Bradycardia

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Sinus Node Dysfunction

Sinus node dysfunction (SND) is often used to describe abnormalities of impulse conduction originating from the Sinoatrial (SA) node. Degenerative changes in sinus node tissue occur throughout the lifespan and can lead to alterations in the generation or conduction of impulses, such as a prolonged pauses or sinus bradycardic episodes. Sinus node dysfunction is most common in individuals over 70 years of age. Sick sinus syndrome (SSS) refers to the symptomatic expression of sinus node dysfunction in patients resulting in fatigue, presyncope, syncope, dizziness, dyspnea, and other outward signs of cardiac output.

Tachycardia- bradycardia syndrome refers to a condition, when an individual has a co-morbid conduction abnormality resulting in a rapid atrial rate, such as atrial fibrillation, atrial flutter, or other supraventricular tachycardia. Upon conversion from the tachycardia, the sinus node fails to efficiently create an impulse resulting in a pause or bradycardia. Patients may or may not be symptomatic with the conversion pause or bradycardia which results.

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Sinus Bradycardia

In some individuals, including trained athletes, a heart rate below 50 bpm is acceptable and a normal variant. Bradycardias can be noted as a manifestation of increased vagal tone, normal aging, and are common in the elderly population as a result of disease progression such as in hypothyroidism. Symptomatic bradycardia is due to reduced cardiac output, which is a function of stroke volume and heart rate. The need for intervention is determined by the presence of symptoms.

Sinus Pause

A sinus pause, or sinus arrest, is the failure of the sinus node to generate an atrial depolarization for a period of time, generally defined as 3 seconds or longer between atrial contractions. Pauses can result from a block of the normal impulse from the sinoatrial tissue or due to failure of the sinus node to depolarize. Nocturnal pauses are commonly related to obstructive sleep apnea, which should be considered in the differential for assessment and in the treatment plan. Pauses are also more common in patients with tachycardia-bradycardia syndrome occurring when the tachyarrhythmia terminates and the sinus node is in recovery. The presence of sinus pauses, in absence of symptoms, does not always warrant

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Table 8.1 Causes of bradycardia

Medications	Tissue disorders	Metabolic	Miscellaneous
Antiarrhythmics	Amyloidosis	Hyper/hypokalemia	Acute MI
Beta blockers	Cardiomyopathies	Hypocalcemia	Autonomic dysfunction
Calcium Channel blockers (non-dihydropyridine)	Ischemic, non-ischemic, infiltrative	Hypothermia	Cardiac surgery
Digoxin	Connective tissue disease	Hypoxia	CABG, TAVR, maze, valve
Interferon	RA, SLE, scleroderma	Ion channel dysfunction	Replacement, ablation
Lithium	Hemochromatosis		Hypothyroidism
Methyldopa	Sarcoidosis		Infection
Opioids	Degenerative fibrosis		Lyme disease, typhoid fever
Risperidone			Dengue fever, malaria
Psychotropic meds			Guillain-Barre
Sympatholytics			Obstructive sleep apnea
Illicit drugs			
Toxins			

Goldberger et al. [1], Kusumoto et al. [2], Semelka and Gera [3]

intervention and can be associated with various physiologic and pathologic conditions as well as extrinsic factors including medications, electrolyte imbalance, increased vagal tone, and others (*see* Table 8.1). Frequent sinus pauses lasting longer than 3 seconds and are symptomatic warrant consideration for pacing support.

Chronotropic Incompetence

Chronotropic incompetence is defined as the inability of the heart rate to adjust appropriately in concordance with increased physical activity or cardiovascular demand. Patients can present with fatigue, lightheadedness, dyspnea on exertion, or syncope associated with activity. Further criteria for diagnosis of chronotropic incompetence, which is well established, includes the failure of the individual to reach 80% of their maximum predicted heart rate at peak exercise. This can be evaluated with exercise stress testing on a treadmill or bicycle. It is important to thoroughly assess individuals in whom there is suspicion for chronotropic incompetence as the condition is also associated with increased risk of coronary artery disease and is seen in approximately one-third of individuals with congestive heart failure [4]. In these patients, pacemaker implantation can provide symptom relief through rate responsive pacing (see Chap. 13).

