DRACO LEARNING

Healthcare Education

ECGS AND BASIC ARRHYTHMIA RECOGNITION

A Comprehensive Textbook for EMTs, CNAs, and Emergency Care Technicians

Draco Learning

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Dedication

To Becky,

my beautiful, amazing wife.

Your love, patience, and unwavering support make everything possible.

This work — and every good thing I do — is for you.

ABOUT THIS TEXTBOOK

This textbook was developed by **Draco Learning**, a division of Draco Inc., to provide Emergency Medical Technicians (EMTs), Certified Nursing Assistants (CNAs), and individuals entering the role of Emergency Care Technician (ECT) with a complete, practical, and comprehensive education in electrocardiography and cardiac arrhythmia recognition. It is intended to serve as both a study guide for initial training and a lasting reference resource throughout your career.

Electrocardiography is one of the most powerful diagnostic tools in medicine. Mastering it opens doors professionally, sharpens your clinical instincts, and — most importantly — makes you a more effective advocate for your patients. This textbook treats you as the intelligent, capable professional you are. Nothing is glossed over. Everything that matters is explained fully, in plain language.

Who This Textbook Is For

This course is specifically designed for:

- **Emergency Medical Technicians (EMTs)** transitioning into or working in hospital-based emergency settings.
- **Certified Nursing Assistants (CNAs)** working or preparing to work in the emergency department or on telemetry units.
- **Emergency Care Technicians (ECTs)** who will be responsible for acquiring 12-lead ECGs and monitoring patients on continuous cardiac monitors.
- **Any healthcare support staff member** whose role includes cardiac monitoring, ECG acquisition, or bedside patient observation.

No prior ECG experience is assumed. If you have never looked at an ECG strip before, this textbook will take you from zero to confident. If you have some experience, this textbook will fill in the gaps and deepen your understanding.

How This Textbook Is Organized

This textbook follows a logical, progressive structure designed to build your knowledge layer by layer:

- **Chapters 1 and 2** build your foundation: how the heart works, its anatomy, and the electrical conduction system that drives the heartbeat.
- **Chapter 3** teaches the practical skill of electrode placement for 4-lead monitoring, 10-electrode setup, and 12-lead ECG acquisition — with detailed anatomical guidance and troubleshooting.
- **Chapters 4 and 5** cover ECG equipment, ECG paper, and how to read and measure the waves and intervals on a strip — the core interpretive skills.
- **Chapter 6** establishes Normal Sinus Rhythm as your gold-standard benchmark.
- **Chapter 7** is the clinical core of this course — a comprehensive, detailed examination of every major cardiac arrhythmia you will encounter in practice.
- **Chapter 8** covers ECG changes in ischemia and acute myocardial infarction (heart attack), including STEMI recognition.
- **Chapter 9** provides a complete step-by-step procedure for performing a 12-lead ECG, with troubleshooting and documentation guidance.
- A comprehensive **Glossary** and **Quick Reference Appendix** are included at the back for rapid review.

Medical and Legal Disclaimer

This textbook is an educational resource intended for healthcare support personnel. The information herein is based on established clinical guidelines and educational standards at the time of publication. Medical knowledge evolves — always follow your facility's current policies, protocols, and the direction of licensed supervising clinicians.

The role of the EMT, CNA, and ECT is to **recognize and report**, not to diagnose or treat. Nothing in this textbook should be interpreted as authorization to independently diagnose cardiac conditions or initiate treatment without appropriate supervision and licensure.

Your Most Important Responsibility

You are a vital link in the chain of survival. Your eyes on the monitor, your hands placing the electrodes correctly, and your voice immediately reporting an abnormality are exactly what saves lives in the emergency department.

Take this training seriously. Every arrhythmia described in this book represents a real patient. Every delay in recognition has real consequences. Your competence matters.

Publisher Information

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CHAPTER 1: THE HEART — ANATOMY, PHYSIOLOGY, AND WHY IT MATTERS FOR ECGS

Learning Objectives

Upon completing this chapter, you will be able to:

1. Describe the location, size, and protective structures of the heart.
2. Identify the four chambers of the heart and explain the function of each.
3. Describe the role of the four cardiac valves.
4. Trace the complete path of blood through the heart and lungs.
5. Explain the difference between systole and diastole.
6. Define the myocardium, endocardium, and epicardium.
7. Explain the coronary circulation and why it matters clinically.
8. Define depolarization and repolarization and relate them to ECG waveforms.
9. Explain the concept of automaticity and its clinical significance.

1.1 The Heart: Location, Size, and Structure

The **heart** is a hollow, muscular, four-chambered organ responsible for circulating blood throughout the entire body. In an average adult, it is approximately the size of a closed fist and weighs between 250 and 350 grams (roughly half a pound). Despite its modest size, the heart performs an extraordinary amount of work — contracting approximately 100,000 times per day, pumping roughly 5 liters of blood per minute at rest, and delivering oxygen and nutrients to every cell in the body without pause for an entire lifetime.

Location

The heart sits in the **mediastinum** (mee-dee-as-TY-num) — the central compartment of the thoracic cavity (the chest). It is located between the lungs, behind the sternum (breastbone), and in front of the thoracic spine. The heart is positioned slightly to the LEFT of the body's midline. Its lower pointed end — called the **apex** (AY-peks) — points downward and to the left, resting just above the diaphragm at approximately the level of the 5th intercostal space. The upper broader end — the **base** — faces upward and to the right.

This anatomical position is why we place chest electrodes on the LEFT side of the chest rather than the right — the left ventricle (the largest, most muscular chamber) dominates the electrical activity and faces toward the left chest wall.

The Pericardium: The Heart's Protective Sac

The heart is enclosed in a tough, double-layered fibrous sac called the **pericardium** (pair-ih-KAR-dee-um). The word "pericardium" comes from the Greek "peri" (around) and "kardia" (heart). The pericardium has two layers:

- **Fibrous pericardium** (outer layer): A tough, non-elastic, fibrous outer covering that anchors the heart to the mediastinum, prevents the heart from over-expanding, and protects it from trauma and infection.
- **Serous pericardium** (inner layer): A thinner, smooth inner layer that itself has two layers — the **parietal layer** (lining the inside of the fibrous pericardium) and the **visceral layer** (which forms the outermost layer of the heart wall itself, also called the **epicardium**).

Between the two layers of the serous pericardium is a thin space — the **pericardial cavity** — which contains 15 to 50 mL of **pericardial fluid**. This fluid acts as a lubricant, reducing friction as the heart beats.

Clinical Connection: Pericarditis and Pericardial Effusion

PERICARDITIS is inflammation of the pericardium. It causes sharp, pleuritic chest pain (worse with breathing and lying flat, relieved by leaning forward). On the ECG, pericarditis produces diffuse ST elevation in a "saddle-shaped" pattern across multiple leads — which must be carefully distinguished from STEMI.

PERICARDIAL EFFUSION occurs when excess fluid accumulates in the pericardial cavity. Large effusions compress the heart from outside, reducing its ability to fill and pump — a life-threatening condition called CARDIAC TAMPONADE. On the ECG, tamponade produces "electrical alternans" — alternating larger and smaller QRS complexes as the heart swings within the fluid.

The Three Layers of the Heart Wall

The wall of the heart is made up of three distinct layers, from outside to inside:

Layer	Description and Function
Epicardium (outer)	The outermost layer of the heart wall. Also serves as the visceral layer of the serous pericardium. Contains fat, coronary arteries and veins, and lymphatic vessels. Provides a smooth, friction-free surface.
Myocardium (middle)	The thick middle layer — the heart muscle itself. Made of specialized cardiac muscle cells (cardiomyocytes) that contract to pump blood. The thickness of the myocardium varies: the left ventricle has the thickest walls (because it works hardest), while the right ventricle and atria are thinner.
Endocardium (inner)	The smooth inner lining of all four heart chambers and the heart valves. Made of thin, smooth cells (endothelium) that allow blood to flow over the surface without clotting. Inflammation of the endocardium is called endocarditis.

1.2 The Four Chambers of the Heart

The heart is divided into four distinct chambers by two structural walls:

- The **interatrial septum** (inter = between, atrial = atria) divides the right and left atria.
- The **interventricular septum** (inter = between, ventricular = ventricles) divides the right and left ventricles — and is the thickest internal wall of the heart.

Together, these septa create a right side and a left side of the heart that function as two separate pumps operating in parallel. The right side handles the pulmonary circulation (to the lungs); the left side handles the systemic circulation (to the entire body).

Right Atrium (RA)

The **right atrium** is the upper-right chamber and serves as the receiving chamber for oxygen-depleted blood returning from the body. It receives blood from three sources:

- The **superior vena cava** (SVC): returns blood from the upper half of the body (head, neck, arms, upper chest).
- The **inferior vena cava** (IVC): returns blood from the lower half of the body (abdomen, pelvis, legs).
- The **coronary sinus**: a venous channel on the posterior surface of the heart that collects blood from the coronary veins (blood that has circulated through the heart muscle itself).

The wall of the right atrium is relatively thin (2–3 mm), reflecting the low pressure required to push blood only into the adjacent right ventricle. The right atrium also contains an important structure called the **sinoatrial (SA) node** — the heart's primary pacemaker, embedded in its upper wall near the junction with the superior vena cava. We will discuss the SA node in detail in Chapter 2.

Right Ventricle (RV)

The **right ventricle** is the lower-right chamber and is responsible for receiving blood from the right atrium and pumping it to the lungs via the **pulmonary artery**. The right ventricle has a crescent-shaped cross-section (wrapping around the left ventricle) and a moderate wall thickness (3–5 mm) — enough to generate the relatively low pressure needed to push blood through the pulmonary circulation, but far less than the left ventricle.

The pulmonary circulation — from the right ventricle to the lungs and back to the left atrium — is a low-resistance, low-pressure circuit. Pressure in the main pulmonary artery is approximately 25/10 mmHg, compared to 120/80 mmHg in the systemic circulation. This pressure difference explains why the right ventricle has a much thinner wall than the left ventricle.

Left Atrium (LA)

The **left atrium** is the upper-left chamber. It receives **oxygen-rich blood** returning from the lungs via the four **pulmonary veins** (two from the right lung, two from the left lung). The pulmonary veins are unique — they are the only veins in the body that carry oxygenated blood.

The left atrium has a slightly thicker wall than the right atrium and operates at a somewhat higher pressure. Within the left atrium is a small pouch called the **left atrial appendage (LAA)** — a finger-like protrusion that is clinically significant because it is the most common site for blood clot formation in patients with atrial fibrillation. When the atria are not contracting effectively (as in A-Fib), blood can pool in the LAA, form clots, and those clots can travel to the brain and cause strokes.

Left Ventricle (LV)

The **left ventricle** is the largest, most powerful chamber of the heart and the one that generates most of the electrical activity visible on the ECG. Its wall is 8 to 15 mm thick — approximately three times thicker than the right ventricle — because it must generate enough pressure (approximately 120 mmHg systolic) to pump blood through the entire systemic circulation, from the aortic valve to the capillaries of every organ and tissue in the body.

The left ventricle has a conical shape, with its apex pointing toward the lower-left. The apex of the heart (and thus the left ventricle) is the point of maximal impulse (PMI) that you can sometimes feel or see pulsating on the anterior chest wall.

Chamber	Key Facts
Right Atrium (RA)	Thin-walled. Receives oxygen-poor blood from body via SVC, IVC, coronary sinus. Contains SA node (pacemaker). Passes blood to RV through tricuspid valve.
Right Ventricle (RV)	Moderately thin-walled. Pumps blood to lungs at low pressure through pulmonary artery. Low-resistance circuit.
Left Atrium (LA)	Slightly thicker than RA. Receives oxygen-rich blood from lungs via 4 pulmonary veins. Left atrial appendage is clot-formation site in A-Fib.
Left Ventricle (LV)	Thickest walls (8-15 mm). Most powerful pump. Generates systemic blood pressure (~120/80). Dominates the ECG signal.

1.3 The Cardiac Valves: One-Way Doors

The heart has four valves — two on the right side and two on the left side — that ensure blood flows in only one direction, prevent backflow (regurgitation), and maintain the efficiency of the cardiac pump. Valves open passively when the pressure behind them exceeds the pressure in front of them, and close when pressure reverses.

Atrioventricular (AV) Valves

The **atrioventricular valves** sit between the atria and ventricles and open during diastole (ventricular filling) and close during systole (ventricular contraction). They are held in place by fibrous strings called **chordae tendineae** (KOR-dee ten-DIN-ee-ee — "heartstrings") attached to muscular projections on the ventricular wall called **papillary muscles**. These structures prevent the valves from being blown backward into the atria during ventricular contraction.

- **Tricuspid valve**: Three leaflets (cusps). Located between the right atrium and right ventricle. Allows blood to flow from RA to RV during diastole; closes during systole to prevent backflow into the RA. "Tricuspid" = three + cusp.
- **Mitral valve** (bicuspid valve): Two leaflets. Located between the left atrium and left ventricle. Allows blood from LA to LV during diastole; closes during systole to prevent backflow into the LA. The mitral valve is the most commonly diseased valve in clinical practice.

Semilunar Valves

The **semilunar valves** are located at the outlet of each ventricle — between the ventricle and its outflow artery. They have three cup-shaped (semilunar = half-moon) leaflets. These valves open during systole (when the ventricles contract and eject blood) and close during diastole (when the ventricles relax) to prevent blood from flowing back from the arteries into the ventricles.

- **Pulmonary valve**: Between the right ventricle and the pulmonary artery. Opens during right ventricular systole to allow blood flow to the lungs.
- **Aortic valve**: Between the left ventricle and the aorta. Opens during left ventricular systole to allow blood to flow into the systemic circulation. The three leaflets of the aortic valve are also where the left and right coronary arteries originate — immediately above the valve.

Why Valves Matter for ECGs

Valve disease can affect the ECG in multiple ways:

• Mitral stenosis (stiff, narrowed mitral valve) causes left atrial enlargement — seen on ECG as a notched, broad P wave (P-mitrale) in Lead II.
• Aortic stenosis (stiff, narrowed aortic valve) forces the left ventricle to work harder, causing left ventricular hypertrophy (LVH) — seen as very tall R waves in V5–V6 and deep S waves in V1–V2.
• Valve problems can also cause arrhythmias, particularly atrial fibrillation (enlarged atria from increased pressure become electrically unstable).

As an ECT, you will not diagnose valve problems, but understanding them helps you understand why a patient's ECG may look unusual.

1.4 Blood Flow Through the Heart: A Step-by-Step Journey

Understanding the complete blood flow pathway is essential for understanding what the ECG is recording. Let's trace a single red blood cell through the entire circuit:

Step 1: Returning to the Right Atrium

After delivering oxygen to the body's tissues, oxygen-depleted blood (now carrying carbon dioxide) travels through the venous system. The blood from the upper body collects into the **superior vena cava**; blood from the lower body into the **inferior vena cava**. Both empty into the **right atrium**. The blood arriving at the right atrium is dark red (because it is low in oxygen) and under very low pressure.

Step 2: Right Atrium to Right Ventricle

When the right atrium contracts (atrial systole), the **tricuspid valve** opens, and blood flows passively — and then with a gentle push from the atrial contraction — down into the **right ventricle**. The atrial contraction provides the final "top-off" of ventricular filling (called the "atrial kick"), contributing approximately 20–30% of the total ventricular filling volume. This is why loss of organized atrial contraction (as in atrial fibrillation) can cause symptoms — the ventricles lose that final filling contribution.

Step 3: Right Ventricle to Lungs (Pulmonary Circulation)

When the right ventricle contracts (ventricular systole), the **tricuspid valve closes** (preventing backflow into the RA) and the **pulmonary valve opens**. Blood is ejected into the **pulmonary artery** (the only artery in the body that carries oxygen-poor blood) and flows into the lungs. This circuit is called **pulmonary circulation** (from the Latin "pulmo," meaning lung).

In the capillary beds surrounding the air sacs (alveoli) of the lungs, two critical gas exchanges occur:

- **Carbon dioxide** (a waste product of cellular metabolism) diffuses from the blood into the air in the alveoli and is exhaled.
- **Oxygen** (from the air you inhale) diffuses from the alveoli into the bloodstream, binding to hemoglobin in the red blood cells.

Step 4: Returning to the Left Atrium

The now oxygen-rich blood (bright red) leaves the lungs through four **pulmonary veins** and flows into the **left atrium**. The pressure in the left atrium is slightly higher than in the right atrium, reflecting the greater demands of the systemic circuit.

Step 5: Left Atrium to Left Ventricle

The **mitral valve** opens, and blood flows from the left atrium into the **left ventricle** — the powerhouse of the heart. Again, the atrial contraction provides the final 20–30% of ventricular filling. This is the same reason A-Fib causes symptoms in patients with poor left ventricular function — they rely more heavily on that atrial kick.

Step 6: Left Ventricle to the Body (Systemic Circulation)

When the left ventricle contracts powerfully, the **mitral valve closes** (preventing backflow into the LA) and the **aortic valve opens**. Blood is ejected into the **aorta** (the largest artery in the body, approximately 2.5 cm in diameter at its base) and distributed throughout the entire systemic circulation. This circuit is called **systemic circulation**.

The pressure generated by the left ventricle is approximately 120 mmHg during systole — enough to push blood to the fingertips, toenails, and brain simultaneously. After delivering oxygen and picking up carbon dioxide and waste products, the blood cycles back to the right atrium and begins again.

1.5 Systole, Diastole, and the Cardiac Cycle

The **cardiac cycle** is the complete sequence of events that occurs during one heartbeat — from the beginning of one contraction to the beginning of the next. The entire cardiac cycle normally lasts approximately 0.8 seconds at a heart rate of 75 bpm.

Key Terms: Systole and Diastole

SYSTOLE (SIS-toh-lee): The phase of active ventricular CONTRACTION. The ventricles squeeze and eject blood. During systole:

- Mitral and tricuspid valves are CLOSED (preventing backflow into atria).
- Aortic and pulmonary valves are OPEN (allowing blood to exit).
- Lasts approximately 0.3 seconds at rest.
- Corresponds to the SYSTOLIC blood pressure reading (top number — e.g., 120 in 120/80).

DIASTOLE (die-AS-toh-lee): The phase of ventricular RELAXATION and FILLING. The ventricles rest and fill with blood. During diastole:

- Aortic and pulmonary valves are CLOSED (preventing backflow from arteries).
- Mitral and tricuspid valves are OPEN (allowing blood to flow from atria).
- Lasts approximately 0.5 seconds at rest.
- Corresponds to the DIASTOLIC blood pressure reading (bottom number — e.g., 80 in 120/80).

At faster heart rates, both systole and diastole shorten. However, diastole shortens proportionally more than systole. This means that at very fast heart rates (e.g., 150–200 bpm), the ventricles have very little time to fill — which is one reason very fast heart rates can cause hemodynamic compromise (low blood pressure, dizziness, shortness of breath) even in patients with otherwise healthy hearts.

The Heart Sounds

The familiar "lub-dub" sound of the heartbeat is produced by the closing of the cardiac valves:

- **S1 ("lub")**: Produced by the closing of the **mitral and tricuspid valves** at the BEGINNING of systole. It marks the start of ventricular contraction.
- **S2 ("dub")**: Produced by the closing of the **aortic and pulmonary valves** at the END of systole (beginning of diastole). It marks the end of ventricular contraction.
- **S3 and S4**: Extra heart sounds heard in certain clinical conditions (heart failure, ventricular stiffness). Their presence may indicate the need for urgent evaluation.

1.6 The Coronary Circulation: The Heart's Own Blood Supply

Despite pumping millions of liters of blood over a lifetime, the heart cannot extract the oxygen it needs from the blood passing through its chambers — the endocardium (inner lining) is too thick, and the chambers are too large. Instead, the heart muscle has its own dedicated arterial blood supply: the **coronary arteries**.

The coronary arteries are the first branches off the aorta. They originate from the aortic root — just above the cusps of the aortic valve — from small openings called the **coronary ostia** (left and right). The coronary arteries fill during **diastole** (when the aortic valve is closed and the myocardium relaxes), not during systole (when the heart is squeezing so hard that blood flow into the coronary arteries is restricted). This is clinically important: when a patient's heart rate is very fast and diastole is very short, the heart has less time to receive its own blood supply.

The Main Coronary Arteries

Artery	Territory Supplied (Clinical Relevance)
Left Main Coronary Artery (LMCA)	Divides into LAD and LCx. Supplies majority of left ventricle. Obstruction here is often called "the widow-maker" — can be rapidly fatal.
Left Anterior Descending (LAD)	Anterior wall of LV, interventricular septum, apex, bundle branches. Most commonly occluded in anterior STEMI. ECG: ST elevation in V1–V4.
Left Circumflex (LCx)	Lateral wall of LV, sometimes posteroinferior wall. ECG: ST elevation in I, aVL, V5, V6.

Right Coronary Artery (RCA)	Inferior wall of LV, right ventricle, SA node (in ~60% of people), AV node (in ~90% of people). ECG: ST elevation in II, III, aVF. Also causes AV block rhythms.

Why Coronary Anatomy Matters for ECG Interpretation

When you see ST elevation in specific leads, you are looking at a "view" of the territory supplied by a specific coronary artery. This tells the cardiologist which artery to look for and open in the cardiac catheterization lab.

ECG changes are not random — they follow the anatomical distribution of the coronary arteries. Learning these patterns helps you understand WHY certain leads change in certain heart attacks.

Inferior STEMI (II, III, aVF) = Right Coronary Artery
Anterior STEMI (V1–V4) = Left Anterior Descending
Lateral STEMI (I, aVL, V5, V6) = Left Circumflex

Coronary Venous Drainage

After delivering oxygen to the myocardium, the blood drains through the **coronary veins** and collects in the **coronary sinus** — a venous channel that runs in the atrioventricular groove on the posterior heart and drains directly into the right atrium. The coronary sinus is visible on the ECG monitor when a patient has right atrial enlargement, and its anatomy is relevant in pacemaker lead placement.

1.7 The Myocardium: Cardiac Muscle Physiology

The myocardium is made of **cardiac muscle cells** called **cardiomyocytes** (KAR-dee-oh-MY-oh-sites). These cells have unique properties that distinguish them from both skeletal muscle and smooth muscle:

- **Striated**: Like skeletal muscle, cardiac muscle has a striped appearance under the microscope, reflecting the organized arrangement of contractile proteins (actin and myosin).
- **Involuntary**: Unlike skeletal muscle, cardiac muscle contracts automatically without conscious control from the brain.
- **Fatigue-resistant**: Cardiac muscle has extremely high concentrations of mitochondria (the energy-producing organelles of cells), enabling it to contract continuously for an entire lifetime without fatiguing.

- **Syncytial (electrically coupled)**: Cardiac muscle cells are connected to each other by specialized junctions called **intercalated discs** (in-TER-kuh-lay-ted), which contain **gap junctions** — channels that allow electrical impulses to pass directly from cell to cell. This makes the myocardium behave like a single coordinated unit: when one cell depolarizes, the impulse spreads rapidly to all adjacent cells. This is called the "functional syncytium" and is what allows a coordinated, wave-like contraction.

The Excitation-Contraction Coupling Process

Understanding how an electrical signal becomes a mechanical contraction is fundamental to understanding arrhythmias and their treatments:

1. An electrical impulse (action potential) arrives at a cardiomyocyte.
2. Sodium ions (Na+) rush into the cell through fast sodium channels — this is the **rapid depolarization phase**. The inside of the cell, which was negative at rest, rapidly becomes positive.
3. Calcium ions (Ca2+) enter the cell through slower L-type calcium channels — the **plateau phase**. This influx of calcium triggers the release of even more calcium from an intracellular store called the **sarcoplasmic reticulum (SR)**.
4. The calcium ions bind to the **troponin-tropomyosin complex** on the contractile proteins, allowing **actin and myosin** to interact and the cell to shorten — **contraction** occurs.
5. Calcium is actively pumped back into the SR and out of the cell. Potassium (K+) exits the cell. The cell returns to its resting negative charge — **repolarization** occurs.
6. The cell is now reset and ready for the next action potential.

This entire sequence — from electrical impulse to mechanical contraction and back to rest — takes approximately 300 milliseconds (0.3 seconds). The ECG traces the electrical portion of this process: the QRS complex represents step 1–4 (depolarization and the beginning of contraction), and the T wave represents step 5 (repolarization).

Key Term: Refractory Period

The refractory period is the time after depolarization during which a cardiac cell CANNOT be re-stimulated to fire again — even if it receives another electrical impulse. This is the heart's built-in protection against extremely rapid, chaotic stimulation.

ABSOLUTE REFRACTORY PERIOD: The cell is completely unresponsive — no stimulus, no matter how strong, can cause re-depolarization. Corresponds roughly to the QRS complex and most of the T wave.

RELATIVE REFRACTORY PERIOD: The cell CAN be stimulated by a very strong stimulus, but this is dangerous — re-stimulation during this period (particularly the peak of the T wave) can trigger lethal arrhythmias. This is called the "vulnerable period" and is the basis of the "R-on-T" phenomenon seen with dangerous PVCs.

The refractory period explains why the heart cannot be driven to impossibly fast rates by external electrical stimulation under most circumstances.

1.8 Depolarization and Repolarization: The Language of the ECG

Every wave on the ECG corresponds to one of these two fundamental electrical events. Understanding them is the key to understanding everything else in this course.

Depolarization — The "Fire" Signal

DEPOLARIZATION (dee-poh-lar-ih-ZAY-shun) is the electrical event in which a resting cardiac cell's internal charge rapidly changes from NEGATIVE to POSITIVE.

Mechanism: Sodium (Na+) and calcium (Ca2+) ions rush INTO the cell through channels in the cell membrane, driven by concentration gradients and electrical forces. This rapid influx makes the inside of the cell positive relative to the outside.

Mechanical result: Depolarization TRIGGERS CONTRACTION. Every time a cardiac muscle cell depolarizes, it physically shortens.

ECG representation: Depolarization of the atria = P wave. Depolarization of the ventricles = QRS complex.

Analogy: Depolarization is like pulling the trigger of a gun. The trigger pull (depolarization) causes the gun to fire (contraction).

Repolarization — The "Reset" Signal

REPOLARIZATION (ree-poh-lar-ih-ZAY-shun) is the electrical event in which a cardiac cell's internal charge returns from POSITIVE back to NEGATIVE (its resting state), making it ready to fire again.

Mechanism: Potassium (K+) ions rush OUT of the cell through potassium channels, restoring the negative resting membrane potential. Calcium is actively pumped back into the sarcoplasmic reticulum.

Mechanical result: Repolarization corresponds to RELAXATION of the muscle.

ECG representation: Ventricular repolarization = T wave. (Atrial repolarization occurs but is hidden within the larger QRS complex.)

Analogy: Repolarization is reloading the gun — the cell resets so it can fire again.

Drugs and electrolyte abnormalities can profoundly affect repolarization, which is why so many medications and conditions alter the appearance of the T wave and QT interval on the ECG. For example, **hypokalemia** (low potassium) slows repolarization, prolonging the QT interval and creating visible U waves. **Hyperkalemia** (high potassium) changes the shape of the action potential, producing peaked T waves, wide QRS complexes, and ultimately a sine wave pattern leading to cardiac arrest.

1.9 Automaticity: The Heart's Independent Pacemaker Cells

Unlike skeletal muscles, which can only contract when instructed by a nerve from the brain (requiring the motor nerve to be intact), the heart generates its own electrical impulses automatically and continuously. This property is called **automaticity** (aw-toh-mah-TIS-ih-tee).

Automaticity resides in a small subset of specialized cardiac cells called **pacemaker cells** or **P cells**. These cells are different from ordinary myocardial contractile cells in one critical way: their resting membrane potential is unstable. Instead of maintaining a stable resting charge, pacemaker cells **spontaneously drift toward depolarization** — their internal charge slowly becomes less negative over time until it reaches a threshold and spontaneously fires an action potential. This gradual drift is called **phase 4 depolarization** (or spontaneous diastolic depolarization).

The rate at which a pacemaker cell fires is determined by how quickly it drifts toward threshold. Cells that drift more quickly fire more often (higher rate). The **sympathetic nervous system** (via norepinephrine/epinephrine) speeds up this drift, increasing the heart rate. The **parasympathetic nervous system** (via acetylcholine released by the vagus nerve) slows this drift, decreasing the heart rate.

Automaticity is present in multiple sites throughout the heart, but at different intrinsic rates:

Pacemaker Site	Intrinsic Rate
SA Node (primary pacemaker)	60–100 bpm
AV Junction (AV node / Bundle of His)	40–60 bpm
Ventricular Purkinje fibers	20–40 bpm

Because the SA node fires fastest, its impulses suppress the slower pacemakers before they can spontaneously fire. This is called **overdrive suppression** — the faster pacemaker "drives" the slower ones, keeping them from taking over. When the SA node fails or slows excessively, the next fastest site (the AV junction) "escapes" suppression and takes over. This is the mechanism behind all escape rhythms.

Chapter 1 Review Questions

1. The left ventricle has the thickest myocardial walls because:

a) It has more chambers than the right ventricle
b) It must generate enough pressure to pump blood through the entire systemic circulation
c) It handles oxygenated blood, which requires more force to process
d) It is located on the left side of the body, which requires more muscle for balance

► **Correct Answer:** b) It must generate enough pressure to pump blood through the entire systemic circulation

Rationale: *The left ventricle generates approximately 120 mmHg of systolic pressure — far more than the right ventricle's ~25 mmHg — because it must push blood through the entire body. This demand requires much thicker, more muscular walls.*

2. During which phase of the cardiac cycle do the coronary arteries primarily fill with blood?

a) Systole — when the ventricles are contracting
b) Diastole — when the ventricles are relaxed
c) During the P wave on the ECG
d) During the QRS complex

► **Correct Answer:** b) Diastole — when the ventricles are relaxed

Rationale: *The coronary arteries primarily fill during diastole, when the myocardium is relaxed and not compressing the vessels, and when the aortic valve is closed (creating a pressure head in the aortic root). Very fast heart rates reduce diastolic time and can limit coronary perfusion.*

3. The "atrial kick" refers to:

a) The sound produced by the mitral valve closing
b) The final 20–30% of ventricular filling provided by atrial contraction
c) The SA node firing in the right atrium
d) The retrograde P wave seen in junctional rhythms

► **Correct Answer:** b) The final 20–30% of ventricular filling provided by atrial contraction

Rationale: *Atrial contraction at the end of diastole provides the final 20–30% of ventricular filling. This is lost in atrial fibrillation (where the atria quiver but don't contract), which is why A-Fib patients with poor LV function often feel significantly worse.*

4. The "vulnerable period" of ventricular repolarization, during which a premature stimulus can trigger ventricular fibrillation, corresponds to:

a) The PR interval
b) The peak of the T wave
c) The QRS complex
d) The isoelectric baseline

► **Correct Answer:** b) The peak of the T wave

Rationale: *The peak of the T wave represents the relative refractory period — when some cells have repolarized and some have not. A stimulus during this time (the R-on-T phenomenon with PVCs) can trigger ventricular fibrillation.*

5. Automaticity means:

a) The heart automatically speeds up during exercise
b) The ability of specialized cardiac cells to spontaneously generate electrical impulses
c) The automatic interpretation function of an ECG machine
d) The involuntary closure of heart valves

► **Correct Answer:** b) The ability of specialized cardiac cells to spontaneously generate electrical impulses

Rationale: *Automaticity is the property of specialized pacemaker cells (particularly the SA node) to spontaneously drift toward depolarization and fire action potentials without external input — allowing the heart to beat independently.*

CHAPTER 2: THE ELECTRICAL CONDUCTION SYSTEM — FROM SPARK TO HEARTBEAT

Learning Objectives

Upon completing this chapter, you will be able to:

10. Identify each component of the cardiac conduction system and describe its function.
11. Trace the normal sequence of electrical activation from SA node to ventricular myocardium.
12. Explain the physiological basis for the AV node delay and its clinical significance.
13. Describe the intrinsic pacemaker rates of each level of the conduction system.
14. Define escape rhythm, overdrive suppression, and enhanced automaticity.
15. Explain the concept of re-entry and why it is important in arrhythmia formation.
16. Relate each structure in the conduction system to its corresponding ECG waveform.
17. Define pulseless electrical activity and explain why it is dangerous.

2.1 The Conduction System: An Elegant and Redundant Design

The heart's electrical conduction system is a specialized network of cells distributed throughout the heart that generates and transmits electrical impulses in a precise, coordinated sequence. It is an elegant biological design: fast enough to produce 60–100 heartbeats per minute, precise enough to ensure the atria contract a fraction of a second before the ventricles, and redundant enough to keep beating even when critical components fail.

The conduction system is often compared to the electrical wiring of a building with a backup generator. The main power source (SA node) runs everything under normal conditions. If it fails, a secondary power source (AV junction) kicks in. If that fails, an emergency backup (ventricular pacemakers) keeps the minimal lights on. The heart almost never simply stops — it always tries to keep beating.

Components of the Conduction System

Component	Location and Role
Sinoatrial (SA) Node	Right atrium, near SVC junction. Primary pacemaker. Rate: 60–100 bpm.

Internodal Pathways	Three tracts in right atrium wall (anterior, middle, posterior) carrying impulse from SA node to AV node.
Bachmann's Bundle	Interatrial pathway from SA node to left atrium. Ensures simultaneous atrial activation.
Atrioventricular (AV) Node	Lower right atrium, near tricuspid valve. Delays impulse 0.12–0.20 sec. Backup pacemaker at 40–60 bpm.
Bundle of His	Begins at AV node, passes through fibrous skeleton. Splits into bundle branches.
Right Bundle Branch (RBB)	Right side of interventricular septum to right ventricle.
Left Bundle Branch (LBB)	Left side of septum. Splits into left anterior fascicle and left posterior fascicle.
Purkinje Fibers	Subendocardial network in both ventricles. Fastest conduction. Last-resort pacemaker at 20–40 bpm.

2.2 The Sinoatrial (SA) Node: The Primary Pacemaker

The **sinoatrial node** (SY-noh-AY-tree-ul) — also called the sinus node — is a small, crescent-shaped structure embedded in the wall of the right atrium, just below the junction of the superior vena cava. In most adults, it measures approximately 1–2 cm long and a few millimeters wide, making it invisible to the naked eye without magnification. Despite its tiny size, it is the most powerful electrical generator in the heart — the conductor of the cardiac orchestra.

The SA node is supplied by the **SA nodal artery**, which arises from the **right coronary artery** in approximately 60% of people, and from the **left circumflex artery** in the remaining 40%. This is clinically important: an inferior wall MI (right coronary artery occlusion) can compromise the SA node's blood supply, causing sinus bradycardia, sinus arrest, or other sinus node dysfunction — a very common finding in inferior MI.

Autonomic Regulation of the SA Node

The SA node's intrinsic firing rate of approximately 60–100 bpm reflects a balance between two competing influences of the autonomic nervous system (ANS):

- **Sympathetic nervous system ("fight or flight")**: Sympathetic nerve fibers release **norepinephrine** (and circulating epinephrine from the adrenal glands), which accelerates phase 4 depolarization — the SA node fires more rapidly. Heart rate increases. This system activates during exercise, stress, pain, fear, fever, and anxiety. Sympathomimetic drugs (epinephrine, dopamine, albuterol) and caffeine mimic this effect.
- **Parasympathetic nervous system ("rest and digest")**: The **vagus nerve** releases **acetylcholine**, which slows phase 4 depolarization — the SA node fires more slowly. Heart rate decreases. This system dominates during rest, sleep, and meditation. Vagal stimulation (bearing down, vomiting, cold water on the face, carotid massage) can markedly slow the heart rate. Beta-blockers, calcium channel blockers, and digoxin enhance parasympathetic tone or directly slow the SA node.

The resting heart rate reflects parasympathetic predominance in most healthy adults — the vagus nerve is constantly applying a "braking" effect. When we stand up, exercise, or become frightened, sympathetic tone increases and this brake is released, accelerating the heart rate.

SA Node Propagation Pattern

When the SA node fires, the depolarization wave spreads outward through the atrial myocardium in all directions from the SA node — like ripples radiating from a stone dropped in water. This wave:

7. Spreads through the **right atrium** directly from the SA node via the atrial myocardium and internodal pathways.
8. Crosses to the **left atrium** via **Bachmann's bundle** — a specialized conduction pathway that runs through the superior interatrial wall. Because of Bachmann's bundle, the left atrium depolarizes almost simultaneously with the right atrium (with only a ~20 ms delay).
9. Converges on the **AV node** as the single access point from the atria to the ventricles.

The P wave on the ECG represents this atrial depolarization wave. Its normal smooth, dome-like shape reflects the orderly spread of activation from the SA node in the right atrium — creating a smooth, biphasic progression as the wave spreads through both atria. If activation starts from a different point in the atria (an ectopic focus), the P wave shape will be different, which is how we can tell that a beat did not originate from the SA node.

