

eMedicine Specialties > Cardiology > Arrhythmias

Atrioventricular Block

Chirag M Sandesara, MD, Fellow, Department of Internal Medicine, Division of Cardiovascular Diseases, University of Iowa Hospitals and Clinics

Brian Olshansky, MD, Professor of Medicine, Department of Internal Medicine, University of Iowa College of Medicine

Updated: Aug 3, 2009

Introduction

Background

Atrioventricular (AV) block occurs when the atrial depolarization fail to reach the ventricles or when atrial depolarization is conducted with a delay. Three degrees of AV block are recognized.

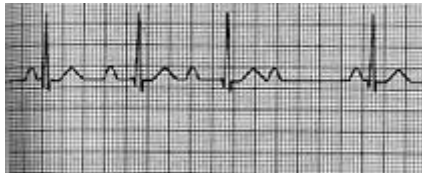
First-degree AV block consists of prolongation of the PR interval on the electrocardiogram (ECG) (>0.20 s in adults and >0.16 s in young children). The upper limit of the reference range for the PR interval is age-dependent in children. All atrial impulses reach the ventricles in first-degree AV block, however, conduction is delayed within the AV node (see Media file 1).



First-degree atrioventricular (AV) block. PR interval is constant and is 0.28 milliseconds.

Second-degree AV block is characterized by atrial impulses (generally occurring at a regular rate) failing to conduct to the ventricles in one of the following 4 ways.

- Mobitz I second-degree AV block (Wenckebach block) consists of progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause. The pause is shorter than the sum of any 2 consecutive conducted beats (R-R interval). An episode of Mobitz I AV block usually consists of 3-5 beats, with a ratio of nonconducted to conducted beats of 4:3, 3:2, and so on (see Media file 2). The block is generally in the AV node but can occasionally occur in the His-Purkinje system and is termed infranodal Wenckebach.



Second-degree atrioventricular (AV) block Mobitz type I (Wenckebach).
Note the prolongation of the PR interval prior to a dropped beat and a shortened PR interval following the dropped beat.

- Mobitz II second-degree AV block is characterized by a constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles, such that either an occasional dropped P wave or a regular conduction pattern of 2:1 (2 conducted and 1 blocked), 3:1 (3 conducted and 1 blocked), and so on is observed (see Media file 3).



Second-degree atrioventricular (AV) block Mobitz type II. A constant PR interval in conducted beats is present. An intraventricular conduction delay also is present.

- High-grade AV block consists of multiple P waves in a row that should conduct, but do not. The conduction ratio can be 3:1 or more and the PR interval of conducted beats is constant. It is a distinct form of complete AV block in that the P waves that conduct to the QRS complexes occur at fixed intervals. For complete AV block, no relationship exists between the P waves and QRS complexes.
- 2:1 AV block could be Mobitz I or Mobitz II, but distinguishing one form from the other is nearly impossible.

Third-degree AV block is diagnosed when no supraventricular impulses are conducted to the ventricles. P waves on the rhythm strip reflect a sinus node rhythm independent from QRS electrocardiographic wave complexes. The QRS complexes represent an escape rhythm, either junctional or ventricular. The escape rhythm originating from the junctional or high septal region is characterized by narrow QRS complexes at a rate of 40-50 beats per minute, whereas escape rhythm from low ventricular sites is characterized by broad QRS complexes at a rate of 30-40 beats per minute. No relationship exists between the rhythm of P waves and the rhythm of QRS complexes. The frequency of P waves (atrial rate) is higher than the frequency of QRS complexes (ventricular rate) (see Media file 4).



Third-degree or complete heart block. The atrial rate is faster than the ventricular rate, and no association exists between atrial and ventricular activity.

AV dissociation is a rhythm identified by atrial and ventricular activation occurring from different pacemakers. AV dissociation does not indicate the presence of AV block and is distinctly different. Ventricular activation may be from either junctional pacemakers or infranodal. AV dissociation can occur in the presence of intact AV conduction, especially when rates of the pacemaker, either junctional or ventricular, exceed the atrial rate. Third-degree AV block can occur with AV dissociation. However, in AV dissociation without AV block, the ventricular rate can exceed the atrial rate and conduction can occasionally occur dependent on the timing between the P wave and the QRS complex.