Atrioventricular Blocks

A disturbance of impulse conduction between the atria and ventricles is known as atrioventricular (AV) block or heart block. This can occur if there is delayed conduction, intermittent loss of conduction, or complete loss of conduction from the atria to the ventricles. AV block/heart block is categorized based on the severity of the impulse conduction disturbance. The types of AV block will be addressed separately below.

First Degree AV Block (See Figs. 8.1 and 8.2)

First-degree heart block is defined as prolonged conduction from the atria to the ventricles with a PR interval greater than 200 ms. This can be secondary to a conduction delay at the AV node and/or the His-Purkinje system. The site of delay can be difficult to differentiate, though one clue is response to exercise. Increased sympathetic tone can increase conduction velocity in AV node

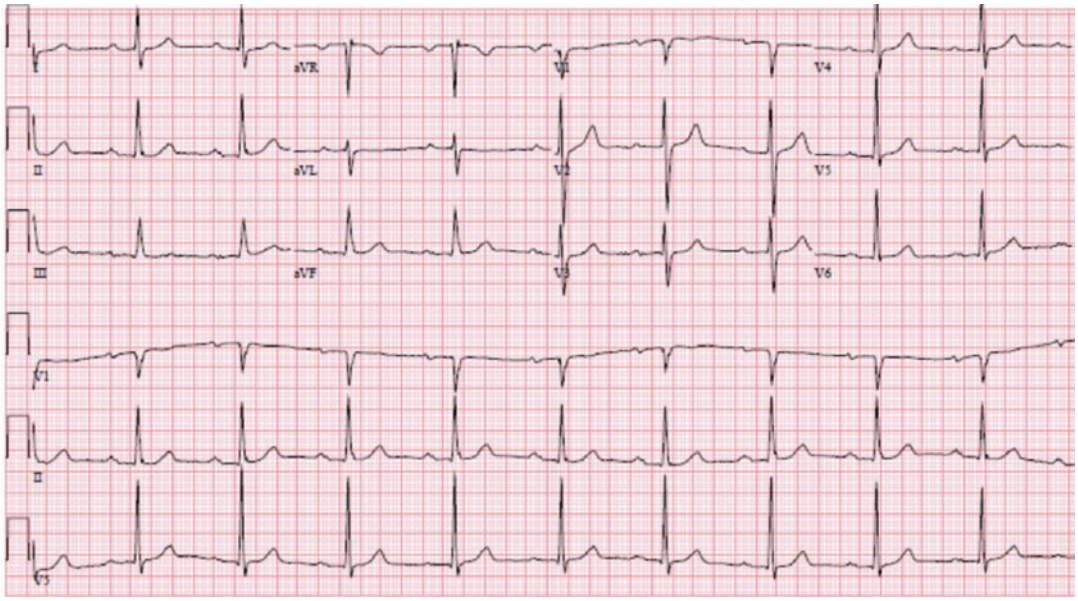

Fig. 8.1 Sinus rhythm with first-degree AV block. PR interval ~ 250 ms

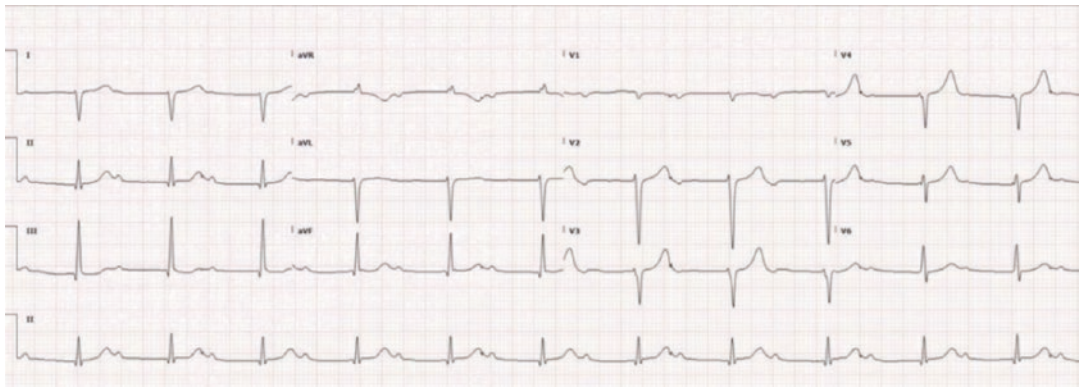

Fig. 8.2 Sinus rhythm with first-degree AV block. PR interval ~420 ms.

