

- The Global Registry of Acute Coronary Events (GRACE) registry enrolled 59,229 patients with acute coronary syndromes (37 percent with STEMI, 33 percent with NSTEMI) at 126 hospitals in 14 countries [17]. Mobitz type II second-degree AV block or third-degree AV block occurred in 2.9 percent of patients (5 percent of STEMI patients, 1.9 percent of NSTEMI patients). When AV block was present, it more commonly occurred at presentation (54 percent versus 46 percent later in the hospitalization). The right coronary artery was the culprit vessel in 65 percent of patients with AV block. Temporal analysis showed a declining incidence of AV block between 1999 and 2007 (-0.2 percent per year).

Conduction disturbances can occur after a "controlled" MI, as occurs with a septal (alcohol) ablation procedure for hypertrophic cardiomyopathy. This issue is discussed elsewhere. (See "Hypertrophic cardiomyopathy: Management of patients with outflow tract obstruction", section on 'Septal reduction therapy'.)

CONDUCTION DISTURBANCES BASED ON INFARCT LOCATION

The management and prognosis of patients with bradyarrhythmias or conduction abnormalities after an MI depends in part upon the location of the infarct. Often the anatomic location of the infarct can be gleaned from the ECG. In other cases, echocardiography or coronary arteriography supplies this information. (See 'Anatomy and electrophysiology' above and 'Management of conduction abnormalities' below.)

For example, the management and prognosis of high-degree AV block differs depending on infarct location:

- High- (second- or third-) degree AV block associated with inferior wall MI is located **proximal to** the His bundle (ie, AV node) in 90 percent of patients [6,20]. For this reason, complete heart block usually results in only a modest and usually transient bradycardia with junctional or escape rhythm rates above 40 beats per minute. It is not uncommon, however, for the junctional escape pacemaker rate to exceed 60 beats per minute. The QRS is usually narrow in this setting.

- High-degree AV block associated with anterior MI is more often located **distal to** the AV node [6]. It is usually symptomatic and has been associated with a high mortality rate due in large part to greater loss of functioning myocardium. The contemporary mortality may

be lower due to improvements in reperfusion therapy and in the management of congestive heart failure and cardiogenic shock, but the risk remains substantial.

Inferior MI — Conduction disturbance in inferior MI may occur upon presentation or after hours or days. Sinus bradycardia, Mobitz type I (Wenckebach), and complete heart block (CHB) are commonly seen, since the SA node, AV node, and His bundle are primarily supplied by the right coronary artery (RCA) [20]. (See 'Anatomy and electrophysiology' above.)

- **Sinus bradycardia** is the most common arrhythmia associated with inferior MI. It is present in up to 40 percent of patients in the first two hours, decreasing to 20 percent by the end of the first day. It is usually attributable to increased vagal tone or increased sensitivity to vagal tone in the first 24 hours after infarction. Transient sinus node dysfunction occurring later may be due to sinus node or atrial ischemia.

- **First-degree AV block** (characterized by prolongation of the PR interval, >200 ms) can arise in the AV node, the bundle of His, or the bundle branches (see "First-degree atrioventricular block"). First-degree AV block at the level of the AV node is common after occlusion of the coronary artery (right or circumflex) which gives rise to the AV nodal artery. RCA occlusion can lead to first-degree AV block via ischemia of the AV node, by enhanced acetylcholine release from the inferoposterior myocardium, or possibly by making the AV node hypersensitive to the action of acetylcholine. First-degree AV block due to occlusion of the RCA with involvement of the AV node is usually transient, generally resolving in five to seven days and requiring no specific therapy.

  Occlusion of the left circumflex artery, which may present as an inferior MI on the ECG, may affect the AV node directly in the 10 percent of individuals in whom it supplies the AV node.

- Inferior MI is typically associated with the more benign **second-degree AV block** of the Wenckebach type (Mobitz type I) (waveform 1A-B); Mobitz type II block is uncommon in this setting, generally occurring with anterior MI (waveform 2).

  The mechanism is similar to that described above for first-degree AV block. Mobitz type I block is usually transient, resolving in most cases within five days. (See "Second-degree atrioventricular block: Mobitz type I (Wenckebach block)".)

- Somewhat more than one-half of cases of **high-degree AV block** in acute MI are present on admission and somewhat less than one-half develop later in the hospitalization [14].

Complete AV block (CHB) with inferior MI generally results from an AV nodal lesion. It is associated with a narrow QRS complex and develops in a progressive fashion from first- to second- to third-degree block. It often results in an asymptomatic bradycardia (40 to 60 beats per minute) and is usually transient, resolving within five to seven days. In the substudy from the TRACE trial described above, the incidence of CHB was significantly higher among patients with an inferior MI than among those with an anterior MI (9.4 versus 2.5 percent) [21]. In the primary percutaneous coronary intervention (PCI) era, one study showed that high-degree AV block occurred in 5.9 percent of patients with right coronary artery occlusion and in 1.5 percent of patients with other infarct-related arteries [14]. Another study from a large United States registry showed a rate of CHB of 3.8 percent of patients with inferior STEMI and 0.9 percent of patients with anterior STEMI [16].

Occasionally, RCA occlusion with inferior MI produces persistent complete AV block. This latter finding suggests concurrent involvement of the left coronary system, resulting in poor collateral flow. In one large United States registry, 11.5 percent of patients with inferior STEMI received a permanent pacemaker prior to discharge [16].

Anterior MI — Conduction disturbances occurring with anteroseptal MI are less frequent but more serious, and the degree of arrhythmic complications is usually directly related to the extent of infarction.

- **First-degree AV block** – Prolongation of the PR interval due to slowed AV nodal conduction rarely occurs in anterior MI, since the AV node is usually supplied by the right or circumflex coronary artery, as opposed to the left anterior descending coronary artery, which is the cause of anterior infarctions in most cases.

- **High- (second- or third-) degree AV block** – Second-degree AV block with anterior MI is usually at the level of the His bundle or below and is usually a Mobitz type II block. The clinical course may be unpredictable, with CHB developing with little warning. (See "Second-degree atrioventricular block: Mobitz type II".)

CHB with anterior MI generally occurs abruptly in the first 24 hours. It can develop without warning or may be preceded by the development of right bundle branch block with either a left anterior fascicular block or left posterior fascicular block, or by alternating bundle branch block (bifascicular or trifascicular block) (waveform 3) [22]. The escape rhythm usually has a wide QRS duration and is unstable, and the event is associated with a high mortality from both arrhythmias and pump failure. Heart block in this setting is thought to result from extensive necrosis that involves the bundle branches traveling within the septum [22].

Less commonly, anterior MI produces first-degree AV block **below** the level of the AV node, a situation that should be suspected if first-degree AV block occurs in the presence of a widened QRS complex.

MANAGEMENT OF CONDUCTION ABNORMALITIES

The initial management of the patient with post-MI conduction abnormalities depends on the location of the MI and the presence and severity of hemodynamic instability (algorithm 1).

- Second-degree AV block, high-grade AV block, or third-degree (complete) AV block in the setting of an anterior MI is unlikely to recover following revascularization; these rhythms are by nature unstable rhythms and more likely to cause, or to be associated with, hemodynamic instability. Patients with newly developed bundle branch block (BBB), or alternating BBB, are also at increased risk of developing hemodynamic instability.

- Second-degree AV block, high-grade AV block, or third-degree (complete) AV block in the setting of an inferior MI may respond to treatment with atropine and/or aminophylline, when the block occurs at the level of the AV node. Following revascularization, conduction abnormalities in the setting of an inferior MI are more likely to resolve and not require permanent pacing.

- Patients with first-degree AV block or isolated BBB rarely develop hemodynamic instability related to their heart rhythm. If hemodynamic instability arises in these settings, a search for other causes is typically warranted.

Hemodynamically unstable patients with Mobitz type II second-degree AV block, high-grade AV block, or third-degree (complete) AV block require immediate pharmacologic therapy and, in many instances, should also receive temporary pacing to increase heart rate and cardiac output. Once the patient is hemodynamically stable, assessment and treatment for any potentially reversible causes (in addition to myocardial ischemia) should occur, followed by a period of continuous ECG rhythm monitoring. Patients whose advanced AV block does not resolve following revascularization and treatment of any other reversible etiologies should undergo placement of a permanent pacemaker [23]. Our approach is in agreement with published professional society guidelines, which discuss the role of temporary cardiac pacing during the peri-infarction period, as well as indications for implantation of a permanent cardiac pacemaker [23,24].