2.3 The AV Node: Gatekeeper, Delay Mechanism, and Backup Pacemaker

The **atrioventricular node** (AV node) is a compact, ovoid structure located in the **floor of the right atrium** near the ostium of the coronary sinus, just above the tricuspid valve ring. It is the only normal electrical connection between the atria and the ventricles. The fibrous skeleton of the heart — the annulus fibrosus — is an electrically inert connective tissue structure that forms a complete barrier between the atria and ventricles everywhere except at the AV node. Without the AV node, an electrical impulse from the SA node simply cannot reach the ventricles through normal pathways.

The AV node serves three critical functions:

Function 1: Filtration (The Gatekeeper)

The AV node filters the number of atrial impulses that reach the ventricles. This is critically important in atrial arrhythmias. In atrial fibrillation, the atria are producing 350–600 chaotic impulses per minute. If all of these reached the ventricles, the ventricular rate would be impossibly fast — 350–600 bpm — which would be incompatible with life. The AV node's refractory properties limit how many impulses can pass through, producing a controlled ventricular rate of 60–150 bpm even during A-Fib.

Function 2: Delay (Optimizing Cardiac Output)

The AV node deliberately slows the conducting impulse by **0.12 to 0.20 seconds** (the AV nodal delay). This is not a malfunction — it is a feature. The delay allows:

- The atria to complete their contraction and fully empty blood into the ventricles before the ventricles contract.
- The ventricles to reach their maximum filling volume (end-diastolic volume) before ejection begins, maximizing stroke volume and cardiac output.

Without this delay, the atria and ventricles would contract simultaneously. The AV valves would close just as the atria were trying to push blood through them — an inefficient and chaotic state that would significantly reduce cardiac output.

On the ECG, the AV nodal delay is represented by the **PR segment** — the flat line between the end of the P wave and the beginning of the QRS complex. Prolongation of the PR interval (> 0.20 sec) indicates a problem with AV node conduction — this is called **first-degree AV block**.

Function 3: Backup Pacemaker

The AV node — or more precisely, the junctional tissue around the AV node and the His bundle — contains pacemaker cells with an intrinsic rate of **40 to 60 bpm**. These cells are normally suppressed by the faster SA node. However, if the SA node fails (sinus arrest) or slows excessively, these junctional pacemaker cells will escape suppression and take over, generating a **junctional escape rhythm** at 40–60 bpm.

The blood supply to the AV node comes from the **AV nodal artery**, which arises from the **right coronary artery** in approximately 90% of people. This is why inferior wall MI (RCA occlusion) is the most common cause of AV blocks — the AV node loses its blood supply, impairs conduction, and can produce first, second, or even third-degree AV block.

2.4 The Bundle of His and Bundle Branches

After passing through the AV node, the impulse enters the **Bundle of His** (pronounced "hiss," named after Wilhelm His Jr., the Swiss cardiologist who first described it in 1893). The Bundle of His is a compact strand of specialized conduction tissue that passes from the AV node through the fibrous skeleton of the heart and travels along the superior border of the interventricular septum.

At the crest of the interventricular septum — just below the membranous septum — the Bundle of His divides into two branches:

Right Bundle Branch (RBB)

The **right bundle branch** is a narrow, discrete bundle that travels along the right side of the interventricular septum toward the apex of the right ventricle, then fans out through the right ventricular myocardium via the Purkinje fibers. It carries the impulse for right ventricular depolarization.

The RBB is long and narrow, which makes it vulnerable to injury (trauma, ischemia, fibrosis). **Right bundle branch block (RBBB)** is a common finding on ECGs and produces a characteristic wide QRS with an "RSR'" pattern ("rabbit ears") in V1 and a broad S wave in Lead I. RBBB can be a normal variant or associated with pulmonary embolism, right heart strain, or structural heart disease.

Left Bundle Branch (LBB)

The **left bundle branch** is shorter and broader than the right, and immediately divides into two fascicles:

- **Left anterior fascicle (LAF)**: Supplies the anterior and superior portions of the left ventricular myocardium. Thin and vulnerable.
- **Left posterior fascicle (LPF)**: Supplies the posterior and inferior portions of the left ventricular myocardium. Broad and relatively protected.

Left bundle branch block (LBBB) produces a characteristic wide, notched QRS that looks very different from RBBB. Importantly, LBBB changes the repolarization pattern so dramatically that **ST analysis for ischemia is unreliable in the presence of LBBB**. A new LBBB in a patient with chest pain is treated as a STEMI equivalent in many hospitals. You should know to flag new LBBB as an urgent finding.

Hemiblocks (Fascicular Blocks)

Blocking of one fascicle of the left bundle branch (anterior or posterior) is called a **hemiblock** (or fascicular block). Hemiblocks alter the direction of ventricular depolarization, causing characteristic axis deviations on the ECG, but do not widen the QRS significantly. They are important to recognize as potential markers of underlying conduction system disease.

2.5 The Purkinje Fiber Network: The Final Highway

The bundle branches terminate in the **Purkinje fiber network** — an extensive subendocardial plexus of highly specialized conduction cells that spreads throughout the inner walls of both ventricles. The Purkinje fibers are the fastest-conducting cells in the heart, capable of propagating impulses at 2 to 4 meters per second — approximately four times faster than ordinary myocardial muscle fibers.

This extraordinary conduction velocity is essential for coordinated ventricular contraction. Without it, different parts of the ventricle would activate at different times, producing a disorganized, inefficient contraction. The Purkinje network ensures that virtually the entire ventricular myocardium depolarizes within a narrow time window (0.06–0.10 seconds) — producing the narrow, sharp QRS complex characteristic of normal ventricular conduction.

The Purkinje fibers activate the ventricles in a specific, optimized sequence:

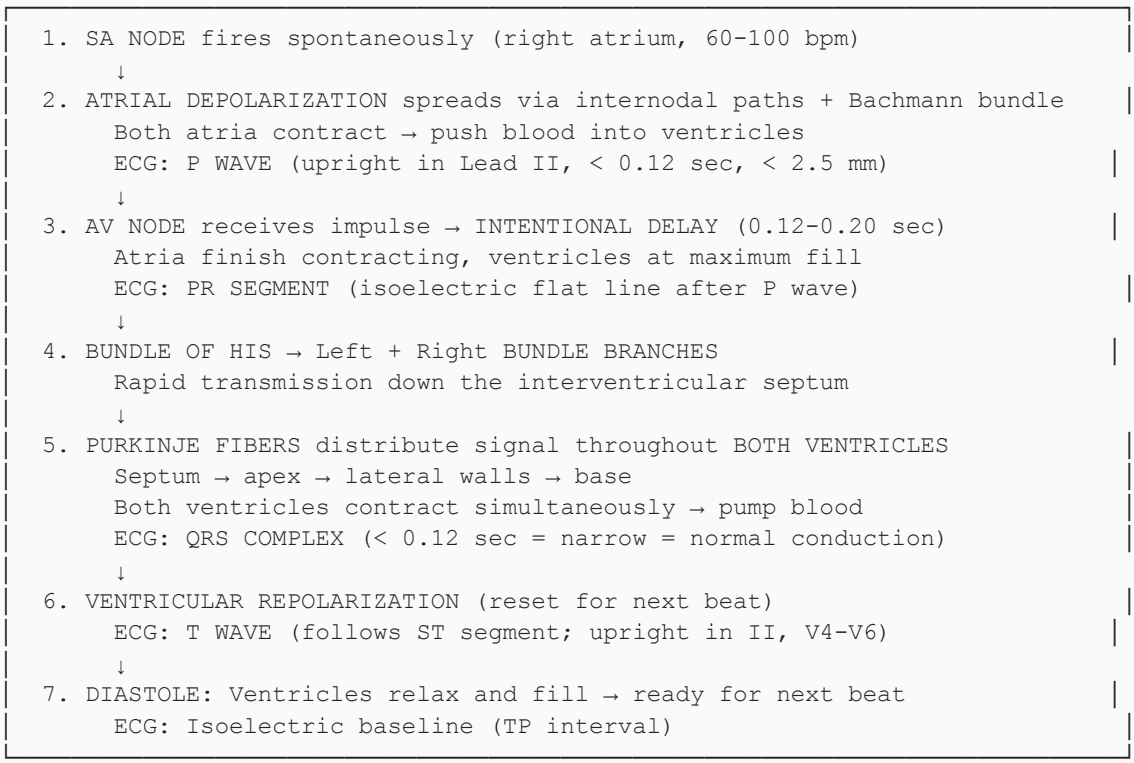
10. The **interventricular septum** depolarizes first, from left to right.
11. The **apex** (bottom tip) of both ventricles activates simultaneously.
12. The activation wave spreads upward toward the **base** (top) of the ventricles.
13. The **posterior base of the left ventricle** is the last to depolarize.

This sequence is optimized to:

- Prevent premature closure of the AV valves (the apex contracts first, pushing blood up toward the outflow tracts).
- Eject blood efficiently upward and out through the aortic and pulmonary valves.
- Minimize energy waste by coordinating the contraction wave with the pressure gradients inside the heart.

2.6 The Complete Normal Conduction Sequence: Summary

Figure 2.1 — Normal Cardiac Electrical Sequence and ECG Correlates



2.7 Intrinsic Pacemaker Rates and the Hierarchy of Automaticity

The heart's conduction system is organized as a hierarchy of backup pacemakers. Each level can take over from the level above it if needed, always at a slower rate. This hierarchy is one of the heart's most important safety features.

Pacemaker Level	Intrinsic Rate
SA Node	60–100 bpm
AV Junction	40–60 bpm
Ventricular / Purkinje	20–40 bpm

Why does the rate decrease at lower levels? Because the slope of phase 4 depolarization (the speed of the spontaneous drift toward the firing threshold) becomes progressively slower at lower levels of the conduction system. The SA node drifts fastest, the AV junction drifts more slowly, and ventricular pacemakers drift most slowly of all.

Overdrive Suppression — Why the SA Node Rules

Under normal conditions, the SA node fires at 60–100 bpm. Its impulses depolarize the AV junction and ventricular pacemakers BEFORE those cells have time to spontaneously reach their own firing threshold. This is called OVERDRIVE SUPPRESSION — the faster pacemaker continuously "resets" the slower pacemakers, preventing them from firing on their own.

When the SA node suddenly stops or slows dramatically, the lower pacemakers don't immediately take over — there is typically a brief pause (called a "sinus pause" or "sinus arrest") while the suppressed pacemaker cells recover from overdrive suppression and begin spontaneously firing again. This pause can cause a brief syncopal (fainting) episode. The longer the pause, the more suppressed the escape pacemaker, and the more severe the symptoms.

2.8 Mechanisms of Arrhythmia Formation

Most arrhythmias arise from one of three underlying mechanisms: **abnormal automaticity**, **triggered activity**, or **re-entry**. Understanding these mechanisms helps you understand why arrhythmias occur and why certain treatments work.

Mechanism 1: Abnormal Automaticity

Normal cardiac cells (non-pacemaker cells) do not spontaneously fire. However, certain conditions can cause them to develop abnormal automaticity — the ability to spontaneously depolarize when they should not. Causes include ischemia (reduced blood flow), electrolyte imbalances (particularly low potassium and low magnesium), drug toxicity (especially digitalis toxicity), and acidosis.

When a non-pacemaker cell develops abnormal automaticity, it becomes an **ectopic focus** (ectopic = out of place) — a pacemaker site outside the normal conduction pathway. Ectopic foci in the atria can cause PACs, atrial tachycardia, or in extreme cases, contribute to atrial fibrillation. Ectopic foci in the ventricles cause PVCs, ventricular tachycardia, or idioventricular rhythms.

Mechanism 2: Triggered Activity

Triggered activity occurs when an abnormal electrical event (called an "afterdepolarization") follows a normal action potential and reaches threshold — triggering another action potential spontaneously, without waiting for the next SA node impulse. Two types exist:

- **Early afterdepolarizations (EADs)**: Occur during the repolarization phase (before the cell fully resets). Associated with prolonged QT interval and responsible for Torsades de Pointes.
- **Delayed afterdepolarizations (DADs)**: Occur after repolarization is complete. Associated with digitalis toxicity and calcium overload states.

Mechanism 3: Re-Entry — The Most Common Mechanism

Re-entry is the most clinically important arrhythmia mechanism and is responsible for most sustained tachyarrhythmias: atrial flutter, SVT, most ventricular tachycardias, and some forms of atrial fibrillation.

Re-entry requires two things: (1) two pathways with different conduction speeds and refractory periods, and (2) a region of unidirectional block. When these conditions are met, an electrical impulse can travel down one pathway, find the other pathway recovered from its refractory period, travel backward up the second pathway, find the first pathway recovered, and cycle in a continuous loop — like a dog chasing its own tail. This circulating loop fires the myocardium repeatedly at whatever rate corresponds to the travel time around the loop.

📋 Clinical Application: Re-Entry and SVT

A 28-year-old woman presents to the ED with sudden-onset palpitations and mild dizziness that started "out of nowhere" 20 minutes ago. Her heart rate is 178 bpm, regular, with a narrow QRS on the monitor. She has had three similar episodes in the past, each resolving spontaneously after several minutes.

This is a classic presentation of SVT (supraventricular tachycardia) caused by AV nodal re-entry (AVNRT). An electrical circuit has established itself within or around the AV node, cycling continuously at ~180 bpm. The narrow QRS confirms that ventricular conduction is occurring via the normal Purkinje system.

Why did it start? Often a PAC (premature atrial contraction) initiates the re-entry circuit by landing at just the right moment — when one pathway is refractory but the other is not.

Your role: Recognize the narrow, regular tachycardia; report it immediately; acquire a 12-lead ECG; prepare the room for possible pharmacological treatment (adenosine) or vagal maneuvers.

2.9 Electrical vs. Mechanical Activity: A Critical Distinction

This principle bears repeating because it has profound clinical implications: **the ECG shows you electrical events, not mechanical events**. Electrical activity normally leads to mechanical contraction — but not always.

Pulseless Electrical Activity (PEA)

PEA is a state of cardiac arrest in which the ECG displays apparently organized electrical activity (which may look like sinus rhythm, a bradycardic rhythm, or almost any organized pattern), but the ventricles are NOT contracting effectively and the patient has NO PULSE.

PEA is CARDIAC ARREST. It requires immediate CPR and treatment of the underlying cause.

Common causes of PEA (remembered by the "H's and T's"):
- Hypovolemia (the most common cause — especially in trauma)
- Hypoxia
- Hydrogen ion excess (acidosis)
- Hypo/Hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade (cardiac)
- Toxins/drugs
- Thrombosis (pulmonary embolism)

Thrombosis (coronary — MI)

This is why you ALWAYS check the patient, not just the monitor. A "normal-looking" rhythm on the screen means nothing if the patient is unresponsive and pulseless.

Chapter 2 Review Questions

1. Why does the AV node delay the electrical impulse?

a) To prevent the heart from beating too fast
b) To allow the atria to finish contracting and maximize ventricular filling before the ventricles contract
c) To filter out abnormal impulses from the SA node
d) To generate its own pacemaker impulses independent of the SA node

► **Correct Answer:** b) To allow the atria to finish contracting and maximize ventricular filling before the ventricles contract

Rationale: *The 0.12–0.20 second AV nodal delay optimizes cardiac output by ensuring the atria fully empty into the ventricles before ventricular systole begins. This delay is represented by the PR interval on the ECG.*

2. The most common cause of AV blocks (first, second, and third degree) is occlusion of which coronary artery?

a) Left anterior descending (LAD)
b) Left main coronary artery (LMCA)
c) Right coronary artery (RCA)
d) Left circumflex (LCx)

► **Correct Answer:** c) Right coronary artery (RCA)

Rationale: *The AV node receives its blood supply from the AV nodal artery, which arises from the RCA in ~90% of people. Inferior MI (RCA occlusion) is the most common cause of AV block.*

3. A Purkinje fiber network that is conducting normally will produce which ECG finding?

a) A wide QRS complex (≥ 0.12 sec)
b) A narrow QRS complex (< 0.12 sec)
c) A long PR interval
d) An absent P wave

► **Correct Answer:** b) A narrow QRS complex (< 0.12 sec)

Rationale: *Normal Purkinje fiber conduction is very rapid, activating both ventricles nearly simultaneously within 0.06–0.10 seconds, producing a narrow QRS. A wide QRS suggests the impulse bypassed the Purkinje system and spread slowly through ordinary myocardial tissue.*

4. Re-entry is responsible for which of the following arrhythmias?

a) Sinus tachycardia
b) Sinus bradycardia
c) Supraventricular tachycardia (SVT) and atrial flutter
d) Sinus arrhythmia

► **Correct Answer:** c) Supraventricular tachycardia (SVT) and atrial flutter
Rationale: *Re-entry — a circulating electrical loop — is the most common mechanism behind SVT, atrial flutter, and many ventricular tachycardias. Sinus rhythms (brady, tachy, arrhythmia) are all normal SA node behaviors.*

5. A patient's ECG monitor shows a rhythm that looks like sinus rhythm at 55 bpm, but the patient is unresponsive and has no palpable pulse. This most likely represents:

a) Normal sinus bradycardia with a sleeping patient
b) Pulseless electrical activity (PEA) — a form of cardiac arrest
c) Junctional escape rhythm requiring observation only
d) An ECG artifact

► **Correct Answer:** b) Pulseless electrical activity (PEA) — a form of cardiac arrest
Rationale: *PEA is organized ECG activity WITHOUT effective mechanical contraction. The patient's unresponsiveness and absent pulse in the presence of an organized ECG rhythm = PEA cardiac arrest. Begin CPR immediately.*

CHAPTER 3: ELECTRODE PLACEMENT — THE FOUNDATION OF EVERY ACCURATE ECG

Learning Objectives

Upon completing this chapter, you will be able to:

18. Define the difference between an electrode and a lead, and explain why this distinction matters clinically.
19. Locate and palpate the sternal angle (Angle of Louis) and use it to count intercostal spaces accurately.
20. Describe the precise anatomical location of each of the 10 electrodes — including the tactile landmarks used to find each position.
21. Apply the complete 12-lead ECG electrode set correctly, in the correct sequence, with proper skin preparation.
22. Identify the anatomical basis for each lead grouping: inferior, anterior, lateral, septal.
23. Describe the implications of even 1–2 intercostal space errors in precordial placement.
24. Apply posterior leads (V7–V9) and right-sided leads (V3R–V6R) when clinically indicated.
25. Recognize the 7 most common electrode placement errors and describe how each affects the ECG.
26. Modify electrode placement appropriately for patients with amputations, mastectomy, obesity, or large breasts.
27. Perform complete, clinically acceptable skin preparation at every electrode site.

3.1 Why Electrode Placement Is the Most Critical Technical Skill in ECG Acquisition

Of all the technical skills involved in acquiring a 12-lead ECG, none is more important — or more frequently performed incorrectly — than electrode placement. Studies have documented that electrode misplacement rates in clinical practice range from 30% to over 50%, depending on the clinical setting. These errors are not trivial: a misplaced precordial electrode by a single intercostal space can produce false ST elevation that mimics a STEMI, false ST depression suggesting ischemia that does not exist, an apparent right bundle branch block in a patient with a normal conduction system, or loss of R wave progression suggesting anterior wall infarction in a patient with a healthy heart.

The consequences of these errors are real and potentially life-altering: unnecessary cardiac catheterization with its associated risks, administration of thrombolytics that carry a 1–2% risk of intracranial hemorrhage, delayed diagnosis of a true STEMI while the team investigates a false

one, or false reassurance in a patient who is actually infarcting. Patient lives depend on technically accurate ECG acquisition.

This chapter provides the most detailed and clinically precise electrode placement guidance available in any educational resource at this level. Study it carefully. Practice it on every patient. Never rush the placement step. A 3-minute investment in correct placement is worth infinitely more than a 1-minute placement that produces an uninterpretable or misleading tracing.

The Foundational Principle of ECG Electrode Placement

Every electrode position in the 12-lead ECG is defined by a specific anatomical landmark that can be FELT on the patient — not estimated, not guessed, not assumed. You must use your hands to PALPATE (feel) the correct location.

Landmarks cannot be assumed from external appearances. Patient body habitus, breast tissue, adipose tissue, and musculature all change how the chest looks without necessarily changing where the underlying ribs and intercostal spaces are. The sternal angle feels the same in a thin 25-year-old athlete and a 320-pound patient — you must palpate it in both.

Visual estimation = errors. Tactile landmark identification = accuracy.

3.2 The Electrode vs. The Lead: Definitions That Matter

Before placing a single electrode, you must understand the difference between an electrode and a lead — two terms that are often (incorrectly) used interchangeably in clinical settings:

Term	Definition
Electrode	The physical adhesive pad attached to the patient's skin. It detects the electrical voltage at the skin surface and converts it into an electronic signal. You can touch and hold an electrode.
Lead	A mathematical calculation comparing the voltage between specific electrode combinations. It creates a "view" of the heart from a specific angle. You cannot physically touch a lead — it exists only as a calculated electrical measurement.

The remarkable efficiency of this system: by strategically placing 10 electrodes on the body and combining their signals in 12 different mathematical ways, we obtain 12 distinct views of the

heart's electrical activity — a comprehensive, three-dimensional electrical portrait obtained non-invasively in approximately 10 seconds.

3.3 The Four Limb Electrodes: Locations, Rationale, and Palpation Technique

The four limb electrodes are placed on the extremities and form the foundation of the frontal plane (limb) leads: Leads I, II, III, aVR, aVL, and aVF. Their placement is conceptually simple but must be consistent to maintain the standardized electrical relationships that define each lead.

Color Coding — Know Your System

Two color-coding systems exist worldwide. Before placing ANY electrodes, confirm which system your machine uses — the label on each lead wire tells you. Applying the wrong wire to an electrode creates a lead reversal artifact that may not be immediately obvious but produces systematically abnormal findings in multiple leads.

Electrode	AHA Standard (United States)
Right Arm	White
Left Arm	Black
Right Leg (Ground)	Green
Left Leg	Red

Memory Aid: AHA Color System

"White is Right" — The RA (Right Arm) electrode is WHITE in the AHA system.

"Smoke (white) rises above Fire (red)" — White/RA is on the right arm (upper right), Red/LL is on the left leg (lower left). The right arm electrode is like white smoke at the top; the left leg electrode is like a red flame at the bottom.

"Christmas colors on the arms" — Green (RL) and Red (LL) on the legs; White and Black on the arms.

IEC alternative: "RYGB" — Red, Yellow, Green, Black going clockwise from RA → LA → LL → RL.

RA — Right Arm Electrode

Standard Location: The medial (inner) surface of the right forearm, approximately 5 to 10 centimeters (2 to 4 inches) proximal to (above) the right wrist.

How to find it: With the patient's right arm relaxed at their side, palm facing upward, look at the inner (medial) surface of the forearm — the side facing the patient's body when the arm is at rest. Identify the crease at the wrist (the wrist flexion crease). Measure approximately two to four finger-widths above this crease. The electrode should lie on the flat, fleshy inner forearm surface, NOT over any bony prominence (wrist bones — the styloid processes), NOT over the cubital fossa (the bend in the elbow where IV lines are placed), and NOT directly over any visible veins (to avoid interfering with IV sites).

What you should feel: A relatively flat, fleshy, non-bony surface. If you feel hard bone beneath your fingertip, move slightly up or down until you are over the muscular belly of the forearm.

For continuous monitoring (alternative placement): Place the RA electrode on the right anterior chest wall, just below the right clavicle, in the infraclavicular fossa (the slight hollow below the clavicle, medial third). This "Mason-Likar" position reduces motion artifact during transport but is NOT used for diagnostic 12-lead acquisition — it changes the electrical axis.

LA — Left Arm Electrode

Standard Location: The medial (inner) surface of the left forearm, approximately 5 to 10 centimeters proximal to the left wrist — the precise mirror image of the RA electrode on the opposite arm.

How to find it: Identical technique to the RA, performed on the left arm. With the patient's left arm relaxed, palm facing upward, locate the wrist crease and measure two to four finger-widths above it on the inner forearm surface. Avoid bony prominences, IV sites, and arterial lines.

Critical note: In patients with an intravenous line or arterial line in either forearm, place the corresponding limb electrode ABOVE the IV site (between the IV insertion point and the body),

not below it. Never place an electrode directly over a functioning IV — this can cause signal interference and potential complications.

RL — Right Leg Electrode (The Ground)

Standard Location: The medial (inner) surface of the right lower leg, approximately 5 to 10 centimeters proximal to (above) the right medial malleolus (the bony bump on the inner side of the right ankle).

How to find it: Locate the medial malleolus — the prominent rounded bony protrusion on the inner (medial) side of the right ankle. Measure approximately two to four finger-widths above this bony landmark, and place the electrode on the flat, fleshy inner shin (medial tibia area). The electrode should be over muscle/subcutaneous tissue, not directly over the tibia (shinbone) itself.

What this electrode does: The RL electrode serves ONLY as an electrical ground (reference point). It does not contribute to any of the 12 cardiac leads — it is never the "positive" or "negative" pole of any lead. Its sole function is to reduce common-mode electrical interference and stabilize the signal baseline. A misplaced or poorly applied RL electrode does not change the cardiac lead polarity, but it will degrade signal quality and increase baseline noise. An absent or disconnected RL electrode typically causes all leads to show dramatically increased baseline artifact or even a flat line.

LL — Left Leg Electrode

Standard Location: The medial (inner) surface of the left lower leg, approximately 5 to 10 centimeters proximal to the left medial malleolus — the precise mirror image of the RL electrode on the opposite leg.

How to find it: Identical technique to RL, on the left leg. Locate the left medial malleolus (inner ankle bone), measure two to four finger-widths above it, and place on the flat inner shin.

Why LL placement matters more than RL: The LL electrode is used in multiple leads: Lead II (RA negative, LL positive), Lead III (LA negative, LL positive), and aVF (LL positive vs. average of RA and LA). An incorrectly placed LL electrode will affect the appearance of ALL inferior leads (II, III, aVF). This is clinically significant because inferior leads are used to diagnose

inferior STEMI, right ventricular infarction, inferior wall ischemia, and inferior Q waves of prior MI.

Placement When Limbs Are Absent or Inaccessible

AMPUTATION: Place the electrode as close to the standard position as possible on the remaining stump. If the patient has a bilateral lower extremity amputation, both leg electrodes may be placed on the lower abdomen or trunk. Document any modified placement clearly on the ECG. The ECG will remain clinically interpretable in most cases.

BILATERAL UPPER LIMB AMPUTATION: Place arm electrodes on the anterior shoulders (infraclavicular area, same as Mason-Likar monitoring position).

CAST OR SPLINT: Place the electrode as close to the standard position as accessible at the edge of the cast. Do NOT remove a cast to place an ECG electrode.

LOWER EXTREMITY EDEMA (severe): Electrodes may not adhere well to edematous skin. Use self-adhesive electrodes designed for difficult skin, and press firmly. In severe cases, wrapping the electrode against the skin with a light gauze wrap for 30 seconds while it adheres can help.

PERIPHERAL ARTERIAL LINE: NEVER place an electrode directly over a femoral arterial line or arterial access site. Place the LL electrode on the lower abdomen ipsilaterally if necessary.

ALWAYS document any nonstandard placement on the ECG tracing. Write the deviation directly on the paper.

3.4 The Six Precordial Electrodes: A Masterclass in Landmark Identification

The precordial (chest) leads are where the most clinically significant electrode placement errors occur, and where the consequences of those errors are most severe. Every precordial electrode location is defined by a combination of intercostal space and an anatomical line. You must be able to reliably and consistently palpate every landmark every time.

The Master Landmark: The Sternal Angle (Angle of Louis)

Every precordial electrode placement begins with one landmark: the **sternal angle** (also called the **manubriosternal junction** or the **Angle of Louis**, named after French physician Antoine Louis). This is the most important anatomical landmark you will use in ECG acquisition, and it is the starting point for counting intercostal spaces to place V1 through V6.

What it is: The sternal angle is the palpable transverse ridge or step-off where the manubrium (upper portion of the sternum) meets the body of the sternum (the longer middle portion). The junction between these two structures creates a small but distinct horizontal ridge that you can feel with your fingertip.

Clinical significance beyond ECG: The 2nd costal cartilage (and therefore the 2nd rib) attaches to the sternum AT the level of the sternal angle. This makes the sternal angle the universal reference point for counting ribs and intercostal spaces on the anterior chest wall.

✓ Step-by-Step: Finding the Sternal Angle by Palpation

1. STEP 1: Position your patient supine (or semi-reclined no more than 30°) with the chest exposed. Stand or sit at the patient's right side.
2.
3. STEP 2: Locate the JUGULAR NOTCH (also called the suprasternal notch) — the visible V-shaped depression at the very top of the sternum, between the two collarbones (clavicles). This is where the clavicles and the manubrium meet. You can see and feel this notch without any pressure.
4.
5. STEP 3: Place your index finger firmly in the jugular notch, so your fingertip is resting in the V-shaped depression.
6.
7. STEP 4: Slowly slide your finger DOWNWARD (toward the patient's feet) along the midline of the sternum, maintaining firm but gentle contact.
8.
9. STEP 5: As you slide approximately 4–5 centimeters below the jugular notch, you will feel a distinct horizontal RIDGE or STEP — a slight bump or ledge that runs across the sternum. Your fingertip will drop slightly as you pass over this ridge. This is the STERNAL ANGLE.
10.
11. STEP 6: CONFIRM: The sternal angle is approximately at the level of the 4th–5th thoracic vertebra posteriorly, and is typically 4–5 cm below the jugular notch. In most adults, you can cover the distance from the jugular notch to the sternal angle with two to three fingers placed side by side.
12.
13. STEP 7: If you cannot feel a clear ridge: In obese patients or patients with significant pectoral muscle development, the ridge may be subtle. Apply firmer pressure and move your fingertip very slowly. If still unclear, press more firmly with the tip of your fingernail (not the pad). The sternal angle is present in virtually all patients — it requires patience to find in those with significant soft tissue overlying it.

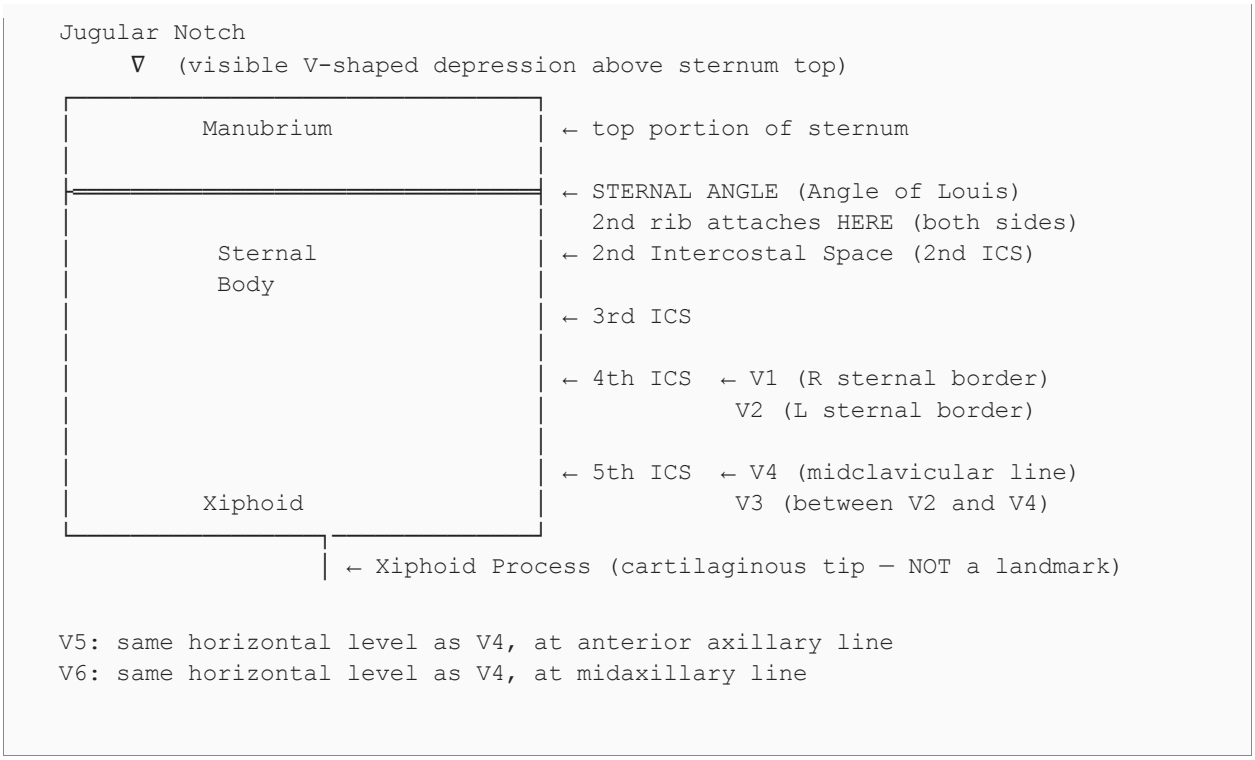
Counting Intercostal Spaces from the Sternal Angle

Once you have confirmed the sternal angle, you can count down to any intercostal space. The technique is precise and must be practiced until it becomes automatic.

✓ Step-by-Step: Counting Intercostal Spaces

1. STEP 1: With your fingertip at the sternal angle, move your finger LATERALLY (sideways) to the RIGHT, staying at the same level. As you move off the sternum onto the right side of the chest, you will feel a slight bump — the 2ND RIB (specifically the 2nd costal cartilage, which attaches to the sternum at the sternal angle).
2.
3. STEP 2: Your finger is now ON the 2nd rib. Feel the rounded firmness of the rib underneath your finger. This is your reference point.
4.
5. STEP 3: Slide your finger DOWNWARD (toward the feet) past the 2nd rib. You will feel the rib slope away and your finger will drop into a soft depression. This depression is the 2ND INTERCOSTAL SPACE (ICS) — the space between the 2nd and 3rd ribs.
6.
7. STEP 4: Continue sliding down. You will feel the next rib — the 3RD RIB — as a firm ridge. Keep going.
8.
9. STEP 5: Slide past the 3rd rib into the next soft depression. This is the 3RD INTERCOSTAL SPACE.
10.
11. STEP 6: Continue down past the 3rd ICS. Feel the 4TH RIB beneath your finger.
12.
13. STEP 7: Slide past the 4th rib. You are now in the 4TH INTERCOSTAL SPACE — the correct level for V1 and V2.
14.
15. STEP 8: For V4: continue down one more level past the 4th ICS, over the 5th rib, into the 5TH INTERCOSTAL SPACE.
16.
17. IMPORTANT: Count SPACES, not ribs. The intercostal space is the soft, resilient area between two ribs. Ribs feel firm and slightly rounded. Intercostal spaces feel soft (you are feeling the intercostal muscles between ribs). If you press on what feels like a firm ridge, you are ON a rib. If you feel into a resilient, slightly soft area, you are IN an intercostal space.
18.
19. COMMON ERROR: Beginning the count from the 1st rib (which is not palpable anteriorly because it is hidden under the clavicle) instead of the 2nd rib. Always begin from the sternal angle → 2nd rib → count down from there.

Figure 3.1 — Chest Anatomy: Landmarks for Precordial Electrode Placement



V1 — 4th Intercostal Space, Right Sternal Border

Location: In the 4th intercostal space (between the 4th and 5th ribs), immediately to the RIGHT of the sternum — right up against the right sternal border.

Step-by-Step Placement:

14. Begin at the sternal angle. Palpate laterally to the right to find the 2nd rib.
15. Count down: 2nd rib → 2nd ICS → 3rd rib → 3rd ICS → 4th rib → 4th ICS. You are now in the 4th intercostal space.
16. Keep your finger at this level and move BACK toward the sternum until your finger is just to the RIGHT of the right sternal edge.
17. Confirm: Your finger should be in a soft space (ICS, not on a rib), immediately adjacent to (touching) the right side of the sternum.
18. Apply the electrode here. The medial edge of the electrode should be touching the right side of the sternum.

What V1 sees: V1 is the most "rightward" precordial electrode. It sits directly over the right side of the interventricular septum and provides the clearest view of:

- Right bundle branch block (RSR' "rabbit ears" in V1)

- Left bundle branch block (wide, deep QS or rS complex)
- Right ventricular hypertrophy (dominant R in V1)
- Posterior wall MI (tall, broad R in V1 — mirror image of posterior infarction)
- Septal ischemia

The most common V1 error: Placing V1 too HIGH — in the 3rd ICS instead of the 4th. This is especially common in female patients, where the presence of breast tissue can draw the clinician's eye upward, or in patients with large pectoral muscles. V1 placed in the 3rd ICS produces:

- A false RSR' pattern mimicking right bundle branch block
- Abnormal R wave progression
- Possible false-positive posterior MI pattern

V2 — 4th Intercostal Space, Left Sternal Border

Location: In the 4th intercostal space, immediately to the LEFT of the sternum — left sternal border. V2 is the direct mirror image of V1 on the opposite side of the sternum.