AV block may also occur in patients with atrial fibrillation. Regular R-R intervals are possible in the presence of AV block (generally at slow regular rates).

Read more on atrial fibrillation at Medscape's Atrial Fibrillation Resource Center.

Pathophysiology

The atrioventricular node (AVN) is part of the conduction system of the heart that allows electrical impulses to be transmitted from the sinus node via atrial tissue (intra-atrial fascicles) to the ventricles. The AV node consists of 3 parts—atrionodal (transitional zone), nodal (compact portion), and nodal-His (penetrating His bundle). The nodal part causes the slowest conduction. The AV node is supplied by the right coronary artery (90%) or by the circumflex artery (10%) and is innervated by both sympathetic and parasympathetic fibers. The AVN receives impulses anteriorly via the

intra-atrial fibers in the septum and posteriorly via the crista terminalis. Impulses arriving at the AVN are transmitted to the ventricle 1:1. As faster impulses arrive, the conduction to the ventricles slows; this is called decremental conduction.

The His-Purkinje system is composed of 2 bundles of Purkinje fibers (the left and right bundle) that conduct electrical impulses to allow rapid ventricular activation. The His-Purkinje system is yet another location where AV block may occur.

First-degree heart block and second-degree Mobitz I AV block are usually caused by a delay at the AV node level, whereas second-degree Mobitz II AV block is generally caused by blockage in the His bundle or lower in the conduction system. Third-degree AV block is caused by conduction disturbances in the AV node or the His-Purkinje system. In most cases of complete AV block, an escape rhythm originates from the ventricles, with wide QRS complexes at a low regular rate of 30-40 beats per minute. A higher anatomic location of the block results in a higher location of the escape rhythm pacemaker, a faster escape rhythm (40-60 beats per min in the region of His bundle), and a narrower QRS duration.

Frequency

United States

First-degree AV block can be found in healthy adults, and its incidence increases with age. At 20 years of age, the PR interval may exceed 0.20 seconds in 0.5-2% of healthy people. At age 60 years, more than 5% of healthy individuals have PR intervals exceeding 0.20 seconds.

Type II second-degree AV block (Mobitz II) is rare in healthy individuals, whereas type I second-degree AV block (Wenckebach) is observed in 1-2% of healthy young people, especially during sleep.

Congenital third-degree AV block is rare—1 case per 20,000 births. This form of heart block, in the absence of major structural abnormalities, is associated with maternal antibodies to Ro (SS-A) and La (SS-B) and secondary to maternal lupus. It is most commonly diagnosed between 18 and 24 weeks' gestation, and may be first, second, or third degree (complete). Mortality approaches approximately 20%; most surviving children require pacemakers.

AV blocks occur more frequently in people older than 70 years, especially in those who have structural heart disease. Approximately 5% of patients with heart disease have first-degree AV block, and about 2% have second-degree AV block. The incidence of third-degree AV block peaks after age 70 years (approximately 5-10%).

International

The international incidence is the same as incidence in the United States.

Mortality/Morbidity

Progressive degrees of AV block carry increasing morbidity and mortality.

AV blocks generally are not associated with major morbidity. However, the low heart rate observed in third-degree or Mobitz II AV block may lead to syncopal episodes with major injuries (eg, head trauma, hip fracture), exacerbation of congestive heart failure, or exacerbation of ischemic heart disease symptoms due to low cardiac output.