thereby shortening the PR interval if that is where the delay is occurring; absent PR shortening would suggest a lower level of delay.

Second-Degree AV Block (See Figs. 8.3 and 8.4)

Second-degree heart block is characterized by intermittent conduction from the atria to the ventricles. There are two separate types:

Mobitz Type I is also referred to as Wenckebach. This is noted on the ECG by progressive PR interval prolongation followed by a single non-conducted P wave.

Mobitz Type II appears on the ECG as a constant PR interval with intact conduction followed by a single non-conducted P wave.

Mobitz I and Mobitz II block refer to ECG patterns. Mobitz I block is more commonly at the level of the AV node and more responsive to changes in autonomic tone, occurring for example frequently with sleep in patients otherwise normal AV conduction. Mobitz II block, conversely, can be both in the AV node and His-Purkinje system, the latter to be suspected when

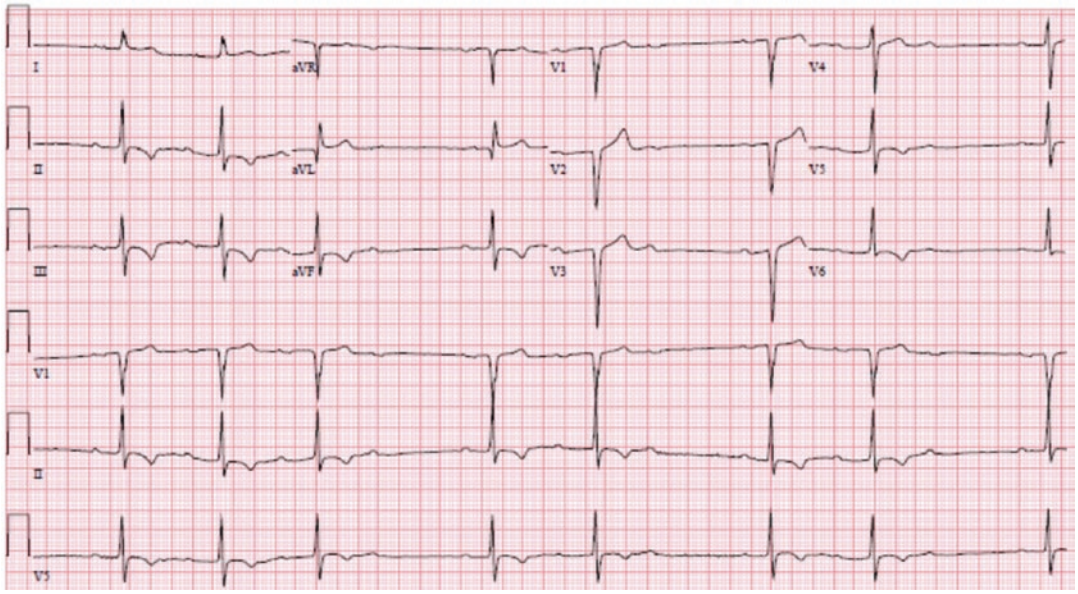

Fig. 8.3 Mobitz Type I (Wenckebach). PR interval gradually prolongs until a QRS is dropped and the next PR interval is shorter than the one prior to the drop

