# Third-degree (complete) atrioventricular block

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Literature review current through: **Jan 2024.**
This topic last updated: **Oct 13, 2022.**

## INTRODUCTION

Atrioventricular (AV) block is defined as a delay or interruption in the transmission of an impulse from the atria to the ventricles due to an anatomical or functional impairment in the conduction system. The conduction disturbance can be transient or permanent, with conduction that is delayed, intermittent, or absent. Commonly used terminology includes:

- **First-degree AV block** – Slowed conduction without missed beats.
- **Second-degree AV block** – Missed beats, often in a regular pattern (eg, 2:1, 3:2), or higher degrees of block, which is further classified into Mobitz type I (Wenckebach) and Mobitz II AV block.
- **Third-degree (complete AV) block** – No atrial impulses reach the ventricle.
- **High-grade AV block** – Intermittent atrial conduction to the ventricle with two or more consecutive blocked P waves but without complete AV block.

The clinical presentation, evaluation, and management of acquired third-degree (complete) AV block will be discussed here. Congenital third-degree (complete) heart block, the etiology of AV block in general, and the management of other specific types of AV block are discussed separately. (See "Etiology of atrioventricular block" and "Congenital third-degree (complete) atrioventricular block" and "First-degree atrioventricular block" and "Second-degree

atrioventricular block: Mobitz type I (Wenckebach block)" and "Second-degree atrioventricular block: Mobitz type II".)

## ETIOLOGY

The potential etiologies of third-degree (complete) AV block are similar to lesser degrees of AV block and include reversible causes (both pathologic and iatrogenic) as well as idiopathic causes ( table 1). Common potentially reversible causes include:

- **Pathologic** – Myocardial ischemia (acute or chronic) involving the conduction system, cardiomyopathy (eg, amyloidosis, sarcoidosis), myocarditis (eg, Lyme disease or COVID-19), endocarditis with abscess formation, hyperkalemia, profound hypothyroidism (myxedema), and hypervagotonia.

- **Iatrogenic** – Medication-related (AV nodal blocking medications), post-cardiac surgery, post-catheter ablation, post-transcatheter aortic valve implantation.

Other pathologic causes may be progressive or irreversible (eg, infiltrative malignancies, neuromuscular diseases). However, in half of more of the cases, no specific reversible causes are identified, and the block is felt to be related to idiopathic progressive cardiac conduction disease with myocardial fibrosis and/or sclerosis that affects the conduction system. A more extensive discussion of the etiology of AV block is presented separately. Congenital complete heart block is generally irreversible. (See "Etiology of atrioventricular block".)

## CLINICAL PRESENTATION AND EVALUATION

The clinical presentation of third-degree (complete) AV block is variable depending upon the rate of the underlying escape rhythm and the presence of comorbid conditions. The evaluation of all patients with suspected third-degree (complete) AV block includes a thorough history, including medications and recent changes in medications, along with a 12-lead electrocardiogram (ECG) and bloodwork (which includes serum electrolytes and thyroid-stimulating hormone [TSH]).

**Clinical history** — All patients with suspected third-degree (complete) AV block should be questioned about any history of heart disease, both congenital and acquired, as well as any recent cardiac procedures that could predispose to AV conduction abnormalities. Patients without known cardiac disease should be questioned about other systemic diseases associated with heart block (eg, amyloidosis, sarcoidosis). Patients who live in an area with endemic Lyme

disease should be questioned about any recent outdoor exposure to ticks or known tick bites. Congenital heart block can be associated with maternal lupus. (See 'Etiology' above and "Congenital third-degree (complete) atrioventricular block".)

Patients with suspected third-degree (complete) AV block that occurs in the setting of acute myocardial ischemia or infarction should undergo concurrent diagnosis and treatment for both conditions. (See "Conduction abnormalities after myocardial infarction", section on 'Management of conduction abnormalities'.)

Patients should provide a full list of medications and be questioned about any recent changes in dosing, with particular attention paid to drugs that alter AV nodal conduction (ie, beta blockers, non-dihydropyridine calcium channel blockers, digoxin, select antiarrhythmic drugs).