Cheng et al found that first-degree AV block (ie, PR interval >200 milliseconds) is associated with an increased risk of atrial fibrillation, pacemaker implantation, and all-cause mortality. In a prospective, community-based cohort of 7,575 individuals from the Framingham Heart Study (mean age, 47 y; 54% women) who underwent routine 12-lead electrocardiography in 1968-1974, 124 individuals had PR intervals >200 msec on the baseline examination. On follow-up of the cohort through 2007, individuals with first-degree AV block had a 2-fold adjusted risk of atrial fibrillation (hazard ratio [HR], 2.06; 95% CI, 1.36-3.12; $P < .001$), a 3-fold adjusted risk of pacemaker implantation (HR, 2.89; 95% CI, 1.83-4.57; $P < .001$), and a 1.4-fold adjusted risk of all-cause mortality (HR, 1.44; 95% CI, 1.09-1.91; $P = .01$). For all 3 outcomes, each 20-msec increment in PR was associated with an increase in risk.^[1]

Race

No racial proclivity exists in AV blocks.

Sex

- A 60% female preponderance exists in congenital third-degree AV block.
- For acquired third-degree AV block, a 60% male preponderance exists.

Age

The incidence of AV block increases with age. The incidence of third-degree AV block is highest in people older than 70 years (approximately 5-10% of patients with heart disease).

Clinical

History

- First-degree AV block is generally not associated with any symptoms and is usually an incidental finding on ECG. People with newly diagnosed first-degree AV block may be healthy individuals with high vagal tone (eg, well-conditioned athletes), or they may have a history of myocardial infarction or myocarditis. First-degree AV block also may represent the first sign of a degenerative process of the AV conduction system.
- Second-degree AV block usually is asymptomatic. However, in some patients, sensed irregularities of the heartbeat, presyncope, or syncope may occur. The latter usually is observed in more advanced conduction disturbances such as Mobitz II AV block. A history of medications that affect AV node function (eg, digitalis, beta-blockers, calcium channel blockers) may be contributory and should be obtained.

- Third-degree AV block frequently is symptomatic with fatigue, dizziness, lightheadedness, presyncope, and syncope being reported most commonly. Syncopal episodes due to slow heart rates are called Morgagni-Adams-Stokes (MAS) episodes in recognition of the pioneer work on syncope by these researchers in the 19th century. Patients with third-degree AV block may have associated symptoms of acute myocardial infarction either causing the block or related to reduced cardiac output from bradycardia in the setting of advanced atherosclerotic coronary artery disease.

Physical

- Routine physical examination does not lead to the diagnosis of first-degree AV block.
- Second-degree AV block may manifest on physical examination as bradycardia (especially Mobitz type II) and/or irregularity of heart rate (especially type I, Wenckebach).
- Third-degree AV block is associated with profound bradycardia unless the site of the block is located in the proximal portion of the AV node. Exacerbation of ischemic heart disease or congestive heart failure caused by AV block related bradycardia and reduced cardiac output may lead to specific clinically recognizable symptoms (eg, chest pain, dyspnea, confusion, pulmonary edema). Cannon *a* waves may be observed intermittently in the jugular venous pulsation when the right atrium contracts against a closed tricuspid valve due to atrioventricular dissociation.

Causes

Delay or lack of conduction through the AV node has multiple causes.

- First-degree and second-degree Mobitz I (Wenckebach) AV blocks may occur in healthy, well-conditioned people as a physiologic manifestation of high vagal tone. Mobitz I (Wenckebach) block also may occur physiologically at high heart rates (especially with pacing) due to increased refractoriness of the AV node, which protects against conducting a fast rhythm to the ventricles.
- AV block may be caused by acute myocardial ischemia or infarction. Inferior myocardial infarction may lead to third-degree block, usually at the AV node level, and by other mechanisms via the Bezold-Jarisch reflex. Anterior myocardial infarction usually is associated with third-degree block due to ischemia or infarction of bundle branches.
- Degenerative changes in the AV node or bundle branches (eg, fibrosis, calcification, infiltration) are the most common cause of nonischemic AV block. Lenegre-Lev syndrome is an acquired complete heart block due to idiopathic fibrosis and calcification of the electrical conduction system of the heart. It is most commonly seen in the elderly and is often described as senile degeneration of the conduction system and may lead to third-degree AV block. In 1999, degenerative changes in the AV conduction system were linked to mutations of the *SCN5A* sodium channel gene (mutations of the same gene may lead to congenital long QT syndrome type 3 and to Brugada syndrome).^[2]

