Second-degree atrioventricular block: Mobitz type I (Wenckebach block)

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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 anatomic 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** – Delayed conduction from the atrium to the ventricle (defined as a prolonged PR interval of >200 ms) without interruption in atrial to ventricular conduction.
- **Second-degree AV block** – Intermittent atrial conduction to the ventricle, often in a regular pattern (eg, 2:1, 3:2), or higher degrees of block, which are further classified into Mobitz type I (Wenckebach) and Mobitz type II second-degree AV block.
- **Third-degree (complete AV) block** – No atrial impulses conduct to 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 Mobitz type I second-degree AV block will be reviewed here. 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 "First-

degree atrioventricular block" and "Second-degree atrioventricular block: Mobitz type II" and "Third-degree (complete) atrioventricular block" and "Congenital third-degree (complete) atrioventricular block".)

DEFINITIONS

In second-degree AV block, some atrial impulses fail to reach the ventricles. Wenckebach described progressive delay between atrial and ventricular contraction and the eventual failure of a P wave to reach the ventricles [1]. Mobitz subsequently divided second-degree AV block into two subtypes, as determined by the findings on the electrocardiogram (ECG) [2]:

- Mobitz type I second-degree AV block (waveform 1), in which progressive PR interval prolongation precedes a nonconducted P wave. The first P wave after block conducts to the ventricle with a shorter PR interval compared with the last P wave before block.
- Mobitz type II second-degree AV block (waveform 2), in which the PR interval remains unchanged prior to a P wave that fails to conduct to the ventricle.
- High-grade AV block, in which two or more consecutive P waves are nonconducted. In contrast to third-degree or complete heart block (waveform 3), however, some P waves continue to be conducted to the ventricle.

Mobitz type I and Mobitz type II second-degree AV block cannot be differentiated from the ECG when 2:1 AV block is present. In this situation, every other P wave is nonconducted, and there is no opportunity to observe for possible PR prolongation that is characteristic of Mobitz type I second-degree AV block. (See 'ECG findings and diagnostic maneuvers' below.)

ETIOLOGY

Mobitz type I second-degree AV block can occur in normal subjects and athletes without underlying cardiac pathology. The potential etiologies of Mobitz type I second-degree AV block also include reversible (both pathologic and iatrogenic) and idiopathic causes that are similar to other degrees of AV block (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), endocarditis with abscess formation, hyperkalemia, and hypervagotonia.

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

In persons who are not highly conditioned athletes in whom no specific reversible cause is identified, the block is often felt to be related to idiopathic progressive cardiac conduction disease with myocardial fibrosis and/or sclerosis that affects the conduction system. (See "Etiology of atrioventricular block".)

Normal subjects and athletes — Mobitz type I second-degree AV block can occur in individuals who have high vagal tone, such as younger persons or highly conditioned athletes at rest [3-9]. The prognosis is excellent in these settings, as progressive block does not appear to occur [4,6-8]. Progression to third-degree (complete) AV block has been reported in infants (occurring in 7 of 16 in one study) [10].

Sinus bradycardia and AV conduction abnormalities are often observed in well-trained endurance athletes. As an example, Mobitz type I second-degree AV block has been described in 2 to 10 percent of long-distance runners [9]. These findings may be related to increased parasympathetic activity associated with training (with a disappearance following detraining or after vagolytic or sympathetic maneuvers) or intrinsic AV nodal mechanisms [8,11].

Underlying heart disease — Mobitz type I second-degree AV block can occur in patients with intrinsic AV nodal disease, myocarditis (including Chagas disease), acute inferior myocardial infarction or ischemia (waveform 4), and cardiac surgery.

- In the majority of patients (approximately 90 percent), the right coronary artery supplies blood to the AV node. Thus, Mobitz type I second-degree AV block (due to ischemia of the AV node) can occur as a complication of an inferior myocardial infarction. The finding of Mobitz type I second-degree AV block in a patient with an inferior myocardial infarction is associated with increased mortality, presumably due to larger infarct size. (See "Conduction abnormalities after myocardial infarction".)

- Mobitz type I second-degree AV block, typically transient, has been described in approximately 3 percent of patients after mitral valve surgery and has been reported in patients with repaired tetralogy of Fallot and after repair of ventricular septal defects [12].

- Mobitz type I second-degree AV block, can be induced by a Valsalva maneuver in patients with Chagas disease who do not have overt cardiac involvement. This may indicate early vagal dysfunction or involvement of the AV node [13]. (See "Chronic Chagas cardiomyopathy: Clinical manifestations and diagnosis".)

PATHOPHYSIOLOGY

Mobitz type I second-degree AV block usually occurs within the AV node, but may also reflect a delay elsewhere in the conduction system (figure 1). Mobitz type I second-degree AV block can be observed in antegrade AV conduction, retrograde ventriculoatrial (VA) conduction across the AV node, or as part of exit block with ectopic and parasystolic pacemakers.

Regardless of the site involved, what follows is a sequence in which there is a gradually increasing PR interval, usually a gradually decreasing R-R interval, and eventually a nonconducted P wave (waveform 1). The following factors are required for this sequence to occur:

- A constant input – The constant input is usually the SA nodal pacemaker that gives rise to atrial depolarization.

- An area of increasing conduction delay and a nonconducted P wave – The PR interval is shortest in the first conducted P wave in the cycle and increases with each ensuing P wave. However, the largest **absolute increase in delay** occurs following the first P wave, a lesser increase in delay occurs following the second P wave, and so forth. The impulse eventually conducts very slowly and block occurs, resulting in a nonconducted P wave (no associated QRS complex). If the pause between the last conducted P wave and the first apparent QRS complex of the next cycle is very long, it may in fact be a junctional escape rather than a conducted P wave. Almost invariably, the second PR interval of the new cycle will be shorter than that of the last conducted P wave that preceded the block.

- An output with a decreasing interval – The output, in this case the QRS-QRS interval (more commonly called the R-R interval), usually decreases with each conducted P wave of the cycle. The shortening R-R interval results from the decreasing increment in delay of AV nodal conduction (eg, the PR interval of beat 1 in a cycle increases by 0.05 seconds [from 0.18 to 0.23], the PR interval of next beat increases by 0.03 seconds [from 0.23 to 0.26], etc).

- Grouped beating – The classic Wenckebach pattern occurs usually with ratios of 3:2, 4:3, or 5:4. This gives rise to a clustering of beats with decreasing R-R intervals that tends to repeat, although mixed ratios do occur fairly frequently. Conduction ratios of over 7:6 usually show an atypical pattern rather than the classic pattern.

- Pause shorter than two input cycles – The R-R interval involving the nonconducted P wave is less than the summed R-R interval of any two previous cycles. This also results from the

incremental conduction delay as the P wave that is not conducted is closer to the preceding QRS than any other in the cycle.

Once conduction ratios exceed 6:5 or 7:6, the progressive increment in PR interval becomes unpredictable and the PR interval remains prolonged but constant. The most common explanation is that the sinus rate changes which, in turn, influences the PR interval through hemodynamic and autonomic effects. The PR interval is still longest in the conducted P wave before, and shortest after, the block (waveform 5).

The site of the Mobitz type I second-degree AV block is in the AV node in the vast majority of cases, with the remaining cases involving the His bundle, bundle branches, or fascicles.

- ECG – The ECG cannot pinpoint with certainty the site of the Mobitz type I second-degree AV block [14,15]. (See 'ECG findings and diagnostic maneuvers' below.)
- His bundle ECG – His bundle ECG, as part of an invasive electrophysiologic (EP) study, can easily confirm the site of the Mobitz type I second-degree AV block. (See 'Electrophysiology study' below.)

CLINICAL PRESENTATION AND EVALUATION

The clinical presentation of Mobitz type I second-degree AV block is typically fairly benign, as uncomplicated Mobitz type I second-degree AV block only rarely produces symptoms. Additionally, in contrast to Mobitz type II second-degree AV block, which can frequently progress to third-degree (complete) AV block, Mobitz type I second-degree AV block most often involves the AV node and rarely progresses to complete heart block. The evaluation of all patients with suspected Mobitz type I second-degree AV block includes a thorough history, including medications and recent changes in medications, along with a 12-lead ECG and bloodwork (which includes serum electrolytes and thyroid-stimulating hormone [TSH]).