In patients under 60 years of age who present with otherwise unexplained heart block, previously undetected cardiac sarcoidosis has been identified in up to 25 to 35 percent of patients [1,2]. Such patients with otherwise unexplained complete heart block should be evaluated for cardiac sarcoidosis [3]. (See "Clinical manifestations and diagnosis of cardiac sarcoidosis".)

**Signs and symptoms** — Nearly all patients with third-degree (complete) AV block will present with some degree of symptoms, though the severity of the symptoms can be quite variable. Symptoms may include:

- Fatigue
- Dyspnea
- Chest pain
- Presyncope or syncope
- Sudden cardiac arrest

Most patients will present with some level of fatigue and/or dyspnea. These symptoms result from the reduced cardiac output associated with the slower ventricular rate (40 beats per minute or less) of most escape rhythms. Infrequently, patients with a faster escape rhythm (50 to 60 beats per minutes) may have minimal or no symptoms. Conversely, patients with a slower escape rhythm (30 beats per minute or less) are more likely to present with syncope. The absence of any escape rhythm may rarely lead to sudden cardiac death.

The new onset of bradycardia associated with third-degree (complete) AV block may also exacerbate comorbid conditions. Patients with underlying coronary heart disease or heart failure may present with abrupt worsening of their typical angina or heart failure symptoms.

Very few patients will be entirely asymptomatic with third-degree (complete) AV block. The complete absence of symptoms is likely to be seen only in relatively young, otherwise healthy patients with a high junctional escape rhythm and a ventricular heart rate greater than 40 beats per minute.

Patients with third-degree (complete) AV block present with bradycardia but otherwise have few specific physical examination findings. Patients may appear pale or diaphoretic related to the abrupt reduction in cardiac output. Patients with underlying heart failure that is exacerbated by the development of heart block may have crackles on lung examination, elevated jugular venous pulsations, and/or peripheral edema.

**Electrocardiographic findings** — Patients with third-degree (complete) AV block will have evidence of atrial (P waves) and ventricular (QRS complexes) activity which are independent of each other on the surface electrocardiogram (ECG) ( waveform 1). In nearly all cases, the atrial rate will be faster than the ventricular escape rate, and there will be no association between the P waves and QRS complexes. As a general rule, the more distal the level of block and the resulting escape rhythm, the slower the ventricular rate will be. Junctional rhythms tend to have a ventricular rate between 40 and 60 beats per minute, while ventricular escape rhythms typically have rates of 40 beats per minute or less and often are unstable.

Escape rhythms occur when a pacemaker other than the sinus node has sufficient time to depolarize, attain threshold, and produce a depolarization. In third-degree (complete) AV block, the escape rhythm that controls the ventricles can occur at any level below that of the conduction block and the morphology of the QRS complex can help to determine the location where this is occurring [4-6].

- If third-degree AV block occurs within the AV node, about two-thirds of the escape rhythms have a narrow QRS complex (ie, a junctional or AV nodal rhythm) ( waveform 1) [7-9].
- Block at the level of the bundle of His is also typically associated with a narrow QRS complex.
- Patients with infrahisian block have a subjunctional escape rhythm with a wide QRS complex ( waveform 2).

If the escape rhythm has a normal QRS duration of less than 120 msec, the block occurs with almost equal frequency in the AV node and the bundle of His [7]. In comparison, involvement of these sites is infrequent with a prolonged QRS; the block in this setting is in the fascicles or bundle branches in over 80 percent of cases [7].

Patients in whom the development of third-degree (complete) AV block exacerbates underlying coronary heart disease may have ECG changes consistent with myocardial ischemia (eg, ST segment and T wave changes).