Management of patients with AV block

Patients with anterior MI — The location of the MI is important in determining the approach to therapy for conduction abnormalities. New conduction abnormalities in the setting of an anterior MI are typically indicative of extensive ischemia/infarction involving the infranodal conduction system and are unlikely to improve with atropine or resolve following revascularization. As such, we proceed directly to temporary cardiac pacing in such patients, with a plan for permanent cardiac pacing if conduction is not restored after the first few days of recovery.

Unstable inferior MI patients — Patients with inferior MI and second-degree AV block, high-grade AV block, or third-degree (complete) AV block who are hemodynamically unstable should be urgently treated (algorithm 1) with atropine and, if hemodynamic instability persists, temporary cardiac pacing (either with transcutaneous or, if immediately available, transvenous pacing). In general, beta agonists (eg, dopamine, epinephrine, dobutamine, etc) should be avoided in hypotensive patients with acute MI and AV block due to the potential for worsening ischemia. For patients with high-grade AV block associated with inferior MI, aminophylline may improve AV conduction and symptoms. Additionally, in the setting of an acute myocardial ischemia, patients should undergo prompt revascularization; following revascularization, most conduction abnormalities will improve or resolve and will not require permanent pacing. (See "Advanced cardiac life support (ACLS) in adults", section on 'Bradycardia'.)

Approach to managing unstable patients with AV block — The most important clinical determination in a patient presenting with AV block is whether or not the patient is hemodynamically unstable due to the resulting bradycardia and reduced cardiac output (algorithm 1). Signs and symptoms of hemodynamic instability include hypotension, altered mental status, signs of shock, ongoing ischemic chest pain, and evidence of acute pulmonary edema. In general, such patients should be treated according to the Advanced Cardiac Life Support protocol for patients with symptomatic bradycardia (algorithm 2), with the exception of avoiding beta agonists (eg, dopamine, epinephrine, dobutamine, etc) in patients with ongoing ischemia due to the potential for worsening ischemia [25]:

- Atropine should be promptly administered if intravenous (IV) access is available, but treatment with atropine should not delay treatment with transcutaneous pacing. The initial dose of atropine is 0.5 mg IV. This dose may be repeated every three to five minutes to a total dose of 3 mg, but should not delay the initiation of temporary cardiac pacing for patients who remain hemodynamically unstable. A favorable response to atropine also suggests that AV block is due to abnormal conduction in the AV node. Atropine is not likely to be effective for patients with an escape rhythm at or below the bundle of His, since the more distal conducting system is not as sensitive to vagal activity.

Caution should be used with administering atropine in the setting of active ischemia and BBB or Mobitz Type 2 AV block because of potential for precipitating ventricular arrhythmia or worsening degree of AV block [23].

- If refractory hypotension occurs after correction of bradycardia with atropine in the setting of an inferior MI, volume depletion or concurrent RV infarction should be suspected. This can be confirmed by right-sided ECG leads or bedside echocardiography. Volume infusion is the treatment of choice in this setting. (See "Right ventricular myocardial infarction".)

- For patients with second- or third-degree AV block associated with symptoms or hemodynamic compromise in the setting of acute inferior MI that persist following atropine, we suggest IV aminophylline to improve AV conduction, increase ventricular rate, and improve symptoms [23]. Several small case series (eight patients or fewer) have shown prompt reversal of AV block in this clinical setting without adverse effects [26]. The typical dose is 250 mg IV or 6 mg/kg IV in 100 to 200 mL of IV fluid over 20 to 30 minutes. Preparations should continue for temporary cardiac pacing in the event that symptoms worsen or do not improve following aminophylline administration.

- Temporary cardiac pacing should be provided for patients with acute MI and persistent unstable bradycardia. In the absence of central venous access, the most immediate way to provide temporary cardiac pacing is via transcutaneous pacing. Transcutaneous pacing is uncomfortable for the patient and may have variable efficacy depending on how well the impulses are transmitted to the myocardium; as such, transcutaneous pacing should be viewed as a temporizing measure until temporary transvenous pacing can be provided. Patients should be monitored by arterial palpation, plethysmography, or intra-arterial monitoring to ascertain that transcutaneous pacing produces a pulse pressure. (See 'Temporary pacing' below and "Temporary cardiac pacing".)

Once a hemodynamically unstable patient has been stabilized, the approach to further management is similar to patients who were initially stable.

Temporary pacing — Our recommendations regarding temporary pacing are in general agreement with those in the 2013 American College of Cardiology Federation/American Heart Association (ACCF/AHA) guideline for the management of patients with ST-elevation MI and in the 2018 American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS) guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay [23,24].

When temporary pacing is required, we recommend transvenous pacing. We do not favor the use of external/transcutaneous cardiac pacing, except in emergency circumstances, because of

the associated physical discomfort and difficulty in ascertaining consistent myocardial capture. If transcutaneous pacing is utilized, it should be used as a temporary bridge to transvenous pacing, while the transvenous pacemaker placement is being arranged. It should be used with appropriate procedural sedation and monitoring for production of a pulse pressure. Placement of transcutaneous pacing pads is also reasonable when observing a patient at risk for development of AV block while a decision regarding temporary transvenous pacing is being made. (See "Temporary cardiac pacing".)

The following points should be considered in deciding whether to place a temporary pacemaker (algorithm 1):

- In patients with inferior MI, third-degree (complete) AV block may be transient and hemodynamically tolerated. We do not routinely insert a pacemaker for third-degree (complete) AV block if the escape rhythm has a narrow QRS and an adequate ventricular rate with stable blood pressure and peripheral perfusion, because this junctional rhythm is usually stable. However, careful monitoring is mandatory and we have a low threshold for pacing should deterioration occur.

- Bradycardia may occur in patients with RV infarction. Hemodynamic status may deteriorate in such patients, since the ischemic RV has a relatively fixed stroke volume and is therefore dependent upon heart rate. Patients with RV infarction associated with second- or third-degree (complete) AV block often benefit hemodynamically from temporary AV sequential pacing. (See "Right ventricular myocardial infarction".)

- New BBB is associated with substantial short-term mortality, particularly in the setting of an anterior MI (>30 percent at 30 days) [9]. However, the incidence of progressive conduction disease (ie, second- and third-degree AV block) is not known, and therefore the potential benefit of prophylactic temporary pacing in this setting is unclear. Temporary transvenous pacing is rarely needed for isolated BBB in the setting of acute MI and efforts should be focused on reperfusion and medical therapy. (See 'Management of patients with BBB' below.)

- For patients who will require temporary pacing for more than 48 hours and are hemodynamically stable for transport to the electrophysiology lab, we generally place an active fixation permanent pacing lead in the RV and externalize the pin for use in an external resterilized permanent pacemaker generator or conventional temporary pacing box. This form of temporary pacing has been shown to be far more stable and better tolerated by patients than conventional temporary pacing wires [27].

Stable inferior MI patients

Second-degree AV block, high-grade AV block, or third-degree (complete) AV block — Patients with inferior MI and second-degree AV block, high-grade AV block, or third-degree (complete) AV block who are hemodynamically stable generally do not require urgent therapy with atropine or temporary cardiac pacing. However, many ventricular escape rhythms are unreliable and potentially unstable, so patients should be continuously monitored with transcutaneous pacing pads in place in the event of clinical deterioration (algorithm 1). Additionally, in the setting of an acute myocardial ischemia, patients should undergo prompt revascularization; following revascularization, most conduction abnormalities will improve or resolve and will not require permanent pacing.

Following revascularization, if second-degree AV block, high-grade AV block, or third-degree (complete) AV block persists, patients should remain on continuous ECG monitoring and should be evaluated for any additional potentially reversible causes of AV block (eg, increased vagal tone, hypothyroidism, hyperkalemia, and drugs that depress conduction).

- Patients with AV block felt to be medication-induced should be observed while the offending agent or agents are withdrawn; such patients may have improvement or resolution of AV block following removal of the medication with or without administration of reversal agents.

- Patients with AV block in the setting of hyperkalemia should receive therapy to reduce serum potassium levels. If AV block subsequently resolves, a permanent pacemaker is not usually needed. (See "Treatment and prevention of hyperkalemia in adults" and "Treatment of primary hypothyroidism in adults".)