Step-by-Step Placement:

19. Use the same level you found for V1 (4th ICS). Simply move to the opposite side of the sternum.
20. Place your finger immediately to the LEFT of the left sternal border, at the same intercostal level as V1.
21. Alternatively: from the sternal angle, palpate laterally to the LEFT to find the 2nd rib on the left side, count down to the 4th ICS, then move back to the left sternal border.
22. The medial edge of the V2 electrode should touch the left side of the sternum.

V1 and V2 together form the "septal leads." They should be placed at the SAME intercostal level on opposite sides of the sternum. If V1 and V2 are at different heights, the relative sizes of their waveforms will be distorted. Confirm that both electrodes are at exactly the same horizontal level by placing your index fingers at both electrode sites simultaneously and verifying they form a horizontal line.

A Common Clinical Shortcut — and Why It Fails

Many clinicians use the nipple as a landmark for V4 (in male patients) or use visual estimation for all precordial leads. This is a significant source of error.

The nipple is NOT a reliable landmark for V4 because:
- In males, nipple position varies by age, body habitus, and skin elasticity — often lying below the 5th ICS or in various lateral positions.
- In females, the nipple is clearly not at the V4 position under any circumstances.
- Studies comparing nipple-based placement to landmark-based placement show significant discordance in the majority of patients.

ALWAYS use the sternal angle → intercostal counting method. Never use the nipple as an ECG landmark.

V4 — 5th Intercostal Space, Midclavicular Line

Location: In the 5th intercostal space at the midclavicular line (MCL). V4 is placed before V3 because V3 is interpolated between V2 and V4.

Finding the Midclavicular Line (MCL):

23. Locate the RIGHT clavicle (in this case we are working on the LEFT chest, so find the LEFT clavicle).
24. Place your finger at the MEDIAL end of the left clavicle — where it meets the sternum (the sternoclavicular joint). You can feel this junction as a firm bump where the clavicle begins.
25. Now locate the LATERAL end of the left clavicle — where it meets the shoulder (the acromioclavicular joint). Find the bony prominence of the shoulder tip (the acromion).
26. Find the MIDPOINT between these two landmarks — halfway along the clavicle from medial end to lateral end.
27. From this midpoint, drop an imaginary VERTICAL LINE straight down toward the feet. This vertical line is the midclavicular line (MCL).

Finding the 5th Intercostal Space:

28. From your V2 position (4th ICS), continue counting down: past the 4th ICS → feel the 5th rib → drop into the 5th ICS.
29. Alternatively, from V1/V2 at the 4th ICS, counting down one more space lands you in the 5th ICS.

Placing V4:

30. Hold the intersection of the 5th ICS (horizontal) and the midclavicular line (vertical).
31. Apply the electrode at this intersection point.
32. Confirm: The electrode should be in a soft intercostal space (not on a rib), and the vertical position should be at the midpoint of the clavicle above it.

What V4 sees: The cardiac apex — the lowest, most anterior point of the left ventricle. V4 provides the clearest view of apical ischemia. Hyperacute (peaked, tall) T waves in V4 are often the first ECG sign of proximal LAD occlusion with apical involvement.

The most common V4 error: Placing V4 too far LATERAL (in the anterior axillary line instead of the midclavicular line). This effectively shifts V4 into V5 territory, creating false R wave progression patterns and potentially missing anterior ischemia.

V3 — Between V2 and V4 (Interpolated Position)

Location: Midway between V2 (left sternal border, 4th ICS) and V4 (5th ICS, MCL). V3 is always placed AFTER V4 is already in position, because its position is defined by the positions of its neighbors.

Step-by-Step Placement:

33. Confirm V2 is correctly placed (left sternal border, 4th ICS).
34. Confirm V4 is correctly placed (5th ICS, MCL).
35. Visually and tactilely identify the midpoint between the V2 electrode and the V4 electrode.
36. Apply V3 at this midpoint.

Critical concept: V3 does NOT have an independent intercostal space or anatomical line definition. Its position is entirely relative to V2 and V4. In some patients (particularly tall patients with a long sternum, or short patients with a compact thorax), V3 may fall in the 4th ICS, the 5th ICS, or somewhere in between. This is normal and expected — do NOT move V3 to a specific intercostal space independent of its neighbors.

What V3 sees: The anterior wall of the left ventricle, in its transitional region between the septum and the apex. V3 is one of the "anterior leads" and is critical for identifying:

- Anterior wall ischemia/STEMI (ST elevation in V3 along with V2, V4)

- Anterior wall Q waves (loss of anterior R wave progression)
- The transition zone (where R and S waves become roughly equal in amplitude)

V5 — Same Horizontal Level as V4, Anterior Axillary Line

Location: At the anterior axillary line (AAL), at the SAME HORIZONTAL LEVEL as V4. This is one of the most frequently misunderstood placements.

Finding the Anterior Axillary Line (AAL):

37. Ask the patient to lower their LEFT arm to their side (relaxed, not raised).
38. Look at the left axilla (armpit) from the front. There is a distinct fold of skin and tissue where the anterior chest wall (pectoral region) meets the upper arm — this is the ANTERIOR AXILLARY FOLD.
39. Feel for this fold by gently lifting the arm slightly and then lowering it — you can feel the anterior muscular fold (the anterior border of the axilla) with your fingers.
40. Drop a vertical imaginary line straight down from the anterior axillary fold. This is the ANTERIOR AXILLARY LINE (AAL).

Placing V5:

41. Maintain the SAME HORIZONTAL LEVEL as V4 — do NOT count down another intercostal space.
42. At the level of V4, move LATERALLY until you reach the anterior axillary line.
43. Apply the electrode at this intersection: same horizontal level as V4, at the AAL.

The Most Critical Concept in Precordial Placement: V4, V5, and V6 Are Horizontal

V4, V5, and V6 must form a perfectly HORIZONTAL line across the chest — they are at the SAME vertical height. Many practitioners incorrectly place V5 and V6 by counting down another intercostal space below V4, which puts them at the 6th ICS — one space too low.

The error of placing V5/V6 too low produces:
- Falsely reduced R wave amplitude in V5/V6
- Apparent poor R wave progression mimicking anterior infarction
- Altered ST segment appearance in lateral leads

To VERIFY horizontal alignment: After placing V4, V5, and V6, place one finger at each electrode site simultaneously (or use a piece of tape as a horizontal reference line). All three should be at exactly the same horizontal level. If V5 or V6 is lower than V4, remove and reposition them.

V6 — Same Horizontal Level as V4, Midaxillary Line

Location: At the midaxillary line (MAL), at the SAME HORIZONTAL LEVEL as V4 and V5.

Finding the Midaxillary Line (MAL):

44. Locate the center (apex) of the axilla — the deepest point of the armpit when the arm is at the side.
45. Drop a vertical imaginary line straight down from the apex of the axilla. This is the MIDAXILLARY LINE.
46. Alternatively: the midaxillary line is midway between the anterior axillary line (V5) and the posterior axillary line (the posterior fold of the axilla, closer to the back).

Placing V6:

47. Maintain the EXACT SAME HORIZONTAL LEVEL as V4 and V5 — critical.
48. At this level, move further lateral until you reach the midaxillary line.
49. Apply the electrode. It will typically be on the lateral chest wall, partially on the side of the thorax.

Practical tip for V6: In many patients, V6 ends up approximately over the midlateral chest wall. When the patient's arm is at rest at their side, V6 is often partially under or beside the arm. Ask the patient to raise their arm slightly during electrode application, then lower it after the electrode is adhered. Confirm V6 is still in the correct position after the arm is lowered.

What V5 and V6 see: The lateral wall of the left ventricle. These leads, along with the limb leads I and aVL, complete the "lateral wall" view group. Left circumflex (LCx) artery occlusion produces ST elevation in the lateral leads (I, aVL, V5, V6). High lateral infarction (I, aVL only) may indicate a first diagonal or obtuse marginal branch occlusion.

3.5 The 12 Leads and Their Anatomical Views of the Heart

Once all 10 electrodes are correctly placed, the ECG machine calculates 12 leads — 12 different electrical views of the heart. Understanding what each lead "sees" is fundamental to interpreting the ECG and understanding why changes in specific leads indicate specific problems.

The Frontal Plane (Limb) Leads

The six limb leads record electrical activity in the frontal plane — the plane that divides the body into front and back, like looking at a face-on view of the body's torso. They view the heart as if standing in front of the patient.

Lead	Positive Electrode
Lead I	LA (Left Arm)
Lead II	LL (Left Leg)
Lead III	LL (Left Leg)
aVR	RA (Right Arm)
aVL	LA (Left Arm)
aVF	LL (Left Leg)

The Horizontal Plane (Precordial) Leads

Lead	Position
V1	4th ICS, R sternal border
V2	4th ICS, L sternal border
V3	Between V2 and V4
V4	5th ICS, midclavicular line
V5	Same level as V4, anterior axillary line
V6	Same level as V4, midaxillary line

Contiguous Lead Groups — The Foundation of STEMI Localization

Leads are grouped by the region of the heart they view. **Contiguous leads** are leads that view the same or anatomically adjacent regions of the heart. ECG changes (ST elevation, ST

depression, T wave inversion, Q waves) that appear in two or more contiguous leads are clinically significant and indicate involvement of the corresponding myocardial territory.

Myocardial Territory	Contiguous Leads
Inferior wall	II, III, aVF
Lateral wall	I, aVL, V5, V6
High lateral wall	I, aVL
Anterior wall	V1, V2, V3, V4
Anterolateral (large territory)	V1–V6 + I, aVL
Posterior wall	V7, V8, V9 (posterior leads)
Right ventricle	V3R, V4R (right-sided leads)

3.6 The Complete 12-Lead ECG Application Procedure

What follows is the complete, clinically validated procedure for applying a 12-lead ECG. This procedure integrates all of the anatomical knowledge above into a practical, step-by-step protocol. Practice this sequence until it becomes automatic — consistency in procedure produces consistency in results.

✓ Pre-Procedure Setup

1. [ ] Verify ECG machine is functioning: powered on, paper loaded and advancing, patient data fields ready.
2. [ ] Inspect all 10 lead wires: check for intact insulation, no visible cracking or fraying, secure connector ends (the most common break point). Replace any damaged wire before proceeding.
3. [ ] Confirm 10 fresh electrode pads are available: check expiration date on packaging (expired electrodes lose conductivity and adhesion). Do not use electrodes from an open/unsealed package that may have dried out.
4. [ ] Identify the color-coding system on the lead wires (AHA or IEC) and confirm you know which electrode goes where.
5. [ ] Ensure the room/environment allows the patient to lie flat safely.

✓ Patient Preparation

1. [ ] Introduce yourself to the patient. Explain the procedure in plain language: "I'm going to place some small stickers on your chest, arms, and legs to record your heart's electrical activity. It takes about 2 minutes and is completely painless."
2. [ ] Obtain verbal consent. If the patient is incapacitated, proceed under implied consent in an emergency.
3. [ ] Position the patient SUPINE — lying flat on their back. The bed or cot should be fully flat, or inclined no more than 15–20 degrees (to accommodate respiratory distress). Greater inclination shifts the cardiac electrical axis and alters the appearance of multiple leads.
4. [ ] Ask the patient to rest both arms flat at their sides — NOT crossed over the chest, NOT holding the sides of the stretcher (muscle tension from gripping creates artifact).
5. [ ] Expose the chest completely. Lower the gown to expose the entire anterior chest. Expose both inner forearms and both inner lower legs.
6. [ ] Maintain patient dignity: use a drape or sheet over areas not immediately being worked on.
7. [ ] Explain privacy measures to the patient and ensure the room door/curtain is closed.

✓ Skin Preparation — The Most Critical and Most Neglected Step

1. Spend adequate time on skin preparation. Poor skin preparation is the single most correctable cause of poor ECG quality.
2.
3. [ ] INSPECT each planned electrode site before touching it:
4. • Excessive hair → CLIP (not shave). One to two passes of a single-use razor or clipper is sufficient.
5. Do NOT dry-shave — creates skin microabrasions that cause stinging and increased impedance.
6. • Oily or lotioned skin → clean with alcohol prep wipe. Allow to DRY COMPLETELY (10–15 seconds).
7. Wet alcohol prevents electrode adhesion. Wait until the skin is visually and tactilely dry.
8. • Diaphoretic (sweaty) skin → dry with a towel using firm patting (not rubbing). Apply electrode quickly.
9. Consider using "diaphoretic skin" electrodes (foam-backed, extra-adhesive) for profusely sweaty patients.
10. • Skin cream, lotion, sunscreen → remove entirely with alcohol wipe. Cream under an electrode = poor signal.
11. • Open wounds → do NOT place electrodes on broken skin. Place nearby or modify placement. Document.
12.
13. [ ] ABRADE the skin at each electrode site using an abrasive electrode preparation pad, rough gauze, or the
14. rough texture on the back of some electrode packaging. Use firm circular motions 5–10 times.

WHY: The outermost skin layer (stratum corneum) is composed of dead, keratinized cells that are poor

electrical conductors. This dead skin layer creates electrical resistance (impedance) between the electrode

and the living conductive tissues beneath. Abrading removes this barrier and dramatically reduces

impedance. Studies show that skin abrasion alone reduces ECG artifact by 50–80%.

You will see slight redness at the site after proper abrasion — this is normal and expected.

[ ] Apply electrode IMMEDIATELY after abrasion before the skin re-humidifies or accumulates new oil.

✓ Electrode Application Sequence — Recommended Order

Apply in this sequence to minimize patient repositioning and ensure systematic coverage:

[ ] Apply RA (White): right inner forearm, 5–10 cm above wrist. Press firmly, smoothing all edges.

[ ] Apply LA (Black): left inner forearm, 5–10 cm above wrist. Mirror of RA.

[ ] Apply RL (Green): right inner lower leg, 5–10 cm above medial malleolus.

[ ] Apply LL (Red): left inner lower leg, 5–10 cm above medial malleolus.

[ ] Now move to the chest. Palpate the jugular notch → slide down to sternal angle → confirm ridge.

[ ] Palpate RIGHT → find 2nd rib → count down → 4th ICS → back to right sternal border.

[ ] Apply V1: right sternal border, 4th ICS.

[ ] Apply V2: left sternal border, 4th ICS (same level as V1, confirmed by horizontal palpation).

[ ] Palpate LEFT clavicle → find medial and lateral ends → identify midpoint → drop MCL line.

[ ] Count down from sternal angle → 5th ICS → intersection with MCL.

[ ] Apply V4: 5th ICS, midclavicular line.

[ ] Apply V3: midway between V2 and V4 (interpolated — place AFTER V4 is confirmed).

[ ] Identify anterior axillary fold (anterior border of axilla) → drop AAL line.

[ ] Apply V5: SAME horizontal level as V4, at the anterior axillary line.

[ ] Identify midaxillary line (center of armpit → vertical line downward).

[ ] Apply V6: SAME horizontal level as V4 and V5, at the midaxillary line.

[ ] Confirm horizontal alignment: V4, V5, and V6 should form a straight horizontal line.

[ ] Connect all lead wires to matching electrodes. Verify each connection is secure.

✓ ECG Acquisition

1. [ ] Enter patient information: full name, date of birth, date and time, indication for ECG.
2. [ ] Review the "quick-look" screen: confirm all 12 leads are showing signal (no flat lines or missing leads).
3. If any lead shows a flat line: check connection, check electrode adhesion, check wire integrity.
4. [ ] Instruct the patient: "Please lie very still, relax all your muscles — arms, legs, and chest.
5. Breathe normally. Do not talk, cough, or move until I tell you we are done. Ready?"
6. [ ] Wait 3–5 seconds after giving instructions to allow the patient to settle and for any movement artifact to resolve.
7. [ ] Press ACQUIRE/RECORD. The machine will record for approximately 10 seconds.
8. [ ] IMMEDIATELY review the printed or displayed tracing for:
9. • Baseline stability (no wandering baseline)
10. • 60-Hz interference (no thick/fuzzy baseline)
11. • Muscle artifact (no irregular high-frequency noise)
12. • All 12 leads visible with waveforms
13. • Calibration mark (1 mV = 10 mm)
14. • Suspicious findings that require immediate reporting
15. [ ] If tracing quality is unacceptable: identify and correct the specific problem, then re-acquire.
16. [ ] If tracing is acceptable: print/transmit to physician; report any immediate findings.

3.7 Recognizing and Correcting the 7 Most Common ECG Acquisition Errors

Error 1: Precordial Leads Too High (Most Common Error Overall)

Frequency: Studies suggest this occurs in 30–50% of ECGs in clinical practice. It is the single most common technical error in ECG acquisition.

What happens: V1 and V2 are placed in the 3rd ICS instead of the 4th ICS. V3 and V4 shift upward accordingly. The entire precordial electrode set is positioned one intercostal space too high.

Why it happens: Clinicians who do not count intercostal spaces from the sternal angle, and instead place V1 and V2 based on visual estimation or tactile "feel" without systematic counting.

ECG consequences: Apparent rSR' pattern in V1 mimicking right bundle branch block. Poor R wave progression mimicking anterior infarction. False ST changes in anterior leads. A 2018

study found that V1/V2 placement 1 ICS too high caused false RBBB patterns in 30% of subjects with otherwise normal ECGs.

Prevention and correction: Always begin with the sternal angle. Always count ribs and intercostal spaces systematically. Never use visual estimation.

Error 2: V5 and V6 Placed Too Low

What happens: V5 and V6 are placed at the 6th ICS instead of being maintained at the same horizontal level as V4.

Why it happens: Practitioners mistakenly believe V5 and V6 are at the next intercostal space below V4 (a logical but incorrect assumption).

ECG consequences: Reduced R wave amplitude in V5/V6, poor R wave progression, altered ST segment appearance in lateral leads, potentially masking lateral wall ischemia.

Prevention: Explicitly mark or note the horizontal level of V4 before placing V5 and V6. Use a straight edge (ruler, piece of tape, or the edge of the electrode package) to draw an imaginary horizontal line from V4 laterally. Place V5 and V6 along this line.

Error 3: RA/LA Limb Lead Reversal

What happens: The RA (White) and LA (Black) lead wires are swapped — RA wire connected to the LA electrode, and vice versa.

Why it happens: Rushing, inattention, or failure to read the wire labels before connecting.

ECG consequences: Lead I becomes inverted (negative P wave and QRS in Lead I). Lead aVR looks like a "normal" Lead I (upright P wave and QRS). This pattern is classic and recognizable. HOWEVER, it can be mistaken for dextrocardia (heart on the right side), lateral STEMI, or other pathology.

How to recognize it on the ECG: Negative P wave in Lead I + P wave inverted in Lead II + aVR shows an upright, normal-appearing complex. The rule: in normal sinus rhythm, the P wave

and QRS should ALWAYS be upright (positive) in Lead I. If they are negative in Lead I, suspect RA/LA reversal first.

Prevention: Read the electrode and wire labels. Develop a consistent sequence for connecting wires. Double-check each connection before acquiring.

Error 4: Limb Electrode Placed on the Trunk (Without Documentation)

What happens: Limb electrodes are placed on the torso (shoulders and lower abdomen) rather than the extremities — the "Mason-Likar" modification — without documenting this deviation.

ECG consequences: The Mason-Likar position shifts the frontal plane axis. Q waves may appear in inferior leads that are not present with standard limb electrode placement. ST segment appearance changes in some leads. A tracing acquired with Mason-Likar electrodes should NOT be used for axis measurement or systematic comparison with a standard ECG.

When Mason-Likar IS appropriate: Continuous monitoring (to reduce motion artifact during ambulation, exercise testing, or transport), NOT for diagnostic 12-lead ECG acquisition.

Error 5: Electrode Placed Over Bony Prominences

What happens: A limb electrode is placed directly over a bony prominence — e.g., directly over the tibial crest (shinbone), over the styloid process of the wrist, or over the malleolus itself.

ECG consequences: Increased impedance, poor electrical contact, and baseline artifact (wandering baseline). Bony tissue has poor electrical conductivity; electrodes need to be placed over muscular or subcutaneous fatty tissue.

Prevention: Always place limb electrodes over the fleshy, muscular surface of the limb, not on bony prominences.

Error 6: Electrode Applied to Skin Not Properly Prepared

What happens: Electrodes placed on wet (sweaty), oily, or lotioned skin without preparation.

ECG consequences: Poor adhesion leads to movement artifact (baseline wander). Wet alcohol under the electrode increases impedance. Lotion creates an insulating layer between electrode and skin.

Error 7: Precordial Lead Wire Connected to Wrong Electrode

What happens: A precordial lead wire (e.g., V3) is connected to the wrong electrode (e.g., V4), producing a positional swap.

ECG consequences: Poor R wave progression, abnormal transition zone, apparent loss of normal voltage progression across the precordial leads.

Prevention: Connect precordial wires in order (V1 → V2 → V3 → V4 → V5 → V6) without skipping. Check that the wire color/label matches the electrode label before moving to the next connection.

3.8 Special Patient Populations

Female Patients and Breast Tissue

Breast tissue presents one of the most common challenges in precordial electrode placement. Breasts are composed primarily of glandular tissue, fat, and connective tissue — none of which affect the underlying rib and intercostal space anatomy. The sternal angle, 4th ICS, 5th ICS, and anatomical lines all exist in exactly the same positions in female patients as in male patients. The challenge is ACCESS.

Standard recommendation: Electrodes should be placed BENEATH the breast, resting on the chest wall — not over the breast tissue. The breast should rest over the electrode after placement. This maintains accurate positional placement based on underlying rib anatomy.

How to achieve under-breast placement:

50. Explain to the patient what you are doing and why. Obtain explicit consent: "To get an accurate reading, the electrode needs to go beneath your breast, resting on your chest wall. I'll need to lift the breast to place it. Is that okay?"
51. If the patient is able, ask her to lift her own breast while you place the electrode.

52. If the patient cannot assist, use the back of your (gloved) hand to gently elevate the breast while placing the electrode with the other hand.
53. After the electrode is adhered, gently lower the breast over the electrode.
54. Confirm the electrode is still in the correct position — breast weight can sometimes displace an incompletely adhered electrode.

Clinical Note: Over-Breast vs. Under-Breast Placement

Some studies have evaluated ECG differences between over-breast and under-breast electrode placement. The general consensus is:

• Under-breast placement (on the chest wall) is the STANDARD and produces more accurate results, particularly for V4 and V5.
• Over-breast placement may reduce amplitude of lateral precordial leads and alter T wave morphology in V5 and V6.
• If over-breast placement is used (due to patient preference or inability to access the chest wall), this MUST be documented on the ECG tracing.

If a patient declines under-breast placement, respect her decision, document the modification, and acquire the best-quality tracing possible. A suboptimal ECG with documented limitation is always preferable to a confrontational encounter.

Patients With Pacemakers or ICDs

The presence of an implanted cardiac device does NOT contraindicate ECG acquisition. Proceed with standard electrode placement. Document the presence of a pacemaker on the ECG tracing. The ECG interpreter will look for pacemaker spikes (vertical lines preceding each paced complex) and assess pacemaker function.

Do NOT avoid the area of the device (typically the left or right upper chest, in the infraclavicular area) when placing V1 and V2 — electrode placement near the device is safe and does not interfere with device function under normal ECG acquisition conditions.

Obese Patients

In patients with obesity, the following modifications and challenges are common:

- **Finding the sternal angle**: May require firmer palpation through subcutaneous tissue. Press firmly with the fingertip (not the fingertip pad) and slide slowly. The sternal angle is a bone-to-bone junction and will still produce a palpable ridge in virtually all patients.

- **Counting intercostal spaces**: More challenging due to subcutaneous tissue over ribs. Apply firm, deliberate pressure and move slowly.
- **Midclavicular line**: The clavicle is palpable in most obese patients — locate its medial and lateral ends explicitly by pressing firmly.
- **V4 placement**: The midclavicular line may appear shifted medially or laterally due to fat redistribution on the chest wall. ALWAYS locate it from the clavicle landmarks, not visually.
- **Electrode adhesion**: Adipose tissue is more mobile and may degrade electrode adhesion. Use self-adhesive electrodes with foam backing. Consider extra skin abrasion.

Pediatric Patients

Electrode placement in children follows the same anatomical landmarks as adults. Key differences:

- The chest is smaller — electrode spacing will be proportionally smaller. V4, V5, V6 may be very close together.
- In children under 8, the right ventricle is relatively larger — an R' in V1 may be a normal finding, not RBBB.
- Heart rates are physiologically higher in children (normal rate: 80–140 in infants; 80–120 in young children; 60–100 in adolescents).
- Use pediatric-sized electrodes if available.

3.9 Posterior Leads (V7–V9) and Right-Sided Leads (V3R–V6R)

The standard 12-lead ECG does not directly image the posterior wall of the left ventricle or the right ventricle. In specific clinical situations — suspected posterior MI or suspected right ventricular infarction — additional leads must be acquired using a modified electrode placement.

When to Apply Posterior Leads

Posterior leads are indicated when:

- ST depression is noted in V1–V3 (particularly with a tall R wave in V1 — the mirror image of posterior ST elevation)
- Inferior STEMI is confirmed and you want to assess for posterior involvement
- High clinical suspicion for posterior MI despite a non-diagnostic standard 12-lead

✓ Applying Posterior Leads (V7, V8, V9) — Step by Step

1. PREPARATION: Note and remember the exact horizontal level of V4 (5th ICS, midclavicular line). You will continue from this horizontal level around to the back.
2.
3. POSITIONING: If possible, ask the patient to lean forward slightly, or gently roll them onto their right side to expose the left posterior chest. Alternatively, slide your hand under the left side of the patient's back and apply electrodes.
4.
5. LANDMARKING: Extend the horizontal line of V4–V6 POSTERIORLY around the left side of the chest.
6.
7. V7 placement: Continue at the SAME HORIZONTAL LEVEL as V4–V6. Move further posterior, to the LEFT POSTERIOR AXILLARY LINE — the posterior fold of the axilla (the fold of tissue at the BACK border of the armpit, on the left side).
8.
9. V8 placement: SAME HORIZONTAL LEVEL. Move further back to the LEFT TIP OF THE SCAPULA (left inferior scapular angle — the bottom tip of the left shoulder blade). V8 is at the left midscapular (subscapular) line.
10.
11. V9 placement: SAME HORIZONTAL LEVEL. Move all the way to the LEFT PARAVERTEBRAL BORDER — immediately to the left of the thoracic spine.
12.
13. ECG ACQUISITION: On the ECG machine, reassign the chest lead inputs:
14. • Connect V4 input wire to the V7 electrode location
15. • Connect V5 input wire to the V8 electrode location
16. • Connect V6 input wire to the V9 electrode location
17. Then print/label the tracing as "V7, V8, V9" (not V4, V5, V6).
18.
19. INTERPRETATION CRITERIA: ST elevation ≥ 0.5 mm in ANY of V7, V8, or V9 = posterior STEMI confirmed.

✓ Applying Right-Sided Leads (V3R–V6R) — Step by Step

1. RIGHT VENTRICULAR INFARCTION occurs in approximately 30–50% of inferior STEMI cases (RCA occlusion).
2. It is critical to identify because management differs significantly (IV fluids required; nitrates and diuretics potentially dangerous).
3.
4. INDICATION: Apply right-sided leads for EVERY patient with confirmed or suspected inferior STEMI.
5.

Right-sided leads are the MIRROR IMAGE of the standard precordial leads, placed on the RIGHT side of the chest.

PREPARATION: The right-sided leads are placed at the SAME anatomical positions as V1–V6 but on the RIGHT side of the chest.

• V1R is where V2 is normally placed (left sternal border, 4th ICS)

• V2R is where V1 is normally placed (right sternal border, 4th ICS)

In practice, V1R and V2R are rarely used clinically. The most important leads are V3R–V6R.

V3R placement: Midway between V2R (right sternal border, 4th ICS) and V4R.

V4R placement (THE MOST IMPORTANT RIGHT-SIDED LEAD): 5th intercostal space, RIGHT MIDCLAVICULAR LINE.

Find the right midclavicular line using the same technique as for V4 but applied to the RIGHT clavicle.

Place at the 5th ICS — the mirror image of standard V4 on the right chest.

V5R placement: Same horizontal level as V4R, right anterior axillary line.

V6R placement: Same horizontal level as V4R, right midaxillary line.

ECG ACQUISITION:

• Connect V3–V6 input wires to the corresponding right-sided electrode positions

• Relabel on the ECG as V3R–V6R

• V4R is the most clinically useful single right-sided lead

INTERPRETATION CRITERIA: ST elevation ≥ 1 mm in V4R = right ventricular infarction confirmed.

(Note: Right-sided lead ST elevation resolves rapidly — usually within 10–12 hours. Acquire these leads EARLY.)

Clinical Application: The Value of Right-Sided Leads

A 71-year-old woman presents by ambulance with chest pressure, vomiting, and near-syncope. Her blood pressure is 82/54 mmHg. Her initial 12-lead ECG shows 3 mm of ST elevation in II, III, and aVF — a classic inferior STEMI pattern. The paramedic notes she has ST depression in I and aVL (reciprocal changes confirming the diagnosis).

The physician calls you to perform right-sided leads. You apply V4R (5th ICS, right midclavicular line) and find 2 mm of ST elevation — a strongly positive V4R.

Clinical significance: This patient has right ventricular infarction. Her hypotension (BP 82/54) is caused by right ventricular failure — the RV is not pumping effectively, reducing left heart filling and causing low cardiac output. She is PRELOAD-DEPENDENT: she needs IV fluids to maintain RV filling pressure.

Contraindicated medications (potentially fatal in this patient):
• Nitroglycerin (reduces preload → collapses RV → cardiac arrest)
• Morphine (reduces preload → same consequence)
• Diuretics (reduces preload → same consequence)

Your V4R finding directly prevents potentially fatal medication errors. Report immediately: "V4R shows 2 mm ST elevation — consistent with right ventricular involvement. The patient is hypotensive and we should avoid nitrates."

3.10 ECG Troubleshooting: What Every ECT Must Know

Even with perfect technique, artifacts and technical problems occur. Being able to rapidly identify and correct them is an essential clinical skill. The following section covers the major artifact types with their specific causes, recognition on the ECG, and correction techniques.

Baseline Wander (Low-Frequency Artifact)

Appearance on ECG: The entire ECG tracing undulates up and down slowly — like riding on ocean waves. The baseline moves continuously between beats, making it impossible to accurately assess the isoelectric line and measure ST changes.

Frequency: The wandering occurs at less than 1 Hz (< 1 cycle per second) — it is a slow, wave-like motion, not a rapid oscillation.

Common causes and specific corrections:

Cause	Correction
Patient breathing too deeply	Instruct to breathe normally and shallowly. Encourage relaxation.
Patient movement (fidgeting, shivering)	Wait for stillness; warm the patient if shivering; treat pain.

Poor electrode adhesion (sweaty, oily skin)	Remove, prep skin thoroughly, replace electrode.
Electrodes placed on bony prominences	Move to fleshy surfaces, reapply.
Limb electrode movement	Move limb electrodes to trunk (Mason-Likar) — note this on the tracing.
Dried or old electrode gel	Replace with fresh electrodes.

60-Hz (AC Power) Interference

Appearance on ECG: The entire tracing has a fine, uniform, regular "fuzz" superimposed on it. The baseline appears thickened. All 12 leads are equally affected simultaneously. At 25 mm/sec paper speed, 60-Hz artifact appears as approximately 2.5 tiny oscillations per millimeter — so fine it resembles a thick, hairy baseline.

Why it happens: The patient (and lead wires) act as an antenna for 60-Hz electrical interference emitted by nearby electrical equipment. When the impedance (resistance) between the electrode and skin is high, the patient's skin-electrode system becomes especially susceptible to picking up environmental electrical noise.

Specific corrections, in order of likelihood of success:

55. Improve skin-electrode impedance: Re-abrade skin, replace electrode, dry sweaty skin. This is the most effective single intervention.
56. Turn off or unplug non-essential electrical equipment: Infusion pumps, electric blankets, heating pads, and powered IV beds are common culprits. Unplugging them from the wall eliminates their electromagnetic field.
57. Reposition lead wires: Ensure lead wires are NOT touching metal bed rails or lying across other wires. Bundling the wires together and routing them away from electrical equipment reduces pickup.
58. Check the ground (RL) electrode: Reapply the right leg ground electrode with fresh skin prep. The ground electrode is the primary noise-rejection point.
59. Use the ECG machine's internal 60-Hz notch filter: Most modern ECG machines have this filter. However, using the filter permanently may mask certain waveform details — it should be a last resort, not a first step. Note on the tracing that the filter was applied.

Somatic Tremor (Muscle Artifact)

Appearance on ECG: Irregular, chaotic, high-frequency oscillations superimposed on the tracing. Unlike 60-Hz artifact (which is perfectly regular), muscle artifact is IRREGULAR in amplitude and frequency. In severe cases, it can completely obscure the QRS complexes and resemble ventricular fibrillation to an untrained eye.

CRITICAL: Tremor Artifact vs. Ventricular Fibrillation

Muscle tremor artifact (especially from Parkinson's disease, severe anxiety, or shivering) can superficially resemble ventricular fibrillation on the monitor — both appear as chaotic, irregular baseline oscillations.

The KEY distinguishing feature: In artifact, if you look carefully, you can usually see ORGANIZED QRS complexes periodically "poking through" the artifact at regular intervals. In true V-fib, there are NO organized QRS complexes — only completely chaotic waveforms.

MOST IMPORTANTLY: Check the patient. If the patient is conscious, talking, or has a pulse — the chaotic tracing is ARTIFACT, not V-fib. Never defibrillate a patient based solely on an ECG finding without confirming the clinical picture.

3.11 Documentation of the ECG Acquisition

Every ECG you acquire is a legal medical document. Complete and accurate documentation is mandatory and protects both the patient and the clinician.

Documentation Element	What to Include
Patient identification	Full legal name, date of birth, medical record number (MRN)
Date and time	Exact date and time of acquisition (not just "this morning")
Clinical indication	Reason for ECG: "chest pain," "syncope," "arrhythmia evaluation," etc.
Placement modifications	Any deviation from standard placement (amputation, wound, mastectomy, Mason-Likar)
Calibration and speed	Note if non-standard settings used (e.g., half-standard, 50 mm/sec)
Immediate findings	Note any immediately apparent abnormalities reported to clinician

Interpreter	Name/credentials of who interpreted the ECG

Chapter 3 Review Questions

1. You are applying V4 to a patient. After locating the sternal angle and counting down intercostal spaces, you find the 5th ICS. Where should V4 be placed along this horizontal level?

a) At the anterior axillary line — the anterior fold of the armpit
b) At the midclavicular line — the vertical line from the midpoint of the clavicle
c) At the midaxillary line — the vertical line from the center of the axilla
d) Immediately adjacent to the left sternal border

► **Correct Answer:** b) At the midclavicular line — the vertical line from the midpoint of the clavicle

Rationale: *V4 is placed in the 5th intercostal space at the midclavicular line (MCL). The MCL is found by locating the medial end (sternoclavicular joint) and lateral end (acromioclavicular joint) of the clavicle and dropping a vertical line from their midpoint.*

2. A 12-lead ECG shows an inverted P wave and inverted QRS complex in Lead I. Lead aVR appears with an upright, normal-appearing P wave and QRS. The patient is alert and has a pulse. What is the MOST LIKELY explanation?

a) Dextrocardia (heart on the right side)
b) RA/LA limb lead reversal
c) A rhythm originating from the left atrium
d) Left bundle branch block

► **Correct Answer:** b) RA/LA limb lead reversal

Rationale: *The classic pattern of RA/LA reversal: inverted (negative) P wave and QRS in Lead I, with aVR appearing like a "normal" Lead I (upright P and QRS). This can mimic dextrocardia, but the precordial lead R wave progression is normal in lead reversal (whereas in true dextrocardia, the precordial leads also show mirror-image patterns). Always verify electrode connections and repeat the ECG before reporting any diagnosis.*

3. While applying precordial leads, you place V4 in the correct position and then place V5 in the NEXT intercostal space below V4 (6th ICS) at the anterior axillary line. What is the consequence of this error?

a) V5 is now in the correct position — this is the standard placement
b) V5 is placed too low, which can reduce lateral R wave amplitude and alter ST appearance
c) V5 is placed too high, causing false anterior ST elevation

d) No clinical consequence — the anterior axillary line position compensates for the level difference

► **Correct Answer:** b) V5 is placed too low, which can reduce lateral R wave amplitude and alter ST appearance

Rationale: *V5 must be at the SAME horizontal level as V4 — NOT in the next intercostal space below. Placing V5 in the 6th ICS (one space too low) moves it away from the standard position, potentially reducing R wave amplitude in V5 and altering lateral lead ST appearances, possibly masking lateral wall ischemia.*

4. A patient presents with an inferior STEMI confirmed by ST elevation in II, III, and aVF. Her blood pressure is 76/48 mmHg. You acquire right-sided leads and find 1.5 mm of ST elevation in V4R. What is the IMMEDIATE clinical significance of this finding?

a) This is a normal finding and requires no special consideration

b) This indicates right ventricular infarction — nitrates and diuretics are potentially dangerous in this patient

c) This pattern confirms a left main coronary artery occlusion

d) This indicates the patient has complete heart block

► **Correct Answer:** b) This indicates right ventricular infarction — nitrates and diuretics are potentially dangerous in this patient

Rationale: *V4R ST elevation ≥ 1 mm confirms right ventricular infarction. RV infarction patients are preload-dependent — they require IV fluids to maintain cardiac output. Nitrates, morphine, and diuretics reduce preload and can precipitate severe hypotension or cardiac arrest. This information must be communicated IMMEDIATELY to the treating team.*

5. During a 12-lead ECG acquisition, the monitor shows a chaotic, high-frequency, irregular baseline artifact in all leads. The patient is sitting up in bed talking to a family member. What is the FIRST step?

a) Immediately defibrillate — this could be ventricular fibrillation

b) Assess the patient: confirm they are conscious and alert, then identify and correct the source of artifact

c) Call a code and begin CPR

d) Administer oxygen and call the physician

► **Correct Answer:** b) Assess the patient: confirm they are conscious and alert, then identify and correct the source of artifact

Rationale: *A patient who is talking and fully conscious CANNOT be in ventricular fibrillation. The chaotic tracing is artifact — most likely muscle tremor from movement or talking. Always check the patient first. Identify the source (patient movement, talking, external electrical interference) and correct it before re-acquiring.*

CHAPTER 4: THE ECG MACHINE, PAPER, AND RECORDING BASICS

Learning Objectives

Upon completing this chapter, you will be able to:

28. Describe the components of an ECG machine and the function of each.
29. Explain ECG paper speed and amplitude standards.
30. Define the dimensions of small and large boxes on ECG paper.
31. Calculate time intervals and voltage amplitudes from ECG paper.
32. Describe the standardization mark and explain why it matters.
33. Explain how to measure heart rate using the large-box method and the 300/150/100 method.
34. Define the isoelectric baseline and explain its significance.