- Infiltrative myocardial diseases resulting in AV block include sarcoidosis, myxedema, hemochromatosis, and progressive calcification related to mitral or aortic valve annular calcification.
- Endocarditis and other infections of the myocardium, such as Lyme disease with active infiltration of the AV conduction system, may lead to varying degrees of AV block.
- Systemic diseases, such as ankylosing spondylitis and Reiter syndrome, may affect the AV nodal conducting tissue.
- Surgical (eg, aortic valve replacement, congenital defect repair) or other therapeutic procedures (eg, AV ablation in patients with supraventricular arrhythmias, alcohol septal ablation in patients with obstructive hypertrophic cardiomyopathy) may cause AV block. Patients with corrected transposition of the great vessels have anterior displacement of the AV node and are prone to develop complete heart block during right heart catheterization or surgical manipulation.
- A variety of drugs may affect AV conduction. The most common drugs include digitalis glycosides, beta-blockers, calcium channel blockers, and other antiarrhythmic agents.

Differential Diagnoses

Myocardial Infarction

Syncope

Other Problems to Be Considered

Differentiate second-degree Mobitz I AV block from Mobitz II AV block.

Differentiate second-degree Mobitz II AV block from third-degree AV block.

Differentiate both Mobitz I and Mobitz II AV block from sinus nodal dysfunction.

Atrial fibrillation with a low heart rate may mask third-degree AV block.

Differentiate third-degree block from AV dissociation where the frequency of the ventricular rhythm exceeds the frequency of the atrial rhythm.

Workup

Laboratory Studies

- Laboratory testing is not usually indicated in patients with AV block.
- Levels of electrolytes and drugs (eg, digitalis) can be checked in the case of second-degree or third-degree AV block when suspicion of increased potassium level or drug toxicity exists.
- In cases when second-degree and third-degree AV block might be a manifestation of acute myocardial infarction, cardiac enzymes should be measured.
- If clinical evaluation suggests systemic illness, appropriate directed laboratory studies for infection, myxedema, or connective tissue disease should be performed.

Imaging Studies

- Routine imaging studies are not helpful in diagnosing AV blocks.
- Imaging studies (eg, echocardiography) might be useful in diagnosing underlying comorbidity (eg, aortic valve stenosis with calcification, wall motion abnormalities in acute ischemia, cardiomyopathy, congenital heart disease such as congenitally corrected transposition of the great vessels).

Other Tests

- Routine ECG recording and cardiac monitoring with careful evaluation of the relationship between P waves and QRS complexes are the standard tests leading to proper diagnosis of AV blocks.
- Identifying episodes of transient AV block with sudden pauses and/or low heart rate causing syncopal episodes may require 24-hour Holter monitoring, multiple ECG recordings, event (loop) ECG recordings, or, in selected cases, monitoring with implantable loop recorders (Reveal, Medtronic, Inc).
- Electrophysiologic testing is indicated in a patient with suspected AV block as the cause of syncope. The invasive recording of AH (atrium-His) and HV (His-ventricle) intervals may determine the degree of conduction abnormality and may guide decisions for pacemaker therapy.

Procedures

- As described, in selected cases, invasive diagnostic procedures may include implantation of loop recorders and electrophysiology testing.

Treatment

Medical Care

Pacing is the treatment of choice in patients with advanced or symptomatic heart block. No effective long-term medical therapy exists.

ACC/AHA/NASPE 2002 guidelines for implantation of cardiac pacemakers and antiarrhythmia devices^[3]

Indications for permanent pacing

First-degree heart block and second-degree Mobitz I AV block do not generally require treatment unless they cause symptoms and are not due to a reversible cause. If a drug overdose is a possible cause, the drug needs to be withheld (and its future use or dosage subsequently should be decreased or reconsidered). Small, uncontrolled trials have suggested some symptomatic and functional improvement by pacing of patients with PR intervals more than 0.30 seconds by decreasing the time for AV conduction. This is rare. Although echocardiographic or invasive techniques may be used to assess hemodynamic improvement before permanent pacemaker implantation, such studies are not required for evaluation of symptoms due to first-degree and second-degree Mobitz I AV block.

