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Basics of the 12-lead ECG: Part 2

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Abstract: A 12-lead ECG is a noninvasive diagnostic tool that detects and records cardiac electrical activity and can identify cardiac pathology. This article discusses select ECG abnormalities including myocardial infarction, bundle-branch blocks, and heart blocks. This is part 2 of a two-part series. Part 1 was published in the November 2023 issue of *Nursing2023*.

Keywords: atrioventricular block, bundle-branch block, cardiology, cardiovascular disease, ECG, heart block, left bundle-branch block, LBBB, myocardial infarction, non-ST-segment elevation myocardial infarction, NSTEMI, right bundle-branch block, RBBB, ST-segment elevation myocardial infarction, STEMI

Case study

A 42-year-old male presented to a clinic for his annual physical exam. He reported nausea and sweating since earlier that morning. He denied any other complaints. The nurse noted that he was diaphoretic. He had a history of type 2 diabetes and hypertension. The nurse recognized this patient's potential for a cardiac event.

The initial interventions included a 12-lead ECG, which revealed ST-segment elevation in leads V1 and V2. The patient's physician was immediately notified and ordered 325 mg of chewable aspirin and supplemental oxygen at 4 liters/minute via nasal cannula to maintain oxygen saturation above 90%.

After reviewing the patient's 12-lead ECG, health history, and presenting signs and symptoms, the nurse and the physician determined that the patient was experiencing an acute ST-segment elevation myocardial infarction (STEMI). The patient was then transported by emergency medical services to the hospital for cardiac catheterization and percutaneous coronary intervention of the left anterior descending artery. The patient tolerated the procedure well and was

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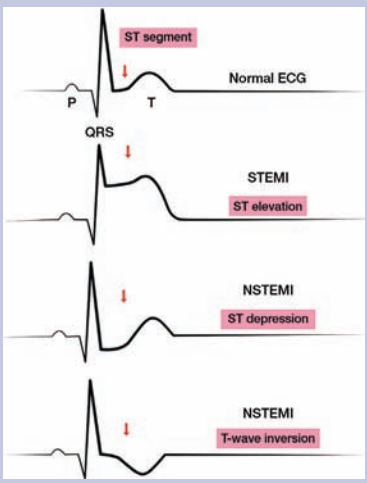
Signs and symptoms of MI[2,3]

Patient variables	Classic signs and symptoms
Men commonly present with more classic signs and symptoms	• Pallor • Diaphoresis • Anxiety • Chest pressure or tightness • Crushing chest pain that increases with exertion • Chest pain radiating into left arm, shoulder, and jaw. • Shortness of breath worse with activity.
Females, older adults, and patients with diabetes or chronic kidney disease commonly present with more atypical symptoms	**Atypical signs and symptoms** • Throat, neck, or jaw pain • Shoulder or back pain • Nausea • Vomiting • Heartburn, indigestion, or abdominal pain • Feeling tired or overwhelmingly fatigued

transferred to the CCU for observation and additional care.

The ECG is a noninvasive diagnostic tool that detects and records cardiac electrical activity and is useful in detecting cardiac pathology.[1] The electrical conduction system of the heart, the anatomy of an ECG complex, ECG lead views, and a systematic method of ECG interpretation were discussed in part 1 of this two-part series. This second installation discusses select ECG abnormalities including myocardial infarction (MI), bundle-branch blocks, and heart blocks.

ST-T wave changes



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Select ECG abnormalities

Myocardial infarction[2]

Acute MI is a potentially life-threatening state caused by a sudden decrease in oxygenation to the cardiac muscle. Time from onset of signs and symptoms to treatment is essential in patients with acute coronary syndromes (ACS), especially those with STEMI. The most common pathophysiologic process for acute MI starts with rupture of eroded atherosclerotic coronary artery plaque causing a decrease in myocardial blood flow. Other acute causes include complete obstruction of coronary artery blood flow because of emboli or vasospasm. Chronic conditions that can cause MI include thyrotoxicosis, hypotension, anemia, and respiratory failure. Use of cocaine and methamphetamine may lead to ACS secondary to increased myocardial oxygen demand. In addition, cocaine can lead to coronary thrombosis, arterial dissection, or arterial spasm.