Clinical history — All patients with suspected Mobitz type I second-degree AV block should be questioned about any history of heart disease, both congenital and acquired, as well as any recent cardiac procedures or medications 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. (See 'Etiology' above.)

Patients with suspected Mobitz type I second-degree 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 without known or suspected cardiac or systemic disease should be questioned about their level of athletic activities and fitness. Such patients should also 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).

Signs and symptoms — Most patients with Mobitz type I second-degree AV block are asymptomatic. Mobitz type I second-degree AV block that occurs in the setting of acute myocardial ischemia or myocarditis may result in clinical deterioration if the resulting ventricular rate is inadequate to maintain cardiac output. Additionally, even in otherwise healthy patients, if the sinus rate is slow and there are fewer conducted beats (2:1 or 3:2 block), there may be a significant reduction in cardiac output resulting in symptoms of hypoperfusion (including fatigue, lightheadedness, syncope, presyncope, or angina) or heart failure.

Patients with Mobitz type I second-degree AV block often present with bradycardia but may have a normal sinus rhythm rate. Additionally, other than the presence of an irregular pulse, there are few specific physical examination findings. Patients may appear pale or diaphoretic if they have bradycardia with a resultant 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.

ECG findings and diagnostic maneuvers — Mobitz type I second-degree AV block is identified by progressive prolongation of the PR interval for several heart beats, followed by a nonconducted P wave (waveform 1).

Mobitz type I second-degree AV block is distinguished from other types of AV block as follows:

- Patients with first-degree AV block have a PR interval that is prolonged (>200 ms) but constant, and each P wave is followed by a QRS interval (waveform 6).
- Patients with Mobitz type II second-degree AV block have a consistent unchanging PR intervals prior to a P wave that suddenly fails to conduct to the ventricles (waveform 2). For patients with second-degree AV block with a ratio of atrial to ventricular conduction that is not 2:1, Mobitz type I and Mobitz type II second-degree AV block are easily distinguished. However, for patients with 2:1 atrial to ventricular conduction, the

distinction between Mobitz type I and Mobitz type II second-degree AV block cannot be made from the surface ECG.

- Patients with third-degree (complete) AV block will have evidence of atrial (P waves) and ventricular (QRS complexes) activity that are independent of each other on the surface ECG (waveform 3). In rare instances, the atrial rate may be exactly twice the ventricular rate, resulting in apparent 2:1 AV block that can mimic second-degree AV block.

If 2:1 AV block is seen on the surface ECG, certain ECG features may aid in differentiating Mobitz type I from Mobitz type II second-degree AV block. Additionally, because of the varying effects of vagal tone on sinus node, AV node, and His-Purkinje system properties, vagal maneuvers can identify the site of a conduction abnormality in patients with second-degree AV block.

- If the PR interval of a conducted beat is very long (>300 ms) or the QRS complex is narrow, the level of block is more likely to be in the AV node, and the rhythm is most likely Mobitz type I second-degree AV block.

- If atropine is administered (typically 0.25 to 0.5 mg IV) and there is enhanced AV nodal conduction resulting in less frequent nonconducted P waves (ie, change from a 2:1 cycle to a 3:2 cycle), this confirms Mobitz type I second-degree AV block. A lack of response to atropine is consistent with but not diagnostic for Mobitz type II second-degree AV block.

- Carotid sinus massage, which increases vagal tone, would be expected to worsen Mobitz type I second-degree AV block by slowing AV nodal conduction. An increased conduction defect (ie, higher-grade block) following carotid sinus massage, which increases vagal tone, implies that the AV node is the site of the abnormality and is consistent with Mobitz type I second-degree AV block. Apparent improvement in AV conduction with the slowing of the sinus node rate (eg, restoration of 1:1 conduction) following carotid sinus massage suggests that the conduction abnormality is below the level of the AV node, consistent with Mobitz type II second-degree AV block. (See "Vagal maneuvers", section on 'Carotid sinus massage'.)

Electrophysiology study — Electrophysiology study (EPS) is not usually performed in patients with Mobitz type I second-degree AV block. EPS is indicated in those patients with syncope or presyncope and 2:1 AV block when the etiology of 2:1 block cannot be discerned with noninvasive maneuvers.

When EPS is performed in such patients, there is typically a progressively longer A-H interval and a stable H-V interval until the final beat of the series in which there is an atrial electrogram with no subsequent His or ventricular electrogram. Less commonly, Mobitz type I second-

degree AV block is observed in the His-Purkinje system (HPS) [16]. In this instance, there will usually be two His potentials (H and H') due to the slowing of conduction, with the H-H' interval prolonging before the H' and subsequent QRS complex are absent with block.

DIAGNOSIS

In nearly all cases, the diagnosis of Mobitz type I second-degree AV block can be made in a patient with an irregular pulse or suggestive symptoms (eg, fatigue, dyspnea, presyncope, and/or syncope) by obtaining a surface ECG.

For patients with 2:1 AV block in whom the distinction between Mobitz type I and Mobitz type II second-degree AV block cannot be made using the surface ECG alone, a long rhythm strip should be obtained or a previous ECG examined to try to find evidence of PR prolongation with nonconducted P waves in a pattern other than 2:1 (eg, 3:2, 4:3, etc). Additionally, carotid sinus massage may be performed, or intravenous atropine administered, to help distinguish the level of AV block. If the diagnosis remains uncertain following these measures, invasive electrophysiology studies can definitively diagnose the type of AV block and accurately identify the level of the block.

MANAGEMENT

The management of Mobitz type I second-degree AV block depends on the presence and severity of any signs and symptoms related to the patient's rate and rhythm (algorithm 1). Symptomatic patients should be treated with ventricular pacing (and, if hemodynamically unstable, atropine) and undergo treatment of any associated potentially reversible causes (eg, myocardial ischemia). In rare cases, cardioneural ablation can be considered for symptomatic patients with enhanced vagal tone [17]. Conversely, asymptomatic patients with Mobitz type I second-degree AV block do not require any specific therapy. Prior to initiating treatment for Mobitz type I second-degree AV block, reversible causes of slowed conduction such a myocardial ischemia, increased vagal tone, and medications should be excluded. (See "Etiology of atrioventricular block".)

Initial management — The initial management of patients with Mobitz type I second-degree AV block depends on the presence or absence of symptoms and the hemodynamic status of the patient (algorithm 1).

Symptomatic and hemodynamically unstable — Patients with Mobitz type I second-degree AV block who are **symptomatic and hemodynamically unstable** should be urgently treated

with atropine and temporary cardiac pacing if not responsive to atropine (either with transcutaneous or, if immediately available, transvenous pacing) (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. Dopamine may be administered in hypotensive patients, while dobutamine is an option for patients with heart failure symptoms. This approach is similar to the patient who presents with unstable Mobitz type II second-degree AV block or unstable third-degree (complete) AV block (algorithm 2) [18]. (See "Second-degree atrioventricular block: Mobitz type II", section on 'Unstable patients' and "Third-degree (complete) atrioventricular block", section on 'Unstable patients'.)

- 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 0.5 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 since the more distal conducting system is not as sensitive to vagal activity.

- 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".)

Symptomatic and hemodynamically stable — Patients with Mobitz type I second-degree AV block who are **symptomatic and hemodynamically stable** do not require urgent therapy with atropine or temporary cardiac pacing (algorithm 1). However, patients should be continuously monitored with transcutaneous pacing pads in place (figure 2) in the event of clinical deterioration.

While stable patients are being monitored, reversible causes of Mobitz type I second-degree AV block such as myocardial ischemia, myocarditis, increased vagal tone, hypothyroidism, and drugs that depress conduction should be excluded in patients prior to implantation of a permanent pacemaker.