**Differential diagnosis of ECG findings** — Third-degree (complete) AV block has a relatively unique appearance on the ECG, with evidence of atrial (P waves) and ventricular (QRS complexes) activity which are independent of each other and an atrial rate faster than the ventricular rate. Rarely, complete AV block can occur in which the atrial rate is exactly twice the ventricular rate (eg, atrial rate of 80 beats per minute with a ventricular rate of 40 beats per minute), in which case the appearance on ECG could be similar to that of second-degree AV (ie, 2:1) block. However, any slight variation in the exact multiples should result in variations on the ECG that allow the distinction between third-degree (complete) AV block and second-degree AV block.

**Electrophysiology study** — With a very few select exceptions, electrophysiologic studies (EPS) are not necessary in patients with complete AV block [10,11]. Among patients with complete AV block, EPS may be indicated when symptoms are not present, the site of block is not apparent, or the block is potentially reversible. (See "Lyme carditis", section on 'Atrioventricular conduction abnormalities'.)

## DIAGNOSIS

In nearly all cases, the diagnosis of third-degree (complete) AV block can be made in a patient with suggestive symptoms (eg, fatigue, dyspnea, presyncope, and/or syncope) by obtaining a surface electrocardiogram (ECG), ideally a full 12-lead ECG but sometime a single-lead rhythm strip is adequate if a full 12-lead ECG cannot be obtained. For the rare patient with a nondiagnostic surface ECG, invasive electrophysiology studies can definitively diagnose third-degree (complete) AV block and accurately identify the level of the block.

## MANAGEMENT

The initial management of the patient with third-degree (complete) AV block depends on the presence and severity of any signs and symptoms related to the ventricular escape rhythm ( algorithm 1). Unstable patients require immediate pharmacologic therapy and, in most 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 should occur, followed by placement of a permanent pacemaker for patients without an identifiable reversible etiology [11].

**Unstable patients** — Patients with third-degree (complete) AV block who are hemodynamically unstable should be urgently treated ( algorithm 1) with atropine, beta-adrenergic agonists, and/or temporary cardiac pacing (either with transcutaneous or, if immediately available, transvenous pacing). Beta-adrenergic agonists may be helpful, particularly in patients with block at the AV node. (See "Advanced cardiac life support (ACLS) in adults", section on 'Bradycardia'.)

The most important clinical determination in a patient presenting with a third-degree (complete) AV block is whether or not the patient is hemodynamically unstable due to the resulting bradycardia and reduced cardiac output. Signs and symptoms of hemodynamic instability include hypotension, altered mental status, signs of shock, ongoing ischemic chest pain, and evidence of acute pulmonary edema. Such patients should be treated according to the Advanced Cardiac Life Support protocol for patients with symptomatic bradycardia ( algorithm 2) [12]:

- Atropine should be promptly administered if intravenous (IV) access is available, but treatment with atropine should not delay treatment with transcutaneous pacing or a chronotropic agent. The initial dose of atropine is 1 mg IV. This dose may be repeated every three to five minutes to a total dose of 3 mg. 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. Atropine is contraindicated in patients with closed angle glaucoma, pyloric stenosis, myasthenia gravis, urinary retention/bladder obstruction, and other conditions.

- Temporary cardiac pacing should be provided. 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. (See "Temporary cardiac pacing".)

- In patients with hypotension associated with third-degree (complete) AV block, we administer dopamine via IV infusion, beginning at a dose of 5 mcg/kg/minute and titrating up to 20 mcg/kg/minute if needed for heart rate and blood pressure augmentation [13]. Our contributors do not use epinephrine in this setting but others do ( algorithm 2).

Isoproterenol can also be used, with an initial infusion of 1 to 5 mcg/min, which is titrated to as high as 20 mcg/min based upon heart rate response.

- In patients with heart failure with reduced ejection fraction (HFrEF) associated with third-degree (complete) AV block, we administer dobutamine via IV infusion, beginning at a dose of 2 to 5 mcg/kg/minute and titrating as needed until an optimal clinical and hemodynamic response is achieved. The usual maintenance dose of dobutamine is 2 to 10 mcg/kg/min (maximum 20 mcg/kg/min).

Once a hemodynamically unstable patient has been stabilized, the approach to further management is the same as for patients who were initially stable. (See 'Stable patients' below.)