Increased age is a risk factor for acute MI. The first MI occurs at an average age of 65.6 years in males and 72 years in females. A family history of MI increases the risk of cardiac events. Modifiable risk factors include nonsteroidal anti-inflammatory drugs, unhealthy diet such as a high consumption of red meat, inactivity, obesity, dyslipidemia, hypertension, diabetes, metabolic syndrome, cigarette smoking, and chronic kidney disease.

On exam, the patient may appear pale, diaphoretic, and anxious. Vital signs may vary depending on the presence and intensity of pain and complications, such as cardiac dysrhythmias and cardiogenic shock. Pulmonary crackles may indicate heart failure or cardiogenic shock (see *Signs and symptoms of MI*).

Chest pain is a common symptom of patients experiencing ACS. Pain is commonly described as pressure or tightness and may become severe, especially in STEMI. Chest pain usually lasts longer than 10 minutes but can exceed hours with a STEMI. Nurses should be aware that not all patients experiencing cardiac events present with the common complaint of chest pain. There are two basic classifications for MI, non-ST segment elevation myocardial infarction (NSTEMI) and STEMI.

NSTEMI

NSTEMI is significantly more common than STEMI and is identified by elevated serum cardiac biomarkers. However, NSTEMI should be considered in any patient presenting with signs and symptoms of myocardial ischemia, even without evidence of ECG changes.[3] The ECG may demonstrate evidence of ischemia, such as ST depression or T-wave inversion without ST-segment elevation or pathologic Q waves (see *ST-T wave changes*). These ST-T wave abnormalities are commonly localized to the leads associated with the region of ischemic myocardium (see *Coronary arteries*). Incomplete occlusion of a coronary artery is generally the cause of NSTEMI.

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Coronary arteries

Coronary arteries and their branches		Portion of myocardium supplied	Portion of conduction system supplied
Right coronary artery		• Right atrium • Right ventricle • Inferior wall of left ventricle (90%)* • Posterior one-third of interventricular septum (90%)*	• Sinoatrial (SA) node (55%)* • Atrioventricular (AV) node and bundle of His (90%)*
Left coronary artery	Left anterior descending (LAD)	• Anterior wall of left ventricle • Portion of lateral wall of left ventricle • Anterior two-thirds of interventricular septum • Left atrium • Lateral wall of left ventricle	• Right and left bundle branches
	Circumflex	• Posterior wall of left ventricle • Inferior wall of left ventricle (10%)* • Posterior one-third of interventricular septum (10%)*	• SA node (45%)* • AV node and Bundle of His (10%)*

* = of population.

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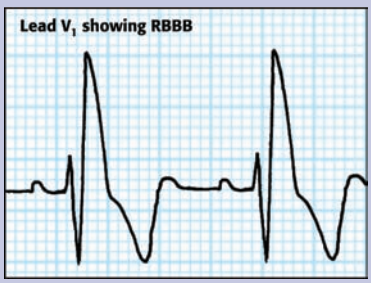
RBBB



In RBBB, the path of the mean QRS vector is changed due to left-to-right slow conduction; lead V1 now records a delayed R wave approaching it, resulting in a positive R wave. The key identifier of RBBB in lead V1 is a QRS complex wider than 0.12 second with a delayed (longer than 0.07 second) positive main R wave. Some RBBBs may display a triphasic waveform ("rabbit ears") consisting of a small r wave, downward S wave, and a second, larger R wave.