Second-degree Mobitz II heart block and third-degree AV block usually require temporary and/or permanent cardiac pacing. Type II second-degree AV block and a wide QRS complex indicate diffuse conduction system disease and constitute an indication for pacing even in the absence of symptoms. Mobitz II with a wide QRS may degenerate into third-degree heart block and is another reason to consider permanent pacing. In the setting of acute anterior myocardial infarction, transcutaneous pacing initially and transvenous pacing subsequently are warranted.

With inferior myocardial infarction, the block usually resolves spontaneously within several days, and only a small percentage of patients require temporary or permanent pacing. Patients with persistent bundle branch block and transient third-degree AV block may benefit from permanent pacing therapy, especially after anterior myocardial infarction. Nonrandomized studies strongly suggest that permanent pacing does improve survival in patients with third-degree AV block, especially if syncope has occurred.

- Class I
 - Third-degree and advanced second-degree AV block at any anatomic level, associated with any one of the following conditions:
 - Bradycardia with symptoms (including heart failure) presumed to be due to AV block (*level of evidence: C*)
 - Arrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia (*level of evidence: C*)
 - Documented periods of asystole greater than or equal to 3.0 seconds or any escape rate less than 40 bpm in awake, symptom-free patients (*level of evidence: B, C*)
 - After catheter ablation of the AV junction (*level of evidence: B, C*): No trials have assessed outcome without pacing, and pacing is virtually always planned in this situation unless the operative procedure is AV junction modification.
 - Postoperative AV block that is not expected to resolve after cardiac surgery (*level of evidence: C*)
 - Neuromuscular diseases with AV block, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb dystrophy (limb-girdle), and peroneal muscular atrophy, with or without symptoms, because unpredictable progression of AV conduction disease may occur (*level of evidence: B*)
 - Second-degree AV block, regardless of type or site of block, with associated symptomatic bradycardia (*level of evidence: B*)
- Class IIa
 - Asymptomatic third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster, especially if cardiomegaly or LV dysfunction is present (*level of evidence: B, C*)
 - Asymptomatic type II second-degree AV block with a narrow QRS: When type II second-degree AV block occurs with a wide QRS, pacing becomes a Class I recommendation (*level of evidence: B*).
 - Asymptomatic type I second-degree AV block at intra- or infra-His levels found on electrophysiologic study performed for other indications (*level of evidence: B*)

- First- or second-degree AV block with symptoms similar to those of pacemaker syndrome (*level of evidence: B*)
- Class IIb
 - Marked first-degree AV block (>0.30 seconds) in patients with LV dysfunction and symptoms of congestive heart failure in whom a shorter AV interval results in hemodynamic improvement, presumably by decreasing left atrial filling pressure (*level of evidence: C*)
 - Neuromuscular diseases such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb dystrophy (limb-girdle), and peroneal muscular atrophy with any degree of AV block (including first-degree AV block) with or without symptoms, because unpredictable progression of AV conduction disease may occur (*level of evidence: B*)

In general, the decision regarding implantation of a pacemaker must be considered with respect to whether or not AV block is permanent. Reversible causes of AV block, such as electrolyte abnormalities, if present, should be corrected first. Some diseases may follow a natural history to resolution (eg, Lyme disease), and some AV block can be expected to reverse (eg, hypervagotonia due to recognizable and avoidable physiologic factors, perioperative AV block due to hypothermia, or inflammation near the AV conduction system after surgery in this region).

Conversely, some conditions may warrant pacemaker implantation owing to the possibility of disease progression even if the AV block reverses transiently (eg, sarcoidosis, amyloidosis, neuromuscular diseases). Finally, permanent pacing for AV block after valve surgery follows a variable natural history, and, therefore the decision for permanent pacing is at the physician's discretion.