In general, a waiting period of several days should be employed to determine potential reversibility of AV block, particularly in patients with acute inferior MI [23]. If no reversible causes are present, definitive treatment of Mobitz type II second-degree AV block, high-grade AV block, or third-degree (complete) AV block involves permanent pacemaker placement in most patients (algorithm 1) [23,28]. Dual-chamber (ie, AV) pacing to maintain AV synchrony is preferred (rather than single chamber RV pacing) in most patients due to the favorable hemodynamic benefits of AV synchrony [23]. Some trials suggest that biventricular cardiac pacing (ie, cardiac resynchronization) is superior to standard dual chamber pacing in patients with heart block and depressed LV systolic function [29,30]. Implantable cardioverter-defibrillators, specifically cardiac resynchronization therapy devices (CRT-Ds), should be considered in patients with AV block and significant LV dysfunction that is likely to be permanent and irreversible despite medical therapy and revascularization. (See "Permanent cardiac pacing: Overview of devices and indications", section on 'Acquired AV block' and "Modes of cardiac pacing: Nomenclature and selection".)

First-degree AV block and Mobitz type I second-degree AV block — Patients with first-degree AV block or Mobitz type I second-degree AV block who are hemodynamically stable and asymptomatic do not require any specific therapy, other than avoidance of AV nodal-blocking agents. We agree with the professional society guidelines which do **not recommend** a pacemaker for most cases of first-degree AV block or Mobitz type I second-degree AV block [23,28].

Management of patients with BBB — Patients with isolated left BBB (LBBB) or right BBB (RBBB) (complete or incomplete) are generally asymptomatic related to their conduction disorder and do not require placement of a pacemaker (temporary or permanent) or any other specific therapy. The one exception to this is for patients with alternating RBBB and LBBB, or RBBB with alternating left anterior fascicular block and left posterior fascicular block, as these indicate a higher likelihood of significant conduction system disease which may progress to unstable AV block. In cases of alternating BBB in the setting of acute MI, we insert a temporary transvenous pacemaker and pursue prompt revascularization. If alternating BBB persists or progresses following revascularization, patients typically receive a permanent pacemaker.

Transient second- or third-degree AV block associated with new persistent BBB after MI is rarely encountered in the primary percutaneous coronary intervention era. Based on small studies in the 1970s showing high mortality in these patients, older versions of pacemaker guidelines recommended routine permanent pacemaker implantation [31]. However, this recommendation has not been adopted in the most recent American or European guidelines [23,28]. Invasive electrophysiology studies looking for severe His-Purkinje conduction disease and/or follow-up event monitoring to guide therapy may be helpful in this rarely-encountered clinical scenario.

PROGNOSIS

High-degree AV block — Advanced (second- or third-degree) AV block is associated with an increase in mortality in patients with an inferior or anterior MI [12-17,21,32]. The increase in mortality risk is seen largely within the first 30 days; among 30-day survivors, subsequent mortality does not appear to be consistently increased in reports of various AMI cohorts [12,13,18,21].

AV block following inferior MI — Although high-grade AV block in patients with an inferior MI is usually transient, it is associated with increased in-hospital mortality.

- Following primary PCI – In one large United States registry in the PCI era, complete heart block (CHB) in the setting of inferior STEMI increased in-hospital mortality twofold [16]. A similar increase in mortality with inferior STEMI was seen in other studies in the PCI era [14,15].

- Following thrombolysis – An increase in mortality with high-degree AV block of about 15 percent at 30 days is seen in patients with an inferior MI treated with a thrombolytic agent [12,21,32-36].

AV block following anterior MI — High-degree AV block in patients with an anterior wall MI is associated with a greater increase in in-hospital and 30-day mortality than seen with an inferior wall infarction, probably due to more extensive myocardial involvement and a higher incidence of hemodynamic complications [12,32].

- Following primary PCI – In the United States National Inpatient Sample registry from the PCI era, CHB in the setting of anterior STEMI increased in-hospital mortality fourfold, versus only twofold in inferior STEMI patients [16]. A similar increase in mortality with CHB and anterior STEMI was seen in other studies in the PCI era [14,15,37]. In a study of 1295 STEMI patients undergoing PCI in Japan, complete AV block occurred in 1.7 percent of patients with anterior STEMI and 10.7 percent of those with nonanterior STEMI [37]. Multivariate analysis showed that CHB was associated with all-cause mortality (hazard ratio [HR] 3.01, 95% CI 1.33-6.09) and major adverse cardiac endpoints (HR 2.23, 95% CI 1.01-4.38) in anterior STEMI patients. In contrast, CHB was not associated with adverse outcomes in patients with nonanterior STEMI.

- Following thrombolysis – In a review of nearly 76,000 patients from four randomized trials of fibrinolytic therapy, 3.2 percent of patients with anterior MI developed AV block and had a significant increase in mortality at 30 days (41 versus 8 percent in those without AV block; odds ratio [OR] 3.0, 95% CI 2.2-4.1) [12].

Bundle branch block — The presence of fascicular or BBB during an acute MI is associated with an increase in in-hospital and long-term mortality [9-11,22,38-47]. However, interpreting the prognostic significance of BBB is complicated for several reasons:

- In contrast to CHB, which is almost always a new finding due to the MI, BBB often precedes the MI. This finding most commonly reflects the prevalence of BBB among patients presenting with acute MI, not the incidence of new BBB due to the infarction, which is generally very low. In the HERO-2 trial, BBB was present on 5.1 percent of initial ECGs, but new BBB developed in only 0.9 percent of patients on a second ECG taken 60

minutes later [9]. The development of a new BBB post-MI is generally felt to be associated with increased mortality [48].

- Chronic and new conduction abnormalities may both predict worse outcomes, but for different reasons. The former is due to more extensive underlying cardiac disease and the latter is due to the association with larger infarctions.

The following observations illustrate the range of findings among patients with BBB. These studies made the assessment of the presence or absence of BBB largely based on the initial ECG. Thus, it could not be determined whether the BBB was pre-existing or associated with the infarction unless there was resolution of the BBB.

- Following primary PCI – Among patients undergoing primary PCI, the presence of BBB on the baseline ECG is associated with increased mortality [48-50]. In a review of 3053 patients in the Primary Angioplasty in Myocardial Infarction (PAMI) trials, LBBB was present in 1.6 percent and RBBB in 3.1 percent [43]. In-hospital mortality was 14.6, 7.4, and 2.8 percent in patients with LBBB, RBBB, and no BBB, respectively.

- Following thrombolysis – In an analysis of 26,003 North American patients entered into the GUSTO-I trial of thrombolytic therapy, the in-hospital mortality was 18 versus 11 percent with and without a BBB [42]. Patients with a BBB were more likely to experience cardiogenic shock (19 versus 11 percent), AV block or asystole (30 versus 19 percent), and to require a pacemaker (18 versus 11 percent). Mortality was higher when the BBB was persistent (20 percent versus 12 and 8 percent in the 24 percent of patients who had partial or complete resolution of the BBB, respectively).

- In a meta-analysis of eight studies that included 105,861 acute MI patients (STEMI and non-STEMI), new LBBB was associated with higher mortality at 30 days (OR 2.10; 95% CI 1.27-3.48) and at one year (OR 2.81; 95% CI 1.64-4.80) [46]. In a meta-analysis of 10 studies that included 63,103 patients (STEMI and non-STEMI), RBBB (new or old) was associated with higher in-hospital (OR 1.94; 95% CI 1.60-2.37) and long-term mortality (OR 1.49; 95% CI 1.37-1.62) [47].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Non-ST-elevation acute coronary syndromes (non-ST-elevation myocardial infarction)" and "Society guideline links: ST-elevation myocardial infarction (STEMI)".)

SUMMARY AND RECOMMENDATIONS

- **Causes of conduction disturbance** – Bradyarrhythmias and conduction disturbances are well-recognized complications of acute myocardial infarction (MI). They are caused by either autonomic imbalance or ischemia/necrosis of the conduction system. (See 'Anatomy and electrophysiology' above.)

- **MI location**

  - **Inferior MI** – Conduction disturbances commonly seen with inferior MI include sinus bradycardia and complete heart block (CHB). Atrioventricular (AV) block with inferior MI generally results from ischemic and autonomic effects on the AV node. It is associated with a narrow QRS complex and develops in a progressive fashion from first- to second- to third-degree block. It often results in an asymptomatic bradycardia (40 to 60 beats per min) and is usually transient, resolving within five to seven days. (See 'Inferior MI' above.)