- Patients with Mobitz type I second-degree 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".)

- Patients with Mobitz type I second-degree 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.

Asymptomatic patients — Patients with Mobitz type I second-degree AV block who are **asymptomatic** do not require any initial treatment (algorithm 1). Similar to symptomatic but hemodynamically stable patients, patients with asymptomatic Mobitz type I second-degree AV block should be evaluated for potentially reversible causes.

- If Mobitz type I second-degree AV block is identified in the hospital setting, patients should be monitored during their hospitalization to ensure stability of the rate and rhythm while undergoing evaluation for potentially reversible causes. Once a patient is defined as being truly asymptomatic and is not considered to be at risk for progression to higher levels of block, continuous monitoring is no longer indicated. Asymptomatic Mobitz type I block itself should not be considered as a reason for hospital admission and should not interfere with other noncardiac treatments in the hospitalized patient.

- If Mobitz type I second-degree AV block is identified in an ambulatory setting, patients should be evaluated for potentially reversible causes and seen for ambulatory follow-up within two to four weeks for a repeat ECG and symptom assessment.

Most asymptomatic patients with Mobitz type I second-degree AV block do not require invasive electrophysiology studies (EPS). However, in patients who have otherwise unexplained syncope or presyncope in whom AV block may be the etiology (and therefore potentially symptomatic AV block), EPS may be considered. (See 'Electrophysiology study' above.)

In asymptomatic patients who have undergone invasive EPS to determine the level of block within the AV node, the decision regarding a pacemaker is based on the level of block:

- For patients whose AV block is identified at the supra-His level (ie, within the AV node), we do not place a permanent pacemaker.

- For patients whose AV block is identified as infranodal, implantation of a permanent pacemaker can be considered based on the potential mortality benefit in asymptomatic patients whose block is identified as infranodal as well as a greater likelihood of progressive AV block and morbidity in patients with infranodal block. (See 'Subsequent

management' below and "Permanent cardiac pacing: Overview of devices and indications", section on 'Acquired AV block'.)

Subsequent management — For the rare patient with Mobitz type I second-degree AV block and symptomatic bradycardia that is not due to a reversible etiology, we recommend implantation of a permanent pacemaker (algorithm 1). For patients with Mobitz type I second-degree AV block and symptomatic bradycardia who require a pacemaker, we implant a dual chamber DDD pacemaker whenever possible in an effort to maintain physiologic AV synchrony (algorithm 3). This approach in symptomatic patients is in agreement with the recommendations of professional society guidelines regarding device-based therapy for arrhythmias [19,20]. (See "Permanent cardiac pacing: Overview of devices and indications", section on 'Acquired AV block'.)

For the large majority of patients with Mobitz type I second-degree AV block and asymptomatic bradycardia, we suggest **not** implanting a permanent pacemaker [20]. This is particularly true in younger patients and those patients in whom the AV block is felt to be supra-Hisian in nature (ie, within the AV node). These patients should have regular follow-up, including a surface ECG, every 6 to 12 months with questioning focused on the interval development of symptoms (eg, fatigue, dyspnea, presyncope, syncope) which could be attributable to the AV block.

For patients with Mobitz type I second-degree AV block who remain asymptomatic, there has been disagreement among clinicians and professional societies regarding optimal management, with the ESC guidelines suggesting pacemaker placement and the ACC/AHA/HRS guidelines recommending against pacemaker placement. There are no randomized trial data to guide this decision. Observational data, with many limitations, provide some support for the use of a permanent pacemaker in such patients:

- Among a cohort of 86 asymptomatic patients with Mobitz type I second-degree AV block seen at a single institution in England from 1968 to 1993 (mean age 69 years, 65 percent male), 39 (45 percent) received a pacemaker for prophylactic purposes [21]. Despite the absence of symptoms, five-year survival was significantly higher among the group who received a pacemaker (87 versus 54 percent in the unpaced group). All mortality occurred in those greater than 45 years of age.

- Among a cohort of 299 patients with Mobitz type I second-degree AV block seen at a single Veterans Affairs Medical Center in the US from 1992 to 2010 (mean age 75 years, 99 percent male), the majority (175 patients) remained asymptomatic, while 124 patients (41 percent) received a pacemaker for symptomatic bradycardia or progression to higher-grade AV block [22]. During an average follow-up of 3.3 years, 190 patients (64 percent)

died. Despite having greater cardiac comorbidity (eg, heart failure, coronary heart disease reduced left ventricular ejection fraction), patients who received a pacemaker had a 46 percent reduction in total mortality compared with those without a pacemaker (hazard ratio 0.54, 95% CI 0.35-0.83). This study, however, was a single-center study in an older-adult, predominantly male population with a high mortality rate.

Our authors and editors feel that these data are insufficient to recommend pacemaker placement in patients with Mobitz type I second-degree AV block at the supra-His level (ie, within the AV node) which is not demonstrated to be infranodal by EP evaluation.

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

- **Definitions** – 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 anatomic or functional impairment in the conduction system. In second-degree AV block, some atrial impulses fail to reach the ventricles. In Mobitz type I second-degree AV block (waveform 1), there is progressive PR interval prolongation for several beats preceding a nonconducted P wave, whereas in Mobitz type II second-degree AV block (waveform 2), the PR interval remains unchanged prior to a P wave that suddenly fails to conduct to the ventricles. (See 'Introduction' above and 'Definitions' above.)

- **Etiology** – Mobitz type I second-degree AV block can occur in normal subjects and athletes without underlying cardiac pathology. The potential etiologies of Mobitz type I second-degree AV block also include reversible (both pathologic and iatrogenic) and idiopathic causes that are similar to other degrees of AV block (table 1). Common potentially reversible causes include certain medications, myocardial ischemia, myocarditis, and cardiomyopathies. (See 'Etiology' above.)

- **Clinical history** – All patients with suspected Mobitz type I second-degree AV block should be questioned about any history of heart disease, both congenital and acquired, as well as any recent cardiac procedures or medications that could predispose to AV conduction abnormalities. (See 'Clinical history' above.)

- **Clinical presentation** – Most patients with Mobitz type I second-degree AV block are asymptomatic. Mobitz type I second-degree AV block that occurs in the setting of acute myocardial ischemia or myocarditis may result in clinical deterioration if the resulting ventricular rate is inadequate to maintain cardiac output. Additionally, even in otherwise healthy patients, if the sinus rate is slow and there are fewer conducted beats (2:1 or 3:2 block), there may be a significant reduction in cardiac output resulting in symptoms of hypoperfusion (including fatigue, lightheadedness, syncope, presyncope, or angina) or heart failure. (See 'Signs and symptoms' above.)

- **ECG findings and diagnostic maneuvers** – Mobitz type I second-degree AV block is identified by progressive prolongation of the PR interval for several heart beats, followed by a nonconducted P wave (waveform 1). If 2:1 AV block is seen on the surface ECG, certain ECG features (eg, PR interval >300 ms, narrow QRS complexes, etc) may aid in differentiating Mobitz type I from Mobitz type II second-degree AV block. Additionally, because of the varying effects of vagal tone on sinus node, AV node, and His-Purkinje system properties, vagal maneuvers can identify the site of a conduction abnormality in patients with second-degree AV block. (See 'ECG findings and diagnostic maneuvers' above and 'Diagnosis' above.)

- **Initial management** – The initial management of patients with Mobitz type I second-degree AV block (algorithm 1) depends on the presence or absence of symptoms and the hemodynamic status of the patient. (See 'Initial management' above.)

  - **Symptomatic patients**

    - **Hemodynamically unstable** – Patients who are symptomatic and hemodynamically unstable should be urgently treated with atropine (0.5 mg intravenously, which may be repeated every three to five minutes to a total dose of

3 mg) and temporary cardiac pacing if not responsive to atropine (either with transcutaneous or, if immediately available, transvenous pacing). (See 'Symptomatic and hemodynamically unstable' above.)