4.1 The ECG Machine: Components and Function

Modern ECG machines are sophisticated electronic devices that detect, amplify, filter, and display or print the tiny electrical signals generated by the heart. The electrical signals reaching the skin surface are extremely small — on the order of 1–3 millivolts (mV) at most — and must be amplified approximately 1,000 times before they can be displayed usefully.

Component	Function
Electrodes	Detect electrical potential (voltage) at the skin surface; convert ionic (body) current to electronic (wire) current via silver/silver-chloride chemistry.
Lead wires	Carry the signal from electrodes to the machine. Color-coded; should be inspected regularly for damage at connector ends.
Differential amplifier	Amplifies the difference between two electrode voltages (the desired cardiac signal) while rejecting common-mode noise (electrical interference affecting both leads equally). This is the primary noise-rejection mechanism.
Analog-to-digital converter	Converts the amplified analog signal into digital data for processing, storage, and transmission. Modern ECGs sample at 500–1000 Hz.

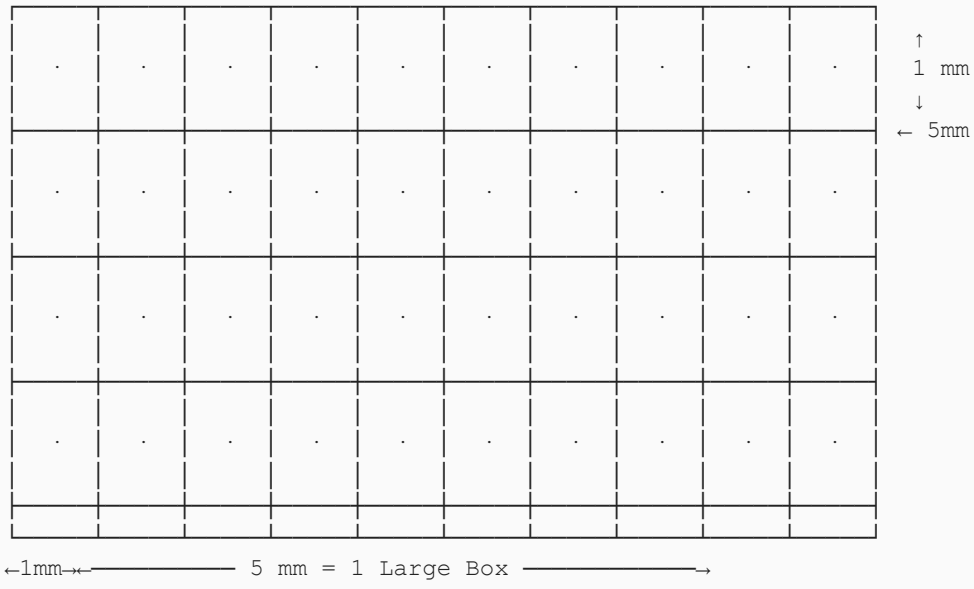
Digital signal processor	Applies filters (baseline filter 0.05–0.67 Hz, muscle filter 25–40 Hz, 60-Hz notch filter), calculates derived leads, performs automatic interpretation.
Display and printer	Displays real-time waveforms; prints the standard 12-lead ECG on thermal paper at standard speed and calibration.

4.2 ECG Paper: The Map of the Heart's Electrical Story

Standard ECG paper is a critical tool for interpretation. It is a continuous strip (or a set of printed strips) with a precise grid printed in red or pink. Every square on that grid has an exact, standardized time and voltage value. Learning to "read" the ECG paper grid is as fundamental as learning to read sheet music before playing an instrument.

The Grid: Small Boxes and Large Boxes

Figure 4.1 — ECG Paper Grid Structure



Grid Unit	Size

Small box (1 thin line)	1 mm × 1 mm
Large box (1 thick line)	5 mm × 5 mm
10 small boxes (horizontal)	10 mm
25 small boxes = 5 large (horiz)	25 mm

Memorize These Values — Everything Else Flows From Them

SMALL BOX: 0.04 seconds wide | 0.1 mV tall
LARGE BOX: 0.20 seconds wide | 0.5 mV tall

To measure ANY time interval: Count the small boxes and multiply by 0.04.
OR: Count large boxes × 0.20, add small boxes × 0.04.

To measure ANY voltage (amplitude): Count small boxes × 0.1 mV.
OR: Count large boxes × 0.5 mV.

The Standardization Mark (Calibration Pulse)

Every diagnostic ECG should include a **standardization mark** — a 1 mV pulse generated by the machine itself that appears as a square-wave signal at the beginning or end of the tracing. The standard calibration produces a deflection exactly **10 mm tall** (10 small boxes = 1 mV). This confirms the machine's amplification is set correctly.

If the calibration mark is shorter than 10 mm: the ECG is recorded at "half standard" (0.5×) amplification — all voltage measurements will appear smaller than actual. This is done for patients with very large-amplitude ECGs to prevent waveforms from colliding with adjacent leads.

If the calibration mark is taller than 10 mm: amplification is above standard. Always check the calibration before commenting on whether waveforms are abnormally tall or small.

Paper Speed

The standard ECG paper speed is **25 mm per second** in the United States. At 25 mm/sec, each large box (5 mm) represents 0.20 seconds, and each small box (1 mm) represents 0.04 seconds.

In some countries (particularly parts of Europe and Asia), 50 mm/sec is used. At 50 mm/sec, the ECG looks "stretched out" horizontally — intervals appear longer and complexes look wider. Always check the paper speed notation on the printed ECG header before measuring intervals.

4.3 Measuring Heart Rate from the ECG Strip

The heart rate can be calculated from the ECG strip using several methods. Each has trade-offs between speed and accuracy.

Method 1: The 300 / Large Box Method (Best for Regular Rhythms)

This is the fastest and most widely used method for regular rhythms:

60. Find an R wave that lands exactly on (or very near) a bold large-box line.
61. Count the number of LARGE BOXES to the NEXT R wave.
62. Divide 300 by the number of large boxes.

Formula: Heart Rate = 300 ÷ (Number of large boxes between R waves)

Large Boxes Between R Waves	Calculated Heart Rate
1 large box	300 bpm
2 large boxes	150 bpm
3 large boxes	100 bpm
4 large boxes	75 bpm
5 large boxes	60 bpm
6 large boxes	50 bpm

7 large boxes	~43 bpm
8 large boxes	~38 bpm
10 large boxes	30 bpm

Memory Tool: 300-150-100-75-60-50

Memorize this sequence: 300 — 150 — 100 — 75 — 60 — 50
These correspond to 1, 2, 3, 4, 5, and 6 large boxes between R waves.

When the R wave falls between large box lines, interpolate: if the R-R interval is between 3 and 4 large boxes (between 75 and 100 bpm), estimate based on position between those two values.

Method 2: The 6-Second Strip Method (Best for Irregular Rhythms)

For irregular rhythms (atrial fibrillation, frequent ectopics, irregular sinus rhythm), the large-box method is inaccurate because R-R intervals vary. The 6-second method gives an average rate:

63. Most ECG strips have marks at 1-second or 3-second intervals at the top of the strip.
64. Count the number of QRS complexes in a 6-second interval (30 large boxes).
65. Multiply by 10 to get beats per minute.

Formula: Heart Rate = (Number of QRS complexes in 6 seconds) × 10

Method 3: Precise Calculation (For Exact Measurements)

For precise heart rate calculation: measure the R-R interval in small boxes, multiply by 0.04 to get the interval in seconds, then divide 60 by that number.

Formula: Heart Rate = 60 ÷ R-R interval (in seconds) = 1500 ÷ R-R interval (in small boxes)

4.4 The Isoelectric Baseline

The **isoelectric baseline** (also called the isoelectric line or TP segment) is the flat horizontal line that represents the ECG during periods when no electrical activity is occurring —

specifically, during the period between the end of the T wave and the beginning of the next P wave (the TP interval).

The isoelectric baseline is the **reference point for measuring all elevations and depressions** of ST segments and J points. When you measure ST elevation, you are measuring how many millimeters the ST segment is elevated ABOVE the isoelectric baseline.

A clean, stable isoelectric baseline is essential for accurate ST analysis. Baseline wander (see Section 3.6) makes it difficult or impossible to accurately measure small ST changes, which is one reason good skin preparation and proper electrode application are so clinically important — not just for aesthetics, but for diagnostic accuracy.

CHAPTER 5: ECG WAVEFORMS, INTERVALS, AND THE FIVE-STEP RHYTHM ANALYSIS METHOD

Learning Objectives

Upon completing this chapter, you will be able to:

35. Name and describe each component of the normal ECG complex: P wave, PR interval, QRS complex, ST segment, T wave, U wave, and QT interval.
36. State the normal duration and amplitude criteria for each waveform and interval.
37. Explain the clinical significance of abnormal P wave morphology.
38. Describe the significance of a prolonged or shortened QT interval.
39. Apply the Five-Step Rhythm Analysis Method to any ECG strip.
40. Define the terms: rate, rhythm, axis, hypertrophy, and ischemia/infarction as used in systematic ECG review.

5.1 The Normal ECG Complex: A Waveform-by-Waveform Guide

Each ECG complex contains a series of named waveforms, segments, and intervals. Learning to identify each one and know its normal values is the foundation of all ECG interpretation. We will review each component in the order it appears on the ECG strip, following the sequence of electrical events in the heart.

The P Wave: Atrial Depolarization

P Wave — Normal Criteria

Duration: < 0.12 seconds (less than 3 small boxes)
Amplitude: < 2.5 mm (less than 2.5 small boxes tall)
Morphology: Smooth and rounded (dome-shaped); not notched, peaked, or biphasic in Lead II
Axis/Direction: Upright (positive) in Leads I and II; inverted (negative) in aVR

Every P wave should look the same as the others in the same lead.
There should be one P wave before every QRS complex.
The P wave should come at regular intervals (PP interval is consistent).

The P wave represents **atrial depolarization** — the wave of electrical activation spreading through both atria from the SA node. A normal P wave is smooth and dome-shaped in Lead II,

reflecting the orderly spread of activation from the SA node in the right atrium through both atria via internodal pathways and Bachmann's bundle.

Abnormal P wave findings and their significance:

P Wave Finding	Possible Significance
Peaked (tall, narrow > 2.5 mm) — "P pulmonale"	Right atrial enlargement (right heart strain — COPD, pulmonary hypertension)
Notched or broad (> 0.12 sec) — "P mitrale"	Left atrial enlargement (mitral valve disease, left heart failure)
Biphasic P wave in V1 (positive then negative)	Left atrial enlargement when the negative portion is deep (> 1 mm) and wide (> 0.04 sec)
No P waves visible	Atrial fibrillation, junctional or ventricular rhythms, severe hyperkalemia
P wave negative in Lead II	Ectopic atrial rhythm (pacemaker not the SA node), LA-RA lead reversal, junctional rhythm
Different P wave morphologies	Multiple ectopic atrial foci (wandering atrial pacemaker, multifocal atrial tachycardia)
P wave after QRS (retrograde)	Junctional rhythm with retrograde atrial conduction

The PR Interval: AV Nodal Conduction Time

PR Interval — Normal Criteria

Duration: 0.12 to 0.20 seconds (3 to 5 small boxes)
Measured from: Beginning of P wave to beginning of QRS complex
Should be: Consistent from beat to beat in sinus rhythm

< 0.12 sec (short PR): Pre-excitation syndrome (Wolff-Parkinson-White), accelerated AV conduction, junctional rhythm
> 0.20 sec (long PR): First-degree AV block (or more severe AV block if PR changes or QRS is dropped)

The PR interval represents the time from the beginning of atrial depolarization (start of P wave) to the beginning of ventricular depolarization (start of QRS complex). It includes:

- The time for atrial depolarization itself (within the P wave)
- The time for the impulse to travel through the AV node (the AV nodal delay — the PR segment between the end of P and the start of QRS)
- The time for the impulse to travel through the Bundle of His and bundle branches (which is included but usually negligible compared to the AV nodal delay)

The PR interval is one of the most clinically important measurements on the ECG. Prolongation (> 0.20 sec) indicates delayed AV conduction — first-degree AV block — which can be a normal variant in well-trained athletes or a sign of medication effects (beta-blockers, digoxin, calcium channel blockers), ischemia, or intrinsic AV nodal disease. Shortening (< 0.12 sec) can indicate accessory pathway conduction (bypassing the AV node), as in Wolff-Parkinson-White syndrome.

The QRS Complex: Ventricular Depolarization

QRS Complex — Normal Criteria

Duration: 0.06 to 0.10 seconds (narrow) — MAXIMUM normal 0.12 sec (3 small boxes)
> 0.12 sec = WIDE QRS — bundle branch block, hyperkalemia, ventricular origin
Amplitude: Varies significantly by lead and patient — no single universal normal

Components:
Q wave: First NEGATIVE deflection before first positive deflection (if present)
R wave: First POSITIVE (upward) deflection
S wave: First NEGATIVE deflection AFTER the R wave
R' (R prime): Second positive deflection (abnormal — seen in RBBB)

The QRS complex represents **ventricular depolarization** — the spread of electrical activation through both ventricles via the bundle branches and Purkinje fibers. It is the most prominent feature of the normal ECG and is associated with ventricular contraction (systole).

Not every QRS complex contains all three components (Q, R, and S). The presence or absence of each component depends on the lead and the patient's cardiac anatomy. A complex consisting of only a negative deflection is called a **QS complex** (no positive deflection at all). A complex with only a positive deflection (no Q or S) is simply called an **R wave**.

Q Waves: Normal vs. Pathological

Q waves are important to understand because they can be either normal (physiological) or abnormal (pathological):

- **Normal "septal" Q waves**: Small, narrow Q waves in I, aVL, V5, V6 representing left-to-right septal depolarization. Duration < 0.04 sec; amplitude < 25% of the following R wave.
- **Pathological Q waves**: Wide (> 0.04 sec = 1 small box) AND/OR deep (> 25% of the R wave height). Represent electrically dead myocardium (old MI). They are permanent in most cases of transmural (full-thickness) infarction.

R Wave Progression in the Precordial Leads

In the precordial leads (V1 through V6), the R wave should gradually get taller as you progress from right to left:

- V1 and V2: Small R waves (mostly negative QRS — deep S wave predominates)
- V3 and V4: Transitional — R and S waves become equal (the "transition zone" where R = S)
- V5 and V6: Tall R waves (mostly positive QRS)

This gradual increase from V1 to V5 is called **normal R wave progression**. Poor R wave progression (R waves failing to grow, or becoming smaller from V1 to V4) is associated with anterior wall MI, left ventricular hypertrophy, or lead misplacement. A dominant (tall) R wave in V1 suggests right ventricular hypertrophy, RBBB, or posterior MI.

The ST Segment: Between Depolarization and Repolarization

ST Segment — Normal Criteria

The ST segment runs from the END of the QRS complex (the J point) to the beginning of the T wave.

Normal: Isoelectric (flat, at baseline level) ± 1 mm (the small clinical tolerance)
Normal in V1-V3: Up to 2 mm of ST elevation can be normal in young males (early repolarization variant)

ABNORMAL ST ELEVATION (above baseline): Ischemia (STEMI, Prinzmetal's angina), pericarditis, left ventricular aneurysm, Brugada syndrome
ABNORMAL ST DEPRESSION (below baseline): Subendocardial ischemia, NSTEMI, digitalis effect, hypokalemia

The **J point** is the point where the QRS complex ends and the ST segment begins. ST elevation is measured at the J point (or 60–80 ms after the J point in some guidelines). The ST segment represents the plateau phase of the ventricular action potential — the brief period after depolarization when the ventricles are uniformly depolarized and before repolarization begins. A normal ST segment should be flat and isoelectric, with no significant elevation or depression relative to the baseline.

The T Wave: Ventricular Repolarization

T Wave — Normal Criteria

Morphology: Rounded, asymmetric (gradual upstroke, steeper downstroke), smooth
Direction (polarity): Should be UPRIGHT (positive) in Leads I, II, V3–V6; INVERTED in aVR
May be upright or inverted in III and V1–V2 as a normal variant
Amplitude: < 5 mm in limb leads; < 10 mm in precordial leads

ABNORMAL T waves: Peaked (hyperkalemia, early MI), inverted (ischemia, RVH, LVH, RBBB, LBBB, cardiomyopathy), flattened (hypokalemia, hypothyroidism)

The T wave represents **ventricular repolarization** — the electrical reset of the ventricular myocardium after contraction. The T wave is in the same direction as the QRS complex in most leads because, although repolarization is the reverse process of depolarization, it occurs in the opposite direction through the myocardium (epicardium repolarizes before endocardium), resulting in the same net vector direction. T wave changes are among the most sensitive but least specific ECG findings — many conditions alter T wave morphology.

The U Wave: A Sometimes-Visible Extra Wave

The **U wave** is a small, rounded wave that may follow the T wave in some leads. It is most visible in V2–V4. Its mechanism is debated but likely represents repolarization of the Purkinje fibers or ventricular papillary muscles. Normal U waves are small (< 1–2 mm) and in the SAME direction as the T wave.

Clinical significance of abnormal U waves:

- **Prominent U waves** (in the same direction as T, taller than normal): Classic sign of **hypokalemia** (low potassium). As potassium falls, U waves become larger and T waves flatten.
- **Inverted U waves** (opposite direction from T wave): May indicate myocardial ischemia or left ventricular volume overload.

The QT Interval: The Complete Ventricular Cycle

QT Interval — Normal Criteria

Measured from: Beginning of QRS complex to END of T wave
Varies with heart rate — MUST be corrected for rate (QTc = corrected QT)

Normal QTc: < 0.44 seconds (440 ms) in men
< 0.46 seconds (460 ms) in women

PROLONGED QTc (> 0.50 sec = HIGH RISK): Torsades de Pointes risk
CAUSES: Medications (amiodarone, sotalol, haloperidol, azithromycin, many others), hypokalemia, hypomagnesemia, hypocalcemia, congenital long QT syndrome, hypothyroidism, hypothermia

SHORT QTc (< 0.36 sec): Hypercalcemia, digoxin effect, congenital short QT syndrome

The QT interval represents the total time for ventricular depolarization AND repolarization — the complete ventricular electrical cycle. The QT interval shortens at faster heart rates (less time between beats = faster repolarization needed) and lengthens at slower rates. This is why it must be corrected for rate before being called normal or abnormal. The most widely used correction formula is **Bazett's formula**: QTc = QT ÷ √RR (where RR is in seconds). Many ECG machines calculate QTc automatically.

Why QT Prolongation Matters

A prolonged QT interval means ventricular repolarization takes longer than normal. During this extended repolarization period, the ventricle is in its relative refractory period — the "vulnerable period" during which a well-timed extra stimulus can trigger TORSADES DE POINTES (TdP), a specific form of polymorphic ventricular tachycardia that can degenerate into ventricular fibrillation.

Torsades de Pointes appears on the ECG as a twisting ribbon of QRS complexes that oscillate around the isoelectric baseline, changing in amplitude and axis. It causes syncope and can be lethal.

As an ECT, ALWAYS report a prolonged QTc (> 500 ms) immediately to the physician or nurse. Ask about any medications the patient is taking — QT-prolonging drugs are one of the most common causes.

5.2 The Five-Step Method of Rhythm Analysis

A systematic approach to every ECG strip is the hallmark of accurate arrhythmia recognition. The "Five-Step Method" provides a consistent framework that ensures you never miss a critical finding. Apply these five steps to EVERY rhythm strip you analyze, in EXACTLY this order. Do not skip steps or reverse the order — the sequence is designed to prevent errors.

The Five Steps — Memorize This Sequence

Step 1: RATE — What is the heart rate? (Fast? Normal? Slow?)
Step 2: RHYTHM — Is the rhythm regular or irregular?
Step 3: P WAVES — Are P waves present? Do they look normal? Is there one P per QRS?
Step 4: PR INTERVAL — Is the PR interval normal (0.12–0.20 sec)? Constant?
Step 5: QRS COMPLEX — Is the QRS narrow (< 0.12 sec) or wide (≥ 0.12 sec)?

Step 1: Rate

Determine the ventricular rate (count the QRS complexes). Classify it as:

- **Bradycardia**: Rate < 60 bpm
- **Normal rate**: 60–100 bpm
- **Tachycardia**: Rate > 100 bpm

Use the large-box method for regular rhythms (300 ÷ large boxes between R waves) or the 6-second method for irregular rhythms (QRS complexes in 6 seconds × 10).

Step 2: Rhythm

Assess whether the rhythm is regular or irregular:

66. Mark the positions of several consecutive R waves on a piece of paper (or use ECG calipers).
67. Compare the R-R intervals across the strip.
68. If all R-R intervals are equal (or vary < 0.04 sec = 1 small box), the rhythm is REGULAR.
69. If R-R intervals vary, the rhythm is IRREGULAR. Further classify: regularly irregular (a predictable pattern of variability) or irregularly irregular (completely random variability — the hallmark of A-Fib).

Step 3: P Waves

Examine the P waves systematically:

- Are P waves present?
- Do all P waves look the same (same size, shape, and axis)?
- Is there exactly ONE P wave for every QRS complex?
- Does each P wave precede its QRS complex (does P come before QRS)?
- Are P waves upright in Lead II? (If not, the rhythm may not be coming from the SA node)

Step 4: PR Interval

Measure the PR interval from the beginning of the P wave to the beginning of the QRS complex. Confirm it is 0.12 to 0.20 seconds (3 to 5 small boxes). Check whether the PR interval is constant from beat to beat (a varying PR interval suggests Wenckebach or other types of AV block).

Step 5: QRS Complex

Measure the QRS duration. Determine whether it is:

- **Narrow** (< 0.12 sec = < 3 small boxes): The impulse traveled through the normal Purkinje system — the origin is probably above or within the bundle of His (supraventricular).
- **Wide** (≥ 0.12 sec = ≥ 3 small boxes): The impulse either (a) traveled through a bundle branch block, (b) originated in the ventricles (bypassing the Purkinje system), or (c) was conducted aberrantly due to the rate or other factors.

After completing all five steps, you have collected enough data to name the rhythm. Compare your findings to the rhythm criteria you will learn in Chapter 7.

Practice Applying the Five Steps

You are monitoring a patient on the cardiac floor. The strip shows: Rate ~75 bpm; Rhythm: Regular; P waves: Present, upright in II, identical morphology, one before every QRS; PR interval: 0.18 sec (consistent); QRS: 0.08 sec (narrow).

Interpretation: All five steps are within normal limits. This is NORMAL SINUS RHYTHM.

Now imagine the same strip, but the PR interval suddenly extends to 0.24 seconds and every P wave is still followed by a QRS. Step 4 is now ABNORMAL: PR > 0.20 sec. Interpretation: FIRST-DEGREE AV BLOCK (with otherwise normal sinus rhythm). The five-step method found the problem immediately.

Chapter 5 Review Questions

1. A patient's ECG shows a QRS duration of 0.14 seconds. This is BEST described as:

a) Normal — within the 0.06–0.12 second range
b) Narrow — indicating normal Purkinje conduction
c) Wide — suggesting bundle branch block or ventricular origin
d) Borderline — no clinical significance

► **Correct Answer:** c) Wide — suggesting bundle branch block or ventricular origin
Rationale: *A QRS ≥ 0.12 seconds (3 small boxes) is WIDE, indicating abnormal ventricular conduction — either a bundle branch block (BBB), an aberrantly conducted beat, or a rhythm originating in ventricular tissue (PVC, V-tach, idioventricular rhythm). A QRS of 0.14 sec is significantly wide.*

2. A patient has frequent, prominent U waves on the ECG. The MOST likely electrolyte abnormality is:

a) Hyperkalemia (high potassium)
b) Hypokalemia (low potassium)
c) Hypercalcemia (high calcium)
d) Hyponatremia (low sodium)

► **Correct Answer:** b) Hypokalemia (low potassium)
Rationale: *Prominent U waves (large U waves in the same direction as T waves) are the classic ECG sign of hypokalemia. As potassium falls, T waves flatten and U waves become increasingly prominent. Severe hypokalemia also prolongs the QT interval and can predispose to dangerous arrhythmias.*

3. In the Five-Step Method, Step 3 assesses P waves. What are you specifically looking for?

a) Whether P waves are larger than QRS complexes
b) Whether P waves are present, look the same, and precede every QRS
c) Whether the PR interval is within normal range
d) Whether P waves cause the QRS to be wide or narrow

► **Correct Answer:** b) Whether P waves are present, look the same, and precede every QRS
Rationale: *Step 3 specifically asks: Are P waves present? Do they look identical? Is there one P before every QRS? Are they upright in Lead II? These questions identify whether the rhythm originates from the SA node and whether AV conduction is occurring normally.*

4. You calculate a QTc of 530 milliseconds on a patient's ECG. You should:

a) Document it as normal and continue monitoring

b) Immediately report it — QTc > 500 ms is a HIGH-RISK finding requiring urgent physician notification

c) Repeat the ECG in 1 hour to confirm

d) Administer magnesium sulfate immediately without physician order

► **Correct Answer:** b) Immediately report it — QTc > 500 ms is a HIGH-RISK finding requiring urgent physician notification

Rationale: *QTc > 500 ms represents significant QT prolongation and substantially increases the risk of Torsades de Pointes (a form of polymorphic VT that can cause sudden cardiac death). This is an urgent finding requiring immediate physician notification.*

CHAPTER 5: READING AND INTERPRETING THE ECG — A SYSTEMATIC APPROACH

Learning Objectives

Upon completing this chapter, you will be able to:

41. Explain the purpose and structure of systematic ECG interpretation.
42. Apply the standard waveform measurements (P wave, PR interval, QRS, ST segment, T wave, QT interval) to any ECG strip.
43. Use the Five-Step Rhythm Analysis Method correctly and in sequence on any rhythm strip.
44. Apply the Six-Step 12-Lead ECG Interpretation Method to systematically analyze a full 12-lead ECG.
45. Describe how to estimate the electrical axis and identify left and right axis deviation.
46. Identify normal and abnormal R wave progression across the precordial leads.
47. Describe the ECG features of left and right ventricular hypertrophy.
48. Distinguish between ischemia, injury, and infarction patterns on the ECG.
49. Apply a structured verbal reporting format to communicate ECG findings accurately and professionally.

5.1 Why Systematic Interpretation Is Non-Negotiable

The human brain is pattern-recognition machinery. Experienced ECG readers appear to glance at a tracing and immediately say "that's V-tach" or "that's an inferior STEMI." This intuitive pattern recognition is real — but it is built on years of SYSTEMATIC analysis performed so many thousands of times that it has become automatic and subconscious. The danger for a beginning learner is trying to emulate the expert's output (the quick "gestalt" read) without building the expert's foundation (thousands of systematic, step-by-step analyses).

Every ECG you encounter during your training and early career must be analyzed systematically, step by step, in the same order every time. This disciplined approach builds the mental template against which deviations (arrhythmias, ischemia, conduction abnormalities) become immediately apparent. It also provides a reliable safety net against missing findings — the expert who occasionally skips steps misses findings that a systematic beginner would catch.

A Commitment to Process

Commit to the following before reading any ECG strip:

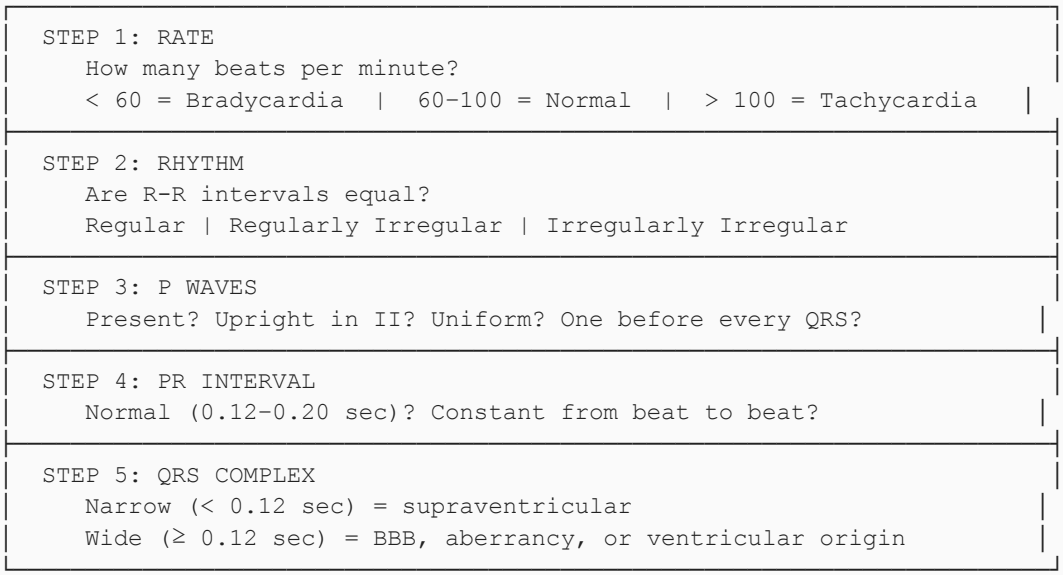
"I will analyze every waveform in sequence. I will not jump to a conclusion before completing all steps. I will name what I see, not what I expect to see."

This commitment — especially during training — is what separates reliable ECT practitioners from those who eventually make dangerous errors.

5.2 The Five-Step Method for Rhythm Strip Analysis

The Five-Step Method is designed for analyzing a single lead rhythm strip — the most common type of ECG analysis performed during continuous cardiac monitoring. Apply these five steps in order, without skipping, to every rhythm strip you encounter.

Figure 5.1 — The Five Steps of Rhythm Analysis

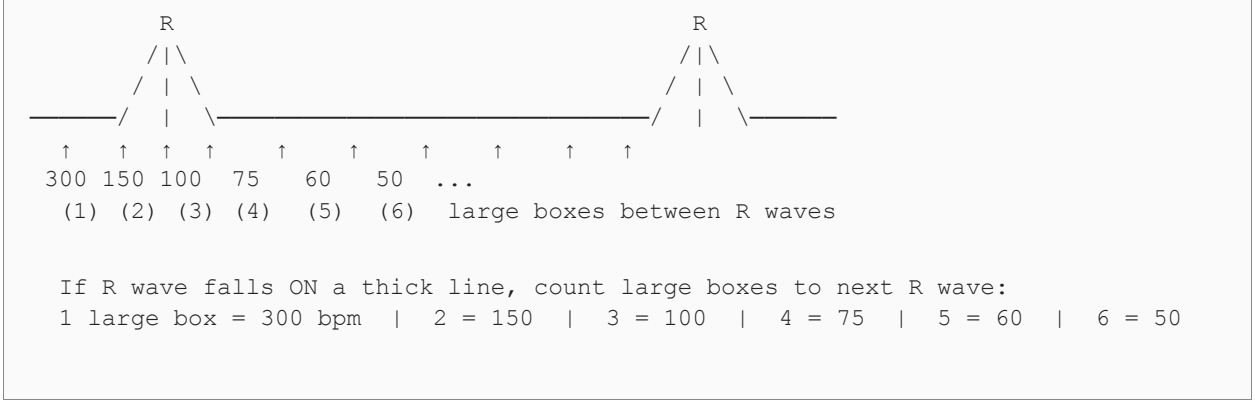


Step 1: Determine the Rate

Heart rate assessment is your first and fastest orientation to the ECG. Within 2–3 seconds of looking at a rhythm strip, you should be able to classify the rate as bradycardic, normal, or tachycardic. This initial classification immediately narrows the differential diagnosis.

Method 1 — The 300 Method (for regular rhythms): Find an R wave that falls exactly on a thick (large-box) vertical line. Count the number of large boxes to the NEXT R wave. Divide 300 by that number.

Figure 5.2 — The 300 Method for Rate Calculation



Method 2 — The 6-Second Method (for irregular rhythms): Count the QRS complexes in a 6-second interval (30 large boxes) and multiply by 10. Most ECG paper has timing marks at the top margin at 1-second or 3-second intervals.

Method 3 — The 1500 Method (most precise): Count the number of SMALL boxes between two consecutive R waves. Divide 1500 by that number. For example, 20 small boxes between R waves = 1500 ÷ 20 = 75 bpm.

Method	Formula
300 Method	300 ÷ large boxes between R waves
6-Second Method	QRS complexes in 6 sec × 10
1500 Method	1500 ÷ small boxes between R waves

Step 2: Assess the Rhythm

Rhythm assessment determines whether the intervals between heartbeats are consistent. Even before identifying the rhythm by name, you must characterize it as regular, regularly irregular, or irregularly irregular.

How to assess rhythm with calipers (the gold standard):

70. Set your calipers to the distance between two consecutive R wave peaks (the R-R interval).
71. Without changing the caliper width, "walk" the calipers across the strip by alternately lifting and placing the caliper tips at each successive R wave.
72. If the caliper tip lands exactly on each R wave peak across the entire strip, the rhythm is REGULAR.
73. If the caliper tip occasionally lands slightly before or after an R wave, note the pattern: is the variation predictable (regularly irregular) or completely random (irregularly irregular)?

How to assess rhythm without calipers:

74. Mark the position of three to five consecutive R wave peaks on a piece of paper using a pen.
75. Slide the paper along the strip to compare R-R intervals at different points in the tracing.
76. If all marks align with subsequent R waves, the rhythm is regular.

Rhythm classifications:

- **Regular**: All R-R intervals are equal (or vary by < 0.04 sec = 1 small box). Normal sinus rhythm, junctional rhythm, V-tach, SVT, atrial flutter with fixed block — all have regular ventricular rhythms.
- **Regularly irregular**: R-R intervals vary, but in a PREDICTABLE, REPEATING PATTERN. Examples: Wenckebach (2° AV block Type I) — a group of progressively shorter R-R intervals followed by a pause; bigeminy — normal beat-PVC-pause-normal beat-PVC-pause repeating.
- **Irregularly irregular**: R-R intervals are COMPLETELY RANDOM with no discernible pattern. This is the defining characteristic of atrial fibrillation. No two consecutive R-R intervals are the same length. There is no pattern, no grouping, no predictability.

Step 3: Analyze the P Waves

P wave analysis is the most diagnostically powerful step in the Five-Step Method. The P wave tells you WHERE the rhythm originates — and that single piece of information narrows the differential enormously. Apply the following checklist systematically to the P waves:

✓ P Wave Analysis Checklist

1. [ ] Are P waves PRESENT? Can you see distinct P waves before each QRS? If no P waves visible → A-Fib, V-tach, junctional tachycardia, or severe hyperkalemia.
2.
3. [ ] Do all P waves look IDENTICAL? Same size, shape, and direction? If P waves vary in morphology → wandering atrial pacemaker, multifocal atrial tachycardia, or ectopic beats mixed with sinus beats.
4.
5. [ ] Are P waves UPRIGHT (positive) in Lead II? This is the most important single P wave criterion. The SA node's depolarization vector points toward Lead II's positive pole, making normal SA node P waves upright in Lead II.
6. • Inverted P wave in Lead II = ectopic pacemaker (not the SA node), retrograde atrial activation (junctional rhythm), or lead reversal.
7.
8. [ ] Is there exactly ONE P wave before EVERY QRS complex? Count: if there are more P waves than QRS complexes, some P waves are being blocked (AV block). If there are more QRS complexes than P waves, the rhythm is not sinus in origin.
9.
10. [ ] Does the P wave PRECEDE each QRS? A P wave that comes AFTER the QRS (retrograde P) indicates the atria were activated BACKWARD from the ventricles (junctional or ventricular rhythm with retrograde conduction).
11.
12. [ ] Is the P wave shape normal? Normal P waves are smooth, dome-shaped, and < 0.12 sec wide, < 2.5 mm tall. Peaked tall P waves (P pulmonale) = right atrial enlargement. Notched wide P waves (P mitrale) = left atrial enlargement.