  - **Anterior MI** – Conduction disturbances occurring with anteroseptal MI are less frequent but more serious, and the degree of arrhythmic complications is usually directly related to the extent of infarction. AV block with anterior MI generally occurs abruptly in the first 24 hours. It can develop without warning or may be preceded by the development of right bundle branch block (RBBB) with either a left anterior or posterior fascicular block pattern (bifascicular or trifascicular block). The escape rhythm usually has a wide QRS complex and is unstable, and the event is associated with a high mortality from both arrhythmias and pump failure. In-hospital and 30-day mortality are higher than seen with an inferior wall infarction. (See 'Anterior MI' above.)

- **Management** – The initial management of the patient with post-MI conduction abnormalities depends on the presence and severity of any signs and symptoms of hemodynamic instability (algorithm 1). Mobitz type II second-degree AV block, high-grade AV block, or third-degree (complete) AV block are often unstable rhythms and more likely to cause, or to be associated with, hemodynamic instability. Patients with newly developed BBB, or alternating BBB, are also at increased risk of developing hemodynamic instability. Patients with Mobitz type I second-degree AV block, first-degree AV block, or isolated BBB rarely develop hemodynamic instability related to their heart rhythm. (See 'Management of conduction abnormalities' above.)

  - Patients with acute MI and second-degree AV block, high-grade AV block, or third-degree (complete) AV block who are hemodynamically unstable should be urgently

treated with atropine (if block is suspected to be at the AV nodal level) and generally temporary cardiac pacing should be instituted (either with transcutaneous or, if immediately available, transvenous pacing).

- For patients with acute inferior MI who are unresponsive to atropine, we suggest intravenous (IV) aminophylline (**Grade 2C**).
- Temporary transvenous pacing is generally warranted in patients with acute MI in the following settings (see 'Temporary pacing' above):
  - Complete (third-degree) AV block.
  - Alternating right and left bundle branch block (LBBB).
  - Right BBB with alternating left anterior and posterior fascicular block.
  - Asystole.
  - Symptomatic bradycardia of any etiology, including sinus bradycardia and Mobitz type I second-degree AV block, if adverse hemodynamic effects are present and the bradyarrhythmia is not responsive to atropine.
  - Mobitz type II second-degree AV block.
  - Bradycardia-induced tachyarrhythmias, such as torsades de pointes.
- In general, a waiting period of several days should be employed to determine potential reversibility of AV block, particularly in patients with acute inferior MI. If no reversible causes are present, definitive treatment of Mobitz type II second-degree AV block, high-grade AV block, or third-degree (complete) AV block involves permanent pacemaker placement in most patients. (See 'Second-degree AV block, high-grade AV block, or third-degree (complete) AV block' above.)

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges the late Mark E. Josephson, MD, who contributed to an earlier version of this topic review.

REFERENCES

1. Sánchez-Quintana D, Cabrera JA, Farré J, et al. Sinus node revisited in the era of electroanatomical mapping and catheter ablation. Heart 2005; 91:189.
2. Rosenbaum M, Elizari MV, Lazzari JO. The Hemiblocks, Tampa Tracings, Tampa 1970.
3. Uhley HN. Some controversy regarding the peripheral distribution of the conduction system. Am J Cardiol 1972; 30:919.
4. Hecht HH, Kossmann CE, Childers RW, et al. Atrioventricular and intraventricular conduction. Revised nomenclature and concepts. Am J Cardiol 1973; 31:232.
5. Demoulin JC, Kulbertus HE. Histopathological examination of concept of left hemiblock. Br Heart J 1972; 34:807.
6. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. N Engl J Med 2003; 348:933.
7. Stiles MK, Brooks AG, Roberts-Thomson KC, et al. High-density mapping of the sinus node in humans: role of preferential pathways and the effect of remodeling. J Cardiovasc Electrophysiol 2010; 21:532.
8. Boyett MR, Honjo H, Kodama I. The sinoatrial node, a heterogeneous pacemaker structure. Cardiovasc Res 2000; 47:658.
9. Wong CK, Stewart RA, Gao W, et al. Prognostic differences between different types of bundle branch block during the early phase of acute myocardial infarction: insights from the Hirulog and Early Reperfusion or Occlusion (HERO)-2 trial. Eur Heart J 2006; 27:21.
10. Go AS, Barron HV, Rundle AC, et al. Bundle-branch block and in-hospital mortality in acute myocardial infarction. National Registry of Myocardial Infarction 2 Investigators. Ann Intern Med 1998; 129:690.
11. Stenestrand U, Tabrizi F, Lindbäck J, et al. Comorbidity and myocardial dysfunction are the main explanations for the higher 1-year mortality in acute myocardial infarction with left bundle-branch block. Circulation 2004; 110:1896.
12. Meine TJ, Al-Khatib SM, Alexander JH, et al. Incidence, predictors, and outcomes of high-degree atrioventricular block complicating acute myocardial infarction treated with thrombolytic therapy. Am Heart J 2005; 149:670.
13. Gang UJ, Hvelplund A, Pedersen S, et al. High-degree atrioventricular block complicating ST-segment elevation myocardial infarction in the era of primary percutaneous coronary intervention. Europace 2012; 14:1639.
14. Auffret V, Loirat A, Leurent G, et al. High-degree atrioventricular block complicating ST segment elevation myocardial infarction in the contemporary era. Heart 2016; 102:40.

15. Kim HL, Kim SH, Seo JB, et al. Influence of second- and third-degree heart block on 30-day outcome following acute myocardial infarction in the drug-eluting stent era. Am J Cardiol 2014; 114:1658.
16. Harikrishnan P, Gupta T, Palaniswamy C, et al. Complete heart block complicating ST-segment elevation myocardial infarction. JACCEP 2015; 1:529.
17. Singh SM, FitzGerald G, Yan AT, et al. High-grade atrioventricular block in acute coronary syndromes: insights from the Global Registry of Acute Coronary Events. Eur Heart J 2015; 36:976.
18. Kosmidou I, Redfors B, Dordi R, et al. Incidence, Predictors, and Outcomes of High-Grade Atrioventricular Block in Patients With ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention (from the HORIZONS-AMI Trial). Am J Cardiol 2017; 119:1295.
19. Misumida N, Ogunbayo GO, Kim SM, et al. Frequency and Significance of High-Degree Atrioventricular Block and Sinoatrial Node Dysfunction in Patients With Non-ST-Elevation Myocardial Infarction. Am J Cardiol 2018; 122:1598.
20. Feigl D, Ashkenazy J, Kishon Y. Early and late atrioventricular block in acute inferior myocardial infarction. J Am Coll Cardiol 1984; 4:35.
21. Aplin M, Engstrøm T, Vejlstrup NG, et al. Prognostic importance of complete atrioventricular block complicating acute myocardial infarction. Am J Cardiol 2003; 92:853.
22. Hindman MC, Wagner GS, JaRo M, et al. The clinical significance of bundle branch block complicating acute myocardial infarction. 1. Clinical characteristics, hospital mortality, and one-year follow-up. Circulation 1978; 58:679.
23. Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. J Am Coll Cardiol 2019; 74:e51.
24. American College of Emergency Physicians, Society for Cardiovascular Angiography and Interventions, O'Gara PT, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013; 61:e78.
25. Panchal AR, Bartos JA, Cabañas JG, et al. Part 3: Adult Basic and Advanced Life Support: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2020; 142:S366.

26. Altun A, Kirdar C, Ozbay G. Effect of aminophylline in patients with atropine-resistant late advanced atrioventricular block during acute inferior myocardial infarction. Clin Cardiol 1998; 21:759.
27. Zei PC, Eckart RE, Epstein LM. Modified temporary cardiac pacing using transvenous active fixation leads and external re-sterilized pulse generators. J Am Coll Cardiol 2006; 47:1487.
28. Brignole M, Auricchio A, Baron-Esquivias G, et al. 2013 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy: the Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J 2013; 34:2281.
29. Yu CM, Chan JY, Zhang Q, et al. Biventricular pacing in patients with bradycardia and normal ejection fraction. N Engl J Med 2009; 361:2123.
30. Curtis AB, Worley SJ, Adamson PB, et al. Biventricular pacing for atrioventricular block and systolic dysfunction. N Engl J Med 2013; 368:1585.
31. Gregoratos G, Cheitlin MD, Conill A, et al. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol 1998; 31:1175.
32. Goldberg RJ, Zevallos JC, Yarzebski J, et al. Prognosis of acute myocardial infarction complicated by complete heart block (the Worcester Heart Attack Study). Am J Cardiol 1992; 69:1135.
33. Harpaz D, Behar S, Gottlieb S, et al. Complete atrioventricular block complicating acute myocardial infarction in the thrombolytic era. SPRINT Study Group and the Israeli Thrombolytic Survey Group. Secondary Prevention Reinfarction Israeli Nifedipine Trial. J Am Coll Cardiol 1999; 34:1721.
34. Berger PB, Ruocco NA Jr, Ryan TJ, et al. Incidence and prognostic implications of heart block complicating inferior myocardial infarction treated with thrombolytic therapy: results from TIMI II. J Am Coll Cardiol 1992; 20:533.
35. Clemmensen P, Bates ER, Califf RM, et al. Complete atrioventricular block complicating inferior wall acute myocardial infarction treated with reperfusion therapy. TAMI Study Group. Am J Cardiol 1991; 67:225.
36. Abidov A, Kaluski E, Hod H, et al. Influence of conduction disturbances on clinical outcome in patients with acute myocardial infarction receiving thrombolysis (results from the ARGAMI-2 study). Am J Cardiol 2004; 93:76.