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STEMI

Specific ECG criteria for the diagnosis of STEMI include new ST-segment elevation at the J-point in two contiguous leads greater than or equal to 0.1 mV in all leads other than leads V2 to V3. For leads V2 to V3, diagnostic criteria include greater than or equal to 2 mm ST-segment elevation in men 40 years of age or older; greater than or equal to 2.5 mm in men less than 40 years, or greater than or equal to 1.5 mm in women regardless of age. ST-segment elevation should prompt an assessment for other ECG changes, an evaluation for signs and symptoms of MI, and evaluation of serum cardiac biomarkers. Identifying ST-segment elevation in patients with or without signs and symptoms is essential to reduce morbidity and mortality.[2]

Bundle-branch blocks

Right bundle-branch block[4]

A right bundle-branch block (RBBB) is generally considered benign without any underlying structural cardiac disorders. An RBBB indicates that the right ventricle is not directly activated by the impulses traveling through the right bundle branch, causing a delay in the ventricular conduction through the bundle of His and the Purkinje fibers. On an ECG, this is depicted as a widened QRS complex and changes in the directional vectors of the R and S waves. These findings are due to the left ventricle's rapid depolarization and the right ventricle's slower depolarization (see *RBBB*). The QRS complex duration is at least 0.12 second. RBBBs are common in older adults and those with hypertension. An RBBB may be a marker of progressive heart disease, including ischemic heart disease, cardiomyopathy, myocarditis, valvular heart disease, and congenital heart disease. It can also be caused by underlying conditions such as pulmonary embolism and pulmonary hypertension.

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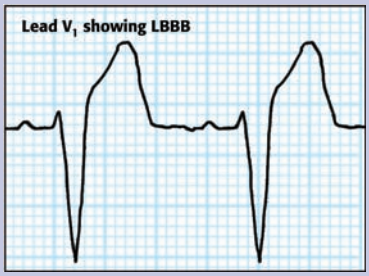
LBBB



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Left bundle-branch block

In a left bundle-branch block (LBBB), electrical and mechanical ventricular dyssynchrony lead to left ventricular remodeling.[5] An LBBB may result from an intraventricular conduction delay, complete conduction block, or a combination.[5] It may be related to conduction system degeneration or myocardial pathology, including acute MI.[3,6] LBBB is associated with heart disease, heart failure, and mortality. The anatomy of an LBBB varies among individuals in size, pattern, number, and location.[5] The ECG will show a prolonged QRS complex at least 0.12 second. A broad notched or slurred R wave will be in leads I, aVL, V5, and V6. The Q wave will be absent in leads I, V5, and V6. The R-peak time is greater than 0.06 second in leads V5 and V6. The ST and T waves progress in opposite directions to the QRS complex (see *LBBB*).[5] LBBB is associated with higher mortality after an MI.

Heart blocks

First-degree atrioventricular (AV) block[7]

First-degree AV block results from a delay in the cardiac electrical impulse traveling through the AV node. Due to this delay, the PR interval is prolonged. A normal PR interval is 0.12-0.20 second; however, in first-degree AV block, the PR interval measures greater than 0.20 second. Given that the PR interval is the only impact seen in first-degree AV block, each PR interval is followed by a QRS complex (*see Sinus bradycardia with first-degree AV block*).

This electrical disturbance is noted more frequently in older adult males. There are several causes of this condition. Occasionally it presents because of a congenital anomaly or due to previous myocardial ischemia or infarction. Inflammatory conditions, such as myocarditis, and chronic autoimmune diseases, such as systemic lupus erythematosus, can also predispose patients to first-degree AV block. Electrolyte imbalances, such as hyperkalemia, may result in this type of heart block. Medications that slow conduction time through the AV node, such as digoxin, calcium channel blockers, and beta blockers, are also known to cause first-degree AV block. Patients with first-degree AV block are generally asymptomatic, and treatment is dependent upon the underlying cause.

Second-degree AV blocks[8]

Type I (Mobitz I or Wenckebach)

The hallmark of this block is a progressive prolongation of the PR interval leading to the eventual drop of a QRS complex (nonconducted P wave). The site of the block is typically the AV node (see *Second-degree AV block, Mobitz I*). This occurs due to the lack of one or more atrial impulses not conducted to the ventricles.