Step 4: Measure the PR Interval

The PR interval is measured from the BEGINNING of the P wave to the BEGINNING of the QRS complex. It represents the total time for the electrical impulse to travel from the SA node through the atria, through the AV node, through the Bundle of His, and begin activating the ventricles.

How to measure the PR interval:

77. Identify a clear, distinct P wave with a well-defined beginning (the upstroke of the P wave from the isoelectric baseline).
78. Count the small boxes from the beginning of the P wave to the beginning of the QRS complex (the first deflection — upward or downward — away from the baseline).
79. Multiply by 0.04 sec: if you count 4 small boxes, the PR interval is 0.16 sec.
80. Normal: 0.12 to 0.20 seconds (3 to 5 small boxes).

PR interval interpretation:

PR Interval	Clinical Significance
< 0.12 sec (< 3 small boxes)	Pre-excitation (Wolff-Parkinson-White syndrome): impulse bypasses AV node via accessory pathway. May also be seen in junctional rhythms with a short RP.
0.12–0.20 sec (3–5 small boxes)	NORMAL AV nodal conduction
> 0.20 sec (> 5 small boxes), CONSTANT	First-degree AV block: delayed but successful AV conduction. Every P wave still reaches the ventricles.
Progressively LENGTHENING	Second-degree AV block, Type I (Wenckebach): progressively longer AV delay until one beat is dropped.
CONSTANT but with sudden DROPPED beats	Second-degree AV block, Type II (Mobitz II): sudden, unpredictable non-conduction with no warning change in PR.
VARIABLE (changes with every beat)	Third-degree (complete) AV block: P waves and QRS complexes are completely independent — the PR "interval" is meaningless.

Step 5: Measure the QRS Complex

The QRS duration tells you whether the ventricles were activated through the NORMAL pathway (Purkinje system, producing a narrow QRS) or through an ABNORMAL pathway (bundle branch block, aberrant conduction, or ventricular origin, producing a wide QRS). This single measurement is often the most important differentiator in emergency arrhythmia recognition.

How to measure QRS duration:

81. Find a lead with a clearly visible, well-defined QRS complex (Lead II is often ideal).
82. Identify the exact point where the QRS begins: the first deflection (upward or downward) from the isoelectric baseline.
83. Identify the exact point where the QRS ends: the return to the baseline (the J point).
84. Count the small boxes from beginning to end of the QRS and multiply by 0.04 sec.

QRS duration interpretation:

QRS Duration	Clinical Significance

0.06–0.10 sec (< 3 small boxes)	Normal: ventricular activation via intact Purkinje system. Rhythm origin is above or within the Bundle of His (supraventricular).
0.10–0.12 sec (borderline)	Borderline wide: incomplete bundle branch block, or beginning of abnormal conduction. Requires further evaluation.
≥ 0.12 sec (≥ 3 small boxes)	WIDE QRS: bundle branch block (RBBB or LBBB), ventricular origin (PVC, V-tach, idioventricular rhythm), hyperkalemia, certain drugs (class IC antiarrhythmics, tricyclic antidepressants), pre-excitation.

The Critical Rule: Wide Complex Tachycardia

When you see a FAST (> 100 bpm) rhythm with a WIDE QRS (≥ 0.12 sec):

ASSUME VENTRICULAR TACHYCARDIA UNTIL PROVEN OTHERWISE.

There are two main causes of wide-complex tachycardia:
1. Ventricular Tachycardia (V-tach): LIFE-THREATENING. Requires immediate treatment.
2. Supraventricular tachycardia (SVT) with aberrant conduction or bundle branch block: usually less immediately dangerous.

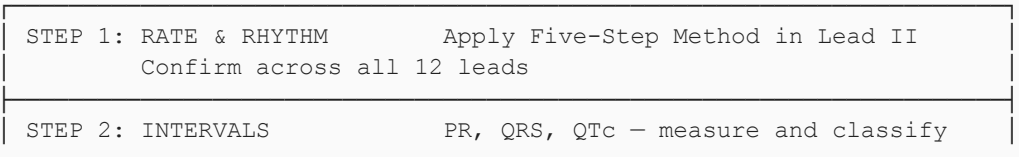
The problem: these two can look IDENTICAL on a rhythm strip, and the clinical stakes are very different.
The solution: in any hemodynamically unstable wide-complex tachycardia, ASSUME V-TACH.
Even if it turns out to be SVT with aberrancy, treating it as V-tach (cardioversion) is safe.
Treating V-tach as SVT (with verapamil or adenosine), however, can be FATAL.

When in doubt: call for help, support the patient, prepare for synchronized cardioversion.

5.3 The Six-Step 12-Lead ECG Interpretation Method

Analyzing a full 12-lead ECG requires a more comprehensive and systematic approach than a single-lead rhythm strip. The Six-Step Method ensures that every diagnostic category is evaluated in a logical, efficient sequence.

Figure 5.3 — Six-Step 12-Lead ECG Interpretation



STEP 3: AXIS	Normal / LAD / RAD — quick screen
STEP 4: R WAVE PROGRESSION	V1 → V6: R growing normally?
STEP 5: HYPERTROPHY	LVH / RVH criteria present?
STEP 6: ST-T CHANGES	Ischemia? Injury? Infarction? Strain?

Step 1: Rate and Rhythm

Begin with Lead II (the best rhythm lead) and apply the Five-Step Method. Then glance across all 12 leads to confirm the rhythm is consistent — occasionally, a lead-specific artifact or anomaly is present, and comparing leads helps identify it. Confirm that P waves are visible and consistent across leads.

Step 2: Intervals

Systematically measure and classify the three critical intervals:

- **PR interval**: Measure in Lead II. Normal 0.12–0.20 sec. Prolonged = AV block. Short = pre-excitation.
- **QRS duration**: Measure in the widest QRS across all leads (usually best seen in Lead II, V1, or V5). < 0.12 sec = narrow; ≥ 0.12 sec = wide.
- **QTc (corrected QT)**: Measure the QT interval (beginning of QRS to end of T wave) in lead II or V5 (where the T wave end is clearest). Apply Bazett's correction: QTc = QT ÷ √(R-R interval in seconds). Normal QTc: < 440 ms in men, < 460 ms in women. QTc > 500 ms = high risk for Torsades de Pointes.

Step 3: Electrical Axis — What It Is and How to Estimate It

The **electrical axis** is the overall direction of ventricular depolarization in the frontal plane — the average direction the depolarization wave travels through the ventricles, expressed as a compass bearing in degrees. In a normal heart, this average vector points downward and to the left (toward the cardiac apex), which is why Lead II — aligned approximately along this direction — typically shows the tallest QRS complexes.

Normal axis: −30° to +90° (in most textbooks; some extend to +105°).

Left axis deviation (LAD): More negative than −30°. Causes: left anterior fascicular block (hemiblock), inferior MI (old), left ventricular hypertrophy, Wolff-Parkinson-White.

Right axis deviation (RAD): More positive than +90° (to +105°). Causes: right ventricular hypertrophy, posterior MI, left posterior fascicular block, normal variant in tall thin individuals.

Extreme axis deviation ("no man's land"): −90° to ±180°. Causes: severe RVH, certain forms of V-tach, bilateral bundle branch block.

The Quick Two-Lead Axis Screen

For practical clinical purposes, the axis can be estimated rapidly using just two leads: Lead I and aVF.

Lead I QRS	aVF QRS
Upright (positive)	Upright (positive)
Upright (positive)	Inverted (negative)
Inverted (negative)	Upright (positive)
Inverted (negative)	Inverted (negative)

Axis Quick-Read Rule

Lead I and aVF act as two perpendicular axes of a compass:
Lead I = the 3 o'clock → 9 o'clock (right to left) axis
aVF = the 12 o'clock → 6 o'clock (top to bottom) axis

If BOTH Lead I and aVF are UPRIGHT → the vector is pointing DOWN and to the LEFT → NORMAL.
If Lead I is UP and aVF is DOWN → the vector is pointing UP and to the LEFT → LEFT AXIS DEVIATION.

Memory tool: "Left when aVF goes up, Lead I goes up = normal. Like two thumbs up."

Step 4: R Wave Progression Across the Precordial Leads

R wave progression refers to the change in R wave amplitude as you move from V1 (rightmost precordial lead) to V6 (leftmost). In a normal heart:

- **V1**: The QRS is primarily negative — a small R wave followed by a deep S wave (rS pattern). This occurs because the major electrical forces move AWAY from V1 (toward the left ventricle).
- **V2**: Still predominantly negative, slightly larger R wave than V1.
- **V3**: The R and S waves begin to equalize.
- **V4**: The R wave becomes dominant — usually the tallest lead in the progression.
- **V5**: Similar to or slightly less than V4.
- **V6**: Predominantly positive R wave, small or absent S wave.

The transition zone: The point where the R wave becomes equal to the S wave (R = S, creating a biphasic complex). Normally this occurs between V3 and V4. If the transition is "early" (before V3), the large R wave in V1/V2 may indicate RVH, posterior MI, or RBBB. If the transition is "late" (V5 or V6), this is **poor R wave progression** — the R waves fail to grow normally across the precordial leads.

Causes of poor R wave progression:

- Anterior wall MI (loss of anterior QRS forces = loss of R waves)
- LBBB (abnormal septal activation)
- Left ventricular hypertrophy
- Precordial electrode placement errors (most common cause in clinical practice — always confirm placement before attributing poor progression to pathology)

Step 5: Ventricular Hypertrophy

Increased workload on a ventricle (due to pressure overload or volume overload) causes the myocardium of that ventricle to hypertrophy — the muscle cells enlarge. More muscle = more electrical voltage generated during depolarization. This increased voltage is visible as increased amplitude (height) of ECG waveforms in leads facing that ventricle.

Left Ventricular Hypertrophy (LVH)

Common causes: Long-standing hypertension (the most common cause), aortic stenosis, hypertrophic cardiomyopathy, aortic regurgitation.

ECG criteria (multiple criteria exist; the Sokolow-Lyon criterion is most widely used):

- **Sokolow-Lyon**: S wave depth in V1 + R wave height in V5 or V6 ≥ 35 mm (3.5 mV). This is the most commonly used voltage criterion.
- **Cornell criterion**: R wave in aVL + S wave in V3 > 28 mm in men, > 20 mm in women.
- **Limb lead criterion**: R wave in aVL ≥ 11 mm.
- **Repolarization changes** (LVH "strain" pattern): ST depression and T wave inversion in lateral leads (I, aVL, V5, V6) — the depolarization-repolarization mismatch of the hypertrophied left ventricle.

Important Limitation of LVH Voltage Criteria

ECG voltage criteria for LVH are NEITHER sensitive nor specific:
• Sensitivity: Only 50–60% of patients with echocardiographically confirmed LVH meet voltage criteria on ECG.
• Specificity: Young athletic individuals with large QRS voltages may meet LVH criteria without true hypertrophy.

Factors affecting ECG voltage: body habitus (thin patients have higher voltage from proximity of heart to chest wall), age (voltage decreases with age), gender.

Echocardiography is the gold standard for diagnosing LVH. ECG findings support — but cannot confirm or exclude — LVH.

Right Ventricular Hypertrophy (RVH)

Common causes: Pulmonary hypertension, chronic obstructive pulmonary disease (COPD), pulmonary embolism (acute right heart strain), pulmonic stenosis, atrial septal defect (right heart volume overload).

ECG criteria:

- Dominant R wave in V1 (R > S in V1) — the right ventricle's increased mass produces a larger rightward vector
- Right axis deviation (axis > +90°)
- Deep S waves in V5, V6
- Right ventricular "strain" pattern: ST depression and T wave inversion in V1–V3 (and sometimes V4)
- P pulmonale: peaked, narrow P waves in Lead II > 2.5 mm (from right atrial enlargement)

Step 6: ST Segment and T Wave Analysis — Ischemia, Injury, and Infarction

The final and often most clinically urgent step in 12-lead interpretation is evaluating the ST segments and T waves for evidence of myocardial ischemia, injury, or infarction. This is the step that will identify STEMIs and save lives.

Measuring the ST Segment

The ST segment is measured at the **J point** — the junction between the end of the QRS complex and the beginning of the ST segment. ST elevation or depression is measured as the vertical distance between the J point and the isoelectric baseline.

Establishing the isoelectric baseline: The most reliable baseline reference point is the **TP segment** — the flat line between the end of the T wave of one beat and the beginning of the P wave of the next. This represents the period of no cardiac electrical activity and is the true "zero" of the ECG. Measure ST elevation/depression from this level.

ST Finding	Pattern Description
ST Elevation (upward)	Convex ("tombstone") upward elevation
ST Elevation (concave)	Concave ("saddle-shaped") upward elevation
ST Depression (horizontal)	Flat downsloping below baseline
ST Depression (downsloping)	ST dips below then continues dropping
ST Depression (upsloping)	Below baseline at J point, rises toward baseline
ST Depression in V1-V3	Mirror-image depression (reciprocal to posterior elevation)

T Wave Abnormalities

T waves are highly sensitive but non-specific indicators of myocardial abnormality. Almost any cardiac or non-cardiac condition can alter T wave morphology.

T Wave Finding	Description
Peaked, tall, symmetric	Narrow peak, tall, symmetric shape

Inverted (negative)	T wave below baseline
Flat	Barely visible, near baseline
Biphasic	Both positive and negative portions
Tall asymmetric	Gradual upstroke, steep downstroke

Wellens Syndrome — A Pattern That Must Not Be Missed

Wellens Syndrome: A Pre-Infarction Warning

Wellens syndrome describes a specific T wave pattern in leads V2-V3 that indicates critical (often 90%+) stenosis of the proximal left anterior descending artery (LAD). It typically occurs in patients who have had recent chest pain that resolved — but the LAD is on the verge of complete occlusion.

Two patterns:
TYPE A (less common): Biphasic T waves in V2-V3 — initially positive (small upward deflection), then deeply negative.
TYPE B (more common): Deeply symmetric T wave inversions in V2-V3 (and sometimes V4).

CRITICAL: In Wellens syndrome, the ECG may be recorded DURING A PAIN-FREE INTERVAL. The patient looks and feels well. But without urgent intervention (cardiac catheterization), they are at very high risk for massive anterior STEMI in the near future.

Do NOT miss this pattern. Any deep symmetric T wave inversions in V2-V3 in a patient with recent chest pain should be reported immediately.

5.4 Reading an ECG Strip: Worked Examples

The following worked examples apply the Five-Step and Six-Step methods to specific clinical scenarios. In clinical practice, you will not have a step-by-step guide next to you — but the internal monologue you develop through repeated practice will replicate these steps automatically.

Worked Example 1: Reading a Rhythm Strip in Lead II

Scenario: A 52-year-old man with chest pain. Lead II rhythm strip, 25 mm/sec, standard calibration.

Sample Strip Description — Normal Sinus Rhythm

```
Step 1 — RATE:
Identify an R wave on a thick line. Count large boxes to next R wave.
Example: 4 large boxes between R waves → 300 ÷ 4 = 75 bpm → NORMAL rate.

Step 2 — RHYTHM:
Mark 5 consecutive R wave peaks. Walk the calipers across the strip.
All R-R intervals equal → REGULAR rhythm.

Step 3 — P WAVES:
Small, rounded positive deflection before each QRS. All look identical.
Upright in Lead II. One P wave before every QRS. → P WAVES NORMAL.

Step 4 — PR INTERVAL:
Count small boxes from start of P to start of QRS.
Example: 4 small boxes × 0.04 = 0.16 sec → NORMAL PR (within 0.12-0.20).

Step 5 — QRS:
Count small boxes from start to end of QRS.
Example: 2 small boxes × 0.04 = 0.08 sec → NARROW QRS (< 0.12).

INTERPRETATION: NORMAL SINUS RHYTHM at 75 bpm.
```

Worked Example 2: Identifying Atrial Fibrillation

Scenario: A 67-year-old woman presents with palpitations. Lead II rhythm strip.

Sample Strip Analysis — Atrial Fibrillation

```
Step 1 — RATE:
QRS complexes are present but not at regular intervals.
Use 6-second method: count QRS complexes in 6 seconds.
Example: 9 complexes in 6 seconds × 10 = 90 bpm average.
Note: rate varies between individual beats.

Step 2 — RHYTHM:
Mark R waves and walk calipers across strip.
R-R intervals vary COMPLETELY randomly — no two the same.
→ IRREGULARLY IRREGULAR rhythm. This is the hallmark of A-Fib.

Step 3 — P WAVES:
No distinct P waves visible. Instead: chaotic, low-amplitude undulation
of the baseline — fibrillatory (f) waves at ~400–600 per minute.
→ NO TRUE P WAVES.
```

```
  Step 4 — PR INTERVAL:
  Cannot measure — no true P waves.

  Step 5 — QRS:
  QRS complexes are narrow (< 0.12 sec). Identical in morphology.
  → NARROW QRS (ventricular conduction normal).

  INTERPRETATION: ATRIAL FIBRILLATION with ventricular rate ~90 bpm.
```

Worked Example 3: Identifying Second-Degree AV Block Type I (Wenckebach)

Scenario: A 74-year-old man with an inferior MI. Rhythm shows grouped beating pattern.

Sample Strip Analysis — Wenckebach (2° AV Block Type I)

```
  Step 1 — RATE:
  Ventricular rate slightly below normal due to some dropped beats.
  Use 6-second method → approximately 50 bpm.

  Step 2 — RHYTHM:
  REGULARLY IRREGULAR. QRS complexes appear in groups, each group
  followed by a pause. Then the grouping pattern repeats.
  P-P intervals are regular (SA node fires consistently).

  Step 3 — P WAVES:
  MORE P WAVES THAN QRS COMPLEXES. Some P waves are not followed
  by a QRS. All P waves look identical and upright in Lead II.

  Step 4 — PR INTERVAL:
  PROGRESSIVE LENGTHENING: measure PR intervals across a group.
  Beat 1: 0.16 sec → Beat 2: 0.20 sec → Beat 3: 0.24 sec → Beat 4: P wave only, no
QRS.
  Then: Beat 1 of next group: 0.16 sec (PR interval RESETS to shortest).
  → PROGRESSIVELY LENGTHENING PR with periodic dropped beats.

  Step 5 — QRS:
  All QRS complexes that do conduct are NARROW (< 0.12 sec).

  INTERPRETATION: SECOND-DEGREE AV BLOCK, TYPE I (WENCKEBACH/MOBITZ I).
```

5.5 The ECT's Verbal ECG Report: Professional Communication

As an ECT, your job is not only to acquire and preliminarily interpret the ECG, but to communicate your findings clearly, professionally, and urgently (when warranted) to the interpreting physician or senior clinician. The following reporting framework ensures complete, accurate, and actionable communication.

The Standard ECG Verbal Report Format

When reporting any ECG finding verbally, use the following structured format:

✓ Verbal ECG Report Template

1. IDENTIFY THE PATIENT: "I am reporting the ECG for [Patient Name], [Age], in [Room/Location]."
2.
3. RHYTHM IDENTIFICATION: "The rhythm appears to be [rhythm name] at approximately [rate] bpm."
4.
5. SUPPORTING FINDINGS: "[The rhythm is regular/irregular.] P waves [are/are not] present. The PR interval is [normal/prolonged/shortened/variable]. The QRS is [narrow/wide]."
6.
7. ST/ISCHEMIA FINDINGS: "The ST segments show [no changes / elevation of X mm in leads Y / depression of X mm in leads Y]. [There are / are not] reciprocal changes."
8.
9. CLINICAL CORRELATION: "The patient [is/is not] symptomatic. Blood pressure is [value]. The patient [has/does not have] chest pain."
10.
11. REQUEST FOR ACTION: "I am requesting [immediate physician evaluation / review at your earliest convenience / cath lab notification per protocol]."

Worked Verbal Report Examples

Example 1 — Routine NSR report:

"This is the ECG for Mr. Johnson, 58 years old in Room 412. The rhythm appears to be normal sinus rhythm at approximately 78 bpm. The rhythm is regular. P waves are present and upright in Lead II. PR interval is 0.16 seconds. QRS is narrow. ST segments appear normal with no significant elevation or depression. The patient is currently asymptomatic and hemodynamically stable."

Example 2 — Urgent STEMI report:

"Dr. Williams, I am calling about Ms. Martinez, 62 years old, in the ED. She presented with acute chest pain. Her 12-lead ECG shows 3 millimeters of ST elevation in leads II, III, and aVF, with reciprocal ST depression in leads I and aVL. This pattern is consistent with an inferior STEMI. Heart rate is 58 bpm in sinus rhythm. Blood pressure is 86 over 52. I have already obtained right-sided leads — V4R shows 1.5 mm of ST elevation. I am requesting your immediate evaluation for cath lab activation."

Example 3 — V-fib/cardiac arrest report:

"Code team — I have ventricular fibrillation on the monitor. The patient is unresponsive with no palpable pulse. CPR is in progress. The defibrillator is being applied now."

Chapter 5 Review Questions

1. Using the 300-method, you find 5 large boxes between consecutive R wave peaks. What is the heart rate?

a) 150 bpm
b) 100 bpm
c) 75 bpm
d) 60 bpm

► **Correct Answer:** d) 60 bpm
Rationale: *300 ÷ 5 = 60 bpm. The 300-method: 1 box = 300, 2 = 150, 3 = 100, 4 = 75, 5 = 60, 6 = 50. At exactly 60 bpm, consecutive R waves are 5 large boxes apart (0.20 sec × 5 = 1.00 sec between beats = 60 beats per minute).*

2. A rhythm strip shows grouped beating — 3 conducted beats followed by a pause, then 3 conducted beats followed by a pause, repeating. The PR interval measures 0.16 sec on the first beat, 0.20 sec on the second beat, and 0.26 sec on the third beat before the dropped beat. This pattern is MOST consistent with:

a) Second-degree AV block, Type II (Mobitz II)
b) Third-degree (complete) AV block
c) Second-degree AV block, Type I (Wenckebach)
d) First-degree AV block

► **Correct Answer:** c) Second-degree AV block, Type I (Wenckebach)
Rationale: *The classic Wenckebach pattern: progressively lengthening PR interval until one P wave is completely blocked (no QRS follows), then the PR resets to its shortest value and the cycle*

repeats. The grouped beating pattern ("footprints of Wenckebach") is characteristic. Mobitz II has a CONSTANT PR before the sudden dropped beat.

3. In the 12-lead ECG, which two leads are used for the quickest screening of electrical axis?

a) Leads I and II
b) Leads I and aVF
c) Leads II and III
d) Leads aVR and aVF

► **Correct Answer:** b) Leads I and aVF

Rationale: *Leads I and aVF are perpendicular to each other in the frontal plane and form the axes of a compass — Lead I (horizontal, right to left) and aVF (vertical, top to bottom). Both upright = normal axis. Lead I up, aVF down = left axis deviation. Lead I down, aVF up = right axis deviation.*

4. The Sokolow-Lyon criterion for left ventricular hypertrophy (LVH) measures:

a) R wave in aVL alone
b) S wave in V1 plus R wave in V5 or V6
c) R wave in V4 minus S wave in V2
d) Total QRS amplitude across all 12 leads

► **Correct Answer:** b) S wave in V1 plus R wave in V5 or V6

Rationale: *The Sokolow-Lyon LVH criterion: S wave depth in V1 + R wave height in V5 or V6 ≥ 35 mm (≥ 3.5 mV). This reflects the increased voltage generated by the hypertrophied left ventricle — deep S in V1 (moving away from the enlarged LV) and tall R in V5/V6 (moving toward it).*

5. Wellens syndrome refers to a pattern of T wave changes in V2-V3 that indicates:

a) Benign early repolarization in a young athlete
b) Critical stenosis of the proximal LAD artery — high risk for imminent anterior STEMI
c) Right ventricular hypertrophy secondary to pulmonary embolism
d) Posterior wall myocardial infarction

► **Correct Answer:** b) Critical stenosis of the proximal LAD artery — high risk for imminent anterior STEMI

Rationale: *Wellens syndrome: deeply inverted or biphasic T waves in V2-V3 (and sometimes V3-V4) indicating critical proximal LAD stenosis. This pattern typically occurs when the patient is PAIN-FREE, creating a dangerous false sense of reassurance. Without urgent catheterization, there is high risk for massive anterior STEMI. It must be recognized and reported immediately.*

CHAPTER 6: NORMAL SINUS RHYTHM — THE STANDARD OF COMPARISON

Learning Objectives

Upon completing this chapter, you will be able to:

50. State all the diagnostic criteria for Normal Sinus Rhythm (NSR).
51. Explain why NSR is used as the baseline for arrhythmia recognition.
52. Identify NSR on a rhythm strip using the Five-Step Method.
53. Describe Sinus Arrhythmia and explain why it is a normal variant.
54. Explain the concept of "sinus rhythm" versus other rhythm origins.

6.1 Normal Sinus Rhythm: The Gold Standard

Normal Sinus Rhythm (NSR) is the cardiac rhythm in which every heartbeat originates from the sinoatrial (SA) node and conducts through the normal pathways in the correct sequence: SA node → atria → AV node → Bundle of His → bundle branches → Purkinje fibers → ventricular myocardium. NSR is the standard against which all arrhythmias are measured — an arrhythmia, by definition, is any rhythm that deviates from NSR.

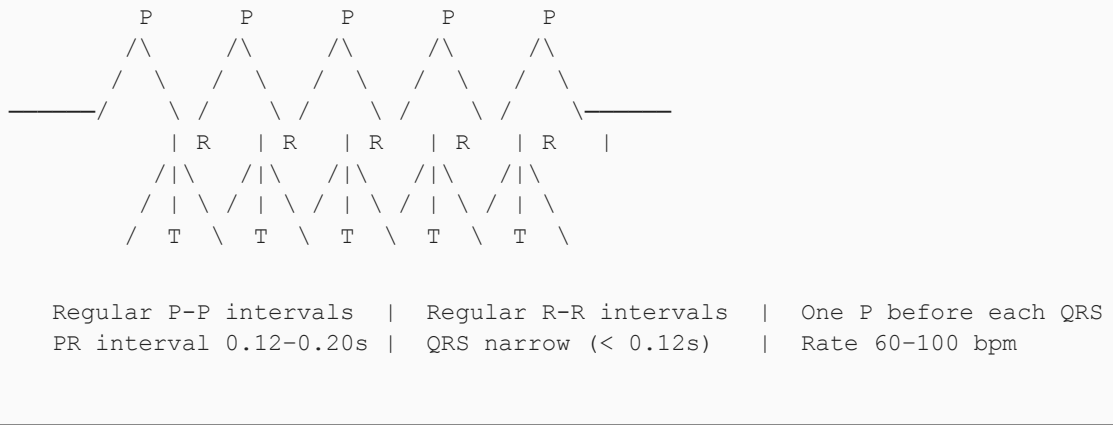
NORMAL SINUS RHYTHM (NSR)	
Rate	60–100 beats per minute
Rhythm	Regular — R-R intervals vary < 0.04 sec (< 1 small box)
P Waves	Present; upright and uniform in Lead II; one P wave before every QRS; all identical in morphology
PR Interval	0.12–0.20 seconds (3–5 small boxes); constant from beat to beat
QRS Duration	< 0.12 seconds (< 3 small boxes) — narrow
Significance	Normal — the desired cardiac rhythm; no treatment needed

6.2 Applying the Five-Step Method to NSR

Let's apply the Five-Step Method systematically to confirm NSR:

- **Step 1 — Rate**: Count the R-R interval. For NSR, the rate is 60–100 bpm. On a typical strip, you should see 3–5 large boxes between R waves (300/5 = 60 bpm to 300/3 = 100 bpm).
- **Step 2 — Rhythm**: Measure multiple R-R intervals with calipers or by marking on paper. In NSR, all R-R intervals should be equal or vary by no more than 1 small box (0.04 sec). If the intervals are perfectly equal, the rhythm is regular.
- **Step 3 — P Waves**: In NSR, there is exactly ONE P wave before each QRS, every P wave is upright (positive) in Lead II, and all P waves look identical (same size, same shape). If any of these conditions is not met, it is not NSR.
- **Step 4 — PR Interval**: Measure from the start of the P wave to the start of the QRS. NSR has a normal, consistent PR interval of 0.12 to 0.20 seconds. The interval should be the same for every beat.
- **Step 5 — QRS**: Narrow QRS (< 0.12 sec) — the ventricles are being activated by the normal Purkinje system. A wide QRS in an otherwise normal sinus rhythm would suggest bundle branch block (but it would still be called "sinus rhythm with bundle branch block" if all other criteria are met).

Figure 6.1 — Normal Sinus Rhythm Schematic



6.3 Sinus Arrhythmia: A Normal Irregular Rhythm

Sinus Arrhythmia is a rhythm that meets all criteria for sinus rhythm (same P waves, normal PR, narrow QRS) except that the rate varies — the R-R intervals vary by more than 0.04 seconds (1 small box). It is important to recognize sinus arrhythmia as a NORMAL, BENIGN variant that requires no treatment.

Sinus arrhythmia is caused by normal **respiratory variation** in vagal tone:

- **During INHALATION**: The lungs expand, activating stretch receptors that briefly inhibit vagal tone (parasympathetic brake released). The SA node speeds up slightly. Heart rate INCREASES.
- **During EXHALATION**: The lungs deflate, increasing vagal tone. The SA node slows down. Heart rate DECREASES.

This respiratory modulation of heart rate is called **respiratory sinus arrhythmia (RSA)**. It is most pronounced in children and young adults with high vagal tone, and tends to diminish with age. Paradoxically, a pronounced sinus arrhythmia is often a sign of good cardiovascular health and autonomic function (high heart rate variability).

SINUS ARRHYTHMIA	
Rate	60–100 bpm (varies with breathing)
Rhythm	IRREGULAR — R-R intervals vary cyclically with respiration (longer on expiration, shorter on inspiration)
P Waves	Present; upright in Lead II; one P before every QRS; all identical
PR Interval	Normal (0.12–0.20 sec); consistent
QRS Duration	Narrow (< 0.12 sec)
Significance	NORMAL — especially in children and young adults; no treatment needed

Chapter 6 Review Questions

1. A rhythm strip shows: rate 78 bpm, regular rhythm, upright P waves before every QRS, PR interval 0.16 sec, narrow QRS. This is BEST interpreted as:

a) Atrial fibrillation
b) Sinus tachycardia
c) Normal sinus rhythm
d) Junctional rhythm

► **Correct Answer:** c) Normal sinus rhythm

Rationale: *All Five-Step criteria are met: rate 60–100 bpm (78), regular rhythm, P waves present and preceding every QRS, PR 0.16 sec (within 0.12–0.20), narrow QRS. This is classic normal sinus rhythm.*

2. A young healthy athlete has a slightly irregular rhythm — the R-R intervals lengthen on expiration and shorten on inspiration, but all P waves are upright, the PR interval is constant, and QRS is narrow. This is MOST LIKELY:

a) Atrial fibrillation requiring treatment
b) Sinus arrhythmia — a normal variant
c) Second-degree AV block type I
d) Wandering atrial pacemaker

► **Correct Answer:** b) Sinus arrhythmia — a normal variant

Rationale: *Sinus arrhythmia is a normal respiratory variation in heart rate with otherwise normal sinus criteria (upright P waves, constant PR, narrow QRS). The correlation with breathing confirms it is respiratory sinus arrhythmia, a common and healthy finding especially in young athletes.*

CHAPTER 7: COMPREHENSIVE ARRHYTHMIA RECOGNITION AND CLINICAL MANAGEMENT

Learning Objectives

Upon completing this chapter, you will be able to:

55. Describe the mechanism, causes, and hemodynamic consequences of each major cardiac arrhythmia.
56. Apply the Five-Step Method to identify each arrhythmia on a rhythm strip.
57. List the defining ECG criteria for every arrhythmia covered in this chapter.
58. Differentiate between clinically similar arrhythmias (e.g., V-tach vs. SVT with aberrancy, Mobitz I vs. Mobitz II).
59. Identify the clinical presentation associated with each arrhythmia, including what the patient looks and feels like.
60. Understand the initial treatment priorities for each arrhythmia category.
61. Use professional verbal reporting language to communicate arrhythmia findings to the clinical team.
62. Recognize arrhythmias that require IMMEDIATE emergency intervention versus those that can be monitored.

This chapter forms the clinical core of your ECG education. Understanding arrhythmias is not simply an academic exercise — it is the skill that will determine whether patients under your care receive timely intervention or experience avoidable deterioration. Every arrhythmia you recognize is an opportunity to potentially save a life.

Each rhythm is presented using a consistent format: mechanism and origin, common causes, hemodynamic impact, definitive ECG criteria, step-by-step strip analysis guide, clinical presentation, differential diagnosis, initial management, a clinical vignette, and reporting language. Study each section completely before moving to the next.

7.1 Normal Sinus Rhythm and Sinus Variants

Normal Sinus Rhythm (NSR)

NORMAL SINUS RHYTHM	
Rate	60–100 bpm

Rhythm	Regular (R-R intervals may vary by ≤ 0.08 sec = 2 small boxes)
P Waves	Present, upright in Lead II, uniform, one before every QRS
PR Interval	0.12–0.20 sec, constant beat-to-beat
QRS Duration	< 0.12 sec (narrow)
ST Segment	Isoelectric — no elevation or depression

Normal sinus rhythm is the benchmark against which all arrhythmias are measured. Every component of the ECG cycle is normal: the SA node generates impulses at 60–100 per minute, conduction proceeds normally through the AV node and bundle branches, and the ventricles are activated via the Purkinje system producing narrow QRS complexes.

Sinus Tachycardia

SINUS TACHYCARDIA	
Rate	> 100 bpm (typically 100–160 bpm at rest; up to 200 bpm with maximum exertion)
Rhythm	Regular
P Waves	Present, upright in Lead II, one before every QRS; may be merged into preceding T wave at very fast rates
PR Interval	0.12–0.20 sec (may shorten slightly at very fast rates due to catecholamine effect on AV node)
QRS	Narrow (< 0.12 sec), unless aberrant conduction present

Mechanism: The SA node increases its rate of spontaneous depolarization in response to physiologic demands or pathologic stressors. This is a NORMAL physiologic response — the SA node is functioning correctly; it is simply being driven faster than 100 bpm by external influences.

Causes — the PATHOLOGIC acronym:

- **P**ain, **A**nxiety, **F**ever, **E**xercise (physiologic causes)
- Hypovolemia/blood loss/dehydration (inadequate cardiac output → compensatory tachycardia)
- Hypoxia (the sinus node is extremely sensitive to oxygen deprivation)

- Anemia (reduced oxygen delivery)
- Thyrotoxicosis/hyperthyroidism
- Medications: epinephrine, atropine, dopamine, salbutamol, caffeine
- Pulmonary embolism (right heart strain, reflex tachycardia)
- Sepsis/infection (vasodilation → decreased preload → compensatory tachycardia)
- Cardiac tamponade (impaired filling → compensatory tachycardia)

Hemodynamic consequences: Sinus tachycardia itself is generally well-tolerated in patients with normal hearts. The problem is that tachycardia SHORTENS diastole — the filling phase of the cardiac cycle. At very fast rates (> 150 bpm), diastolic filling time becomes critically shortened, reducing stroke volume and potentially causing demand ischemia (the heart works harder while receiving less time for coronary perfusion). In patients with coronary artery disease, sinus tachycardia can precipitate angina or NSTEMI.

Critical Teaching Point: Sinus Tachycardia Is a SYMPTOM

Sinus tachycardia is almost NEVER the primary problem — it is the body's response to a primary problem.

You should NEVER treat sinus tachycardia by trying to slow the heart rate without first identifying and treating the underlying cause.

Examples:
• Patient with sepsis: heart rate 130 bpm → treating with beta-blockers to slow the HR would worsen hypotension
• Patient with massive pulmonary embolism: HR 140 → treating the HR instead of the PE could be fatal
• Patient with severe pain: HR 115 → HR will normalize when pain is treated

Your job: IDENTIFY sinus tachycardia, ASSESS the patient, IDENTIFY the cause, REPORT to the clinical team.

Clinical presentation: The patient may complain of palpitations, a racing heart, or "my heart is beating fast." At higher rates (> 130 bpm), patients may report chest tightness, lightheadedness, or shortness of breath. Depending on the underlying cause, the patient may appear anxious, pale, diaphoretic, feverish, or in pain.