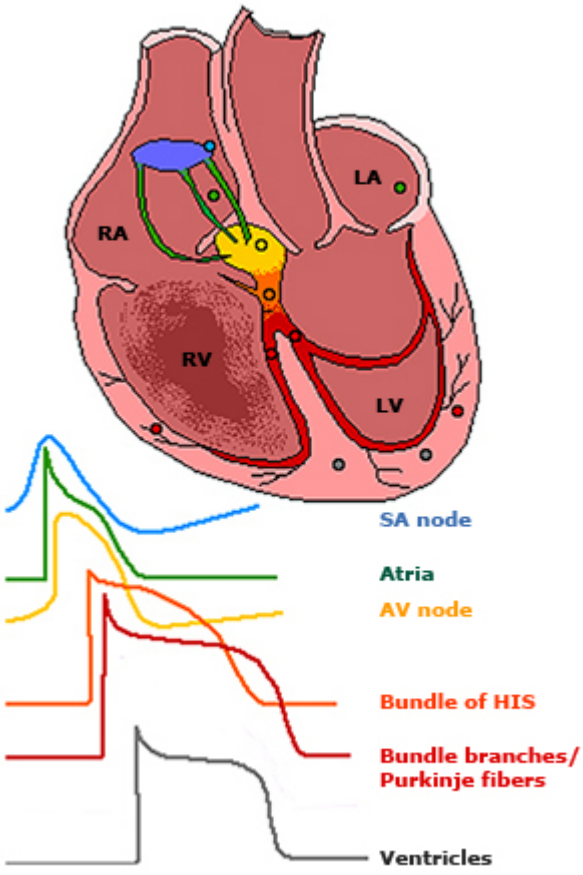
37. Kawamura Y, Yokoyama H, Kitayama K, et al. Clinical impact of complete atrioventricular block in patients with ST-segment elevation myocardial infarction. Clin Cardiol 2021; 44:91.
38. Godman MJ, Lassers BW, Julian DG. Complete bundle-branch block complicating acute myocardial infarction. N Engl J Med 1970; 282:237.
39. Hindman MC, Wagner GS, JaRo M, et al. The clinical significance of bundle branch block complicating acute myocardial infarction. 2. Indications for temporary and permanent pacemaker insertion. Circulation 1978; 58:689.
40. Scheinman MM, Gonzalez RP. Fascicular block and acute myocardial infarction. JAMA 1980; 244:2646.
41. Dubois C, Piérard LA, Smeets JP, et al. Short- and long-term prognostic importance of complete bundle-branch block complicating acute myocardial infarction. Clin Cardiol 1988; 11:292.
42. Sgarbossa EB, Pinski SL, Topol EJ, et al. Acute myocardial infarction and complete bundle branch block at hospital admission: clinical characteristics and outcome in the thrombolytic era. GUSTO-I Investigators. Global Utilization of Streptokinase and t-PA [tissue-type plasminogen activator] for Occluded Coronary Arteries. J Am Coll Cardiol 1998; 31:105.
43. Guerrero M, Harjai K, Stone GW, et al. Comparison of the prognostic effect of left versus right versus no bundle branch block on presenting electrocardiogram in acute myocardial infarction patients treated with primary angioplasty in the primary angioplasty in myocardial infarction trials. Am J Cardiol 2005; 96:482.
44. Ricou F, Nicod P, Gilpin E, et al. Influence of right bundle branch block on short- and long-term survival after acute anterior myocardial infarction. J Am Coll Cardiol 1991; 17:858.
45. Brilakis ES, Wright RS, Kopecky SL, et al. Bundle branch block as a predictor of long-term survival after acute myocardial infarction. Am J Cardiol 2001; 88:205.
46. Al Rajoub B, Noureddine S, El Chami S, et al. The prognostic value of a new left bundle branch block in patients with acute myocardial infarction: A systematic review and meta-analysis. Heart Lung 2017; 46:85.
47. Xiang L, Zhong A, You T, et al. Prognostic Significance of Right Bundle Branch Block for Patients with Acute Myocardial Infarction: A Systematic Review and Meta-Analysis. Med Sci Monit 2016; 22:998.
48. Melgarejo-Moreno A, Galcerá-Tomás J, Consuegra-Sánchez L, et al. Relation of New Permanent Right or Left Bundle Branch Block on Short- and Long-Term Mortality in Acute Myocardial Infarction Bundle Branch Block and Myocardial Infarction. Am J Cardiol 2015; 116:1003.

49. Widimsky P, Rohác F, Stásek J, et al. Primary angioplasty in acute myocardial infarction with right bundle branch block: should new onset right bundle branch block be added to future guidelines as an indication for reperfusion therapy? Eur Heart J 2012; 33:86.
50. Vivas D, Pérez-Vizcayno MJ, Hernández-Antolín R, et al. Prognostic implications of bundle branch block in patients undergoing primary coronary angioplasty in the stent era. Am J Cardiol 2010; 105:1276.

Topic 45 Version 30.0

GRAPHICS

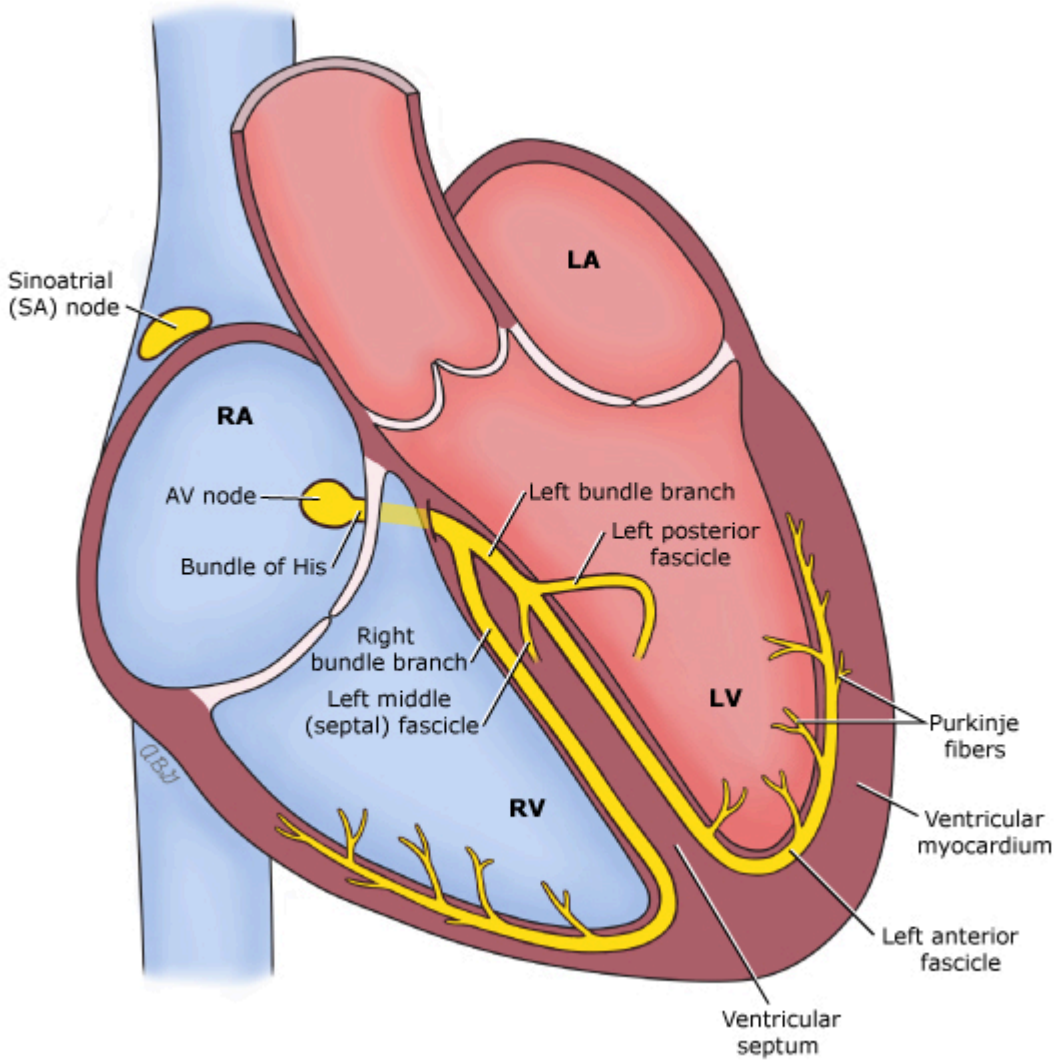
Action potentials generated by different parts of conduction system