Causes for Wenckebach are varied. Medications such as beta-blockers, non-dihydropyridine calcium channel blockers, and digoxin may be implicated. Myocardial ischemia involving the conduction system, especially, acute inferior MI or ischemia can be a cause. Infectious causes include myocarditis secondary to Lyme disease. Electrolyte imbalances such as hyperkalemia and hypermagnesemia may also be causative. Patients with this conduction defect are usually asymptomatic but may occasionally feel "skipped" beats. Treatment is aimed at correcting underlying causes.

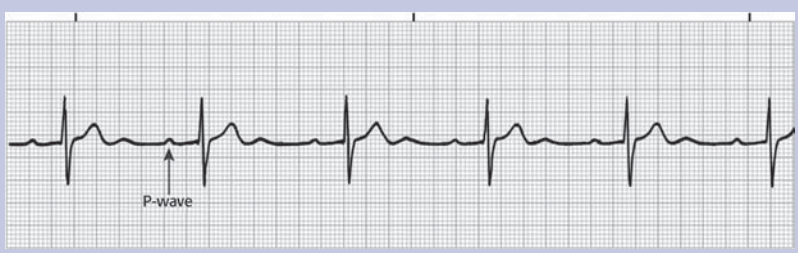
Second-degree AV block, Type II (Mobitz II)

The etiology of this block is below the AV node, in the bundle of His

Sinus bradycardia with first-degree AV block

Rhythm: Regular
Rate: 47 to 50 beats/minute
P waves: Sinus P waves present; one P wave to each QRS complex
PR interval: 0.32 second (remains consistent)
QRS complex: 0.08 to 0.10 second
Comment: A U-wave is present.

First-degree AV block is the least serious form of heart block, produces no signs or symptoms, and requires no specific treatment. Rarely does this form of heart block progress to a higher degree of AV block. Drugs causing AV block should be reviewed and discontinued if indicated.



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or in the bundle branches.[8] The PR interval is consistent, and P waves will maintain a consistent rate. However, not all P waves will be followed by a QRS complex, indicating conduction loss (see *Second-degree AV block, Mobitz II with 2:1 AV conduction*). The QRS, when present, may be wide (0.12 second or greater).

Causes of Mobitz II include structural abnormalities such as fibrosis or cardiomyopathies, ischemia, particularly anterior wall MI, medications as mentioned under Wenckebach, inflammatory conditions such as myocarditis and endocarditis, and hyperkalemia.

Patients are usually symptomatic and may be hemodynamically unstable with signs and symptoms including syncope. Hemodynamically unstable patients require continuous cardiac monitoring and treatment according to advanced cardiovascular life support (ACLS) guidelines, which may include temporary cardiac pacing. Permanent cardiac pacing may ultimately be necessary. Patients exhibiting Mobitz II are more likely to progress to third-degree AV block.

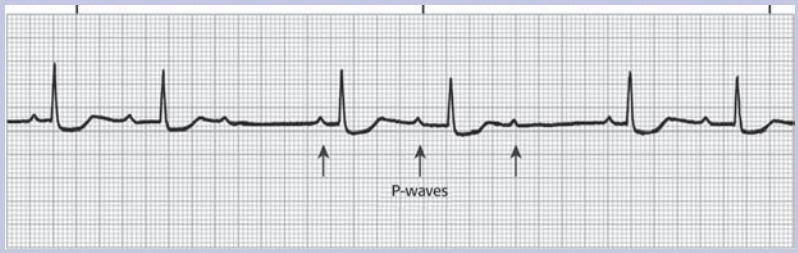
Third-degree AV block (complete heart block)[7]

Third-degree AV block or complete heart block occurs when there is no conduction of electrical impulses from the atria to the ventricles. Patients with third-degree AV block will have P waves and QRS complexes independent of each other (see *Third-degree AV block*). The cardiac rhythm is maintained from an ectopic focus, meaning by pacemaker cells outside of the normal electrical pathway. Conducted impulses in third-degree AV block are generally considered escape rhythms. The heart rate in these patients is typically below 40 beats/minute.