- **Hemodynamically stable** – Patients who are symptomatic and hemodynamically stable do not require urgent therapy with atropine or temporary cardiac pacing. However, patients should be continuously monitored with transcutaneous pacing pads in place (figure 2) in the event of clinical deterioration. (See 'Symptomatic and hemodynamically stable' above.)

- **Asymptomatic patients** – Patients who are asymptomatic do not require any initial treatment. Reversible causes of slowed conduction such as myocardial ischemia, increased vagal tone, and medications should be excluded. If no reversible causes are present, and the patient is asymptomatic, no specific therapy is required. (See 'Asymptomatic patients' above.)

- **Subsequent management** (algorithm 1) (See 'Subsequent management' above.)

  - **Symptomatic patients** – For the rare patient with Mobitz type I second-degree AV block and symptomatic bradycardia that is not due to a reversible etiology, we recommend implantation of a permanent pacemaker (**Grade 1A**). We implant a dual chamber DDD pacemaker whenever possible in an effort to maintain physiologic AV synchrony.

  - **Asymptomatic patients** – For the large majority of patients with Mobitz type I second-degree AV block and asymptomatic bradycardia, we suggest **not** implanting a permanent pacemaker (**Grade 2B**). This is particularly true in younger patients and those patients in whom the AV block is felt to be supra-Hisian in nature (ie, within the AV node). These patients should have regularly follow-up, including a surface ECG, every 6 to 12 months.

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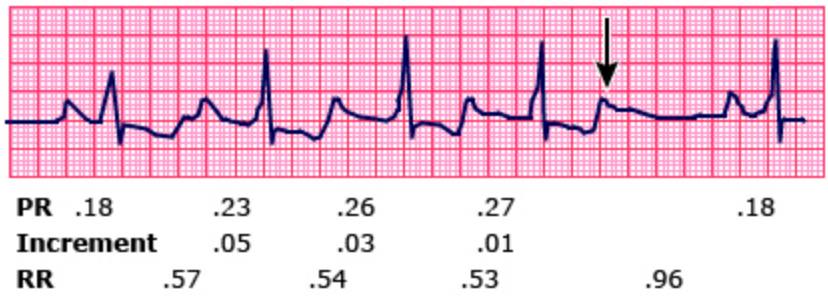
Topic 909 Version 38.0

GRAPHICS

Electrocardiogram showing Mobitz type I (Wenckebach) atrioventricular block

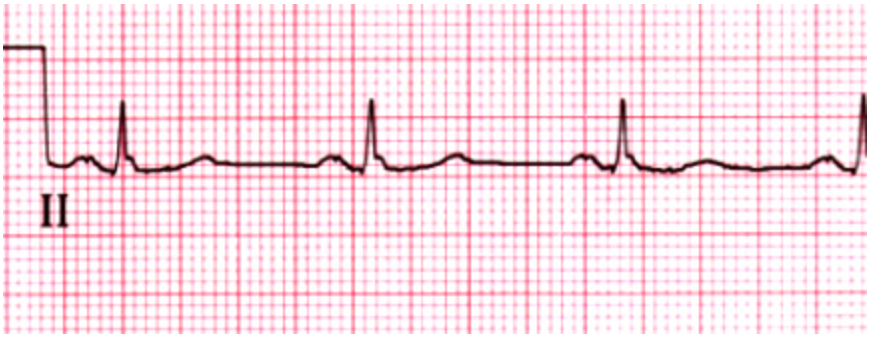


Single-lead electrocardiogram showing Mobitz type I (Wenckebach) second-degree atrioventricular block with 5:4 conduction. The characteristics of this arrhythmia include: a progressively increasing PR interval until a P wave is not conducted (arrow), a progressive decrease in the increment in the PR 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

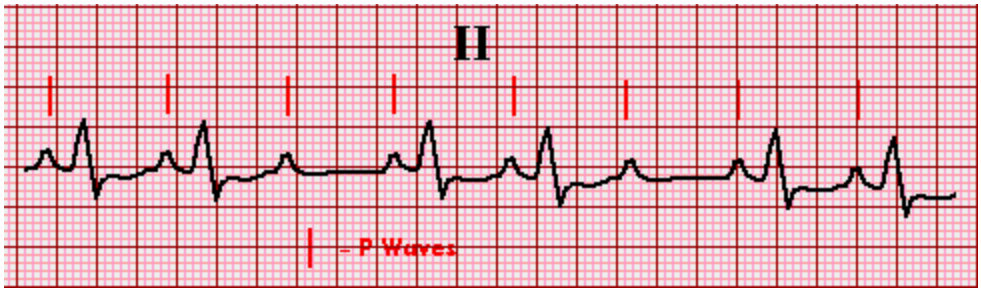


Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

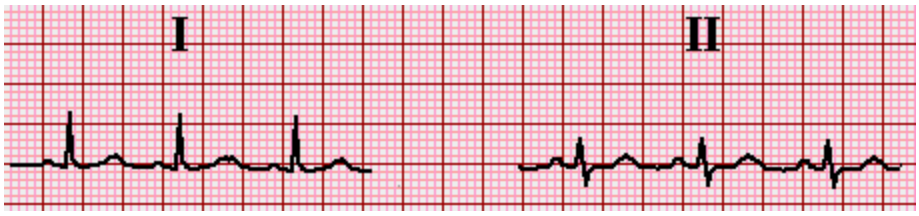
Single lead electrocardiogram (ECG) showing Mobitz type II second degree atrioventricular (AV) block



The third and sixth P waves are not conducted through the AV node (there is no associated QRS complex). The PR interval is constant prior to and after the non-conducted beats.

Graphic 58649 Version 4.0

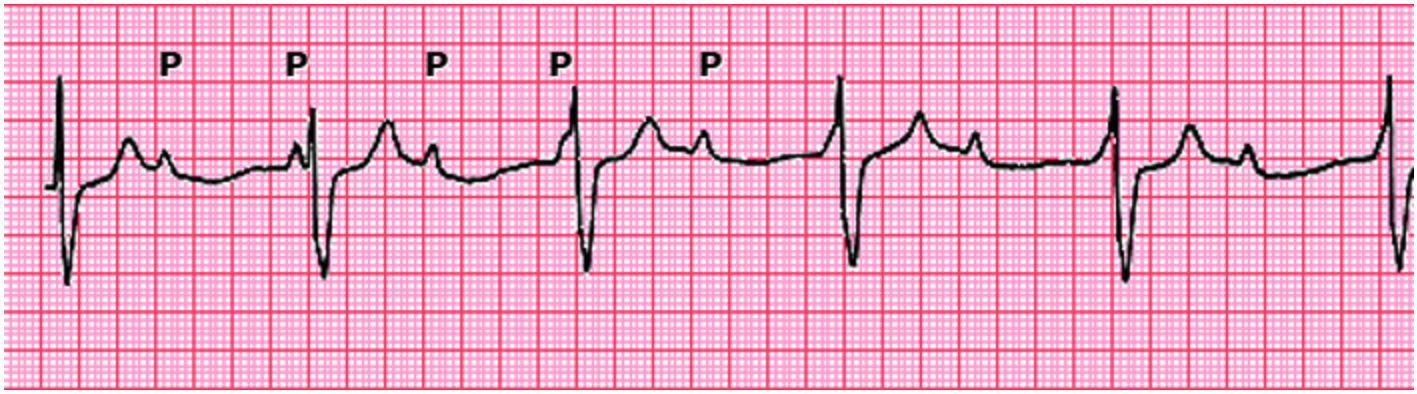
Sinus rhythm



The normal P wave in sinus rhythm is slightly notched since activation of the right atrium precedes that of the left atrium. The P wave is upright in a positive direction in leads I and II. A P wave with a uniform morphology precedes each QRS complex. The rate is between 60 and 100 beats per minute and the cycle length is uniform between sequential P waves and QRS complexes. In addition, the P wave morphology and PR intervals are identical from beat to beat.