**Stable patients** — Patients with third-degree (complete) AV block who are hemodynamically stable 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). In addition, most stable patients continue to have symptoms related to the bradycardia and will require identification and treatment of any reversible causes or permanent therapy with an implantable pacemaker.

While stable patients are being monitored, evaluation and treatment should proceed as follows:

- Reversible causes of third-degree (complete) AV block such as myocardial ischemia, increased vagal tone, hypothyroidism, hyperkalemia, and drugs that depress conduction, should be excluded in patients prior to implantation of a permanent pacemaker.

  - Patients with third-degree (complete) AV block in the setting of an acute myocardial infarction should be treated with temporary pacing and revascularization; following revascularization, most conduction abnormalities will improve or resolve and will not require permanent pacing. (See "Conduction abnormalities after myocardial infarction", section on 'Summary and recommendations'.)

  - Patients with third-degree (complete) AV block felt to be medication-induced should be observed while the offending agent or agents are withdrawn; such patients will often have improvement or resolution of AV block following removal of the medication. If the medication is deemed necessary, permanent pacing is indicated.

  - Patients with third-degree (complete) AV block in the setting of hyperkalemia should receive therapy to reduce serum potassium levels; similarly, patients with hypothyroidism should receive thyroid replacement therapy. If third-degree (complete)

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".)

- Patients with Lyme carditis and associated heart block frequently do not require permanent cardiac pacing. Third-degree (complete) AV block typically improves to lesser degrees of AV block within one week, and more minor conduction disturbances usually resolve within six weeks. As such, while these patients may initially require temporary cardiac pacing, permanent cardiac pacing should be reserved for patients with persistent third-degree (complete) AV block following an adequate course of therapy for Lyme disease. (See "Lyme carditis".)

- Patients who develop complete heart block after TAVR generally require permanent pacemakers, and should initially be treated with temporary transvenous pacing. Guidelines for patients with less severe conduction abnormalities after TAVR have been developed [14]. (See "Transcatheter aortic valve implantation: Complications".)

- Transient vagally mediated heart block, often seen on hospital telemetry during sleep, generally does not require specific intervention. Pacing is controversial in vagally mediated syncope, particularly in younger patients.

If no reversible causes are present, definitive treatment of third-degree (complete) AV block generally involves permanent pacemaker placement [10,11]. Dual-chamber (ie, atrioventricular) to maintain AV synchrony is preferred (rather than single chamber right ventricular pacing) in most patients due to the favorable hemodynamic benefits of AV synchrony [11]. Some trials suggest that biventricular cardiac pacing (ie, cardiac resynchronization) is superior to standard dual chamber pacing in patients with heart block [15,16]. Implantable cardioverter-defibrillators, specifically cardiac resynchronization therapy devices (CRT-Ds), should be considered in patients with complete AV block and significant left ventricle dysfunction. Conducting system pacing (His-bundle or left-bundle pacing) has emerged as an alternative to CRT pacing [17,18]. Leadless pacing may be appropriate in selected patients. (See "Permanent cardiac pacing: Overview of devices and indications" and "Modes of cardiac pacing: Nomenclature and selection".)

## 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: Arrhythmias in adults"

and "Society guideline links: Cardiac implantable electronic devices".)

## INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Bradycardia (The Basics)" and "Patient education: Heart block in adults (The Basics)")

## SUMMARY AND RECOMMENDATIONS

- **Definition and etiology** – In third-degree (complete) atrioventricular (AV) block, no atrial impulses reach the ventricle. Third-degree (complete) AV block can occur in the AV node or in the infranodal specialized conduction system. The potential etiologies of third-degree (complete) AV block include reversible causes (both pathologic and iatrogenic) as well as idiopathic causes. Common potentially reversible causes include myocardial ischemia, medications, and cardiac procedures. (See 'Introduction' above and 'Etiology' above and "Etiology of atrioventricular block".)