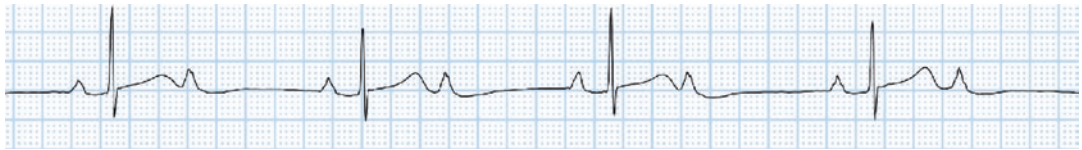

Fig. 8.4 Mobitz Type II. PR interval of conducted beats is stable, every other beat is dropped and QRS is narrow indicating block in the AV node

conduction disease (bundle branch or fascicular block) is present. This form of AV block is less responsive to changes in autonomic tone.

Advanced/High-Grade AV Block (Fig. 8.5)

High-grade AV block, which is differentiated from third-degree (complete) AV block, denotes intermittent conduction from the atria to the ventricles. When conducting from the atria to the ventricles, the PR interval is constant; however, there can be two or more consecutive non-conducted P waves (unlike second-degree heart block type II, where there is only a single non-conducted P wave). The mechanism of block is like Mobitz II AV block, though this finding is reflective of more advanced disease.

Third Degree/Complete Heart Block (Fig. 8.6)

Third-degree heart block is characterized by the absence of conduction between the atria to the ventricles. This is also frequently described as a complete dissociation between the atria and the ventricles. The ventricular rate is consequently driven by an escape rhythm. Portions of the conduction system have enhanced automaticity with impulse generation at rates that classically slow as the site of escape moves lower in the conduction system. Escape rhythms from the AV node below the level of block are often quite stable and