Clinical Vignette: Sinus Tachycardia

A 34-year-old man is admitted following a motor vehicle accident with a known femur fracture. His heart rate on the monitor is 124 bpm, regular. Blood pressure is 88/58 mmHg. The rhythm strip shows upright P waves in Lead II, PR interval 0.16 sec, narrow QRS. The ECT recognizes this as sinus tachycardia and immediately reports: "Doctor, Mr. Davis is in sinus tachycardia at 124 bpm with a blood pressure of 88/58. Given his mechanism of injury, I am concerned about hemorrhage. Requesting immediate evaluation." The underlying cause — internal bleeding — is treated with IV fluids and surgical hemostasis. The heart rate normalizes as volume is restored.

Sinus Bradycardia

SINUS BRADYCARDIA	
Rate	< 60 bpm
Rhythm	Regular
P Waves	Present, upright in Lead II, uniform, one before every QRS
PR Interval	0.12–0.20 sec, constant
QRS	Narrow (< 0.12 sec)

Mechanism: The SA node depolarizes at a rate below 60 bpm. Like sinus tachycardia, this may be a normal physiologic variant (especially in trained athletes and during sleep) or a pathologic finding.

Causes:

- **Normal physiologic**: Athletic conditioning (well-trained hearts have high vagal tone); sleep; vasovagal response
- **Increased vagal tone**: Pain, vomiting, carotid sinus massage, bearing down (Valsalva), inferior MI (the RCA supplies the SA node in 60% of patients)
- **Medications**: Beta-blockers, calcium channel blockers (diltiazem, verapamil), digoxin, amiodarone, opioids
- **Metabolic**: Hypothyroidism, hypothermia (rate slows 6-8 bpm per 1°C decrease)
- **SA node disease (sick sinus syndrome)**: Degenerative disease of the SA node; most common in elderly patients

Hemodynamic consequences: The impact of sinus bradycardia on the patient depends entirely on the cardiac output equation: CO = Heart Rate × Stroke Volume. If stroke volume

increases proportionally (as in well-trained athletes), cardiac output is maintained and the patient is completely asymptomatic. If stroke volume CANNOT compensate — as in patients with poor ventricular function, acute MI, or medication overdose — cardiac output falls and the patient may develop hypotension, dizziness, syncope, altered mental status, or even cardiac arrest.

The critical question for sinus bradycardia: Is the patient SYMPTOMATIC?

	Asymptomatic Bradycardia
Heart rate	Any rate < 60
Blood pressure	Normal
Mental status	Alert and oriented
Symptoms	None
Action required	Assess and monitor, identify cause

Sinus Arrhythmia

SINUS ARRHYTHMIA	
Rate	60–100 bpm (usually; rate may be faster with tachycardic variant)
Rhythm	Irregularly irregular — but with a RESPIRATORY pattern (speeds up with inhalation, slows with exhalation)
P Waves	Present, upright in Lead II, uniform, one before every QRS
PR Interval	0.12–0.20 sec, may slightly vary
QRS	Narrow (< 0.12 sec)

Sinus arrhythmia is a NORMAL finding, particularly in children, young adults, and athletes. During inhalation, intrathoracic pressure decreases, venous return increases, and reflex sympathetic activation slightly increases the sinus rate. During exhalation, vagal tone increases and the rate slows. The key distinguishing feature is that the rate change is CYCLICAL and RESPIRATORY — it correlates directly with breathing.

Clinical significance: None. Sinus arrhythmia requires no treatment. It actually indicates a healthy, responsive autonomic nervous system.

How to confirm: Ask the patient to briefly hold their breath. During breath-holding, the R-R interval variation typically disappears and the rhythm becomes regular. This confirms the respiratory origin of the variation.

7.2 Atrial Arrhythmias

Premature Atrial Complexes (PACs)

PREMATURE ATRIAL COMPLEXES (PACS)	
Rate	Underlying rhythm rate (PACs are individual ectopic beats, not sustained rhythms)
Rhythm	IRREGULAR — the PAC occurs EARLIER than the next expected sinus beat
P Waves	The ectopic P wave (P') DIFFERS in morphology from sinus P waves — may be inverted, peaked, biphasic, or notched, depending on the site of ectopy in the atria
PR Interval	Usually 0.12–0.20 sec, but may be slightly shorter or longer than the underlying sinus PR
QRS	Usually narrow (< 0.12 sec) — ventricular conduction is typically normal. If the PAC conducts with aberrancy (reaches the bundle branches while they are still partially refractory), the QRS will be wide and may mimic a PVC.
Compensatory Pause	INCOMPLETE (non-compensatory): The SA node is usually RESET by the PAC, so the next sinus beat occurs earlier than it would if a PVC occurred.

Mechanism: An ectopic focus in the atrial tissue depolarizes prematurely — before the SA node fires. Because the impulse still travels through the normal AV node and conduction system, the resulting QRS is usually narrow and normal-appearing. The early beat disturbs the SA node's timing, resetting its cycle.

Causes: PACs are extremely common and can occur in completely healthy individuals. Triggers include caffeine, alcohol, tobacco, fatigue, stress, electrolyte imbalances (particularly hypokalemia and hypomagnesemia), stimulant medications, atrial enlargement, and cardiac disease. Frequent PACs (> 6–10 per minute) can be a harbinger of sustained atrial arrhythmias, especially atrial fibrillation.

Clinical significance: Isolated PACs in an otherwise healthy person require no specific treatment. Frequent PACs in a patient with structural heart disease may warrant further evaluation. Patients often feel PACs as a "skipped beat," "extra beat," or "flutter" in the chest.

Atrial Flutter

ATRIAL FLUTTER	
Atrial Rate	250–350 bpm (classic "typical" atrial flutter: 300 bpm)
Ventricular Rate	Depends on AV conduction ratio: 2:1 block (most common) = ~150 bpm; 3:1 = ~100 bpm; 4:1 = ~75 bpm
Rhythm	Ventricular rhythm is REGULAR (with fixed block) or REGULARLY IRREGULAR (with variable block)
P Waves	NO true P waves. Instead: characteristic SAWTOOTH FLUTTER WAVES (F waves) — negative, saw-toothed deflections at 300 bpm, most visible in II, III, aVF, and V1. Continuous, without an isoelectric baseline between them.
PR Interval	Cannot measure in traditional sense; the F-R relationship varies with the conduction ratio
QRS	Narrow (< 0.12 sec) unless aberrant conduction

Mechanism: Atrial flutter is a macro-reentrant tachycardia — a large circular electrical circuit in the right atrium (in "typical" or isthmus-dependent flutter) that continuously rotates at 300 times per minute. The AV node cannot conduct all 300 impulses per minute to the ventricles (it would be physiologically dangerous if it did), so it blocks a proportion of the flutter waves. The most common conduction ratio is 2:1, meaning for every 2 flutter waves, 1 is conducted to the ventricles — producing a ventricular rate of approximately 150 bpm.

The 150 bpm Rule in Atrial Flutter

Any patient with a regular tachycardia at approximately 150 bpm (140–160 bpm) should be suspected of having atrial flutter with 2:1 block until proven otherwise.

At 2:1 conduction, one flutter wave is hidden (obscured) within the QRS complex, making the flutter waves difficult to see without careful examination of the ST segment in inferior leads.

How to unmask flutter waves:

- Carotid sinus massage or adenosine temporarily increases AV block
- This slows the ventricular rate, making the flutter waves clearly visible
- The rhythm "reveals itself" — the sawtooth pattern becomes obvious

WARNING: If you give adenosine to what you think is SVT at 150 bpm, and it is actually atrial flutter, the adenosine will transiently slow the ventricular rate, reveal the flutter waves, then wear off — and the flutter will resume. This diagnostic maneuver can be both therapeutic and revealing.

Causes: Atrial flutter is almost always associated with structural heart disease or other predisposing conditions: coronary artery disease, hypertensive heart disease, cardiomyopathy, valvular heart disease, pericarditis, post-cardiac surgery, pulmonary embolism, COPD, thyrotoxicosis.

Hemodynamic consequences: At 2:1 conduction (ventricular rate ~150 bpm), patients frequently experience symptoms of decreased cardiac output: palpitations, dyspnea, lightheadedness, chest discomfort, and pre-syncope. Atrial flutter, like atrial fibrillation, carries a risk of atrial thrombus formation and stroke.

Clinical Vignette: Atrial Flutter

A 71-year-old woman with a history of hypertensive heart disease presents with 2 hours of palpitations and mild shortness of breath. Her BP is 104/62. The ECT acquires a 12-lead. Heart rate is 152 bpm, regular. In the inferior leads (II, III, aVF), a regular sawtooth pattern is visible at approximately 300 per minute with negative deflections below the isoelectric line. Two flutter waves can be counted for every QRS complex. The ECT reports: "Dr. Jones, Mrs. Thompson has what appears to be atrial flutter at approximately 150 bpm with 2:1 block. Blood pressure is 104/62. She is symptomatic with palpitations and mild dyspnea." The patient is prepared for rate control management and anticoagulation evaluation.

Atrial Fibrillation (A-Fib)

ATRIAL FIBRILLATION	
Atrial Rate	350–600 "fibrillatory impulses" per minute — so rapid and chaotic that coordinated atrial contraction is absent
Ventricular Rate	Variable. Uncontrolled A-Fib: 100–180 bpm. Rate-controlled A-Fib: 60–100 bpm. "Slow" A-Fib (with AV nodal disease or rate-controlling medications): < 60 bpm

Rhythm	IRREGULARLY IRREGULAR — this is the DEFINING, PATHOGNOMONIC feature. No two R-R intervals are equal. The rhythm is completely chaotic.
P Waves	ABSENT. No true P waves. Instead: chaotic fibrillatory (f) waves — irregular, low-amplitude undulations of the baseline. Best seen in V1 and II. The baseline appears to "shimmer" or undulate continuously.
PR Interval	Cannot be measured — no true P waves
QRS	Narrow (< 0.12 sec) in the absence of bundle branch block or aberrant conduction. QRS complexes are identical in morphology (they are the same ventricular activation each time — just arriving at random intervals).

Mechanism: Atrial fibrillation is caused by multiple, rapidly rotating micro-reentrant circuits ("rotors") throughout both atria. These circuits fire at 350–600 per minute, creating completely chaotic electrical activity. The atria quiver rather than contract in an organized fashion — "the bag of worms" description. The AV node acts as a gatekeeper, allowing only a fraction of these impulses to reach the ventricles, and which impulses are conducted is entirely random — hence the perfectly irregular ventricular response.

Causes: Atrial fibrillation is the most common sustained cardiac arrhythmia in clinical practice (affecting approximately 6 million Americans). Risk factors and causes include:

- Age (risk doubles with each decade after 55)
- Hypertension (most common modifiable risk factor)
- Coronary artery disease and prior myocardial infarction
- Heart failure (dilated left atrium from elevated filling pressures)
- Valvular heart disease (especially mitral stenosis and mitral regurgitation — both cause left atrial enlargement)
- Thyrotoxicosis (thyroid hormone is directly arrhythmogenic to the atria)
- Pulmonary embolism (acute right heart strain and pressure elevation)
- Obstructive sleep apnea
- Alcohol ("holiday heart syndrome" — even acute intoxication can trigger A-Fib)
- Electrolyte abnormalities (hypokalemia, hypomagnesemia)

Hemodynamic consequences — two major risks:

1. Loss of atrial kick: Normal atrial contraction contributes approximately 20–30% of ventricular filling. In A-Fib, the atria quiver rather than contract, eliminating this "atrial kick." In

patients with reduced ventricular function or diastolic dysfunction (stiff ventricles), this loss can reduce cardiac output by up to 30%, precipitating heart failure decompensation.

2. Thromboembolic risk: Because the atria are not contracting effectively, blood pools — particularly in the left atrial appendage (a small pouch-like structure that is particularly vulnerable to thrombus formation). Clot formation in the left atrial appendage can then embolize to the brain, causing ischemic stroke. The annual stroke risk in untreated A-Fib ranges from 1–15% depending on additional risk factors (assessed by the CHA_2DS_2-VASc score).

A-Fib: The Stroke Connection

Atrial fibrillation is responsible for approximately 15–20% of all ischemic strokes — making it one of the most preventable causes of stroke.

The risk is highest in patients with:

- Age ≥ 65
- Prior stroke or TIA (transient ischemic attack)
- Congestive heart failure
- Hypertension
- Diabetes mellitus
- Female sex (additional risk factor)
- Vascular disease (MI, peripheral artery disease)

Anticoagulation (warfarin, direct oral anticoagulants) dramatically reduces stroke risk in high-risk A-Fib patients. When you identify a new A-Fib or report that a patient has A-Fib, you are directly supporting a decision process that may prevent that patient from having a devastating stroke.

Clinical Vignette: New-Onset Atrial Fibrillation

An 68-year-old man with a history of hypertension and type 2 diabetes is admitted for "heart racing and feeling weak" for the past 6 hours. The ECT acquires a 12-lead. Rate is irregular at approximately 108 bpm by 6-second counting method. No P waves are visible — only an irregular undulating baseline. R-R intervals are completely random. QRS is narrow. The ECT reports: "Doctor, Mr. Williams appears to be in atrial fibrillation with an uncontrolled ventricular rate of approximately 108 bpm. No P waves are visible. QRS is narrow. He reports palpitations and weakness for 6 hours. Blood pressure is 138/84." The physician evaluates and discusses rate control, rhythm control, and anticoagulation. The ECT notes that with the patient's history of hypertension and diabetes, his CHA_2DS_2-VASc score is at least 3 — placing him in the high stroke-risk category.

Supraventricular Tachycardia (SVT)

PAROXYSMAL SVT (PSVT / AVNRT / AVRT)	
Rate	150–250 bpm (typically 160–200 bpm)
Rhythm	REGULAR — extremely characteristic of SVT. The rate is fast and the rhythm is perfectly regular.
P Waves	Often NOT VISIBLE — buried within the QRS complex or in the ST segment immediately after the QRS. If visible, they are inverted (retrograde) in Lead II, III, and aVF.
PR Interval	If P waves are visible, they are very SHORT (because retrograde conduction is rapid and backward)
QRS	Narrow (< 0.12 sec) in most cases. Wide if aberrant conduction is present — creating the dangerous "wide-complex tachycardia" differential with V-tach.
Onset	Paroxysmal (sudden) onset and termination — a hallmark feature.

Mechanism: The most common form of SVT is AV nodal reentrant tachycardia (AVNRT), which accounts for approximately 60% of all SVT cases. A reentrant circuit forms within or immediately around the AV node — using two pathways with different conduction speeds and refractory periods. Once the circuit is established (typically triggered by a PAC), the circuit spins continuously at 160–200 per minute, simultaneously activating the ventricles (downward) and the atria (backward = retrograde). Because the atria and ventricles are activated nearly simultaneously, the retrograde P wave is buried within or immediately after the QRS complex.

The second most common form is AV reentrant tachycardia (AVRT) using an accessory pathway (as in Wolff-Parkinson-White syndrome). Less commonly, SVT may arise from an ectopic atrial focus (ectopic atrial tachycardia).

Causes: SVT can occur in patients with no structural heart disease. Common triggers include caffeine, alcohol, nicotine, stimulant medications, fatigue, stress, and electrolyte abnormalities. In patients with Wolff-Parkinson-White syndrome, the accessory pathway predisposes to AVRT.

Clinical presentation: SVT typically presents dramatically. The patient reports sudden onset of palpitations ("my heart just suddenly started pounding"), chest tightness, shortness of breath, lightheadedness, and occasionally near-syncope. Some patients have recurrent episodes and can predict them. The blood pressure may be maintained or decreased depending on the rate and the patient's underlying cardiac function. The rapid rate limits diastolic filling time and can precipitate chest pain in patients with coronary artery disease.

Clinical Vignette: SVT

A 28-year-old woman in the emergency department reports "my heart suddenly started racing while I was watching TV — I felt like I might pass out." She appears pale and anxious. HR is 192 bpm, BP 98/64, respirations 24. The monitor shows a perfectly regular narrow-complex tachycardia at 192 bpm. No P waves are visible — they appear to be buried in the QRS. The ECT reports: "Dr. Chen, Ms. Rivera is in SVT at 192 bpm with a blood pressure of 98/64. The rhythm is regular and narrow complex. No P waves visible." While the physician evaluates vagal maneuvers (Valsalva, carotid sinus massage), the ECT prepares adenosine as directed. The adenosine terminates the SVT and returns the patient to normal sinus rhythm.

7.3 Ventricular Arrhythmias

Ventricular arrhythmias arise from within the ventricular myocardium or the distal conduction system below the Bundle of His. Because the impulse does not use the normal Purkinje system, ventricular activation is SLOW and DISORGANIZED — producing the hallmark WIDE QRS complex (≥ 0.12 sec) with abnormal morphology. Ventricular arrhythmias range from benign isolated ectopic beats to immediately life-threatening rhythms requiring emergency defibrillation.

Premature Ventricular Complexes (PVCs)

PREMATURE VENTRICULAR COMPLEXES (PVCS)	
Rate	Underlying rhythm rate (PVCs are single ectopic beats, not a sustained rhythm)
Rhythm	The underlying rhythm is interrupted by an early beat (the PVC), creating an IRREGULAR rhythm
P Waves	The PVC beat has NO preceding P wave — it originates in the ventricles, bypassing the atria entirely. A retrograde P wave may occur after the QRS if conduction travels backward to the atria.
PR Interval	Not applicable to the PVC beat itself; the underlying sinus beats have a normal PR
QRS of PVC	WIDE (≥ 0.12 sec) and BIZARRE — abnormal morphology. The QRS of the PVC looks dramatically different from the normally conducted QRS complexes.
T Wave of PVC	The T wave is in the OPPOSITE direction from the QRS — called T wave discordance. If the QRS is predominantly negative (wide negative deflection), the T wave will be positive, and vice versa.

Compensatory Pause	FULL (complete) compensatory pause follows the PVC: the SA node was NOT reset by the PVC, so the next sinus beat occurs exactly when it would have if the PVC had not occurred. The total interval from the beat BEFORE the PVC to the beat AFTER is exactly TWICE the normal R-R interval.

Mechanism: A ventricular cell or group of cells depolarizes spontaneously before the SA node fires, producing an early, wide, bizarre complex. The abnormal morphology reflects the aberrant ventricular activation — the impulse must travel slowly through working myocardium (not Purkinje fibers) to reach all ventricular tissue.

Causes: PVCs are extremely common — found in up to 70% of healthy adults on 24-hour monitoring. In healthy individuals with no structural heart disease, they are almost always benign. However, in the context of cardiac disease, PVCs take on greater significance:

- Electrolyte disorders: hypokalemia, hypomagnesemia, hypocalcemia — MOST COMMON REVERSIBLE CAUSE
- Myocardial ischemia or acute MI (PVCs in the setting of acute MI require close monitoring)
- Heart failure (frequent PVCs indicate increased arrhythmia risk)
- Digoxin toxicity (frequently causes PVCs, often bigeminal)
- Stimulants: caffeine, cocaine, methamphetamine
- Hypoxia
- Medications that prolong the QT interval (predispose to triggered PVCs from R-on-T phenomenon)

PVC Patterns — Clinical Significance

PVC Pattern	ECG Appearance
Isolated PVCs	Single PVC in an otherwise normal rhythm; occasional
Bigeminy	Alternating sinus beat and PVC (every other beat is a PVC)
Trigeminy	Every third beat is a PVC (sinus-sinus-PVC repeating)
Couplet	Two consecutive PVCs
Triplet / Salvo	Three consecutive PVCs (short run of V-tach)
R-on-T phenomenon	PVC falls on the T wave of the preceding beat

Multifocal PVCs	PVCs of different morphologies — some point up, some down; all wide and bizarre but varying in shape

R-on-T Phenomenon: The Most Dangerous PVC Pattern

When a PVC falls during the T wave of the preceding beat — specifically the DOWNSLOPE of the T wave (the "vulnerable period" of ventricular repolarization) — it can trigger ventricular fibrillation.

Why the T wave downslope is dangerous:
At this moment, some ventricular muscle cells have repolarized and are ready to fire again, while others are still in the relative refractory period. This heterogeneity of repolarization means a premature stimulus can cause SOME areas to fire while others remain refractory, creating multiple, chaotic re-entrant circuits — V-Fib.

CLINICAL ACTION: If you observe an R-on-T PVC, notify the physician IMMEDIATELY.
This is a high-risk finding that requires urgent assessment and management.

Ventricular Tachycardia (V-Tach)

MONOMORPHIC VENTRICULAR TACHYCARDIA	
Rate	> 100 bpm (typically 100–250 bpm; most commonly 140–200 bpm). By definition, "V-tach" is ≥ 3 consecutive PVCs at a rate ≥ 100 bpm.
Duration	Non-sustained V-tach (NSVT): < 30 seconds, self-terminating. Sustained V-tach: ≥ 30 seconds, or requiring intervention before 30 seconds.
Rhythm	REGULAR — this is a key distinguishing feature from V-Fib
P Waves	May be present (the SA node continues firing independently) but are DISSOCIATED from the QRS complexes (AV dissociation). P waves are usually buried in the wide QRS complexes or barely visible at irregular intervals.
QRS	WIDE (≥ 0.12 sec) and BIZARRE morphology. All QRS complexes look identical to each other (monomorphic = same shape = same ectopic focus each time).
AV Dissociation	CRITICAL FINDING: The atria and ventricles are beating INDEPENDENTLY. P waves march at the sinus rate (~60–100). QRS complexes march at the faster V-tach rate (~140–200). When the sinus impulse accidentally occurs at exactly the right moment, it can conduct to the ventricles and produce a normal-looking QRS in the middle of the V-tach (FUSION BEAT or CAPTURE BEAT) — this is PATHOGNOMONIC for V-tach.

AV Dissociation, Fusion Beats, and Capture Beats — Definitive Proof of V-Tach:

- **AV dissociation**: The ventricles and atria are beating independently — the most important diagnostic criterion separating V-tach from SVT with aberrancy.
- **Fusion beat**: A QRS complex that is "in between" normal and wide — the sinus impulse and the ventricular impulse reach the ventricles at the same moment and FUSE into a hybrid QRS. This proves the wide complex rhythm is ventricular in origin.
- **Capture beat**: Occasionally during V-tach, the sinus impulse "captures" the ventricles at exactly the right time, producing a completely NORMAL NARROW QRS in the middle of the wide-complex tachycardia. This is PATHOGNOMONIC (100% specific) for V-tach.

Hemodynamic consequences: V-tach is a LIFE-THREATENING emergency because:

85. The rapid rate severely impairs diastolic filling and cardiac output
86. The loss of coordinated AV timing (the atrial kick) further reduces cardiac output by 20–30%
87. Many patients with V-tach have underlying structural heart disease (reduced ejection fraction), making their hearts particularly vulnerable to rapid rates
88. V-tach can degenerate into V-Fib at any time

Clinical presentation spectrum:

Category	Patient Appearance
Stable V-tach	Alert, awake, complaining of palpitations, chest tightness, lightheadedness. Able to speak in full sentences.
Unstable V-tach	Pale, diaphoretic, confused, very weak. Unable to speak clearly. May have chest pain.
Pulseless V-tach	Unresponsive. No palpable pulse. Not breathing normally.

The Rule for Wide-Complex Tachycardia: ASSUME V-TACH

Any wide-complex tachycardia (rate > 100 bpm, QRS ≥ 0.12 sec) must be treated as V-tach until definitively proven otherwise.

Why? Because the consequences of UNDER-treating V-Tach (treating it as SVT with aberrancy) can be FATAL:

- Verapamil and diltiazem given to V-tach patients can cause cardiovascular collapse
- Adenosine is generally safer to try but may still accelerate V-tach in some cases

The "three Cs" that suggest V-tach over SVT-with-aberrancy:
CONCORDANCE: All QRS complexes in precordial leads point the SAME direction (all positive or all negative)
CAPTURE/FUSION BEATS: Definitive evidence of V-tach
CAPTURE of AV dissociation on the ECG: P waves marching independently

When you are unsure: call for help, support the patient hemodynamically, prepare for cardioversion.

Clinical Vignette: V-Tach

A 68-year-old man with a history of inferior MI 4 years ago is on a telemetry floor. The monitor alarms. The ECT rushes to assess: on the monitor, there is a wide-complex tachycardia at approximately 170 bpm. QRS complexes are wide and bizarre at about 0.16 sec. The rhythm is regular. The patient is pale and diaphoretic, reporting chest pain. BP by cuff is 76/48. The ECT immediately activates the emergency response: "CODE — Room 412. Mr. Brown has a wide-complex tachycardia at 170 bpm. He is hemodynamically unstable with a BP of 76/48 and chest pain. I believe this is ventricular tachycardia." While the team arrives, the ECT applies defibrillator pads, prepares for synchronized cardioversion, and ensures IV access is established.

Torsades de Pointes (TdP)

TORSADES DE POINTES	
Rate	200–300 bpm
Rhythm	Irregular — the rhythm of TdP is chaotic and shifting
QRS	Wide, bizarre, continuously CHANGING in amplitude and axis — the QRS complexes appear to twist around the isoelectric baseline. The name "Torsades de Pointes" is French for "twisting of the points" — referring to this characteristic twisting visual pattern.
Preceding QTc	CRITICAL: TdP is ALWAYS preceded by a prolonged QT/QTc interval. The long QT creates the vulnerable window that allows TdP to be triggered.
Onset	Frequently triggered by a short-long-short sequence (a PVC followed by a long compensatory pause, then another early beat = the R-on-T mechanism in the setting of QT prolongation)

Mechanism: TdP is a form of polymorphic V-tach that occurs specifically in the context of a prolonged QT interval. The prolonged QT reflects delayed ventricular repolarization — different areas of the ventricle repolarize at different times, creating a LARGE vulnerable window during which an early impulse (a PVC) can initiate multiple circulating re-entrant circuits, producing the characteristic twisting pattern.

Causes of prolonged QT (and therefore TdP risk):

- **Medications** (most common cause): antiarrhythmics (quinidine, sotalol, amiodarone), antibiotics (azithromycin, fluoroquinolones), antipsychotics (haloperidol, quetiapine), antihistamines, methadone — resource: CredibleMeds.org maintains a continuously updated list
- **Electrolyte abnormalities**: Hypokalemia, hypomagnesemia, hypocalcemia
- **Congenital long QT syndrome**: Romano-Ward syndrome, Jervell and Lange-Nielsen syndrome — may cause sudden cardiac death in young, otherwise healthy individuals
- **Bradycardia**: Slower heart rates lengthen the QT interval

Clinical significance: TdP is immediately life-threatening. It typically occurs in self-terminating bursts (10–60 beats) that cause palpitations, presyncope, or syncope. However, TdP can degenerate into V-Fib and cardiac arrest. Treatment centers on REMOVING THE CAUSE (stopping the offending medication, correcting electrolytes) and IV MAGNESIUM SULFATE (which shortens the QT and terminates TdP). Overdrive pacing may be used to suppress TdP by speeding the heart rate (which shortens the QT interval).

Ventricular Fibrillation (V-Fib)

VENTRICULAR FIBRILLATION	
Rate	Cannot be counted — there are no organized beats. Coarse V-Fib: large amplitude, irregular chaotic waveforms. Fine V-Fib: low amplitude, may resemble asystole; associated with worse outcomes.
Rhythm	COMPLETELY CHAOTIC — no regularity whatsoever
P Waves	None
QRS	None — there are no organized QRS complexes, only continuous chaotic electrical activity
T Waves	None — cannot be identified

Mechanism: Ventricular fibrillation is the result of multiple simultaneous re-entrant circuits throughout the ventricular myocardium, creating completely disorganized electrical activity. Without coordinated depolarization, the ventricles cannot contract meaningfully — they quiver (fibrillate) and generate NO cardiac output. The patient is in CARDIAC ARREST.

V-Fib = Cardiac Arrest. Immediate Action Required.

Ventricular fibrillation is the MOST COMMON initial rhythm in adult sudden cardiac arrest.

When V-Fib appears on the monitor:

1. IMMEDIATELY verify: Is the patient unresponsive? Is there a pulse? (A motion artifact can mimic V-Fib on the monitor — ALWAYS CHECK THE PATIENT, not just the monitor.)

2. If unresponsive with no pulse: CALL A CODE. Begin CPR immediately.

3. Defibrillation is the ONLY effective treatment for V-Fib. For every minute without defibrillation, survival decreases by 7–10%. TIME TO DEFIBRILLATION IS THE MOST IMPORTANT DETERMINANT OF SURVIVAL.

4. The AED or manual defibrillator delivers UNSYNCHRONIZED (asynchronous) shock to V-Fib (not synchronized, because there is no organized QRS to synchronize to).

5. Continue CPR immediately after shock delivery — do not pause to check for a rhythm until after 2 minutes of high-quality CPR.

Clinical presentation: The patient with V-Fib is UNRESPONSIVE, APNEIC (not breathing normally), and PULSELESS. This is sudden cardiac death unless immediately reversed by defibrillation and CPR.

Causes: V-Fib is most commonly precipitated by acute myocardial infarction (especially within the first hour — the "zone of danger"), but can also be caused by severe electrolyte disorders, hypothermia, drowning, severe drug toxicity, and progression from V-tach.

Asystole

ASYSTOLE	
Rate	Zero — no electrical activity
Rhythm	None
P Waves	Absent (may rarely see occasional P waves = "P-wave asystole" or "atrial standstill" — atria still firing but no ventricular activity)
QRS	Absent

ECG Appearance	Flat line — isoelectric baseline with no waveforms. Confirm in TWO leads before declaring asystole.

Mechanism: Complete absence of cardiac electrical activity. The heart has no functioning pacemaker at any level. This represents either the END STAGE of another cardiac arrest rhythm (V-Fib that has failed to be defibrillated and has deteriorated into asystole) or, less commonly, a primary failure of all cardiac pacemaker activity.

The Most Important Rule About Asystole: Confirm in Two Leads

Fine ventricular fibrillation can look EXACTLY like asystole in a single lead.

V-Fib is TREATABLE with defibrillation. Asystole is NOT treatable with defibrillation.

If you defibrillate fine V-Fib while thinking it is asystole: you may shock the patient into asystole when they had a potentially shockable rhythm.

The protocol: If you see a flat line on the monitor:
1. Confirm the patient is truly unresponsive (rule out artifact).
2. Check lead connections and electrode contact.
3. Switch to a SECOND LEAD and confirm the flat line.
4. If confirmed asystole in two leads: begin CPR, do NOT defibrillate.

Survival from asystole is very poor unless there is an immediately reversible cause (the "Hs and Ts"). Reversible causes of asystole include:

The "Hs"	The "Ts"
Hypovolemia	Tension pneumothorax
Hypoxia	Tamponade (cardiac)
Hydrogen ion excess (severe acidosis)	Toxins/drug overdose
Hyper/Hypokalemia (electrolyte disorders)	Thrombosis, pulmonary (massive PE)
Hypothermia	Thrombosis, coronary (massive MI)

Pulseless Electrical Activity (PEA)

Definition: PEA is a cardiac arrest state in which organized electrical activity IS present on the ECG (the monitor shows what appears to be a normal or near-normal rhythm), but there is NO palpable pulse and NO effective cardiac output. The electrical system is functioning; the mechanical system is not.

Why PEA is deceptive and dangerous: An ECT looking at the monitor sees what appears to be a sinus rhythm at 80 bpm — but the patient is unresponsive and pulseless. If the ECT focuses only on the monitor and fails to assess the patient, a cardiac arrest can be missed. This is why ALWAYS CHECKING THE PATIENT is a fundamental rule.

Causes: PEA is ALWAYS caused by an underlying reversible condition — the same Hs and Ts as asystole. The most common causes in clinical settings:

- Hypovolemia (massive blood loss, severe dehydration)
- Tension pneumothorax (air trapped in pleural space collapses the heart and great vessels)
- Cardiac tamponade (blood/fluid in pericardial sac prevents ventricular filling)
- Massive pulmonary embolism (blocks right ventricular outflow)
- Severe acidosis
- Severe hyperkalemia

Treatment: CPR plus IDENTIFYING AND TREATING THE UNDERLYING CAUSE. Unlike V-Fib and V-Tach, PEA is NOT treated with defibrillation (there is no chaotic rhythm to defibrillate). Treatment is entirely aimed at reversing the underlying cause while maintaining CPR.

7.4 Atrioventricular (AV) Conduction Disorders

AV blocks represent a spectrum of impaired conduction between the atria and ventricles. The anatomical level of the block (AV node vs. infranodal) and the severity of block (delayed vs. intermittent vs. complete) determine the clinical significance. Understanding this spectrum is essential — some AV blocks are benign, while others require permanent pacemaker implantation to prevent death.

First-Degree AV Block

FIRST-DEGREE AV BLOCK

Rate	Depends on underlying sinus rate (usually 60–100 bpm)
Rhythm	Regular
P Waves	Present, upright in Lead II, uniform, one before EVERY QRS — all P waves conduct
PR Interval	> 0.20 sec (> 5 small boxes) — PROLONGED but CONSTANT from beat to beat
QRS	Narrow (< 0.12 sec) — ventricular conduction is normal once the impulse passes through the AV node

Mechanism: EVERY P wave conducts to the ventricles — no beats are dropped. However, conduction through the AV node is abnormally slow, producing a PR interval greater than 0.20 seconds. Despite the alarming name ("block"), first-degree AV block is NOT a true block — it is simply a delay.

Causes: Increased vagal tone (athletes, inferior MI), beta-blockers, calcium channel blockers, digoxin, AV nodal disease, electrolyte abnormalities, Lyme disease carditis.

Clinical significance: Isolated first-degree AV block is almost always BENIGN and requires no specific treatment. However, it should be noted in the ECG report because: (1) it may progress to higher-degree block, especially with medications; (2) very prolonged PR intervals (> 0.30 sec) may contribute to AV dyssynchrony at certain heart rates.

Second-Degree AV Block, Type I — Wenckebach / Mobitz I

2ND-DEGREE AV BLOCK, TYPE I (WENCKEBACH)	
Rate	Atrial rate: regular (SA node fires consistently). Ventricular rate: slightly below atrial rate due to dropped beats.
Rhythm	REGULARLY IRREGULAR — grouped beating pattern. QRS complexes appear in clusters followed by a pause (the dropped beat). Then the cluster repeats.
P Waves	Present, upright, uniform. MORE P WAVES THAN QRS COMPLEXES (some P waves do not conduct to the ventricles).
PR Interval	PROGRESSIVELY LENGTHENS from beat to beat within each group — gets longer with each successive beat until one P wave is completely blocked (no QRS follows). Then the PR RESETS to the shortest value and the cycle begins again.

QRS	Narrow — the QRS complexes that do conduct are normal and narrow. The dropped beat has only a P wave.

Mechanism: At the level of the AV node, each successive impulse finds the node increasingly fatigued from the previous conduction. Each beat takes slightly longer to traverse the AV node until one impulse arrives when the AV node is completely refractory and cannot conduct at all — producing the "dropped beat." After the pause (rest period), the AV node recovers and the cycle restarts.

Location: The block is WITHIN the AV node. The clinical significance of this location: AV nodal cells have a rich blood supply (from the RCA in 60% of patients) and can recover or receive pharmacologic support with atropine.

Causes: Inferior wall MI (the AV node is supplied by the RCA in most patients; inferior STEMI frequently involves AV nodal ischemia), increased vagal tone, medications (beta-blockers, calcium channel blockers, digoxin), myocarditis, athletic heart syndrome.

Clinical significance: Usually BENIGN. Most patients with Wenckebach are asymptomatic or have only mild symptoms. The ventricular rate is usually adequate. The dropped beats are regularly spaced and predictable. In the setting of inferior MI, Wenckebach typically resolves as the ischemia resolves (within 24–72 hours).

Treatment: Usually monitoring only. Symptomatic patients may receive atropine (increases AV nodal conduction). Temporary pacing is rarely needed.

Second-Degree AV Block, Type II — Mobitz II

2ND-DEGREE AV BLOCK, TYPE II (MOBITZ II)	
Rate	Atrial rate regular. Ventricular rate: slower than atrial rate, depends on conduction ratio.
Rhythm	REGULARLY IRREGULAR — QRS complexes appear at regular intervals, but with sudden unexpected pauses.
P Waves	MORE P WAVES THAN QRS COMPLEXES. The ratio of P waves to QRS complexes is FIXED (e.g., 2:1, 3:1) in any given period — though it can change.

PR Interval	CONSTANT from beat to beat — DOES NOT LENGTHEN before the dropped beat. The beat simply FAILS to conduct WITHOUT WARNING — the PR is the same on the beat before the dropped beat as on every other beat.
QRS	Wide (≥ 0.12 sec) in most cases — because the block is BELOW the Bundle of His, and the QRS complexes that do conduct show aberrant ventricular activation

Mechanism: The block is BELOW the AV node — in the Bundle of His or in the bundle branches. This is a critical distinction from Wenckebach. In Mobitz II, the AV node conducts perfectly (constant PR) — but the infranodal conduction system periodically fails to transmit the impulse to the ventricles. Because the failure is BELOW the His bundle, there is no warning — the beat simply does not conduct.