- **Clinical presentation** – The clinical presentation of third-degree (complete) AV block varies depending upon the rate of the underlying escape rhythm and the presence of comorbid conditions. Nearly all patients with third-degree (complete) AV block will present with some degree of symptoms, though the severity of the symptoms can be quite variable. Most patients will present with some level of fatigue and/or dyspnea, and very few patients will be entirely asymptomatic. (See 'Signs and symptoms' above.)

- **ECG findings** – Patients with third-degree (complete) AV block will have evidence of atrial (P waves) and ventricular (QRS complexes) activity which are independent of each other on the surface ECG. In complete heart block, the escape rhythm can occur at any level below that of the conduction block, and the morphology of the QRS complex can help to determine the location at which this is occurring. If third-degree AV block occurs within the AV node or the bundle of His, the escape rhythm tends to have a narrow QRS complex, whereas AV block occurring below the bundle of His (ie, infrahisian block) results in a subjunctional escape rhythm with a wide QRS complex. (See 'Electrocardiographic findings' above.)

- **Diagnosis** – In nearly all cases, the diagnosis of third-degree (complete) AV block can be made in a patient with suggestive symptoms (eg, fatigue, dyspnea, presyncope, and/or syncope) by obtaining a surface ECG, ideally a full 12-lead ECG but sometime a single-lead rhythm strip is adequate if a full 12-lead ECG cannot be obtained. (See 'Diagnosis' above.)

- **Initial management** – Initial treatment of the patient with third-degree (complete) AV block depends on the presence and severity of any signs and symptoms related to the ventricular escape rhythm ( algorithm 1). (See 'Management' above.)

  - **Hemodynamically unstable** – Unstable patients require immediate pharmacologic therapy and, in most instances, temporary pacing to increase heart rate and cardiac output. Atropine (initial dose 1 mg intravenously [IV]) should be promptly administered if IV access is available, but treatment with atropine should not delay treatment with transcutaneous pacing or a chronotropic agent. Temporary cardiac pacing should be provided. In the absence of central venous access, the most immediate way to provide temporary cardiac pacing is via transcutaneous pacing (see 'Unstable patients' above and "Temporary cardiac pacing"). Beta-adrenergic agonists may also be helpful in some patients with complete heart block.

  - **Hemodynamically stable** – Patients with third-degree (complete) AV block who are initially hemodynamically stable 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. (See 'Stable patients' above.)

- **Subsequent management** – Once the patient is hemodynamically stable, reversible causes of third-degree (complete) AV block such as myocardial ischemia, increased vagal tone, hypothyroidism, hyperkalemia, and drugs that depress conduction, should be

excluded in patients prior to implantation of a permanent pacemaker. (See 'Stable patients' above.)

Once reversible causes of heart block have been excluded, permanent pacing is generally indicated. (See 'Stable patients' above.)

## ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Leonard Ganz, MD, FHRS, FACC, who contributed to earlier versions of this topic review.



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Topic 911 Version 39.0

## GRAPHICS

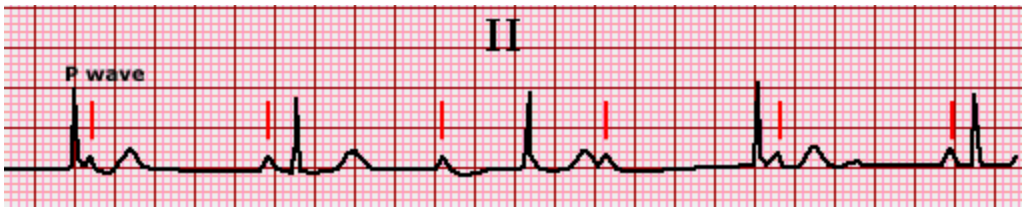
### Major causes of atrioventricular (AV) block