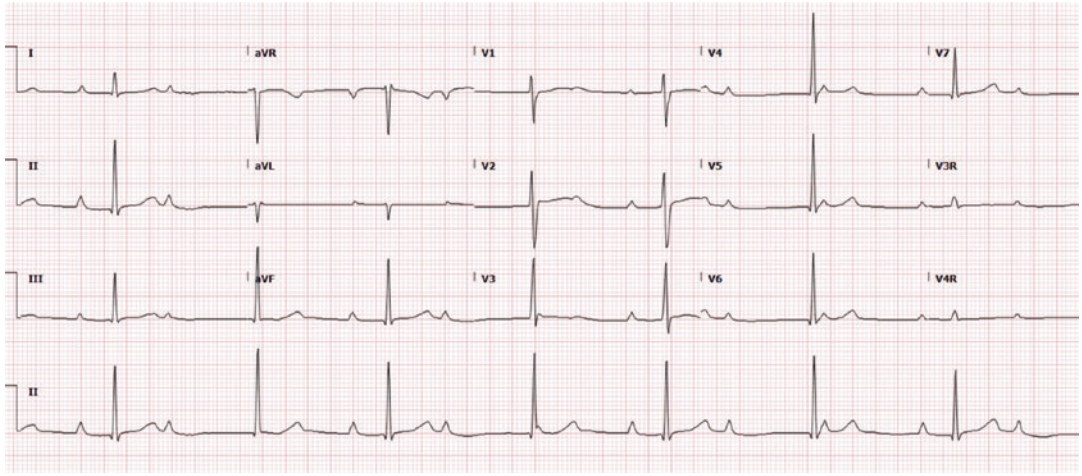

Fig. 8.5 Advanced or high-grade heart block

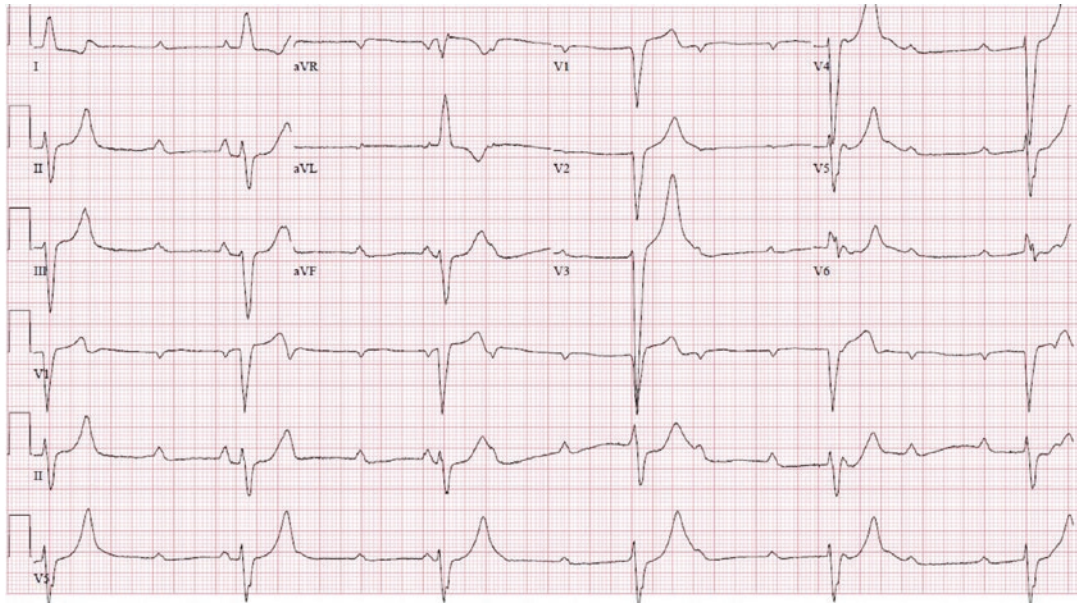

Fig. 8.6 Third-degree/complete heart block with LBBB escape rhythm

marked by a QRS complex nearly identical to baseline. Escape rhythms from the lower conduction system or ventricle, or wide complex escapes, are much less stable and high risk for hemodynamic instability. Placement of temporary pacemakers should be strongly considered in patients with wide escape rhythms, hemodynamically unstable heart rates, or presenting with syncope. Often, patients with complete heart block have compensatory hypertension due to increased SVR. Do not treat the hypertension. Vasodilating the patient may cause hemodynamic instability due to fixed cardiac output from the bradycardia.

Conduction System Abnormalities

Additional conduction abnormalities occur within the bundle branch system. The normal QRS interval ranges from 0.06–0.10 seconds; however, if a bundle branch block is present, the QRS duration is 0.12 seconds or longer. Despite the conduction delay associated with a bundle branch block, many patients remain asymptomatic. Changes in morphology related to bundle branch blocks can be best identified in various leads on the 12-lead ECG. In addition to the widened QRS interval, the QRS complex will have an abnormal shape. Furthermore, as depolarization is abnormal in a BBB, repolarization is also abnormal which results in T-wave inversion. Most commonly, this is noted in opposing deflection patterns of the QRS complex and T-waves of the same beat.

Right Bundle Branch Block (Fig. 8.7)

The electrical impulse of a right bundle branch block (RBBB) is conducted through the intraventricular septum down the faster conducting left bundle branch to the left ventricle. However, due to a block in conduction within the right bundle, the impulse reaches the RV through myocardial cell-to-cell conduction which is much slower. Electrocardiographic signs of RBBB include an initial small r wave in V_1 and V_2 (the right precordial leads) and a small q wave in leads I, aVL, V_5, and V_6 as septal depolarization from the left bundle is unaffected. Often the rSR' pattern that results in V_1 and V_2 is coined "rabbit ears" given the appearance of the two points on the R waves.