Mobitz II: The Most Dangerous Form of Second-Degree AV Block

MOBITZ II IS A MEDICAL EMERGENCY requiring urgent evaluation for pacemaker placement.

Why is Mobitz II so dangerous?

1. It can progress WITHOUT WARNING to complete (third-degree) heart block — potentially causing sudden loss of cardiac output and syncope or cardiac arrest.
2. The site of block is BELOW the His bundle — an area with poor collateral blood supply and unreliable escape rhythms.
3. The escape rhythm that takes over in Mobitz II patients who progress to complete block is a VENTRICULAR escape rhythm at 20–40 bpm — extremely slow, wide, and unreliable.
4. Atropine is typically INEFFECTIVE (may paradoxically worsen infranodal block).

ANY patient with Mobitz II requires:

- Immediate physician notification
- Discontinuation of any AV-blocking medications
- Preparation for temporary transcutaneous or transvenous pacing
- Urgent cardiology consultation for permanent pacemaker evaluation

Second-Degree AV Block with 2:1 Conduction

A special case deserving separate mention: when every other P wave is blocked (2 P waves for every 1 QRS = 2:1 conduction ratio), it is IMPOSSIBLE to determine from the ECG alone whether this is Type I (Wenckebach) or Type II (Mobitz II). You cannot see the PR progressively lengthening because there is only ONE conducted beat per cycle — there is nothing to compare the PR interval to.

How to differentiate 2:1 block type:

- Wide QRS → strongly suggests Mobitz II (infranodal block)
- Narrow QRS → more likely Wenckebach (AV nodal block), but not definitive
- Look at LONGER rhythm strips — occasionally a 3:2 ratio appears, revealing the PR pattern
- Clinical context: anterior MI → Mobitz II likely (septal perforating arteries supply the His bundle); inferior MI → Wenckebach more likely

Third-Degree (Complete) AV Block

THIRD-DEGREE (COMPLETE) AV BLOCK	
Rate	ATRIAL rate: 60–100 bpm (sinus node fires normally). VENTRICULAR rate: determined by the ESCAPE pacemaker: junctional escape = 40–60 bpm (narrow QRS); ventricular escape = 20–40 bpm (wide QRS).
Rhythm	Atrial rhythm: regular. Ventricular rhythm: regular. BUT THEY ARE INDEPENDENT OF EACH OTHER.
P Waves	Present, regular, upright in Lead II — the SA node fires normally. However, NO P WAVES CONDUCT TO THE VENTRICLES.
PR Interval	COMPLETELY VARIABLE — there is NO relationship between P waves and QRS complexes. The "PR intervals" appear to change with every beat because the P waves and QRS complexes are marching at completely different rates.
QRS	Depends on the level of the escape pacemaker: Narrow = junctional escape (block is at the AV node level, Bundle of His is intact); Wide = ventricular escape (block is below the His bundle)

Mechanism: Complete failure of conduction between the atria and ventricles. Not a single atrial impulse reaches the ventricles. The ventricles are maintained by a RESCUE ESCAPE PACEMAKER at a lower level in the conduction system. The SA node continues to fire at its normal rate, the escape pacemaker fires at its much slower intrinsic rate, and the two are completely independent — this is called **AV dissociation**.

Clinical presentation: The clinical severity depends entirely on the VENTRICULAR ESCAPE RATE:

- Junctional escape (40–60 bpm, narrow QRS): Patients may be mildly symptomatic with fatigue, dyspnea on exertion, or lightheadedness — but the rate may be adequate enough to maintain reasonable cardiac output at rest

- Ventricular escape (20–40 bpm, wide QRS): Patients are usually severely symptomatic — profound hypotension, syncope (Stokes-Adams attacks), heart failure, or cardiac arrest

Complete Heart Block: Immediate Action Required

Third-degree AV block with a wide-QRS ventricular escape rhythm is a MEDICAL EMERGENCY.

The ventricular escape pacemaker at 20–40 bpm is:
• UNRELIABLE: can fail at any time, causing cardiac arrest
• RATE-FIXED: cannot increase with activity or physiologic demand
• DRUG-RESISTANT: atropine does NOT reliably increase the ventricular escape rate in complete block

Management:
1. Immediate physician notification
2. IV atropine (may work if block is at AV node level; often ineffective)
3. Transcutaneous (external) cardiac pacing: apply pacing pads, set rate ~70–80 bpm, increase output until capture
4. Dopamine or epinephrine infusion as a bridge
5. Urgent transvenous temporary pacing
6. Cardiology consultation for permanent pacemaker

7.5 Junctional Rhythms

Junctional rhythms arise from the AV junction — the AV node and Bundle of His. These cells have an intrinsic rate of 40–60 bpm and serve as the heart's secondary pacemaker when the SA node fails or fires too slowly. Because the His-Purkinje system is intact, the QRS complex is typically NARROW (< 0.12 sec) — this is the key differentiator between junctional and ventricular rhythms.

The P wave in junctional rhythms is characteristically INVERTED in Lead II, III, and aVF (negative deflection) because the atria are activated BACKWARD (retrograde) from the AV junction — the activation wave travels from the AV node upward to the atria, opposite to the normal top-down direction.

Junctional Rhythm Type	Rate
Junctional Escape Rhythm	40–60 bpm

Accelerated Junctional Rhythm	60–100 bpm
Junctional Tachycardia	> 100 bpm

JUNCTIONAL RHYTHM — KEY ECG FEATURES	
Rate	Escape: 40–60 bpm; Accelerated: 60–100 bpm; Tachycardia: > 100 bpm
Rhythm	Regular
P Waves	INVERTED in Lead II, III, aVF (retrograde conduction). May appear: BEFORE the QRS (with short PR < 0.12 sec), WITHIN the QRS (hidden), or AFTER the QRS (retrograde P in ST segment)
PR Interval	If P wave precedes QRS: < 0.12 sec. If P within QRS: no measurable PR. If retrograde P after QRS: RP interval visible.
QRS	NARROW (< 0.12 sec) — the hallmark of junctional rhythm differentiating it from ventricular rhythms

7.6 Bundle Branch Blocks (BBB)

When conduction through either the right or left bundle branch fails, the affected ventricle must be activated slowly via working myocardium rather than the Purkinje system — producing a wide QRS complex (≥ 0.12 sec) with abnormal morphology. Identifying bundle branch blocks is essential because: (1) they alter the QRS and ST-T wave morphology in ways that can mimic or mask ischemia; (2) new bundle branch blocks may indicate myocardial ischemia; (3) LBBB in the context of symptoms is treated as a STEMI equivalent.

Right Bundle Branch Block (RBBB)

RIGHT BUNDLE BRANCH BLOCK (RBBB)	
QRS Duration	≥ 0.12 sec (≥ 3 small boxes)
Key Finding in V1	RSR' pattern ("rabbit ears") — a broad, notched R wave in V1, often described as "M-shaped." The second R (R') is the delayed right ventricular activation.
Key Finding in V6	Wide, deep S wave (slurred S wave) in the lateral leads (V5, V6, Lead I) — the last portion of ventricular activation moves rightward, creating a terminal S wave in left-facing leads

T Wave	T wave DISCORDANT from QRS in V1: if the terminal R' is tall (positive), the T wave is inverted (negative). This is NORMAL for RBBB — do not interpret as ischemia.
P Wave & PR	Normal — RBBB is a ventricular conduction defect, not an AV conduction defect

Memory aid for RBBB: In V1, the QRS looks like the letter "M" or has a "rabbit ears" pattern. In V6, there is a prominent S wave. A simple memory tool: "MaRRoW" — in RBBB, V1 is the right side and shows M shape; V6 shows W (double downstroke = broad S). M → RBBB in V1.

Causes: RBBB may be a normal variant in older individuals (isolated RBBB without heart disease). Pathologic causes include: pulmonary embolism (acute right heart strain — new RBBB during PE is a poor prognostic sign), right heart failure, congenital heart disease (atrial septal defect), anterior MI (septal perforating arteries supply the right bundle branch), myocarditis.

Clinical significance: Isolated, chronic RBBB without other cardiac findings is generally benign. However, NEW RBBB in a symptomatic patient (especially with chest pain or dyspnea) requires immediate evaluation.

Left Bundle Branch Block (LBBB)

LEFT BUNDLE BRANCH BLOCK (LBBB)	
QRS Duration	≥ 0.12 sec (often ≥ 0.14 sec — broader than RBBB)
Key Finding in V1	Broad, all-negative QRS — either a QS pattern (single deep negative) or rS pattern (tiny initial R then deep S). No normal septal Q wave.
Key Finding in V6	Broad, notched, all-positive QRS — a wide, "M-shaped" or "plateau" R wave (tall and wide, with a notch or plateau on the upstroke) with NO S wave. The lateral leads (I, V5, V6, aVL) are all broadly positive.
ST-T Changes	DISCORDANT throughout: T waves and ST segments are opposite to the QRS in EVERY lead. V1 shows ST elevation with negative QRS (DO NOT mistake this for STEMI). V6 shows ST depression with positive QRS (DO NOT mistake for lateral ischemia). These changes are NORMAL for LBBB.

LBBB: The ECG Masquerade

LBBB produces major distortions of the ECG that make standard interpretation of NEARLY EVERY finding unreliable:

1. ST segment analysis: Normal LBBB creates ST changes that mimic both ST elevation AND ST depression in different leads — making the detection of acute ischemia extremely difficult.

2. Q wave analysis: LBBB eliminates the normal septal Q waves (which are diagnostic of old inferior or lateral MI) and can create false Q waves.

3. LVH criteria: Standard LVH voltage criteria cannot be applied in LBBB.

4. NEW OR PRESUMABLY NEW LBBB in a patient with acute chest pain is treated as a STEMI EQUIVALENT and activates the cath lab — even without ST elevation meeting standard STEMI criteria.

5. SGARBOSSA CRITERIA: A set of ECG criteria for diagnosing acute MI IN THE PRESENCE OF LBBB:
- ST elevation ≥ 1 mm CONCORDANT with (in the same direction as) the QRS complex: 5 points
- ST depression ≥ 1 mm in V1, V2, or V3 (concordant ST depression): 3 points
- ST elevation ≥ 5 mm DISCORDANT from the QRS (excessively elevated beyond what LBBB normally causes): 2 points

A score of ≥ 3 points has high specificity for acute MI in LBBB.

MEMORY AID for LBBB vs RBBB:
"WiLLiaM MaRRoW" — LBBB: V1 shows W, V6 shows M (WiLLiaM). RBBB: V1 shows M, V6 shows W (MaRRoW).

7.7 Arrhythmia Quick-Reference Summary

Rhythm	Rate
Normal Sinus Rhythm	60–100
Sinus Tachycardia	> 100
Sinus Bradycardia	< 60
PAC	Varies
Atrial Flutter	~150 V rate
Atrial Fibrillation	100–180

SVT (AVNRT)	150–250
PVC	Underlying rate
V-Tach	100–250
V-Fib	N/A
Asystole	0
PEA	Variable
1st Degree AV Block	60–100
2nd Degree Type I	Slowed
2nd Degree Type II	Slowed
3rd Degree (Complete)	Escape rate
Junctional Escape	40–60
RBBB	Varies
LBBB	Varies

Chapter 7 Review Questions

1. A 70-year-old man is on a telemetry unit following an inferior MI. His rhythm strip shows a regularly irregular rhythm with grouped beating (3 conducted beats, then a pause, repeating). The PR interval measures 0.16 sec on the first beat, 0.22 sec on the second, and 0.28 sec on the third. The beat following each pause has no QRS. QRS complexes are narrow. This rhythm is BEST described as:

a) Third-degree (complete) AV block with junctional escape rhythm
b) Second-degree AV block, Type II (Mobitz II)
c) Second-degree AV Block, Type I (Wenckebach)
d) Sinus arrhythmia with PACs

► **Correct Answer:** c) Second-degree AV Block, Type I (Wenckebach)
Rationale: *The classic Wenckebach pattern: progressively lengthening PR intervals (0.16 → 0.22 → 0.28 sec) until one P wave is completely blocked (no QRS). Then the cycle resets. This occurs*

frequently in inferior MI because the RCA supplies the AV node in 60% of patients. The narrow QRS and AV nodal location are consistent with Wenckebach. Mobitz II would show a CONSTANT PR interval before the suddenly dropped beat.

2. You are monitoring a 78-year-old woman in the cardiac ICU. The monitor alarm sounds. The ECG shows completely chaotic, irregular electrical activity with no identifiable P waves, QRS complexes, or T waves. The baseline undulates randomly at varying amplitude. The patient is unresponsive and you cannot detect a carotid pulse. Your IMMEDIATE priority action is:

a) Obtain IV access and administer amiodarone 300 mg IV push

b) Confirm asystole in a second lead before proceeding

c) Call a code, begin CPR, and prepare for immediate unsynchronized defibrillation

d) Administer adenosine 6 mg IV push to terminate the arrhythmia

► **Correct Answer:** c) Call a code, begin CPR, and prepare for immediate unsynchronized defibrillation

Rationale: *The ECG describes ventricular fibrillation (V-Fib): chaotic, irregular activity with no organized waveforms. The patient is confirmed in cardiac arrest (unresponsive, pulseless). V-Fib is a shockable rhythm. The immediate priorities are: call a code, begin high-quality CPR, and defibrillate as soon as the defibrillator is ready. Amiodarone is appropriate after defibrillation if V-Fib persists. Adenosine is for SVT, not V-Fib. Asystole confirmation is relevant only if the screen shows a flat line.*

3. A wide-complex tachycardia at 185 bpm is noted on the monitor. The QRS is 0.16 sec. The rhythm is regular. Occasionally, what appear to be narrow QRS complexes are visible amid the wide complexes — each appearing just slightly different from the wide complexes (partial normalization). The patient is pale, diaphoretic, and has a BP of 74/50. Which finding is PATHOGNOMONIC (100% specific) for ventricular tachycardia?

a) QRS duration ≥ 0.12 sec during tachycardia

b) Positive QRS concordance across precordial leads

c) Capture beats and fusion beats visible during the tachycardia

d) Heart rate above 150 bpm

► **Correct Answer:** c) Capture beats and fusion beats visible during the tachycardia

Rationale: *Capture beats (a sinus impulse completely "capturing" the ventricles and producing a normal narrow QRS in the middle of the wide-complex tachycardia) and fusion beats (hybrid QRS formed when sinus and ventricular impulses simultaneously activate the ventricles) are pathognomonic for V-tach. They prove AV dissociation — the atria and ventricles are beating independently. Wide QRS and concordance are supportive of but not specific for V-tach. The patient is unstable and requires immediate synchronized cardioversion.*

4. Lead II rhythm strip analysis: the R-R intervals are completely and randomly variable — no two consecutive intervals are the same length. The baseline shows continuous low-amplitude irregular undulation (no distinct P waves). QRS complexes are narrow and uniform. Heart rate averages 112 bpm by 6-second count. This rhythm is:

a) Sinus arrhythmia with respiratory variation

b) Atrial flutter with variable block

c) Multifocal atrial tachycardia (MAT)

d) Atrial fibrillation with rapid ventricular response

► **Correct Answer:** d) Atrial fibrillation with rapid ventricular response

Rationale: *Atrial fibrillation: the defining feature is an IRREGULARLY IRREGULAR rhythm — completely random R-R intervals with NO pattern. The baseline shows fibrillatory (f) waves rather than true P waves. QRS complexes are narrow (normal ventricular conduction). Rate of 112 bpm qualifies as "rapid ventricular response" (uncontrolled). Atrial flutter has a sawtooth pattern and usually regular rhythm (fixed block) or regularly irregular. MAT shows distinct P waves of at least 3 different morphologies. Sinus arrhythmia has a RESPIRATORY pattern and visible sinus P waves.*

5. A new LBBB is identified in a 62-year-old man presenting with crushing substernal chest pain radiating to the left arm, diaphoresis, and nausea for 90 minutes. Standard STEMI criteria (ST elevation ≥ 1 mm in two or more contiguous leads) are NOT clearly present. What is the appropriate clinical response?

a) Observe the patient and repeat ECG in 6 hours; STEMI criteria are not met so cath lab activation is premature

b) Treat as a STEMI equivalent and notify the physician immediately for urgent cath lab activation

c) Administer aspirin only and discharge with cardiology follow-up

d) Apply the Sgarbossa criteria and activate the cath lab only if score ≥ 10 points

► **Correct Answer:** b) Treat as a STEMI equivalent and notify the physician immediately for urgent cath lab activation

Rationale: *New or presumably new LBBB in a patient with acute chest pain symptoms consistent with MI is treated as a STEMI EQUIVALENT per ACC/AHA guidelines — regardless of whether ST elevation meeting standard criteria is visible. LBBB fundamentally alters ST-T wave patterns, making standard STEMI criteria unreliable. The Sgarbossa criteria are used to identify STEMI within existing LBBB (a score of ≥ 3, not 10, has clinical utility). The correct action is immediate physician notification and cath lab activation — time is muscle, and delays are deadly.*

CHAPTER 8: ISCHEMIA, INJURY, AND INFARCTION — THE STEMI AND THE 12-LEAD ECG

Learning Objectives

Upon completing this chapter, you will be able to:

63. Describe the pathophysiology of acute coronary syndrome (ACS) and the distinction between NSTEMI and STEMI.
64. Explain the ECG changes associated with ischemia, injury, and infarction.
65. Identify the pattern of ECG changes in an acute STEMI — from hyperacute T waves through Q wave formation.
66. Identify which leads correspond to which coronary artery and which wall of the left ventricle.
67. Explain reciprocal changes and why they reinforce the STEMI diagnosis.
68. Define the criteria for ST elevation required to diagnose STEMI.
69. Apply the concept of STEMI equivalents (new LBBB, posterior STEMI pattern).
70. Describe the ECT's role in STEMI identification and reporting.

8.1 Acute Coronary Syndrome: The Clinical Spectrum

Acute Coronary Syndrome (ACS) is an umbrella term for conditions caused by sudden reduction or cessation of blood flow through one or more coronary arteries. The underlying mechanism is nearly always plaque rupture: a vulnerable atherosclerotic plaque in the coronary artery wall ruptures, exposing its contents to the bloodstream. Platelets aggregate at the rupture site and a blood clot (thrombus) forms, partially or completely blocking the artery.

ACS exists on a spectrum:

Condition	Pathology
Unstable Angina	Partial occlusion; ischemia only; no cell death
NSTEMI (Non-ST-Elevation MI)	Partial occlusion; subendocardial infarction
STEMI (ST-Elevation MI)	Complete occlusion; transmural (full-thickness) infarction

Why ECG Recognition of STEMI Saves Lives

Time is muscle. Every minute a coronary artery is completely occluded, approximately 2 million cardiac muscle cells die. The damage is largely irreversible.

The goal is "door-to-balloon" time ≤ 90 minutes — from the moment the patient arrives at the hospital to the moment the blocked artery is reopened in the cardiac catheterization lab.

The ECG is the FASTEST diagnostic tool available. A 12-lead ECG can be acquired, transmitted, and interpreted in under 10 minutes. As an ECT, your ability to quickly and accurately acquire and recognize a STEMI ECG directly shortens the time to treatment and saves heart muscle.

In pre-hospital settings, many systems now transmit the pre-hospital 12-lead ECG directly to the receiving hospital, allowing cath lab activation BEFORE the patient arrives — further reducing door-to-balloon time.

8.2 The ECG Evolution of an Acute STEMI

The ECG changes of an acute STEMI evolve in a predictable sequence over the hours and days following the onset of ischemia. Recognizing where a patient is in this sequence helps estimate the timing of the event.

Time After Occlusion	ECG Change
Minutes (hyperacute phase)	HYPERACUTE T WAVES — tall, broad, peaked, symmetric T waves. This is the FIRST ECG sign of acute MI.
Minutes to hours	ST ELEVATION — the ST segment rises above the baseline in the territory of the occluded artery. J-point elevation, often described as "tombstone" when very tall.
Hours	RECIPROCAL CHANGES — ST depression appears in leads anatomically opposite to the infarct zone. These are mirror images of the ST elevation.
Hours to 1–2 days	T WAVE INVERSION — after ST elevation resolves (partially), T waves become inverted in the affected leads.
Hours to days	Q WAVE FORMATION — pathological Q waves appear as dead myocardium becomes electrically silent. The dead tissue no longer contributes a positive vector — the surviving tissue's vector dominates, pulling the early QRS negative.

Weeks to months	ST returns to baseline. Q waves persist. T waves may normalize partially.

8.3 STEMI Criteria: The Numbers You Need to Know

STEMI Diagnostic Criteria (ACC/AHA)

ST ELEVATION is measured at the J POINT (the junction of the QRS and ST segment) in two or more CONTIGUOUS leads.

CRITERIA (new ST elevation at the J point):
- ≥ 1 mm in ALL leads EXCEPT V1-V4
- ≥ 2 mm in V1-V4 in MEN ≥ 40 years
- ≥ 2.5 mm in V1-V4 in MEN < 40 years
- ≥ 1.5 mm in V1-V4 in WOMEN (all ages)

STEMI EQUIVALENTS (same urgency as STEMI, may not meet traditional elevation criteria):
- New (or presumably new) LBBB with chest pain
- Posterior STEMI pattern (ST depression in V1-V3 + tall R waves — see posterior leads protocol)
- De Winter T waves (ST depression + tall symmetric T waves in anterior leads — LAD occlusion)

ALWAYS measure ST elevation from the ISOELECTRIC BASELINE, not from adjacent waves.

8.4 STEMI Localization: Which Wall, Which Artery

ST Elevation in These Leads	Infarct Location
II, III, aVF	Inferior wall
V1, V2, V3, V4	Anterior wall (septal-anterior)
I, aVL, V5, V6	Lateral wall
V1–V4 + I, aVL (widespread)	Anterior-lateral (large territory)
ST depression in V1–V3 + tall R (R > S in V1)	Posterior wall (requires posterior leads)
V3R, V4R (right-sided leads)	Right ventricle (with inferior STEMI)

Reciprocal Changes: Confirming the STEMI

Reciprocal changes are **ST depressions** that appear in leads anatomically opposite to the STEMI territory. They are "mirror images" of the ST elevation — if you could flip the ECG in the affected leads upside down, you'd see the same picture as the direct leads.

STEMI Location	Direct Leads (ST elevation)
Inferior STEMI	II, III, aVF
Anterior STEMI	V1–V4
Lateral STEMI	I, aVL, V5–V6
Posterior STEMI	V7, V8, V9 (if posterior leads done)

Clinical Vignette: Inferior STEMI with RV Involvement

A 67-year-old man calls 911 reporting severe crushing chest pain radiating to his jaw and left arm, with nausea. He is diaphoretic and pale.

Pre-hospital 12-lead ECG findings: Rate 58 bpm, regular. ST elevation 3 mm in II, III, aVF (inferior leads). ST depression 2 mm in I and aVL (reciprocal changes confirming inferior STEMI). The QRS in lead III is larger than in lead II — suggesting a more rightward infarct territory (RCA proximal occlusion).

You apply right-sided leads: V4R shows 1.5 mm of ST elevation — confirming right ventricular involvement.

You transmit the ECG to the hospital. The cath lab is activated before arrival. The patient's door-to-balloon time will be under 60 minutes.

Key points: (1) Inferior STEMI + right-sided V4R elevation = RV infarct. (2) Avoid nitroglycerin (preload-dependent RV). (3) Reciprocal changes in I and aVL confirm true STEMI (not pericarditis or early repolarization). (4) Pre-hospital ECG transmission saves lives.

8.5 STEMI Mimics — When It Looks Like STEMI but Isn't

Several conditions can produce ST elevation on the ECG that is NOT caused by an acute coronary occlusion. Being aware of these mimics prevents unnecessary cath lab activations, while the clinical context (patient history, symptoms, risk factors) helps distinguish them:

Mimic	Key ECG Distinguishing Features
Early repolarization (normal variant)	Concave ("saddle") ST elevation, most prominent in V2–V4 and inferior leads; J-point elevation; notching at J point; no reciprocal changes; resolves with tachycardia
Pericarditis	Diffuse ST elevation in MULTIPLE lead groups (not contiguous territory); concave ("saddle") ST; PR depression (PR lower than baseline); no reciprocal changes (rare)
Left ventricular aneurysm	Persistent ST elevation in territory of old MI; Q waves present; patient has history of prior large MI
LBBB	Discordant ST elevation in V1-V3 (opposite to main QRS direction) — the "expected" ST changes of LBBB; NOT a true STEMI pattern UNLESS Sgarbossa criteria met
Brugada syndrome	ST elevation in V1-V3 with coved (convex upward) morphology followed by T wave inversion; not a STEMI but a channelopathy associated with sudden cardiac death
Hyperkalemia	ST elevation (usually concave), with peaked T waves, wide QRS, sine-wave pattern

8.6 The ECT's Role in STEMI — Activation and Communication

Your role as an ECT in STEMI care is not to diagnose — it is to acquire a high-quality 12-lead ECG as quickly as possible, recognize a possible STEMI pattern, and communicate your findings immediately so that the physician or advanced clinician can confirm the diagnosis and activate the cath lab.

✓ STEMI Protocol — ECT Role Checklist

1. Acquire the 12-lead ECG within 10 minutes of patient arrival (or on first clinical contact in pre-hospital settings).
2. Ensure all 10 electrodes are correctly placed — even 1 mm of ST elevation in the wrong lead due to misplacement can falsely trigger a cath lab activation.
3. Immediately examine the ECG for ST changes in contiguous leads.
4. Look for reciprocal changes — they CONFIRM the STEMI.
5. If STEMI criteria are met: IMMEDIATELY alert the physician, charge nurse, or paramedic supervisor.

6. Do NOT delay to "see what happens" or wait for symptoms to worsen.
7. If the initial ECG is non-diagnostic and symptoms persist: repeat the 12-lead every 5–15 minutes.
8. In pre-hospital settings: transmit the ECG electronically to the receiving hospital if your system supports it.
9. Document the time the ECG was acquired and when it was interpreted.
10. Continue monitoring. If the patient suddenly deteriorates (V-tach, V-fib): initiate code protocol.

What to Say When You Find a Possible STEMI

Speak clearly, directly, and urgently:

"Dr. [Name] / Charge Nurse — I have a patient, [Name], age [age], with chest pain. The 12-lead ECG shows ST elevation of [X] mm in leads II, III, and aVF, with reciprocal ST depression in I and aVL. This is consistent with an INFERIOR STEMI. I am requesting immediate evaluation."

Do NOT say: "The ECG looks a little funny" or "Something might be wrong." Be specific, direct, and urgent. Use the words "ST elevation" and "consistent with STEMI." These words trigger the response.

Chapter 8 Review Questions

1. ST elevation in leads II, III, and aVF with ST depression (reciprocal changes) in leads I and aVL is MOST consistent with:

a) Anterior STEMI (LAD occlusion)
b) Lateral STEMI (LCx occlusion)
c) Inferior STEMI (RCA occlusion)
d) Posterior STEMI (LCx or posterior descending)

► **Correct Answer:** c) Inferior STEMI (RCA occlusion)

Rationale: *Leads II, III, and aVF are the inferior leads — they view the inferior wall of the left ventricle, which is supplied by the RCA in ~90% of people. Reciprocal ST depression in I and aVL confirms the inferior territory and helps distinguish true STEMI from pericarditis.*

2. The FIRST ECG sign of acute myocardial infarction is:

a) Pathological Q wave formation
b) T wave inversion
c) Hyperacute T waves — tall, broad, peaked T waves
d) ST elevation

► **Correct Answer:** c) Hyperacute T waves — tall, broad, peaked T waves

Rationale: *Hyperacute T waves are the earliest ECG change in acute MI, appearing within minutes of coronary occlusion. They are tall, broad, and symmetric. ST elevation appears minutes to hours later. This is the most commonly missed stage of acute MI because it is subtle and the patient may not yet appear critically ill.*

3. A patient with chest pain has ST depression in V1, V2, and V3, along with a tall R wave in V1 (R > S). What should you do next?

a) Reassure the patient — ST depression in V1-V3 is a normal finding

b) Obtain posterior leads (V7, V8, V9) to evaluate for posterior STEMI

c) Obtain right-sided leads to evaluate for RV infarction

d) Document the finding and continue monitoring without action

► **Correct Answer:** b) Obtain posterior leads (V7, V8, V9) to evaluate for posterior STEMI

Rationale: *ST depression in V1-V3 with tall R waves is the classic "mirror image" of a posterior STEMI (because V1-V3 are pointing in the opposite direction from the posterior wall). Posterior leads (V7-V9) are required to confirm — ST elevation ≥ 0.5 mm in any of V7-V9 = posterior STEMI.*

CHAPTER 10: CLINICAL PRACTICE SCENARIOS — APPLYING YOUR KNOWLEDGE

The following case-based scenarios are designed to bridge the gap between knowledge and clinical performance. Each scenario presents a realistic patient encounter as you would experience it in a clinical setting. Read each scenario carefully, analyze the ECG findings, and form your interpretation before reviewing the Answer and Discussion section.

These scenarios integrate anatomy, physiology, ECG technique, arrhythmia recognition, and clinical communication. They reflect the real complexity of patient care: the patient who is anxious and moving, the strip that has artifact, the rhythm that could be two different things. Developing comfort with ambiguity — and the discipline to act safely when uncertain — is an essential clinical skill.

Before You Begin Each Scenario

As you read each scenario:

1. Identify the RATE using the most appropriate method.
2. Determine the RHYTHM (regular, regularly irregular, or irregularly irregular).
3. Analyze the P WAVES (present? upright in II? uniform? one before every QRS?).
4. Measure the PR INTERVAL (normal? prolonged? variable? shortening?).
5. Assess the QRS COMPLEX (narrow or wide? uniform morphology?).
6. Form your INTERPRETATION and determine the clinical urgency.
7. Determine what you would SAY to the physician — practice the verbal report format.

Scenario 1: The Athletic Patient

Patient Presentation

A 22-year-old collegiate cross-country runner presents for a pre-participation sports physical. She denies any symptoms, takes no medications, and has no cardiac history. During the ECG portion of the physical, the following findings are noted on her 12-lead: heart rate 48 bpm. The rhythm is regular. P waves are upright in Lead II, uniform, one before every QRS. PR interval 0.18 sec. QRS narrow at 0.08 sec. The R-R interval varies slightly when the patient breathes in vs. breathes out.

ECG Analysis:

- Rate: 48 bpm — below 60 bpm = bradycardia
- Rhythm: Slightly irregular, correlating with respiration = sinus arrhythmia component

- P waves: Normal sinus P waves — all criteria met
- PR interval: 0.18 sec — normal
- QRS: Narrow — normal ventricular conduction

Interpretation: Sinus bradycardia (48 bpm) with sinus arrhythmia in a well-trained athlete.

Discussion: This ECG is NORMAL for an endurance athlete. High aerobic fitness increases resting vagal tone, which physiologically slows the SA node. Resting heart rates of 40–55 bpm are not uncommon in well-conditioned athletes and require no investigation or treatment in the absence of symptoms. The sinus arrhythmia is also a healthy finding, reflecting intact autonomic respiratory reflexes. The key principle: ALWAYS consider the clinical context. The same ECG in a 75-year-old man with syncope would require immediate evaluation.

What you would say: "The ECG shows sinus bradycardia at 48 bpm with sinus arrhythmia. Given her athletic conditioning and the absence of symptoms, this appears to be a physiologic variant consistent with an athletic heart. The rhythm is regular and all intervals are normal."

Scenario 2: The Palpitating Executive

Patient Presentation

A 45-year-old business executive presents to the emergency department at 11 PM reporting his "heart started racing about 30 minutes ago" while reviewing documents. He drinks 5–6 cups of coffee daily and smokes one pack of cigarettes per day. He is diaphoretic and anxious. Vital signs: HR 188 bpm, BP 102/68, SpO2 98%. The monitor shows a perfectly regular tachycardia at 188 bpm. The QRS is narrow (0.08 sec). No P waves are visible — the area immediately after each QRS shows a subtle negative deflection that appears to be buried in the tail of the QRS.

ECG Analysis:

- Rate: 188 bpm — rapid tachycardia
- Rhythm: Regular — perfectly so. This is highly characteristic of SVT.
- P waves: Not visible as distinct pre-QRS deflections. A subtle inverted (retrograde) deflection appears immediately after each QRS.
- PR interval: Cannot be measured in traditional sense
- QRS: Narrow — 0.08 sec

Interpretation: Supraventricular tachycardia (SVT) — most likely AVNRT (AV nodal reentrant tachycardia).

Discussion: The hallmarks of SVT here are: (1) perfectly regular tachycardia, (2) narrow QRS (ventricular activation is normal), (3) P waves not visible or retrograde in the ST segment. The retrograde P wave immediately after the QRS is the "pseudo-S wave" in inferior leads — a classic finding of AVNRT. The patient's history (caffeine, nicotine, stress, sudden onset) is classic. The blood pressure of 102/68 with diaphoresis indicates hemodynamic compromise.

What you would say: "Doctor, Mr. Harrison is in SVT at 188 bpm. The rhythm is perfectly regular, QRS is narrow, and no distinct P waves are visible before the QRS — there appears to be a retrograde P wave in the ST segment. He is symptomatic with diaphoresis. Blood pressure is 102/68. Requesting immediate evaluation."

Additional teaching point: If the physician attempts vagal maneuvers (Valsalva, carotid sinus massage) and the patient converts to normal sinus rhythm — you will witness the rhythm abruptly "snap" back to a normal rate. This dramatic termination is characteristic of SVT. If adenosine is administered, briefly increased AV block will occur (a momentary asystole or very slow rhythm followed by restoration of sinus rhythm or reveal of an underlying atrial flutter).

Scenario 3: The Irregular Heartbeat

> **Patient Presentation**
>
> *A 72-year-old woman with a history of hypertension and "heart trouble" presents with 3 days of fatigue and mild shortness of breath. She reports occasional palpitations. A routine 12-lead ECG is obtained. Findings: The ventricular rate averages 76 bpm by 6-second count. The rhythm is completely irregular — no two consecutive R-R intervals are identical. No distinct P waves are visible. The baseline shows fine, low-amplitude irregular undulation. QRS complexes are narrow and uniform at 0.08 sec.*

ECG Analysis:

- Rate: ~76 bpm average (variable — use 6-second method)
- Rhythm: IRREGULARLY IRREGULAR — the defining feature of A-Fib
- P waves: Absent. Fibrillatory (f) waves visible on baseline.
- QRS: Narrow, uniform — normal ventricular conduction

Interpretation: Atrial fibrillation with controlled ventricular response (~76 bpm).

Discussion: The "controlled" ventricular rate (60–100 bpm) suggests this patient may already be on rate-controlling medications (beta-blockers, calcium channel blockers, or digoxin). Her symptoms of fatigue and dyspnea for 3 days raise the possibility that this A-Fib may be relatively new (or that her rate control has been suboptimal). The clinical priorities here are: (1) assess hemodynamic stability; (2) evaluate for thromboembolic risk using CHA_2DS_2-VASc score; (3) determine if rate or rhythm control is appropriate; (4) ensure anticoagulation is addressed.

What you would say: "Doctor, Mrs. Chen has what appears to be atrial fibrillation with a controlled ventricular rate averaging 76 bpm. The rhythm is irregularly irregular with no distinct P waves. QRS is narrow. She has 3 days of fatigue and mild shortness of breath. Blood pressure is 128/76."

Scenario 4: The Post-MI Telemetry Alarm

Patient Presentation

It is 3 AM on the telemetry floor. You are monitoring a 65-year-old man admitted yesterday with an acute inferior STEMI, which was treated with primary PCI and stenting of the right coronary artery. His post-procedure course has been stable. The telemetry alarm sounds. On the monitor, you see: heart rate 58 bpm, regularly irregular rhythm. In Lead II, you count 4 P waves for every 3 QRS complexes. All P waves look identical and are upright. The PR interval measures: 0.18 sec (beat 1), 0.24 sec (beat 2), 0.30 sec (beat 3), then a lone P wave with no QRS, followed by a reset: 0.18 sec on the next beat. QRS complexes are narrow at 0.08 sec.

ECG Analysis:

- Rate: 58 bpm ventricular rate — slightly slow
- Rhythm: Regularly irregular — grouped beating with pause
- P waves: More P waves than QRS complexes (4:3 pattern), all upright in Lead II
- PR interval: PROGRESSIVELY LENGTHENS then drops — classic Wenckebach
- QRS: Narrow — ventricular conduction intact

Interpretation: Second-degree AV block, Type I (Wenckebach / Mobitz I).