| **Physiologic and pathophysiologic** |
|---|
| Increased vagal tone |
| Progressive cardiac conduction system disease |
| With fibrosis and/or sclerosis (Lenegre disease) |
| With calcification (Lev disease) |
| Ischemic heart disease, including acute myocardial infarction |
| Cardiomyopathy |
| Infiltrative processes (eg, sarcoidosis, amyloidosis, hemochromatosis, malignancy, etc) |
| Other non-ischemic cardiomyopathies (eg, idiopathic, infectious, etc) |
| Infections (eg, viral myocarditis, Lyme carditis) |
| Congenital AV block |
| Related to structural congenital heart disease |
| As part of neonatal lupus syndrome |
| Other |
| Hyperkalemia, severe hypo- or hyperthyroidism, trauma, degenerative neuromuscular diseases |
| **Iatrogenic** |
| Drugs |
| Beta blockers, calcium channel blockers, digoxin, adenosine, antiarrhythmic drugs |
| Cardiac surgery |
| Post valvular surgery, post surgical correction of congenital heart disease |
| Transcatheter aortic valve implantation |
| Catheter ablation of arrhythmias |
| Transcatheter closure of VSD |
| Alcohol septal ablation for HCM |

VSD: ventricular septal defect; HCM: hypertrophic cardiomyopathy.

Graphic 62885 Version 6.0

## Third degree (complete) atrioventricular block with narrow QRS escape rhythm




The P waves are completely dissociated from the QRS complexes. The QRS complexes are narrow, indicating a junctional escape rhythm. The atrial and ventricular rates are stable; the former is faster than the latter.

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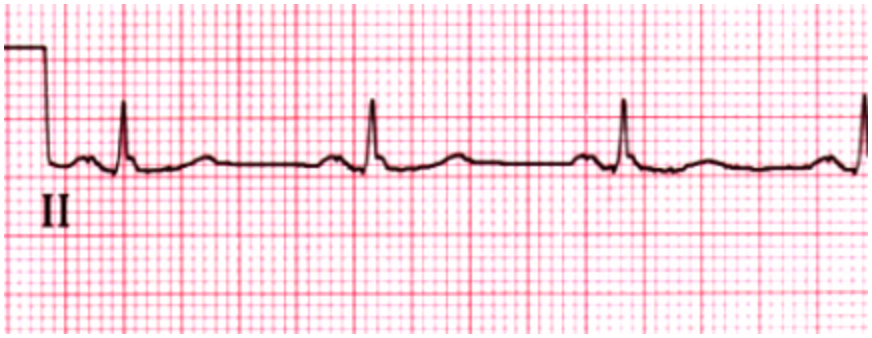
Graphic 65545 Version 5.0

## 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.

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*Courtesy of Morton F Arnsdorf, MD.*

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Graphic 59022 Version 3.0

## Third-degree (complete) atrioventricular block with wide QRS escape rhythm


The P waves are completely dissociated from the QRS complexes and the PR intervals are variable. The atrial or PP rate (75 beats per minute) is faster than the ventricular or RR rate (30 beats per minute), establishing complete atrioventricular blockade as the etiology. The QRS complexes are wide indicating that the escape rhythm is ventricular.

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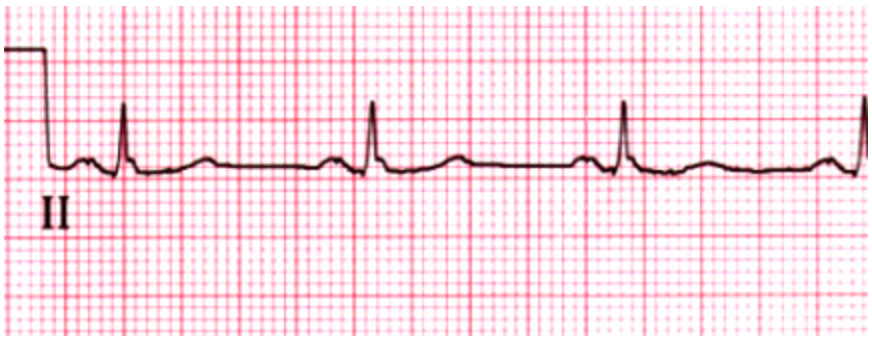
Graphic 51446 Version 6.0

## 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.

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*Courtesy of Morton F Arnsdorf, MD.*