Right bundle branch blocks are the most common conduction disorder of the ventricular conduction system and can occur without any underlying cause or obvious pathology. Acutely, a RBBB can be seen in patients with ischemia or anterior MI and in additional associated conditions such as heart failure, valvular heart disease, and pulmonary embolism. Notably, the presence of a RBBB in a patient without significant heart disease does not increase risk of cardiovascular (CV) death; thus, if the patient is otherwise asymptomatic, no treatment is indicated. In patients with known CV disease, having a RBBB is an independent risk factor for all-cause mortality. In the setting of acute MI or the post MI period, a RBBB is associated with increased risk of mortality.

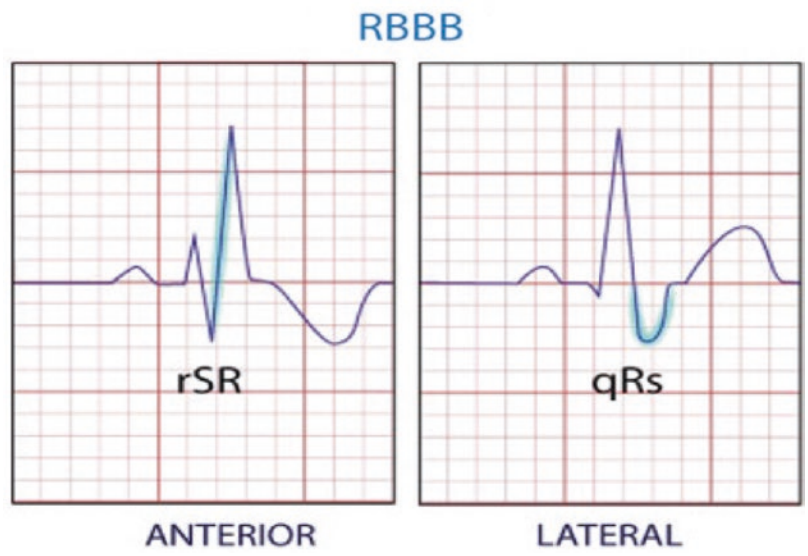

Fig. 8.7 Right bundle branch block morphology (Cardiology, Chang [5]: Springer)

Left Bundle Branch Block (Fig. 8.8)

In a left bundle branch block (LBBB), electricity moves quickly down the right bundle branch to depolarize the right ventricle. Next the impulse is carried more slowly across the interventricular septum and finally to the left ventricle, which is in opposition to normal depolarization. Electrocardiographic characteristics of a LBBB include a deep, wide QRS complex in lead V_1 and a large R wave in lead V_6, as well as a wide QRS complex with a T-wave in the opposite direction from the primary QRS deflection [6]. Often a left axis deviation is also present.

Conditions associated with LBBB include myocardial infarction (MI), hypertension, severe aortic stenosis (AS), Lenégre disease (a primary degenerative disease of the conduction system), and various cardiomyopathies. Cardiac surgery and trans-catheter aortic valve replacement as well as primary amyloidosis are all associated with increased risk of conduction disease. Left bundle branch blocks are more common in the elderly and in those with heart disease and should signal the need for further investigation as to causation such as echocardiography or stress testing. Of note, if a LBBB is found in the presence of acute MI, the patient has much greater risk of developing complete heart block leading to implantation of a pacemaker.

Due to the mechanism of the delayed conduction in a LBBB, the right ventricle receives the electric impulse before the left ventricle. In time, this can cause remodeling of the left ventricle due to alterations in left ventricular perfusion, mechanics, and workload. In the setting of heart failure, the presence of a LBBB is an independent predictor of mortality despite other risks associated with underlying disease, gender, or age. In comorbid heart failure with reduced ejection fraction, <35%, a QRS interval greater than 150 ms, and Class II or greater symptoms of heart failure, cardiac resynchronization therapy (CRT) is reasonable and may be recommended.