Graphic 69872 Version 2.0

Single-lead electrocardiogram (ECG) showing sinus rhythm with third-degree (complete) AV block

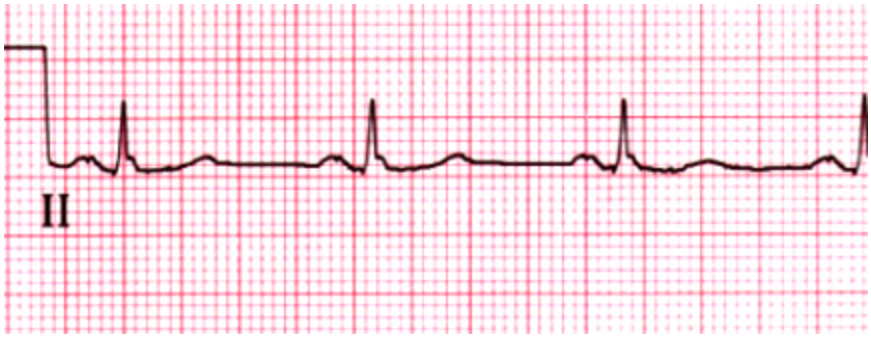


Sinus rhythm with third-degree (complete) heart block. There is independent atrial (as shown by the P waves) and ventricular activity, with respective rates of 83 and 43 beats per minute. The wide QRS complexes may represent a junctional escape rhythm with underlying bundle branch block or an idioventricular pacemaker.

Courtesy of Ary Goldberger, MD.

Graphic 72863 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

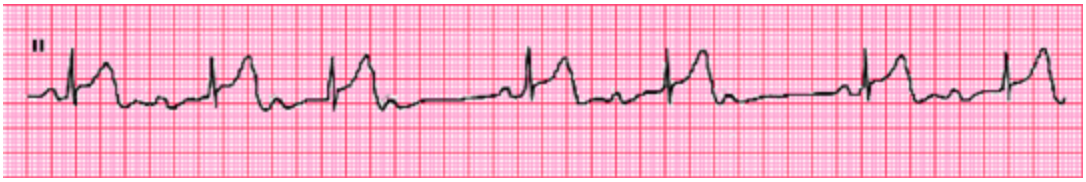
Major causes of atrioventricular (AV) block

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

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

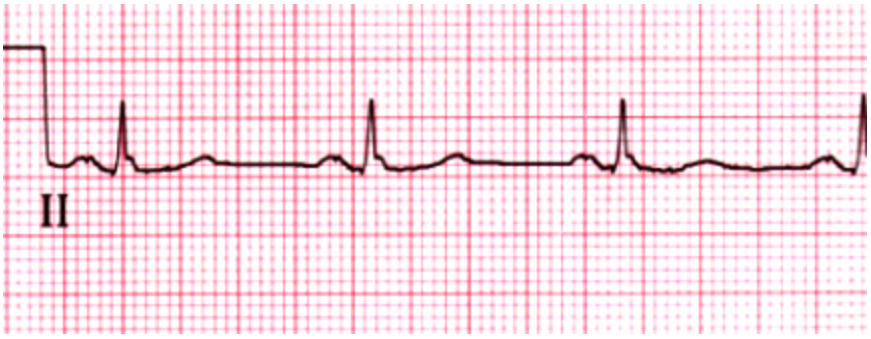


This rhythm strip shows a Mobitz type I (Wenckebach) atrioventricular block with 4:3 and 3:2 conduction and progressive prolongation of the PR intervals of conducted beats. The marked ST segment elevation suggests acute inferior wall ischemia or infarction that may be responsible for the arrhythmia.

Courtesy of Ary Goldberger, MD.

Graphic 62040 Version 3.0

Normal rhythm strip

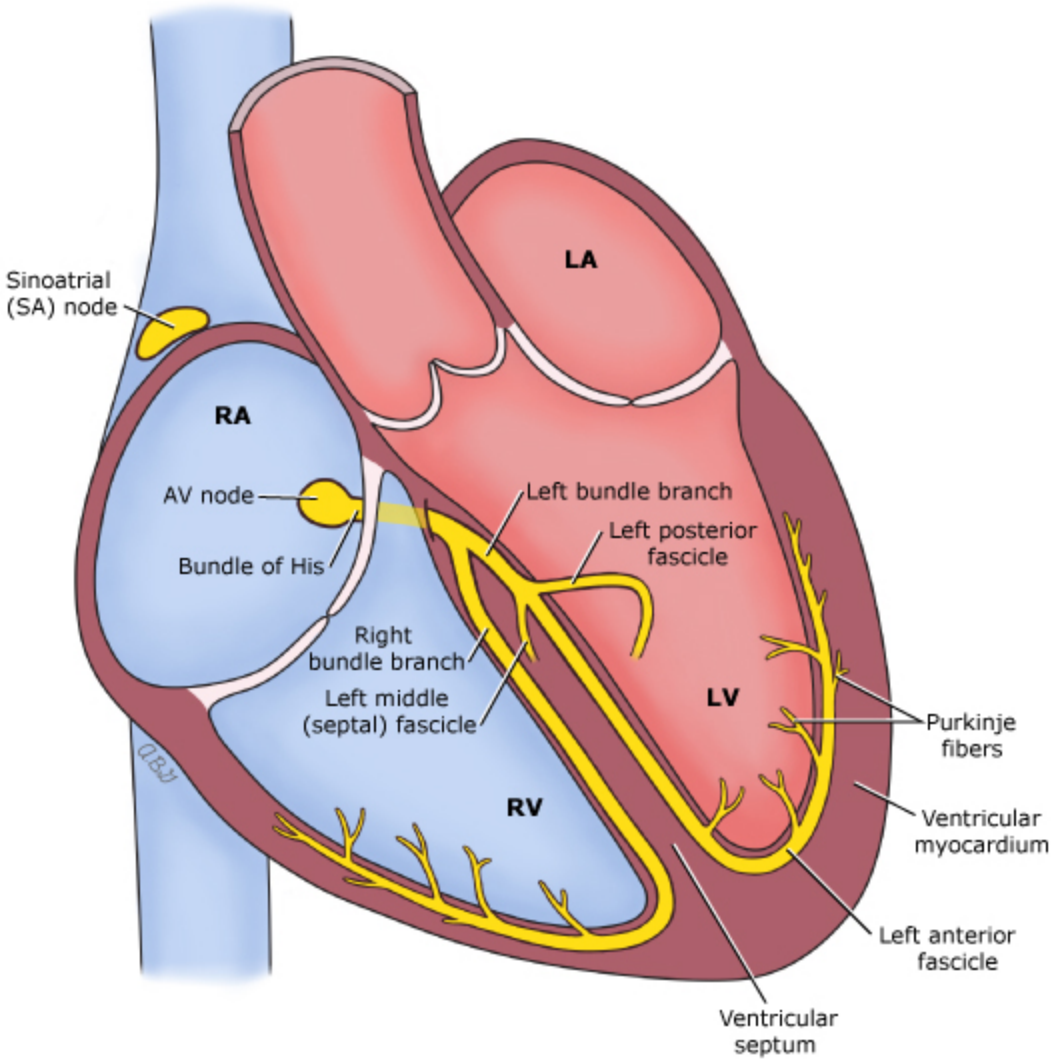


Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Normal conduction system

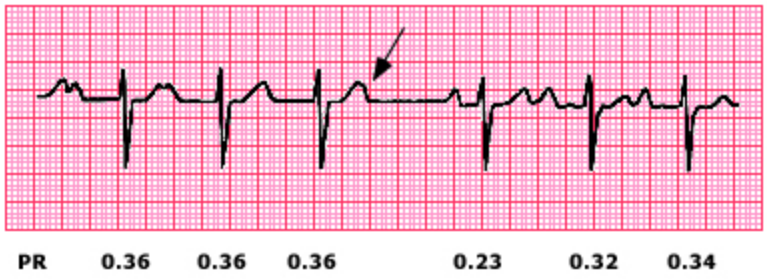


Schematic representation of the normal intraventricular conduction system (His-Purkinje system). The Bundle of His divides into the left bundle branch and right bundle branch. The left bundle branch divides into anterior, posterior, and, in some cases, median fascicles.