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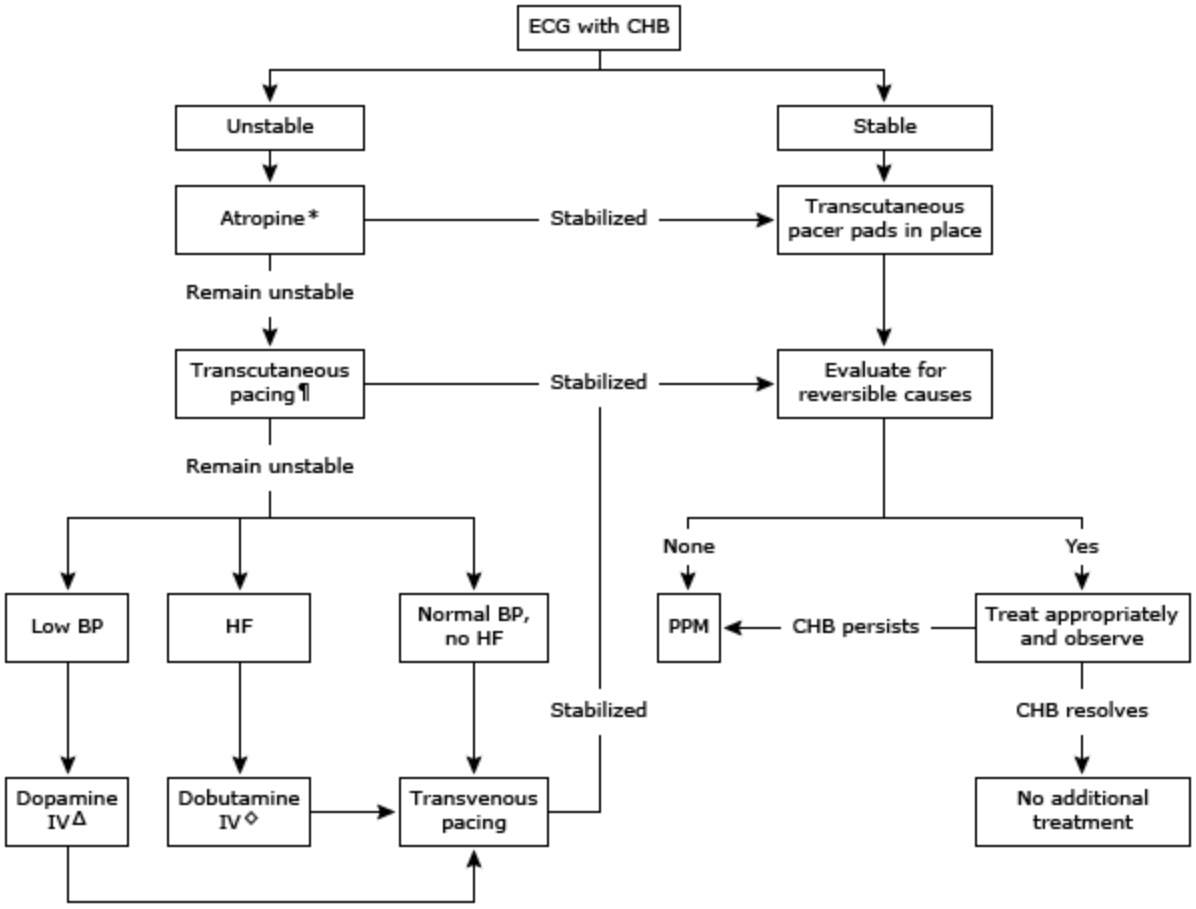
Graphic 59022 Version 3.0

## Algorithm showing the treatment approach to the patient with complete heart block




ECG: electrocardiogram; CHB: complete heart block; BP: blood pressure; HF: heart failure; IV: intravenous; PPM: permanent pacemaker.

* The initial dose of atropine is 1 mg IV. This dose may be repeated every 3 to 5 minutes to a total dose of 3 mg.