Second-degree AV block; Mobitz I

Rhythm: Regular (atrial); irregular (ventricular)
Rate: 71 beats/minute (atrial) 50 beats/minute (ventricular)
P waves: Sinus *P* waves present
PR interval: Progressively lengthens from 0.20 to 0.32 second
QRS complex: 0.06 to 0.08 second
Comment: ST-segment depression is present.

In second-degree AV block, Mobitz I (also known as Mobitz type I and Wenckebach), the initial impulse leaves the sinus node, travels through the AV node, and is conducted to the ventricles, but each successive impulse has increasing difficulty passing through the AV node until finally an impulse does not pass through and is not conducted to the ventricles.



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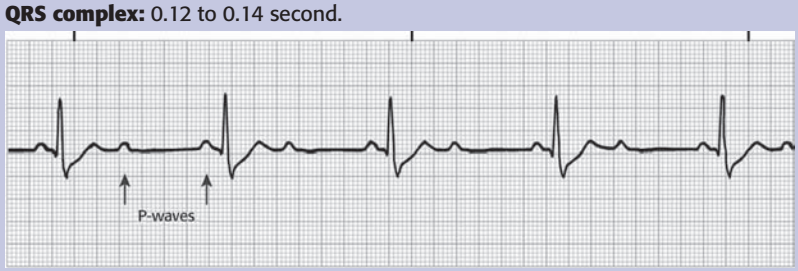
Second-degree AV block, Mobitz II with 2:1 AV conduction

Rhythm: Regular (atrial and ventricular rhythm)
Rate: 79 beats/minute (atrial); 41 beats/minute (ventricular)
P waves: Two sinus *P* waves to each QRS
PR interval: 0.16 second (remains consistent)
QRS complex: 0.12 to 0.14 second.



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Causes for third-degree heart AV block are similar to those for second-degree AV blocks. Inferior and anterior MIs are of particular concern for causing third-degree AV block. Patients are usually symptomatic and can exhibit signs and symptoms including hypotension, lightheadedness, syncope, angina, and loss of consciousness. Hemodynamically unstable patients should be treated as per ACLS guidelines and may require permanent cardiac pacing.

Conclusion

The 12-lead ECG is a diagnostic tool often used to rule-in and rule-out cardiac events. Recognizing basic abnormalities such as RBBB, LBBB, STEMI, NSTEMI, and heart blocks as well as patient

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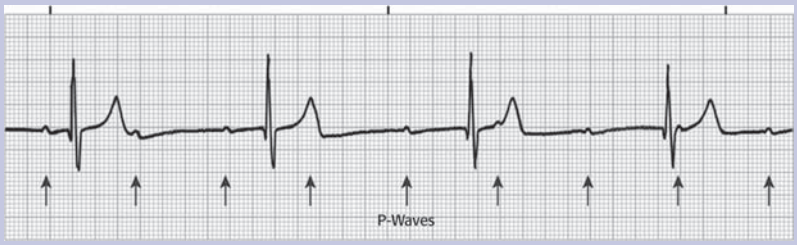
Third-degree AV block

Rhythm: Regular (atrial); regular (ventricular)
Rate: 75 beats/minute (atrial); 33 to 34 beats/minute (ventricular)
P waves: Sinus P-waves; no relationship to QRS complexes; found hidden in QRS complexes, ST segments, and T-waves
PR interval: Varies greatly
QRS complex: 0.12 second



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signs and symptoms is important to ensure quality patient care. Nurses can positively impact patients' morbidity and mortality by timely recognition and intervention. ■

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The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NURSE.0000991560.56637.b6

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