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

Graphic 63340 Version 6.0

Electrocardiogram (ECG) showing atypical Mobitz type I (Wenckebach) atrioventricular (AV) block

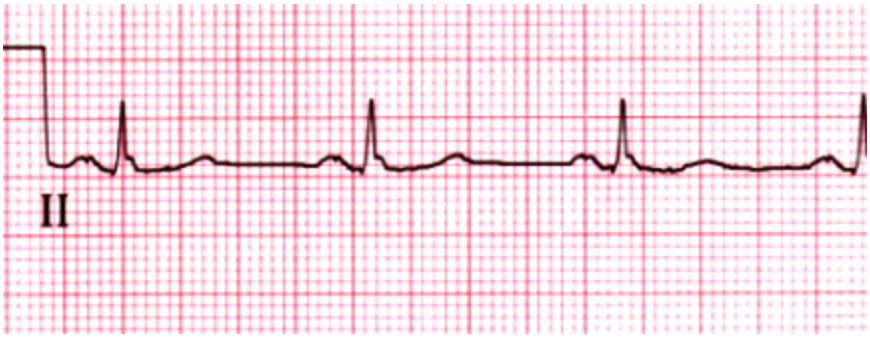


Single lead electrocardiogram (ECG) strip showing an atypical Mobitz type I (Wenckebach) AV block with 18:17 ratio. The last three cycles of the group, the skipped beat (with the P wave lost in the T wave; arrow), and the first three cycles of the next group are shown. The last three cycles had a PR interval of 0.36 sec while the first three cycles showed PR intervals of 0.23, 0.32 and 0.34 sec with a decreasing R-R interval. This demonstrates the importance of comparing the PR interval of the last beat before the dropped QRS to the PR interval of the first and second beats of the new cycle. The PR interval is the longest in the beat before the dropped beat, shortest in the first beat of the cycle, and increases in the second beat.

Courtesy of Morton Arnsdorf, MD.

Graphic 78766 Version 3.0

Normal rhythm strip

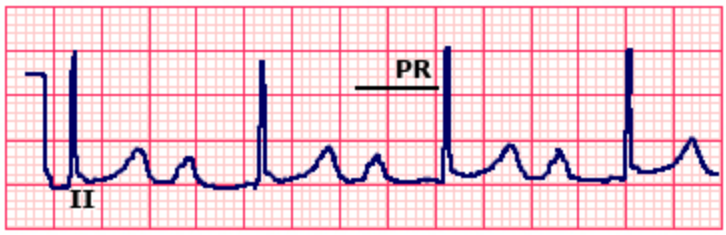


Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Single-lead electrocardiogram (ECG) showing first-degree atrioventricular (AV) block I

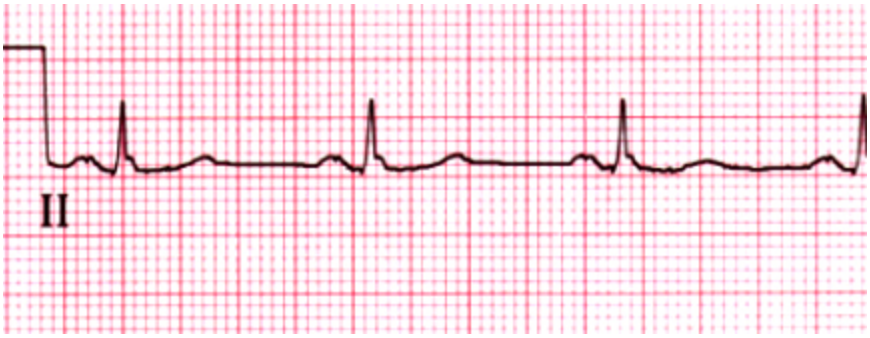


Electrocardiogram of lead II showing normal sinus rhythm, first-degree atrioventricular block with a prolonged PR interval of 0.30 seconds, and a QRS complex of normal duration. The tall P waves and P wave duration of approximately 0.12 seconds suggest concurrent right atrial enlargement.

Courtesy of Morton Arnsdorf, MD.

Graphic 67882 Version 6.0

Normal rhythm strip

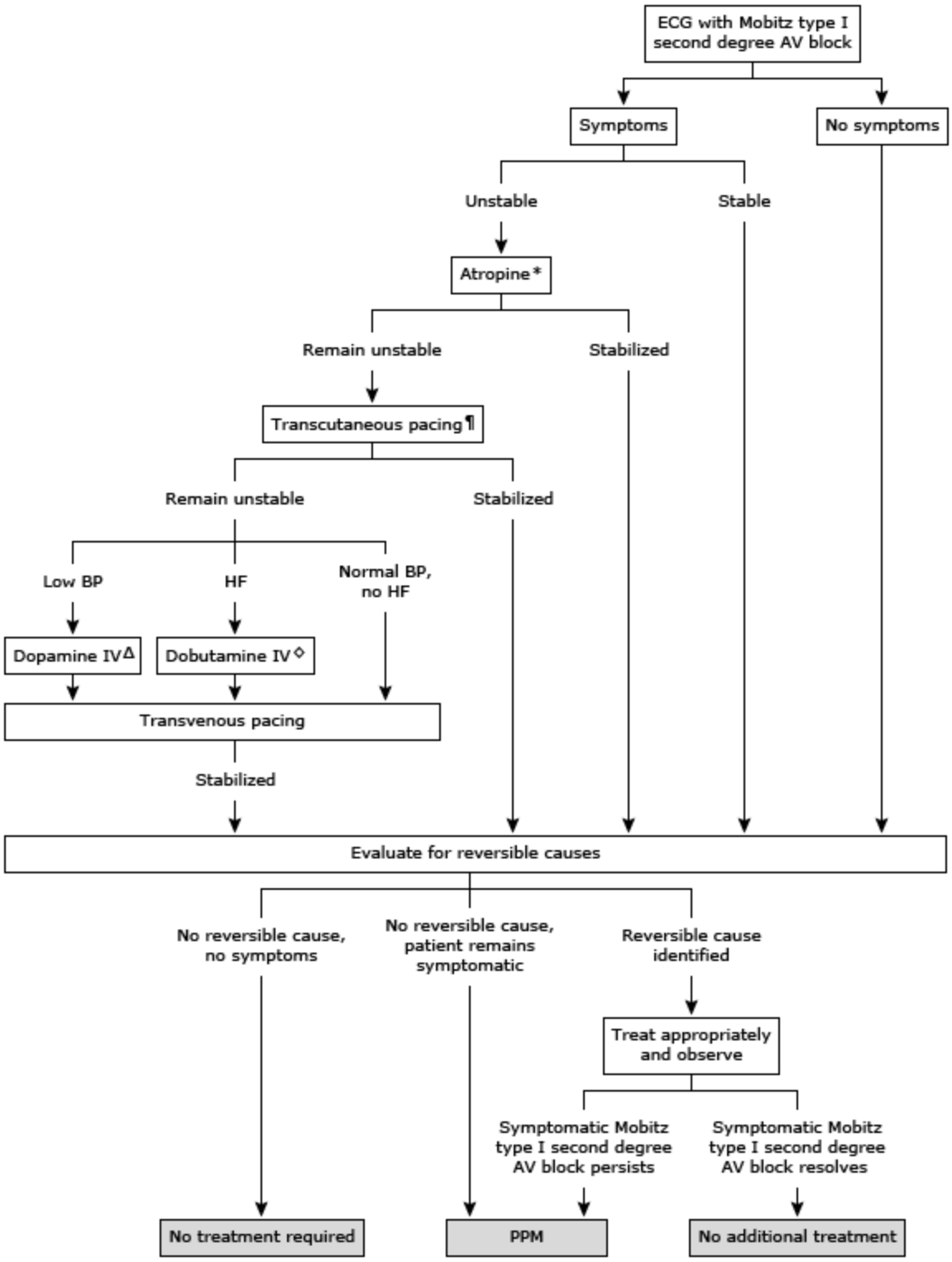


Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Algorithm management Mobitz I second degree AV block in adults



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

* 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.