Left Anterior Fascicular Block (LAFB)

The left bundle branch further divides into the anterior and posterior fascicles. Conduction down the left bundle depolarizes the interventricular septum and causes a septal Q-wave in leads I, aVL, and V6. If the anterior fascicle is not conducting, the impulse proceeds from posterior to anterior resulting in a profound Left axis. The Q wave is present in the lateral leads as stated above. A small R wave is present with a very

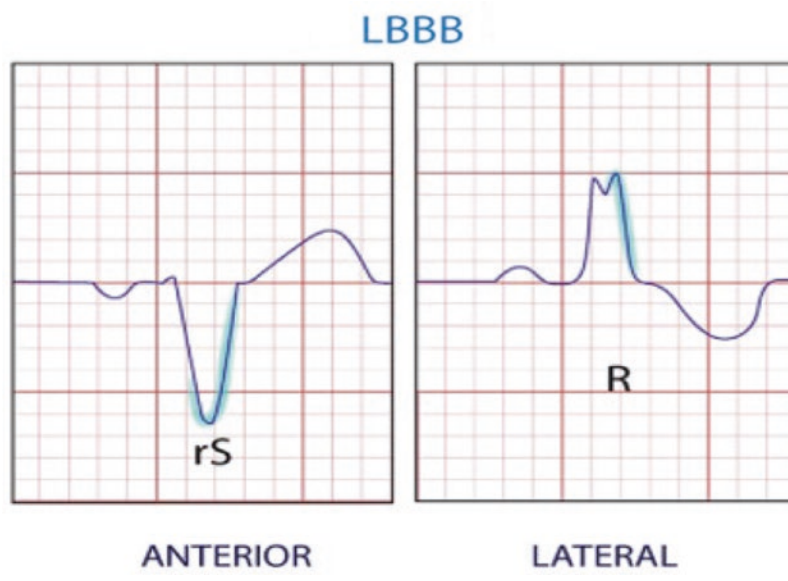

Fig. 8.8 Left bundle branch block morphology (Cardiology, Chang [5]: Springer)

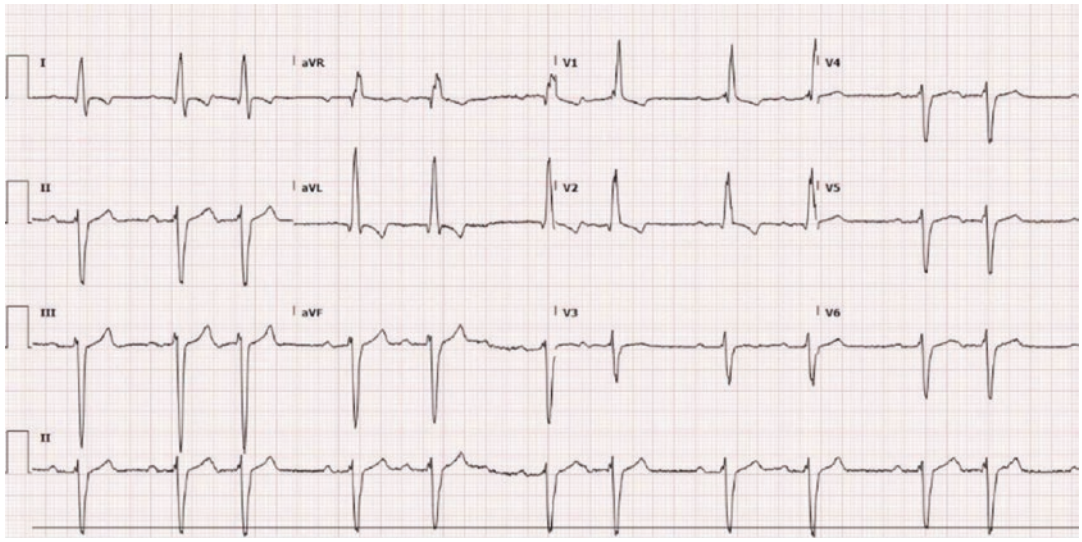

Fig. 8.9 First degree AV block with LAFB and RBBB

negative deflection in leads II and III as well. If a RBBB is present with a left axis, consider a LAFB which is called bifascicular block. If first degree block is also seen, it is called trifascicular block and has a higher risk of symptomatic bradycardia (*see* Fig. 8.9).