The sinoatrial (SA) and atrioventricular (AV) nodes generate a slow action potential, mediated by calcium ions. In comparison, the tissues of the atria, ventricles, and the His-Purkinje system generate a fast action potential mediated by sodium ions. Sequential activation of these structures results in the characteristic waveforms visible on the surface electrocardiogram (ECG). The AV node and bundle of His are small structures; as a result, no electrical activity is recorded on the surface ECG during their activation.

Graphic 61989 Version 4.0

Normal conduction system

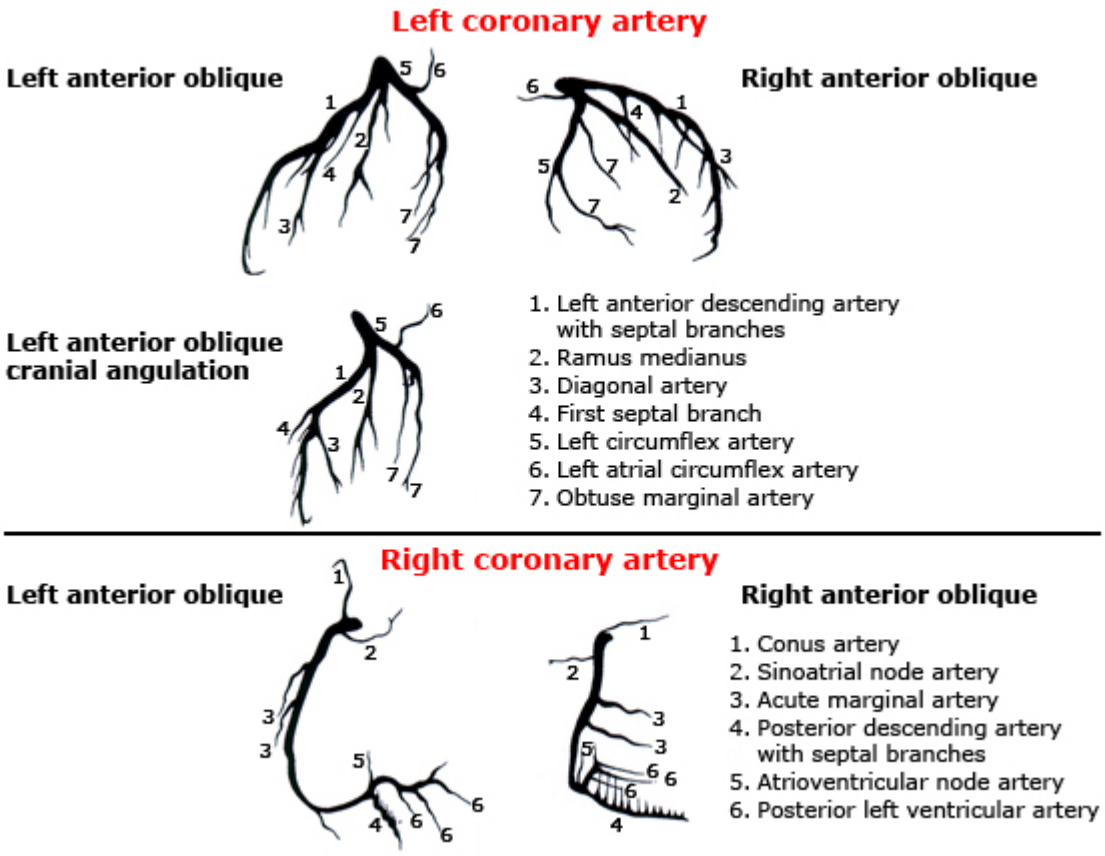


Schematic representation of the normal intraventricular conduction system (His-Purkinje system). The Bundle of His divides into the left bundle branch and right bundle branch. The left bundle branch divides into anterior, posterior, and, in some cases, median fascicles.

AV: atrioventricular; RA: right atrium; LA: left atrium; RV: right ventricle; LV: left ventricle.

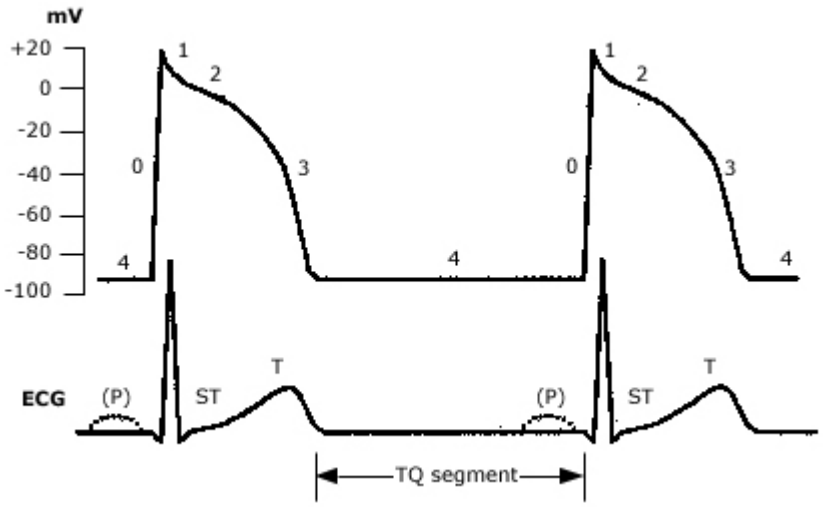
Graphic 63340 Version 6.0

Coronary artery anatomy



Graphic 70956 Version 2.0

Relationship between fast sodium-mediated myocardial action potential and surface electrocardiogram

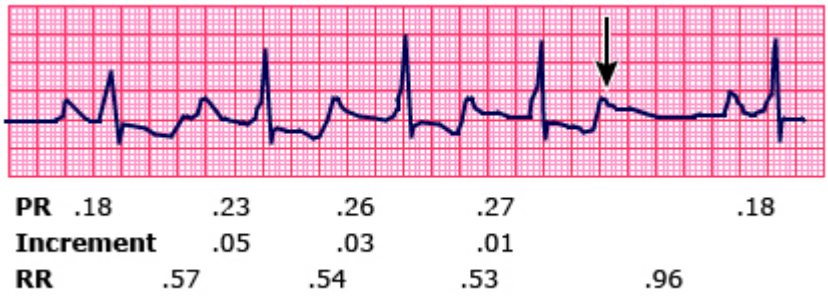


Each phase of the myocardial action potential (numbers, upper panel) corresponds to a deflection or interval on the surface ECG (lower panel). Phase 4, the resting membrane potential, is responsible for the TQ segment; this segment has a prominent role in the ECG manifestations of ischemia during exercise testing.

ECG: electrocardiogram.

Graphic 64133 Version 4.0

Electrocardiogram showing Mobitz type I (Wenckebach) atrioventricular block

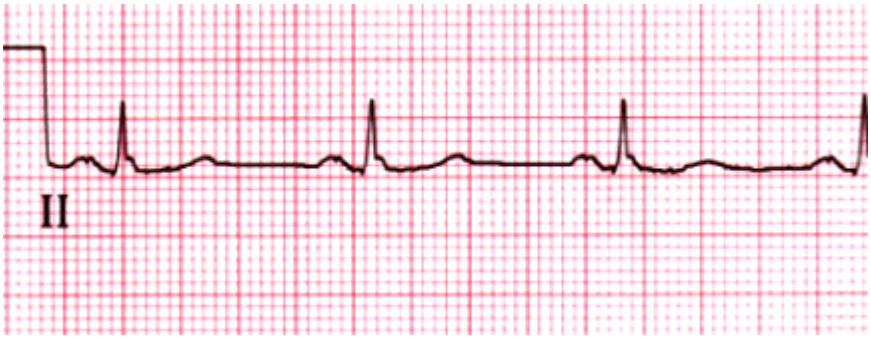


Single-lead electrocardiogram showing Mobitz type I (Wenckebach) second-degree atrioventricular block with 5:4 conduction. The characteristics of this arrhythmia include: a progressively increasing PR interval until a P wave is not conducted (arrow), a progressive decrease in the increment in the PR interval, a progressive decrease in the RR interval, and the RR interval that includes the dropped beat (0.96 s) is less than twice the RR interval between conducted beats (0.53 to 0.57 s).