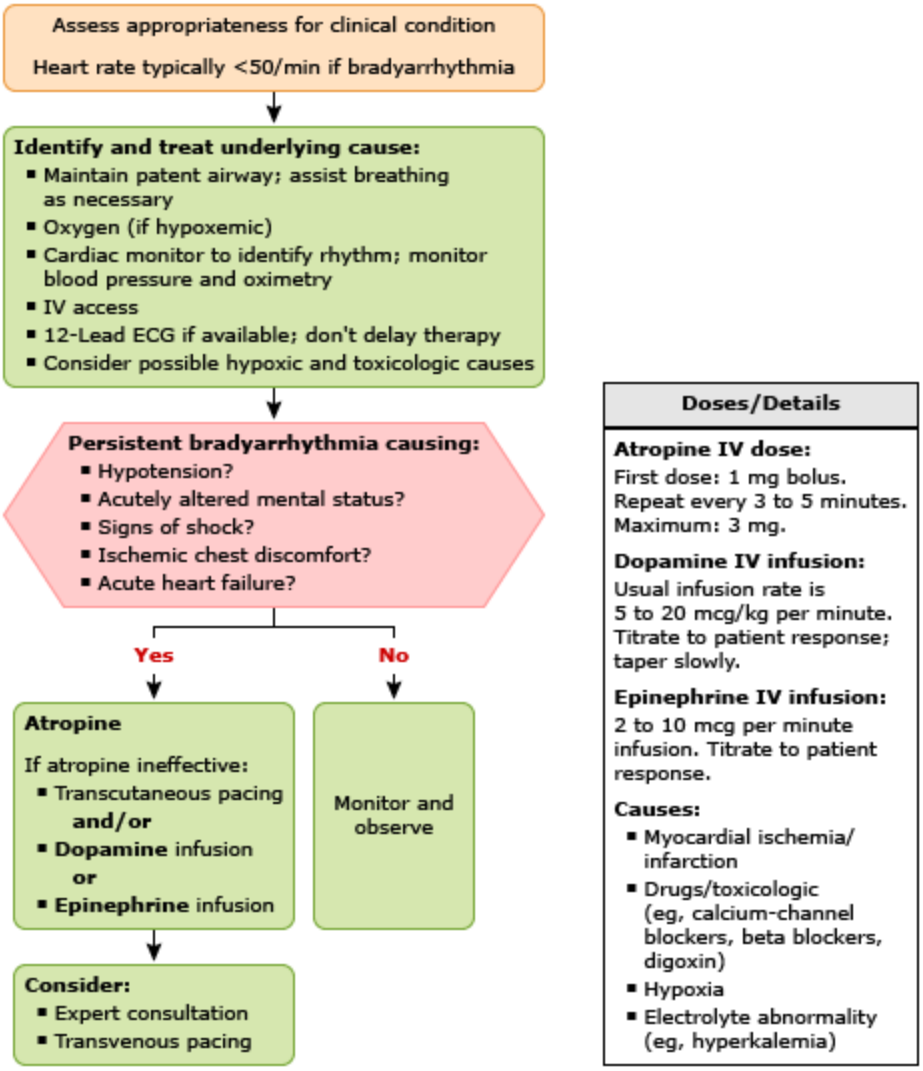
¶ While transcutaneous pacing may be initially successful in stabilizing the patient, it may not be consistently reliable. Central venous access should be considered in the event that urgent transvenous pacing is required.

Δ Dopamine IV infusion typically begins at a dose of 3 mcg/kg/minute and can be titrated up to 20 mcg/kg/minute if needed for heart rate and blood pressure augmentation.

◇ Dobutamine IV infusion typically begins at a dose of 5 mcg/kg/minute and can be titrated up to 20 mcg/kg/minute if needed for heart rate and blood pressure augmentation.

Graphic 109617 Version 1.0

Adult bradycardia algorithm 2020 update



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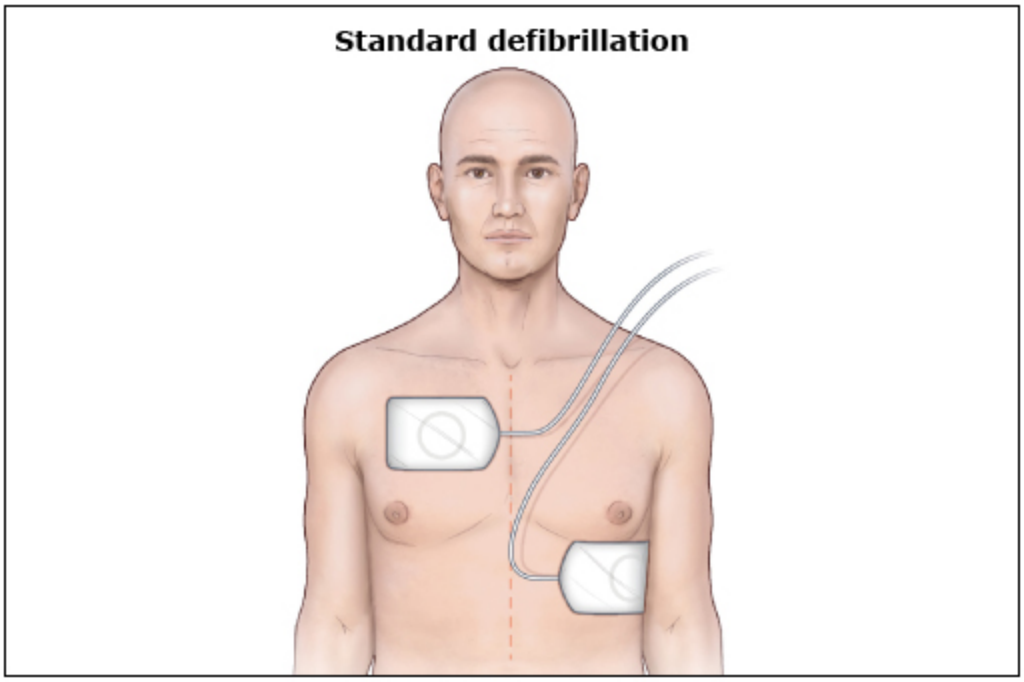
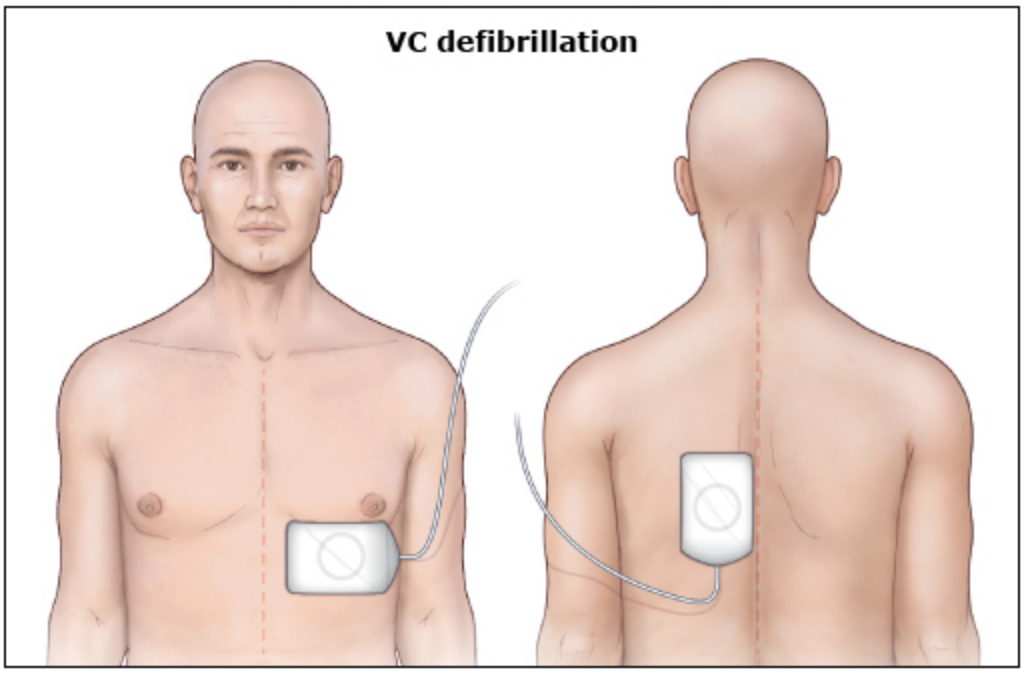
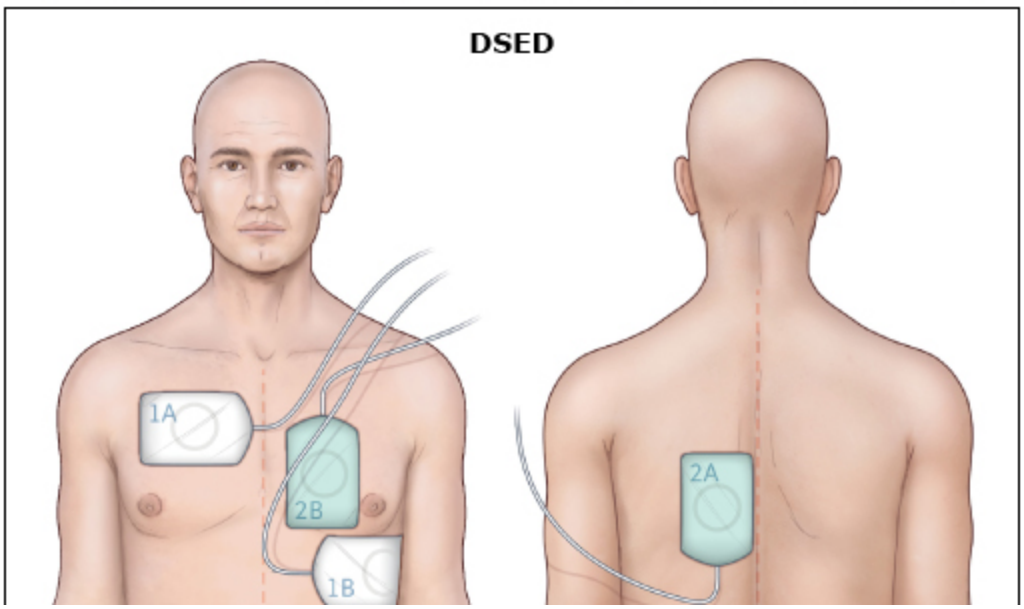
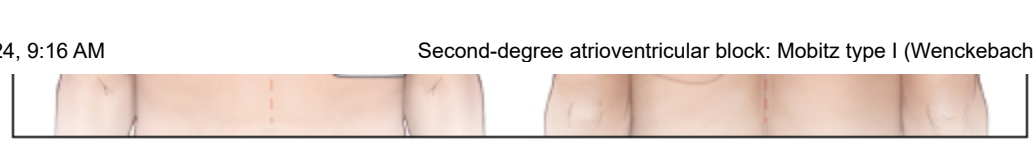
Graphic 130748 Version 11.0