Evaluation and Management of Conduction Abnormalities

To evaluate causes of bradycardia, a thorough history and physical exam should be performed. When taking the history, emphasis should be placed on evaluation of the timing and relationship of symptoms to activity, meals, stress, position changes, and other triggers. A list of current medications, both prescription and non-prescription, are also vital to evaluating causation. Furthermore, the family history and complete cardiac history should be investigated. A comprehensive physical examination including carotid pulses and auscultation for bruits, peripheral pulses, heart sounds (to assess for structural/valvular disease), obtaining orthostatic vitals, and assessing for signs of systemic illness are of importance.

Following the history and physical exam, a 12-lead electrocardiogram (ECG) is the most helpful, initial test. Interpretation of the ECG rhythm with attention to interval lengths and waveforms and can further direct diagnosis and management. Based on the patient's history, symptoms, physical exam, and ECG findings, further evaluation with ambulatory cardiac monitoring may be completed. Home monitoring allows for a quantitative evaluation of arrhythmia as well as correlates the patient's symptoms with the timing and type of arrhythmia. The type of monitoring is dependent upon the frequency of symptoms. If the individual is experiencing daily symptoms, home monitoring through a wearable device is reasonable, noting more accurate diagnosis with longer recordings of 2–4 weeks as compared to a 24-to-48-hour monitoring window. For patients with less frequent symptoms of arrhythmia, or if syncope is the chief symptom, an implantable loop recording device should be considered. Furthermore, based on clinical history, if chronotropic incompetence is suspected, ambulatory heart monitoring and exercise stress testing is appropriate. As previously discussed, patients who have nocturnal sinus pauses should be evaluated for sleep apnea.

Laboratory studies to identify causes of bradycardia are directed by findings noted within the history and physical. Given potential underlying etiologies of thyroid disease, metabolic syn-

dromes, electrolyte imbalance, and infectious processes, a metabolic panel including magnesium, a complete blood count, and thyroid function testing are reasonable to assess. Guidelines also support that individual with conduction disease be screened with Lyme titers, if they reside in endemic regions. For younger patients presenting with advanced blocks, further investigation for connective tissue diseases such as sarcoidosis and amyloidosis is warranted. Lastly, in patients with conduction disorders caused by genetic mutations including SCN5A sodium channel and LMNA (Lamin A/C) mutations, it is recommended that their first-degree relatives likewise undergo gene testing (Table 8.1).

For patients with incidentally diagnosed first-degree AV block and second-degree type I AV block, no immediate pacing intervention is required. Consideration of exercise stress testing, particularly with second-degree type I AV block is recommended to ensure adequate chronotropic competence. A transthoracic echocardiogram is recommended to evaluate for structural heart disease. If this is unrevealing for abnormality and the patient is asymptomatic, observation without further intervention can be considered. Ambulatory monitoring (2–4 weeks) is appropriate to ensure that there is no further underlying AV nodal disease such as second-degree type II AV block or high-degree AV block.

For patients with symptomatic second-degree type II AV block, high-degree AV block or third-degree AV block temporary pacing may need to be considered while further workup for reversible causes is performed. Furthermore, if no reversible cause is identified or if the patient will require AV nodal blocking agents for management of comorbidities, then permanent pacing is indicated. Temporary and permanent pacing will be discussed in Chap. 13.

Pearls

- Symptoms are the defining factors determining whether bradycardia needs treatment.
- Look for underlying causes of bradycardia before recommending permanent pacing.
- Type 1 and Type II Mobitz I do not need treatment.
- March out the P waves and then the R waves to determine their relationship.
- Normal PR is <200 ms.
- Normal QRS 0.6–0.10.

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