Types of cardiac pacemakers implanted in patients with heart block may include ventricular (usually VVI) or dual chamber (usually DDD) modes of pacing. The cardiologist or electrophysiologist should make the decision regarding the most optimal mode of pacing.

Surgical Care

Pacemaker implantation is a routine surgical procedure, generally performed with local anesthesia in the electrophysiology lab. The procedure usually requires an overnight observation period in the hospital.

Consultations

Consultation with a cardiologist or cardiac electrophysiologist is indicated in the case of advanced heart block or unexplained syncope. An electrophysiologist must be consulted when invasive electrophysiology study is needed to determine the level and/or magnitude of conduction disturbance.

Activity

- Advanced heart block, such as Mobitz II or third-degree AV block, may become more symptomatic with increased activity, where an actual increase in block and decrease in effective heart rate may occur.

- Exercise may be used to evaluate 2:1 heart block and differentiate Mobitz I (where the conducted rate increases) from second-degree Mobitz type II heart block (where the block becomes more significant and often symptomatic).
- Restrictions after permanent pacemaker implantation include restricted weight lifting with the ipsilateral hand/arm to the pacemaker until healing occurs (approximately 6 wk). Contact sports are restricted unless a protective shield is worn over the implanted pacemaker. Electromagnetic interference, from power lines and arc welding for example, may cause inhibition of pacing. This is problematic for patients who are pacemaker dependent.

Medication

Long-term medical therapy is not indicated in AV block. Permanent pacing is the therapy of choice in advanced AV block, and it does not require concomitant medical therapy. Sometimes AV nodal blocking medications that contribute to heart block can be discontinued if not necessary. Temporary transcutaneous or transvenous pacing is the treatment of choice for an emergency involving a slow heart rate (and for asystole) caused by AV blocks. Atropine administration (0.5–1.0 mg) may improve AV conduction in emergencies where bradycardia is caused by a proximal AV block (located in the AV node) but may worsen conduction if the block is in the His-Purkinje system.

Anticholinergic agents

The goal is to improve conduction through the AV node by reducing vagal tone via muscarinic receptor blockade. This is only effective if the site of block is within the AV node. For patients with suspected infranodal block, this therapy is ineffective and may make the level of the block worse if it is in the His bundle or below.

Atropine (Atropair, Atropisol)

Increases AV conduction. Insufficient dose may cause paradoxical slowing of the heart rate.

Dosing

Adult

0.5-1 mg IV bolus; not to exceed 0.04 mg/kg (maximum 0.04 mg/kg)

Pediatric

0.1 mg IV; usual bolus 0.02 mg/kg IV

Interactions

Coadministration with other anticholinergics has additive effects; pharmacologic effects of atenolol and digoxin may increase with atropine; levodopa, phenothiazine, and agents with cholinergic mechanisms decrease effect; thiazides and amantadine increase effect and toxicity

Contraindications

Documented hypersensitivity; thyrotoxicosis; narrow-angle glaucoma; tachycardia

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Avoid in Down syndrome and/or children with brain damage to prevent hyperreactive response; avoid in coronary heart disease, tachycardia, congestive heart failure, cardiac arrhythmias, and hypertension; caution in peritonitis, ulcerative colitis, hepatic disease, and hiatal hernia with reflux esophagitis; in prostatic hypertrophy, dysuria may occur with prostatism, and catheterization may be required; in acute MI and congestive heart failure, may cause an exacerbation of symptoms and precipitate ventricular arrhythmias

Isoproterenol (Isuprel)

Has beta1- and beta2-adrenergic receptor activity. Binds beta-receptors of heart, smooth muscle of bronchi, skeletal muscle, vasculature, and alimentary tract. Has positive inotropic and chronotropic actions.