Pad placement for defibrillation


Standard defibrillation


VC defibrillation


DSED
1A
2B
1B
2A



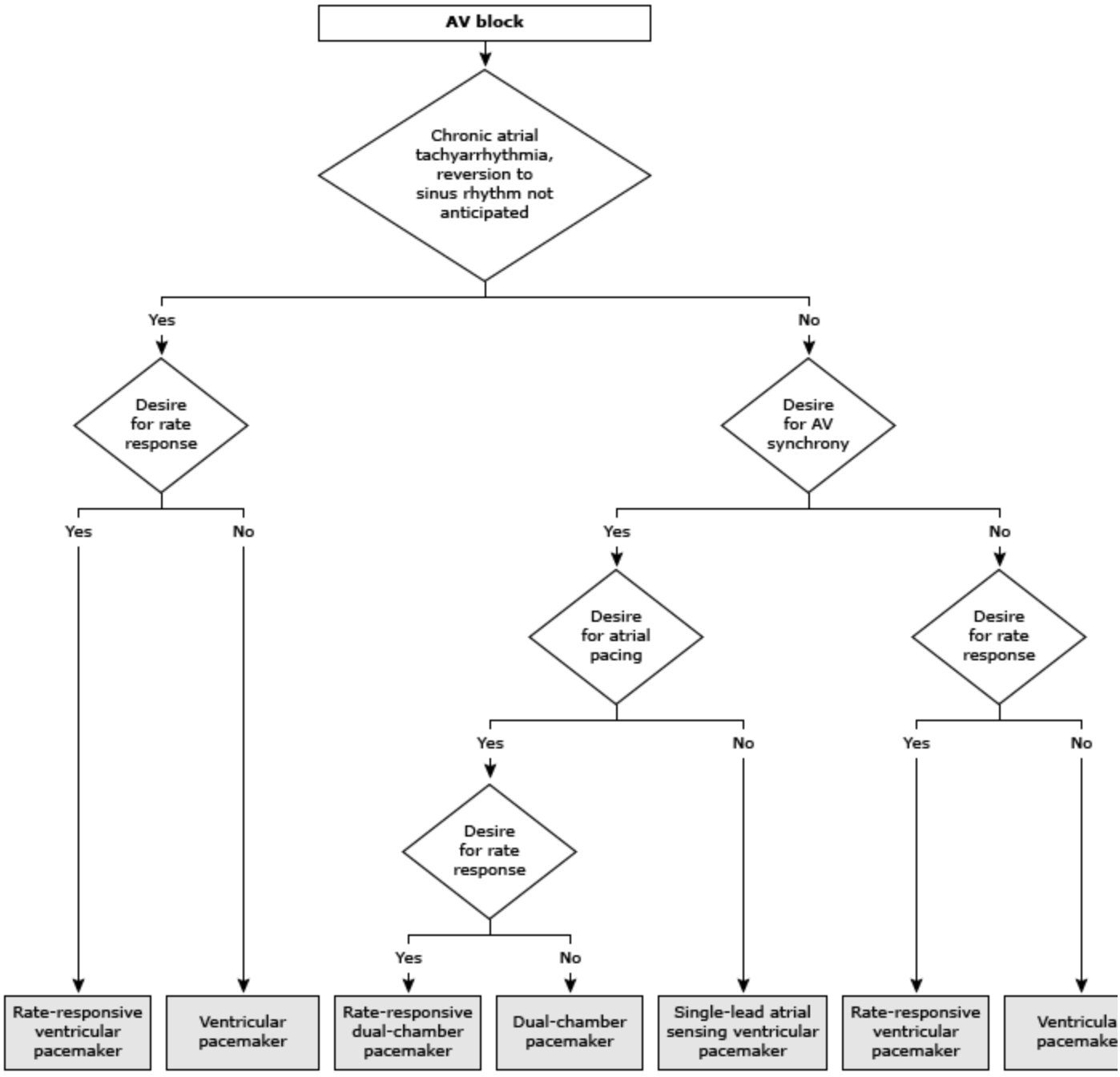
Vector change defibrillation consists of altering the direction of the vector. In clinical practice, this typically means moving the electrode pads from their standard position to an anterior-posterior position, as shown above. Double sequential external defibrillation (DSED) consists of rapid sequential shocks from 2 defibrillators.

DSED: double sequential external defibrillation; VC: vector change.

From: Cheskes S, Verbeek PR, Drennan IR, et al. Defibrillation strategies for refractory ventricular fibrillation. N Engl J Med 2022; 387:1947. Copyright © 2022 Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.

Graphic 103268 Version 3.0

Selection of pacemaker systems for patients with atrioventricular block



Decisions are illustrated by diamonds. Shaded boxes indicate type of pacemaker.

AV: atrioventricular.

Graphic 71370 Version 4.0