¶ While transcutaneous pacing may be initially successful in stabilizing the patient, it may not be consistently reliable. Central venous access should be considered in the event that urgent transvenous pacing is required.

Δ Dopamine IV infusion typically begins at a dose of 5 mcg/kg/minute and is titrated as needed to achieve an optimal clinical and hemodynamic response (maximum dose of 20 mcg/kg/minute).

◇ Dobutamine IV infusion typically begins at a dose of 2 to 5 mcg/kg/minute and is titrated as needed to achieve an optimal clinical and hemodynamic response. The usual maintenance dose of dobutamine is 2 to 10 mcg/kg/minute (maximum dose of 20 mcg/kg/minute).

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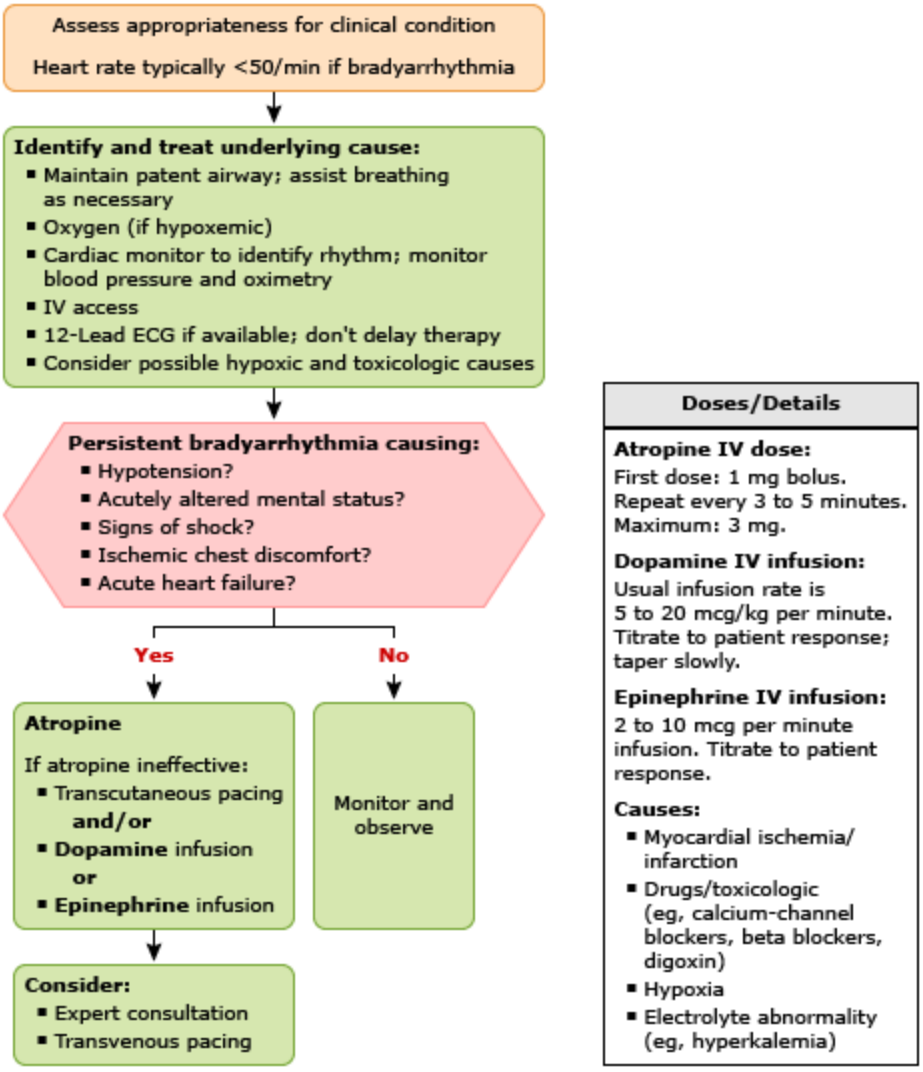
Graphic 103365 Version 7.0

## Adult bradycardia algorithm 2020 update

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Graphic 130748 Version 11.0

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