Courtesy of Morton Arnsdorf, MD.

Graphic 73051 Version 7.0

Normal rhythm strip

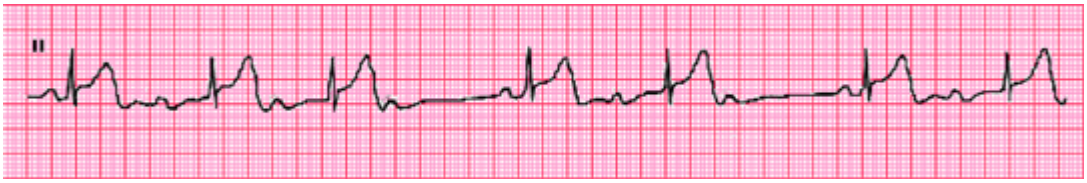


Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Electrocardiogram (ECG) showing concurrent Mobitz type I (Wenckebach) atrioventricular (AV) block and inferior myocardial infarction (MI)

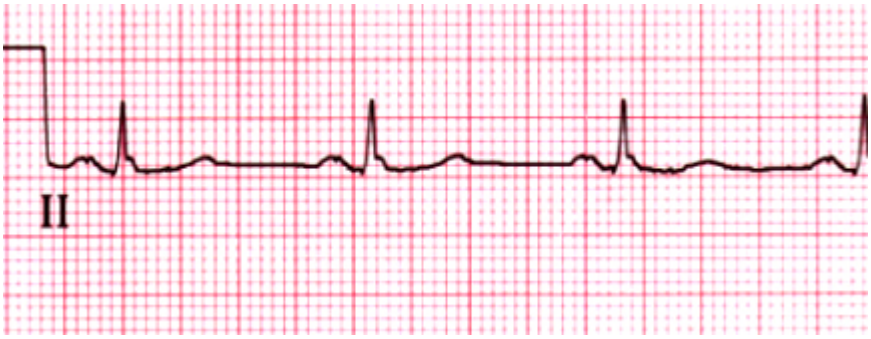


This rhythm strip shows a Mobitz type I (Wenckebach) atrioventricular block with 4:3 and 3:2 conduction and progressive prolongation of the PR intervals of conducted beats. The marked ST segment elevation suggests acute inferior wall ischemia or infarction that may be responsible for the arrhythmia.

Courtesy of Ary Goldberger, MD.

Graphic 62040 Version 3.0

Normal rhythm strip

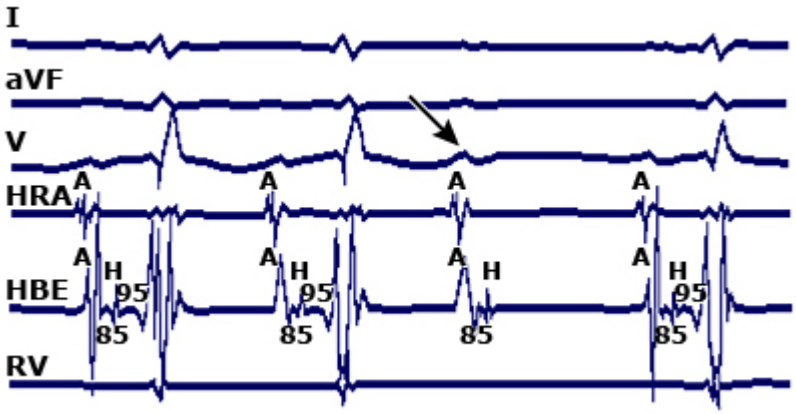


Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Electrocardiographic and electrophysiologic features of Mobitz type II second-degree atrioventricular block

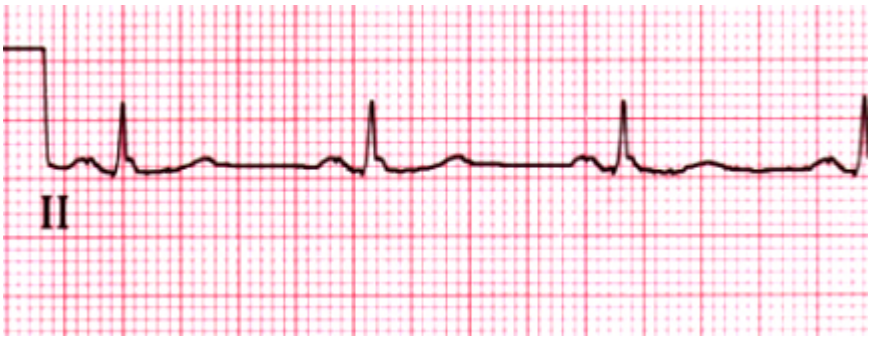


The PR and RR intervals are constant, but the third atrial beat (A) is not conducted (arrow). His bundle electrocardiography (HBE) shows constant AH (85 ms) and HV (95 ms) intervals and normal AH but no HV conduction in the nonconducted beat. The last finding indicates that the block is distal to the His bundle, in contrast with the more proximal location of Mobitz type I atrioventricular block.

Adapted from: Josephson ME, Clinical Cardiac Electrophysiology: Techniques and Interpretations, 2nd ed, Lea & Febiger, Philadelphia 1993.

Graphic 79539 Version 8.0

Normal rhythm strip

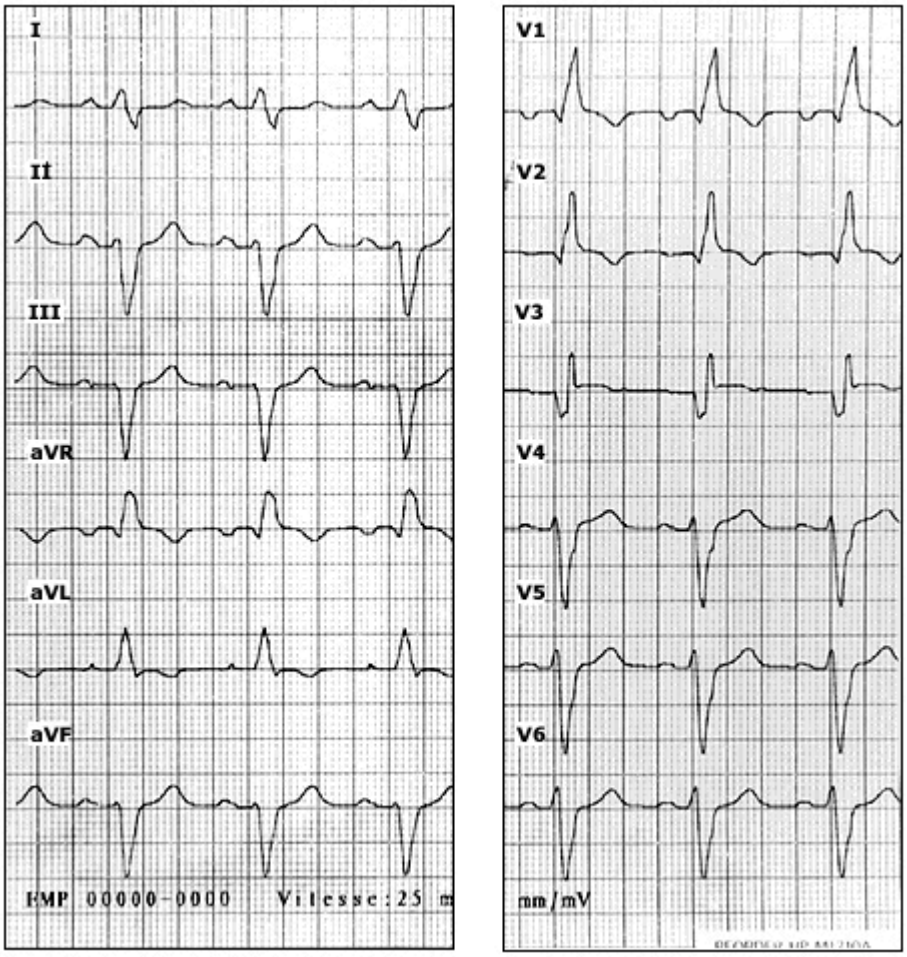


Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

12-lead electrocardiogram (ECG) showing bifascicular block with right bundle branch block and left anterior fascicular block