Dosing

Adult

Dilute 1 mL of 1:5000 solution (0.2 mg) to 10 mL with sodium chloride or 5% dextrose injection and administer an initial dose of 0.02-0.06 mg IV (1-3 mL of diluted solution); for subsequent doses, administer 0.01-0.2 mg IV (0.5-10 mL of diluted solution) to achieve heart rate of 90-100 beats/min

Alternatively, dilute 10 mL of 1:5000 solution (2 mg) in 500 mL of D5W or dilute 5 mL of 1:5000 solution (1 mg) in 250 mL of D5W and administer 5 mcg/min (1.25 mL/min of diluted solution) to achieve heart rate of 90-100 beats/min

Pediatric

Not established

American Heart Association recommends an initial infusion rate of 0.1 mcg/kg/min, with usual range being 0.1-1 mcg/kg/min

Interactions

Bretylium increases action of vasopressors on adrenergic receptors, which may in turn result in arrhythmias; guanethidine may increase effect of direct-acting vasopressors, possibly resulting in severe hypertension; tricyclic antidepressants may potentiate pressor response of direct-acting vasopressors

Contraindications

Documented hypersensitivity; tachyarrhythmias; tachycardia or heart block caused by digitalis intoxication; ventricular arrhythmias that require inotropic therapy; angina pectoris

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

By increasing myocardial oxygen requirements while decreasing effective coronary perfusion, isoproterenol may have a deleterious effect on the injured or failing heart; in some patients, presumably with organic disease of the AV node and its branches, isoproterenol paradoxically may worsen heart block or precipitate Adams-Stokes attacks; caution in coronary artery disease, coronary insufficiency, diabetes, or hyperthyroidism and sensitivity to sympathomimetic amines; if heart rate exceeds 110 beats per min, decreasing infusion rate or temporarily discontinuing infusion may be advisable

Follow-up

Further Inpatient Care

- Patients with first-degree and benign second-degree Mobitz I AV block do not require hospitalization.
- Patients with symptomatic second- or third-degree AV block require hospitalization with telemetry monitoring. Transcutaneous or transvenous pacing should be utilized, and indications for permanent pacing need to be determined.

Further Outpatient Care

- Patients with first- and second-degree Mobitz I AV block may require follow-up ECGs or Holter monitoring to determine the likelihood and rate of progression of the AV conduction disorder.
- Patients with implanted pacemakers require routine follow-up to monitor pacemaker function.

Inpatient & Outpatient Medications

No long-term medication is needed.

Transfer

Symptomatic patients with advanced second- or third-degree AV block with slow heart rate require temporary pacing while being transferred to a specialized medical center.

Complications

- Sudden death due to asystole or secondary to polymorphic ventricular tachyarrhythmias
- Cardiovascular collapse with syncope, aggravation of ischemic heart disease, congestive heart failure, exacerbation of renal disease
- Head and musculoskeletal injuries during syncopal episodes
- Pacing therapy (temporary or permanent) may be complicated acutely by tamponade, hemothorax, or pneumothorax. Dysfunction of the pacemaker, lead fracture, and malfunction (eg, inappropriate capture or sensing) are infrequent complications of pacing therapy. Infection of the pacemaker or lead wires is a rare, but important, short-term and long-term complication of pacemaker implantation.

Prognosis

Patients treated with permanent pacing due to AV blocks have an excellent prognosis. Patients with advanced AV blocks who are not treated with permanent pacing remain at high risk of sudden cardiac death.

Patient Education

Patients with implanted pacemakers require additional education, with particular emphasis on situations involving exposure to magnetic and electrical fields (eg, airport security gates) and training regarding transtelephonic monitoring of pacemaker function.

Miscellaneous

Medicolegal Pitfalls

- Failure to interpret the ECG correctly and make the proper diagnosis of AV block
- Failure to initiate temporary pacing in a timely fashion in emergency situations of AV block with low heart rate or progressive AV block during myocardial infarction
- Failure to hospitalize a patient in a unit with continuous cardiac monitoring
- Failure to recommend and implant a permanent pacemaker in the setting of class I (and usually class II) indications

- Failure to restrict driving in a patient who chooses not to receive pacemaker therapy for high-grade AV block