Discussion: This is the classic presentation of Wenckebach after inferior MI. The RCA supplies the AV node in 60% of patients, and inferior infarctions commonly produce transient AV nodal ischemia with Wenckebach pattern. This arrhythmia is expected and, in this context, usually self-limiting — resolving as the AV nodal ischemia resolves over 24–72 hours. The narrow QRS confirms the block is at the AV node (not infranodal), which is reassuring. The ventricular rate of 58 bpm is adequate.

Critical distinction: If the same patient instead showed a PR interval that was CONSTANT beat-to-beat (e.g., always 0.18 sec) before suddenly dropping a beat — that would be Mobitz II, requiring IMMEDIATE notification and preparation for pacing.

What you would say: "Doctor, Mr. Garcia is showing second-degree AV block, Wenckebach pattern, in the post-inferior STEMI period. Ventricular rate is 58 bpm. QRS is narrow. He appears comfortable and his blood pressure is 118/72. I wanted to make you aware of this change on the monitor."

Scenario 5: The Collapsing Patient

Patient Presentation

You are working in a step-down unit. A 58-year-old woman with ischemic cardiomyopathy (ejection fraction 30%) suddenly grabs her bed rail and says "I feel dizzy" — then slumps back and becomes unresponsive. The monitor alarm sounds immediately. The screen shows a fast, wide-complex, regular tachycardia at approximately 195 bpm. QRS complexes are wide (0.18 sec) and bizarrely shaped. You cannot palpate a carotid pulse.

ECG Analysis:

- Rate: 195 bpm
- Rhythm: Regular
- P waves: Cannot identify distinct P waves amid the wide complexes
- QRS: WIDE — 0.18 sec — and bizarre in morphology
- Patient: Unresponsive, no pulse

Interpretation: Pulseless Ventricular Tachycardia (Pulseless V-Tach) → CARDIAC ARREST.

Discussion: This patient has decompensated from stable V-Tach (if she was ever in it) to PULSELESS V-Tach. Despite a wide-complex tachycardia on the monitor, the PATIENT defines this as cardiac arrest — no consciousness, no pulse. Pulseless V-Tach is treated IDENTICALLY to V-Fib: immediate defibrillation with an unsynchronized (asynchronous) shock.

WHY unsynchronized in pulseless V-Tach? Because synchronized cardioversion requires identifying a QRS to synchronize with — and when the patient has no effective perfusing rhythm, there is no time to synchronize. Deliver an immediate unsynchronized shock.

Your immediate actions:

89. CALL A CODE. "Code Blue — Step-Down Unit, Room 520!"
90. Begin HIGH-QUALITY CPR immediately. 100–120 compressions per minute, full chest recoil.
91. Apply defibrillator pads if not already on the patient.
92. Charge defibrillator to 200 joules (biphasic) as directed by protocol.
93. Clear and deliver UNSYNCHRONIZED shock.
94. Resume CPR immediately after shock — do not pause to check rhythm for at least 2 minutes.

Scenario 6: The "Artifact vs. V-Fib" Dilemma

> 📋 **Patient Presentation**
>
> *A 44-year-old post-surgical patient is on continuous monitoring on the surgical floor. At 10:35 PM, the telemetry screen suddenly shows completely chaotic, large-amplitude waveforms — indistinguishable from coarse ventricular fibrillation. The alarm screams "VENTRICULAR FIBRILLATION." You run to the room.*

What you find: The patient is sitting upright, eating ice chips from a cup, fully alert and conversing normally. He says "that alarm keeps going off — is something wrong?"

Discussion — The MOST IMPORTANT lesson in this scenario:

ALWAYS ASSESS THE PATIENT, NOT JUST THE MONITOR

The single most dangerous habit in cardiac monitoring is treating the monitor alarm as the primary source of truth.

The sequence must ALWAYS be:

1. Look at the monitor → note the apparent rhythm
2. IMMEDIATELY go to the patient → assess responsiveness, pulse, breathing
3. Then interpret the rhythm in the context of the patient's clinical status

In this scenario: the monitor showed V-Fib, but the patient was alert and eating. This is ARTIFACT — not V-Fib.

Common causes of V-Fib-like artifact: patient movement or tremor, lead wire disconnected/loose electrode, patient shivering or having a seizure-like movement, electromagnetic interference.

The opposite is equally true: NEVER dismiss a V-Fib alarm without seeing the patient.

The rule: "If the patient is awake and talking, the rhythm on the monitor cannot be V-Fib or pulseless V-Tach — it must be artifact." These rhythms produce NO cardiac output and therefore NO consciousness.

Appropriate response: After confirming the patient is hemodynamically stable and alert, systematically troubleshoot the artifact:

95. Check all electrode contacts — peel back and re-prep any poorly adhering electrodes
96. Check for loose or damaged lead wires
97. Ask the patient if he was moving during the alarm — note that motion causes artifact
98. Re-acquire a clean baseline rhythm strip and document the normal underlying rhythm
99. Document in the chart: "Monitor alarm for apparent V-Fib at 10:35 PM. Patient assessment: alert, oriented, in no distress, with normal vital signs. Rhythm confirmed as [NSR / underlying rhythm] after artifact correction."

Scenario 7: The Subtle STEMI

📋 Patient Presentation

A 55-year-old woman presents to the emergency department with "indigestion and back pain" for the past 2 hours. She took antacids at home with no relief. She appears mildly uncomfortable. Vital signs: HR 78 bpm, BP 138/84, SpO2 99%. Her 12-lead ECG is obtained. You note: Sinus rhythm at 78 bpm. In the inferior leads (II, III, aVF), the ST segments appear elevated approximately 2 mm above baseline with a slightly convex (tombstone) morphology. In Leads I and aVL, the ST segments appear depressed approximately 1.5 mm. The precordial leads appear normal.

ECG Analysis:

- Rate and Rhythm: Normal sinus rhythm at 78 bpm — NOT a reassuring finding. Many STEMI patients present in sinus rhythm with normal rates initially.
- ST Elevation: 2 mm in II, III, aVF — inferior leads — STEMI territory
- ST Depression in I and aVL: This is RECIPROCAL ST DEPRESSION — a critical finding that confirms the ST elevation is ischemic (not a normal variant). Reciprocal changes occur in leads facing the OPPOSITE wall from the infarction.
- Inferior STEMI territory: Right coronary artery (RCA) in most patients, or left circumflex in a minority ("left dominant" circulation)

Interpretation: Inferior STEMI with reciprocal changes in I and aVL.

Do NOT Be Fooled by Atypical Presentations

Women, diabetics, and elderly patients frequently present with ATYPICAL symptoms of acute MI:

- "Indigestion," nausea, or vomiting (inferior MI often causes vagal symptoms)
- Back pain, shoulder pain, or jaw pain — not classic "crushing chest pain"
- Fatigue, weakness, or feeling "not right"
- Shortness of breath without chest pain

The ECG does not lie even when the symptoms are atypical.
A woman with "indigestion" and inferior ST elevation is having a heart attack — period.

Your job: obtain the ECG, identify the findings, and report immediately.
Do not minimize or delay because the story does not sound "typical."

What you would say: "Doctor — URGENT. Mrs. Perez has ST elevation in leads II, III, and aVF measuring approximately 2 mm, with reciprocal ST depression in leads I and aVL. The pattern is consistent with an acute inferior STEMI. Heart rate 78, BP 138/84. I recommend immediate evaluation for cath lab activation. I am also obtaining right-sided leads to assess for RV involvement."

Follow-up action: In inferior STEMI, RIGHT-SIDED LEADS (V4R specifically) should be obtained to rule out right ventricular infarction — which occurs in up to 30–50% of inferior STEMIs. An RV infarction fundamentally changes management: nitroglycerin and diuretics (which reduce preload) can cause catastrophic hypotension in RV infarction patients who are preload-dependent. This is why your right-sided leads could save this patient's life.

Scenario 8: The High-Degree Block

Patient Presentation

A 79-year-old man with a history of sick sinus syndrome is brought to the ED by family after fainting at dinner. He had no warning symptoms. On arrival, he is groggy and confused, with a blood pressure of 72/48. His skin is cold and diaphoretic. The monitor shows: atrial rate approximately 80 bpm (regular P waves in Lead II). Ventricular rate: 32 bpm, also regular but SLOWER than the atrial rate. The QRS complexes are wide (0.18 sec) and bizarre. P waves appear to "march through" the QRS complexes with no fixed relationship — the PR interval changes with every beat.

ECG Analysis:

- Two independent rates: atrial 80 bpm, ventricular 32 bpm
- AV dissociation: P waves and QRS complexes have no relationship
- Wide QRS (0.18 sec): ventricular escape rhythm below the His bundle
- Rate 32 bpm: severely bradycardic
- Hemodynamically UNSTABLE: BP 72/48, confusion, cold skin

Interpretation: Third-degree (complete) AV block with wide-QRS ventricular escape rhythm at 32 bpm. HEMODYNAMICALLY UNSTABLE.

Discussion: This man is in cardiogenic shock from complete heart block. His heart is beating at 32 bpm with a wide, unreliable ventricular escape rhythm. The AV node is not conducting a single atrial impulse to the ventricles. Atropine is unlikely to be effective (the block is below the His bundle, atropine affects the AV node). He needs TRANSCUTANEOUS (external) PACING immediately as a bridge to transvenous temporary pacing and permanent pacemaker implantation.

What you would say: "EMERGENCY — I need a physician NOW at bedside. Mr. Collins has complete heart block at 32 bpm with a wide QRS escape rhythm. Blood pressure is 72/48. He is confused with cold, diaphoretic skin. He is hemodynamically unstable. I am applying transcutaneous pacing pads now pending physician orders."

Scenario 9: Recognizing a Fatal Drug Effect

Patient Presentation

A 67-year-old woman with schizophrenia and a history of hypertension is admitted for a urinary tract infection. She is receiving IV haloperidol (an antipsychotic) for agitation. On day 2, the telemetry monitor shows: sinus rhythm at 74 bpm. The rhythm is regular. P waves, PR interval, and QRS are all normal. However, the QT interval (measured from the beginning of the QRS to the end of the T wave) measures 0.54 seconds. QTc calculated = 0.60 sec. The T waves appear broad and slightly low-amplitude.

ECG Analysis:

- Rate/Rhythm: NSR at 74 bpm — normal
- QRS: Normal, narrow
- QT interval: 0.54 sec — PROLONGED (normal < 0.44 sec in women)
- QTc: 0.60 sec — CRITICALLY PROLONGED (> 0.50 sec = high risk for TdP)
- Cause: Haloperidol is a well-known QT-prolonging medication

Interpretation: Normal sinus rhythm with critically prolonged QTc (0.60 sec) — HIGH RISK FOR TORSADES DE POINTES. Likely drug-related (haloperidol).

Discussion: The QTc of 0.60 seconds is critically prolonged. QTc > 0.50 seconds is associated with greatly increased risk of Torsades de Pointes (TdP), a form of polymorphic V-tach that can degenerate into V-Fib. The patient has no symptoms yet — but this is a PREVENTABLE complication if acted upon promptly. The ECT identifies this before TdP occurs, potentially preventing cardiac arrest.

What you would say: "Doctor, I need to alert you to a finding on Mrs. Kim's telemetry. She is in normal sinus rhythm, but her QTc is measuring 0.60 seconds — critically prolonged. She is currently receiving IV haloperidol. I am concerned about the risk for Torsades de Pointes. Requesting immediate evaluation."

Expected management: Stop the offending medication (haloperidol), check electrolytes (supplement potassium and magnesium aggressively — target K^+ > 4.5 mEq/L, Mg^{2+} > 2.0 mg/dL), increase monitoring frequency, have IV magnesium sulfate and defibrillator immediately available.

Scenario 10: The Unfolding Code

Patient Presentation

A code blue is called in the emergency department. A 61-year-old man with no cardiac history collapsed in the waiting room. CPR is in progress on arrival of the team. The monitor shows coarse ventricular fibrillation. The team delivers 200J biphasic defibrillation. After 2 minutes of CPR, the monitor is checked. The rhythm now shows: completely regular, wide QRS complexes at 42 bpm. There are no P waves. The patient remains unresponsive and pulseless (confirmed by the team leader).

ECG Analysis after shock:

- Rate: 42 bpm — slow
- Rhythm: Regular — one of the few reassuring features
- P waves: None
- QRS: Wide (0.16 sec), bizarre
- Patient: Pulseless — no cardiac output despite organized rhythm

Interpretation: Pulseless Electrical Activity (PEA) — wide-complex idioventricular rhythm at 42 bpm, no pulse. (Alternatively, this could be an organized ventricular escape rhythm post-defibrillation, also without pulse — the management is the same.)

Discussion: The monitor shows an organized rhythm — but the patient has NO PULSE. This is PEA. The key teaching point: DO NOT SHOCK an organized rhythm. Defibrillation is for V-Fib and pulseless V-Tach (chaotic or fast ventricular rhythms). Shocking a patient in PEA or asystole will not help and may harm. The correct response: CONTINUE CPR and aggressively search for and treat reversible causes — the Hs and Ts.

What you contribute at the code: As the ECT, your role is to accurately identify the rhythm, differentiate it from other possibilities, and clearly communicate to the code team leader. "Team leader — after the shock, the rhythm is now organized: wide-QRS complexes at approximately 42 bpm, no P waves visible. No pulse on pulse check. This appears to be PEA — organized electrical activity without perfusion."

Scenario 11: Reading Between the Lines — The Concealed Flutter

Patient Presentation

> *A 68-year-old man with a history of valvular heart disease presents with 1 day of palpitations and mild chest discomfort. Vitals: HR 152 bpm, BP 106/70. The monitor shows a regular tachycardia at 152 bpm. The QRS is narrow (0.09 sec). There appear to be no visible P waves. The house intern diagnoses "SVT" and asks you to prepare adenosine. Before administering, you study the rhythm more carefully in the inferior leads (II, III, aVF).*

What you notice on careful inspection: In the ST segment of each QRS complex, and in the space between QRS complexes, there is a subtle but regular SAWTOOTH pattern — negative, downward notching occurring at exactly twice the QRS rate (approximately 300 per minute). The pattern is most visible in Lead II and aVF. The negative deflections are regular and continuous with no isoelectric baseline between them.

ECG Analysis:

- Rate: 152 bpm ventricular rate; flutter waves at ~304 per minute
- Rhythm: Regular ventricular rate — consistent with fixed 2:1 conduction
- P waves: Absent — replaced by sawtooth FLUTTER WAVES at ~300/min in inferior leads
- QRS: Narrow

Interpretation: Atrial FLUTTER with 2:1 conduction block (not SVT).

Discussion: At 2:1 conduction, one flutter wave is hidden INSIDE each QRS complex, making flutter waves appear absent unless carefully sought. The ventricular rate in 2:1 flutter is almost exactly 150 bpm (half of 300) — and any regular tachycardia at 140–160 bpm should raise suspicion for atrial flutter with 2:1 block.

Adenosine IS sometimes used in this setting — not to treat the flutter, but as a DIAGNOSTIC tool: it temporarily increases AV block, briefly slowing the ventricular rate and revealing the flutter waves clearly. However, after adenosine wears off, the flutter resumes.

The distinction between SVT and atrial flutter matters clinically: flutter has a thromboembolic risk similar to atrial fibrillation (anticoagulation is required if flutter has been present > 48 hours before cardioversion) and requires different long-term management than SVT.

Scenario 12: The ECG and the Electrolytes

Patient Presentation

A 58-year-old man with end-stage renal disease on dialysis misses two consecutive dialysis sessions. He presents to the emergency department with profound weakness, unable to stand. His last dialysis was 5 days ago. Vital signs: HR 54 bpm, BP 98/62. The 12-lead ECG shows: sinus rhythm at 54 bpm. The QRS complexes are WIDE (0.18 sec) and appear "rounded" or "slurred" — they lack the sharp, narrow appearance of normal QRS complexes. The T waves are TALL, NARROW, PEAKED, and SYMMETRIC — strikingly prominent in multiple leads, especially the precordial leads. The P waves are barely visible and appear small and flattened.

ECG Analysis:

- Rate: 54 bpm — bradycardic
- QRS: Wide and slurred — abnormal ventricular conduction
- T waves: Tall, peaked, narrow, symmetric — HYPERKALEMIA CLASSIC SIGN
- P waves: Flattened/absent — as K^+ rises, atrial paralysis occurs
- Clinical context: Missed dialysis, renal failure = HYPERKALEMIA until proven otherwise

Interpretation: Severe hyperkalemia with ECG changes. This ECG shows the classic "sine wave" progression of hyperkalemia.

Serum K+ (mEq/L)	ECG Changes
5.5–6.0	Tall, peaked, narrow T waves (earliest sign)
6.0–6.5	PR interval prolongation; P wave flattening begins
6.5–7.0	QRS widening; P wave may disappear
7.0–8.0	Sine wave pattern: widening QRS merges with T wave
> 8.0	V-Fib or asystole

What you would say: "URGENT, Doctor — Mr. Thompson has an ECG showing classic features of severe hyperkalemia: peaked, narrow T waves, widened QRS at 0.18 seconds, and barely visible P waves. He is bradycardic at 54 bpm with a blood pressure of 98/62. He has missed two dialysis sessions. I am requesting immediate evaluation for emergency treatment of hyperkalemia."

Emergency treatment sequence (for physician orders): IV calcium gluconate (immediate cardiac membrane stabilization), sodium bicarbonate, insulin + dextrose, albuterol nebulization, Kayexalate (slower), and emergent dialysis.

Self-Assessment: Final Competency Checklist

Use this checklist to assess your readiness for independent ECG monitoring practice. You should be able to confidently answer "yes" to every item before clinical practice.

✓ ECT Core Competency Checklist

1. [ ] I can identify all 12 leads (limb leads I, II, III; augmented leads aVR, aVL, aVF; precordial leads V1–V6) and explain what cardiac region each lead "sees."
2.
3. [ ] I can place all 10 electrodes for a standard 12-lead ECG by palpating anatomical landmarks WITHOUT looking at a reference guide.
4.
5. [ ] I can perform effective skin preparation including abrasion technique and describe why it reduces artifact.
6.
7. [ ] I can explain the ECG paper grid: the value of each small box (0.04 sec, 0.1 mV) and each large box (0.20 sec, 0.5 mV).
8.
9. [ ] I can identify and name every waveform on an ECG strip: P wave, QRS complex (and its components Q, R, S), J point, ST segment, T wave, U wave.
10.
11. [ ] I can apply the Five-Step Method systematically to any rhythm strip without skipping steps.
12.
13. [ ] I can accurately calculate heart rate using the 300-method, 1500-method, and 6-second method.
14.
15. [ ] I can identify all of the following rhythms from a single-lead rhythm strip: NSR, sinus tachycardia, sinus bradycardia, sinus arrhythmia, PACs, atrial flutter, atrial fibrillation, SVT, PVCs (isolated, bigeminy, trigeminy, couplets, R-on-T), V-tach, V-fib, asystole, 1st-degree AV block, 2nd-degree AV block Type I (Wenckebach), 2nd-degree AV block Type II (Mobitz II), 3rd-degree (complete) AV block, junctional escape rhythm, RBBB, LBBB.
16.

17. [ ] I can distinguish Wenckebach from Mobitz II and explain why this distinction is clinically critical.

18.

19. [ ] I can explain what PEA is, why it is dangerous, and why it cannot be treated with defibrillation.

20.

21. [ ] I can identify the ST changes of an inferior STEMI in a 12-lead ECG and name the expected reciprocal changes.

22.

23. [ ] I can explain why right-sided leads are obtained in inferior STEMI and what finding indicates RV infarction.

24.

25. [ ] I can differentiate V-tach from SVT with aberrancy and explain the clinical consequences of misidentification.

26.

27. [ ] I can assess the QTc interval and identify when QTc prolongation requires urgent notification.

28.

29. [ ] I can provide a professional verbal ECG report using the standard structured format.

30.

31. [ ] I understand the immediate actions for every life-threatening arrhythmia: V-Fib (CPR + defib), pulseless V-Tach (CPR + defib), stable V-Tach (immediate physician notification), complete heart block (physician notification + prepare for pacing), STEMI (physician notification + cath lab activation).

32.

33. [ ] I understand that I ALWAYS assess the PATIENT first, and the monitor is a tool — not a diagnosis.

CHAPTER 9: PERFORMING THE 12-LEAD ECG — COMPETENCY AND CLINICAL PRACTICE

Learning Objectives

Upon completing this chapter, you will be able to:

71. Demonstrate proficiency in the complete 12-lead ECG acquisition procedure.
72. Explain the importance of patient comfort and communication during ECG acquisition.
73. Describe documentation requirements for ECG acquisition.
74. Explain the safety considerations for ECG acquisition.
75. Describe how to identify and correct common ECG acquisition errors.
76. Apply the quality assurance framework to all ECG acquisitions.

9.1 Clinical Competency Standards for ECG Acquisition

Proficiency in 12-lead ECG acquisition is a core clinical competency for the Emergency Care Technician. A technically perfect ECG requires not only correct electrode placement but also effective patient communication, proper skin preparation, attentive equipment management, and thorough documentation. This chapter consolidates the clinical performance standards you will be evaluated against.

✓ Complete 12-Lead ECG Performance Checklist — Clinical Standard

1. BEFORE STARTING:
2. [ ] ECG machine on, paper loaded, patient data entered (name, DOB, date/time).
3. [ ] All lead wires present and intact; check connectors for damage.
4. [ ] 10 fresh electrode pads available (check expiration date).
5. [ ] Explained procedure to patient; consent obtained.
6. [ ] Patient in supine or slightly reclined position, arms at sides.
7.
8. SKIN PREPARATION:
9. [ ] Inspected all electrode sites for hair, lotion, moisture, skin breakdown.
10. [ ] Clipped excessive hair where needed.
11. [ ] Cleaned oily or sweaty skin with alcohol wipe; allowed to dry completely.
12. [ ] Lightly abraded skin at each site to reduce impedance.
13.
14. LIMB ELECTRODE APPLICATION:
15. [ ] RA (White): Right inner forearm, 5-10 cm above wrist.

16. [ ] LA (Black): Left inner forearm, 5-10 cm above wrist.
17. [ ] RL (Green): Right inner lower leg, 5-10 cm above ankle.
18. [ ] LL (Red): Left inner lower leg, 5-10 cm above ankle.
19. [ ] All electrodes pressed firmly, edges completely sealed.
20.
21. PRECORDIAL ELECTRODE APPLICATION:
22. [ ] Located sternal angle (Angle of Louis) by palpation.
23. [ ] Palpated 2nd rib and counted to 4th ICS.
24. [ ] V1: 4th ICS, right sternal border — PLACED.
25. [ ] V2: 4th ICS, left sternal border — PLACED.
26. [ ] Located 5th ICS and midclavicular line.
27. [ ] V4: 5th ICS, midclavicular line — PLACED.
28. [ ] V3: Between V2 and V4 — PLACED.
29. [ ] V5: Same horizontal level as V4, anterior axillary line — PLACED.
30. [ ] V6: Same horizontal level as V4, midaxillary line — PLACED.
31. [ ] All electrodes firmly applied, properly connected.
32.
33. ACQUISITION:
34. [ ] Instructed patient to lie still, relax muscles, breathe normally, not talk.
35. [ ] Confirmed all leads showing signal (no flat lines on quick-look).
36. [ ] Acquired 10-second tracing.
37. [ ] Reviewed tracing immediately for artifact, lead reversals, missing leads.
38. [ ] Tracing acceptable — OR — identified problem and repeated acquisition.
39.
40. POST-ACQUISITION:
41. [ ] Tracing labeled with patient name, DOB, date, time, indication.
42. [ ] Tracing printed / transmitted to physician.
43. [ ] Reviewed for ST changes / obvious arrhythmias — reported abnormal findings.
44. [ ] Electrodes removed if study complete; patient made comfortable.
45. [ ] Documented ECG acquisition in medical record.

9.2 Safety Considerations

ECG acquisition is generally a safe, non-invasive procedure. However, several safety considerations must always be observed:

- **Electrical safety**: The ECG machine must be properly grounded. Never use damaged lead wires. Do not acquire an ECG if the patient is in contact with water or standing water on the floor. Ensure the ECG machine is hospital-grade and regularly maintained.

- **Skin integrity**: Do not place electrodes over open wounds, skin breakdown, or active rashes. Document any modified placements.
- **Patient dignity**: Always maintain appropriate draping. Only expose the skin necessary for electrode placement. Offer same-sex chaperones if appropriate per facility policy.
- **Latex allergy**: Check for latex allergies before applying latex-containing electrodes. Latex-free electrodes are widely available.
- **Implanted devices**: Pacemakers and ICDs do not contraindicate ECG acquisition. Note the presence of a pacemaker on the ECG tracing — it affects interpretation.
- **Emergency situations**: In a rapidly deteriorating patient, apply the monitoring leads first (for continuous rhythm monitoring) before completing the full 12-lead. A rhythm strip in 30 seconds is more valuable than a perfect 12-lead in 5 minutes.

9.3 Documentation Requirements

Thorough documentation of every ECG acquisition is a legal, clinical, and quality assurance requirement:

Documentation Element	Requirement
Patient identification	Full name, date of birth, medical record number on the ECG printout
Date and time of acquisition	Exact time — this is a medicolegal document and may determine diagnosis timing
Clinical indication	Reason ECG was obtained (e.g., "chest pain," "syncope," "arrhythmia evaluation")
Lead placement modifications	Any deviation from standard placement (amputation, wound, mastectomy) must be noted
Machine settings	Paper speed (if not standard 25 mm/sec) and calibration (if not standard 10 mm/mV)
Interpreter	Who reviewed the ECG and what was their interpretation
Actions taken	What was done with the result (transmitted, reported, filed)

GLOSSARY OF KEY TERMS

The following glossary defines all major terms introduced in this textbook. Terms appear in alphabetical order. Each term includes its pronunciation guide (where applicable) and its clinical significance. Use this glossary as a quick reference when reviewing chapter material or preparing for assessments.

Absolute Refractory Period: The phase after depolarization when no stimulus, however strong, can re-depolarize a cardiac cell. Corresponds to the QRS and early T wave.

Action Potential: The complete electrical cycle of a cardiac cell — from resting membrane potential through depolarization and back to repolarization.

Acute Coronary Syndrome (ACS): An umbrella term for unstable angina, NSTEMI, and STEMI — all caused by sudden coronary artery compromise.

Arrhythmia: Any cardiac rhythm that deviates from normal sinus rhythm in rate, regularity, or origin of impulse.

Atrial Fibrillation (A-Fib): A cardiac arrhythmia characterized by chaotic, disorganized atrial electrical activity, absence of true P waves, and irregularly irregular ventricular response.

Atrial Flutter: A rapid, organized atrial arrhythmia with characteristic sawtooth flutter waves at ~300 bpm, typically conducted to ventricles with 2:1 block (ventricular rate ~150 bpm).

Atrioventricular (AV) Block: Impaired conduction through the AV node; classified as first, second (Type I or II), or third degree (complete).

Atrioventricular (AV) Node: The specialized electrical structure in the lower right atrium that delays the cardiac impulse (0.12–0.20 sec) before passing it to the ventricles.

Automaticity: The ability of specialized cardiac cells to spontaneously generate electrical impulses without external stimulation.

Biphasic P Wave: A P wave that is both positive and negative in the same lead; associated with left atrial enlargement when the negative portion is deep and wide in V1.

Bundle Branch Block (BBB): Impaired conduction through the right (RBBB) or left (LBBB) bundle branch, producing a characteristic wide (≥ 0.12 sec) QRS complex.

Bundle of His: The compact conduction pathway connecting the AV node to the bundle branches, traveling through the fibrous cardiac skeleton.

Capture Beat: A narrow QRS complex occurring within a run of ventricular tachycardia when the SA node momentarily captures (conducts to) the ventricles — diagnostic of V-tach.

Cardiac Cycle: The complete sequence of electrical and mechanical events constituting one heartbeat, from one systole to the next.

Cardiomyocyte: A cardiac muscle cell. The functional unit of the myocardium.

Coronary Artery: Blood vessels supplying oxygenated blood to the myocardium. The two main coronary arteries (left and right) arise from the aorta just above the aortic valve.

Depolarization: The electrical event in which a cardiac cell's resting charge changes from negative to positive, triggering contraction. Atrial depolarization = P wave; Ventricular depolarization = QRS complex.

Diastole: The phase of ventricular relaxation and filling; the bottom number of blood pressure; when coronary arteries primarily fill.

ECG (Electrocardiogram): A graphic recording of the heart's electrical activity obtained from electrodes placed on the skin surface.

Ectopic Focus: A site outside the SA node that generates spontaneous electrical impulses, producing abnormal beats (PACs, PVCs) or abnormal rhythms.

Electrode: The physical adhesive pad attached to the skin that detects electrical voltage. 10 electrodes are used in a standard 12-lead ECG.

Escape Rhythm: A rhythm produced by a lower pacemaker (AV junction or ventricle) that "escapes" suppression when the SA node fails or slows excessively.

Fascicular Block (Hemiblock): Block of one division (fascicle) of the left bundle branch — left anterior hemiblock or left posterior hemiblock.

First-Degree AV Block: Prolonged but consistent AV conduction (PR interval > 0.20 sec) with all P waves successfully conducting to ventricles.

Fusion Beat: A QRS complex produced by simultaneous activation from two different sources (e.g., SA node and ventricular focus) — diagnostic of V-tach when seen during a wide-complex tachycardia.

Hyperacute T Wave: The earliest ECG sign of acute MI — tall, broad, peaked, symmetric T waves appearing within minutes of coronary occlusion.

Hyperkalemia: Elevated serum potassium; ECG findings include peaked T waves, widened QRS, loss of P waves, and potentially a sinusoidal (sine wave) pattern.

Hypokalemia: Low serum potassium; ECG findings include T wave flattening, prominent U waves, and QT/QU prolongation.

Idioventricular Rhythm: An escape rhythm arising from ventricular pacemaker cells at 20–40 bpm when both SA node and AV junction have failed. Wide QRS.

Intercalated Disc: Specialized junctions between cardiac muscle cells containing gap junctions that allow rapid electrical coupling, enabling the heart to function as a syncytium.

Isoelectric Line (Baseline): The flat line on the ECG representing periods of no electrical activity; used as the reference point for measuring ST elevation or depression.

J Point: The point where the QRS complex ends and the ST segment begins; the reference point for measuring ST elevation or depression.

Junctional Rhythm: A rhythm arising from the AV junction (40–60 bpm) with absent, inverted, or retrograde P waves.

Lead: A mathematical combination of electrode signals that provides a specific "view" of the heart's electrical activity. The 12-lead ECG provides 12 views from 10 electrodes.

Left Bundle Branch Block (LBBB): Block of the left bundle branch; produces wide QRS with broad R in I/V5/V6 and deep QS in V1; makes standard STEMI analysis unreliable.

Mediastinum: The central chest compartment containing the heart, great vessels, trachea, and esophagus.

Mitral Valve: The two-leaflet (bicuspid) atrioventricular valve between the left atrium and left ventricle.

Myocardium: The muscular middle layer of the heart wall; the cardiac muscle responsible for pumping blood.

Normal Sinus Rhythm (NSR): A cardiac rhythm originating in the SA node at 60–100 bpm, conducting normally through all pathways, producing a narrow QRS with upright P waves in Lead II.

NSTEMI: Non-ST-Elevation Myocardial Infarction — a heart attack confirmed by elevated troponin but without diagnostic ST elevation on the ECG.

Overdrive Suppression: The mechanism by which the fastest pacemaker (SA node) suppresses slower pacemakers by depolarizing them before they can spontaneously fire.

P Wave: The ECG waveform representing atrial depolarization. Normal: < 0.12 sec, < 2.5 mm, upright in Lead II.

Pericardium: The fibrous sac enclosing the heart; contains pericardial fluid that lubricates the heart.

Premature Atrial Contraction (PAC): An early beat originating from an ectopic atrial focus; characterized by an ectopic P wave followed by a narrow QRS.

Premature Ventricular Contraction (PVC): An early beat originating from the ventricular myocardium; characterized by no preceding P wave, a wide bizarre QRS, and a full compensatory pause.

PR Interval: The ECG interval from the beginning of the P wave to the beginning of the QRS; represents AV nodal conduction time. Normal: 0.12–0.20 sec.

Purkinje Fibers: The terminal, rapidly conducting branches of the cardiac conduction system within the ventricular walls; fastest conducting cells in the heart.

QRS Complex: The ECG waveform representing ventricular depolarization. Normal duration: < 0.12 sec (narrow). Wide QRS indicates bundle branch block or ventricular origin.

QT Interval: The ECG interval from the beginning of the QRS to the end of the T wave; represents complete ventricular depolarization + repolarization. Must be corrected for rate (QTc).

Re-entry: The most common arrhythmia mechanism — a circulating electrical loop sustained by two pathways with different conduction properties and a zone of unidirectional block.

Reciprocal Changes: ST depression in leads anatomically opposite to an area of ST elevation; mirror images of the infarct zone; confirm the diagnosis of STEMI.

Refractory Period: The time after depolarization when a cardiac cell cannot (absolute) or is difficult to (relative) re-stimulate.

Repolarization: The electrical event returning a cardiac cell to its resting negative charge after depolarization, allowing relaxation and resetting. Ventricular repolarization = T wave.

Right Bundle Branch Block (RBBB): Block of the right bundle branch; produces wide QRS with RSR' ("rabbit ears") pattern in V1 and broad S wave in Lead I.

Sinoatrial (SA) Node: The primary pacemaker of the heart, located in the right atrium near the SVC junction; normal intrinsic rate 60–100 bpm.

Sinus Arrhythmia: A normal variant in which the heart rate varies cyclically with breathing — faster on inhalation, slower on exhalation. All other sinus criteria are normal.

Sinus Bradycardia: A sinus rhythm with a rate < 60 bpm. May be normal (athletes) or pathological.

Sinus Tachycardia: A sinus rhythm with a rate > 100 bpm; almost always a physiological response to an underlying condition.

ST Segment: The ECG interval from the J point to the beginning of the T wave; normally isoelectric. Elevation or depression indicates ischemia, injury, or infarction.

STEMI: ST-Elevation Myocardial Infarction — a cardiac emergency caused by complete coronary artery occlusion, producing ST elevation in contiguous leads. Requires emergency reperfusion.

Supraventricular Tachycardia (SVT): A fast (> 100 bpm), regular, narrow-complex tachycardia originating above the bundle of His. Most commonly caused by AV nodal re-entry.

Syncytium: Cardiac muscle behaving as a single unit due to electrical coupling through gap junctions in intercalated discs.

Systole: The phase of ventricular contraction and blood ejection; the top number of blood pressure.

T Wave: The ECG waveform representing ventricular repolarization. Normal: rounded, asymmetric, upright in I, II, V3-V6.

Third-Degree AV Block (Complete Heart Block): Complete failure of AV conduction — atria and ventricles beat independently at different rates (AV dissociation). An emergency.

Torsades de Pointes: A form of polymorphic ventricular tachycardia characterized by QRS complexes that twist around the isoelectric baseline, caused by QT prolongation.

Tricuspid Valve: The three-leaflet atrioventricular valve between the right atrium and right ventricle.

U Wave: A small wave after the T wave, most visible in V2–V4; prominent in hypokalemia.

Vagus Nerve: The primary parasympathetic nerve supply to the heart; slows the SA node and AV node when stimulated.

Ventricular Fibrillation (V-Fib): Completely chaotic, disorganized ventricular electrical activity with no effective cardiac output. Cardiac arrest requiring immediate CPR and defibrillation.

Ventricular Tachycardia (V-Tach): Three or more consecutive beats originating from the ventricular myocardium at > 100 bpm; wide, bizarre QRS. Pulseless V-tach = cardiac arrest.

Wenckebach (Mobitz I): A form of second-degree AV block in which the PR interval progressively lengthens until a P wave is blocked, then the cycle repeats.

Wolff-Parkinson-White (WPW) Syndrome: A condition caused by an accessory conduction pathway (Bundle of Kent) bypassing the AV node; produces short PR, delta wave, and predisposes to SVT.

QUICK REFERENCE APPENDIX — RHYTHM SUMMARY CARDS

This appendix provides at-a-glance rhythm summary cards for all arrhythmias covered in this textbook. Use these cards for rapid review during clinical rotations and for pre-examination study.

Rhythm	Rate
Normal Sinus Rhythm (NSR)	60–100
Sinus Bradycardia	< 60
Sinus Tachycardia	> 100
Sinus Arrhythmia	60–100
PAC	Variable
PVC	Variable
Atrial Fibrillation	Ventricular 60–150+
Atrial Flutter	Ventricular ~150 (2:1)
SVT	150–250
Junctional Escape	40–60
AIVR	40–100
V-Tach	100–250
V-Fib	None
Asystole	0
1st Degree AVB	Normal
2nd Degree Type I (Wenckebach)	Ventricular slower
2nd Degree Type II (Mobitz II)	Ventricular slower

3rd Degree (Complete) AVB	Ventricle 20–60