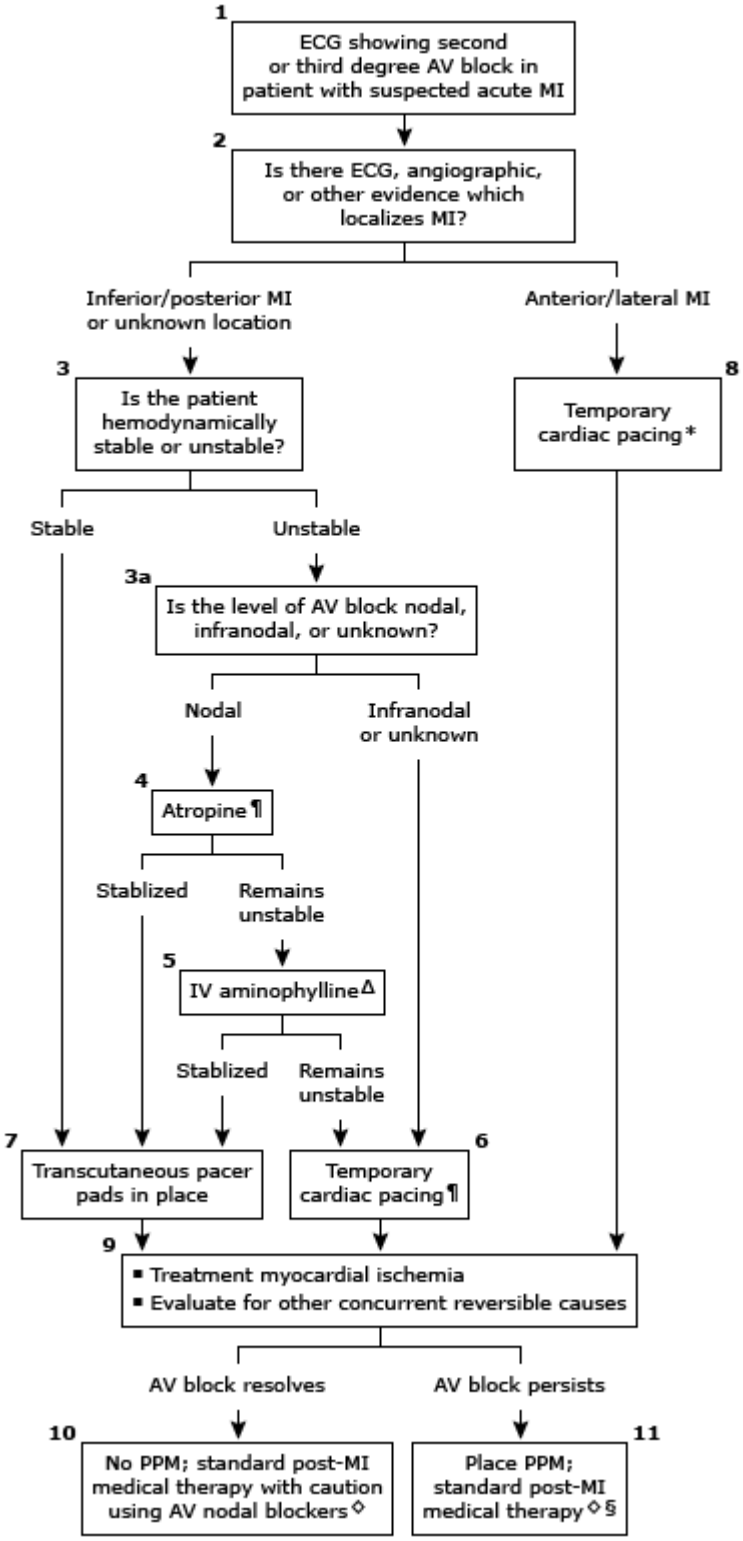


The 12-lead ECG from a patient with a history of an anteroseptal myocardial infarction (Q waves seen in lead V1-V3) shows bifascicular block with RBBB and LAFB. A typical RBBB is seen with a QRS duration of 0.16 seconds and an rSR' configuration in lead V1 and a deep S wave in V6. The QRS complexes in leads II, III, and avF are negative, with a rS morphology, diagnostic of a pathologic left axis deviation, known as LAFB.

ECG: electrocardiogram; RBBB: right bundle branch block; LAFB: left anterior fascicular block.

Graphic 69886 Version 4.0

Treatment of peri-myocardial infarction conduction abnormalities



ECG: electrocardiogram; AV: atrioventricular; MI: myocardial infarction; PPM: permanent pacemaker.

* Transcutaneous pacing is frequently the quickest way to provide temporary cardiac pacing, but is unreliable for extended periods of treatment and uncomfortable for the patient. While transcutaneous

pacing may be initially successful in stabilizing the patient, central venous access should be established and transvenous pacing provided in the vast majority of patients requiring temporary cardiac pacing.

¶ The initial dose of atropine is 0.5 mg IV. This dose may be repeated every three to five minutes to a total dose of 3 mg. If the patient does not respond promptly and/or clinically deteriorates, proceed directly to temporary pacing.

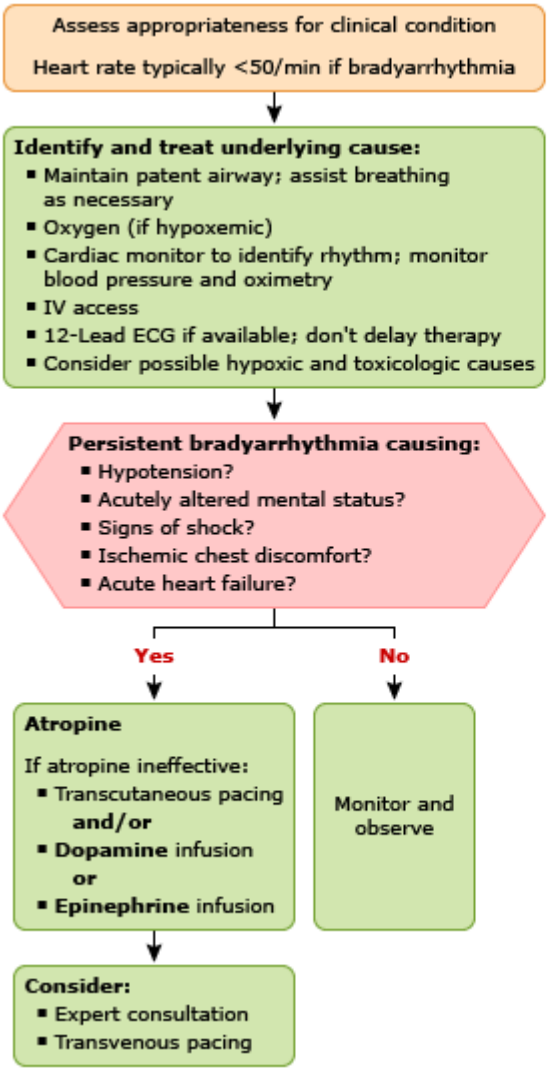
Δ The typical aminophylline dose is 250 mg IV bolus. In patients with a prior heart transplant, a higher weight-based dose is preferred (6 mg/kg IV mixed in 100 to 200 mL of IV fluid adminstered over 20 to 30 minutes).

◇ Refer to UpToDate content on management of acute MI.

§ In general, a waiting period of several days should be employed to determine potential reversibility of AV block, particularly in patients with acute inferior MI. If no reversible causes are present, definitive treatment involves PPM placement in most patients.

Graphic 120722 Version 1.0

Adult bradycardia algorithm 2020 update



Doses/Details
Atropine IV dose: First dose: 1 mg bolus. Repeat every 3 to 5 minutes. Maximum: 3 mg.
Dopamine IV infusion: Usual infusion rate is 5 to 20 mcg/kg per minute. Titrate to patient response; taper slowly.
Epinephrine IV infusion: 2 to 10 mcg per minute infusion. Titrate to patient response.
Causes: ▪ Myocardial ischemia/ infarction ▪ Drugs/toxicologic (eg, calcium-channel blockers, beta blockers, digoxin) ▪ Hypoxia ▪ Electrolyte abnormality (eg, hyperkalemia)

Graphic 130748 Version 11.0