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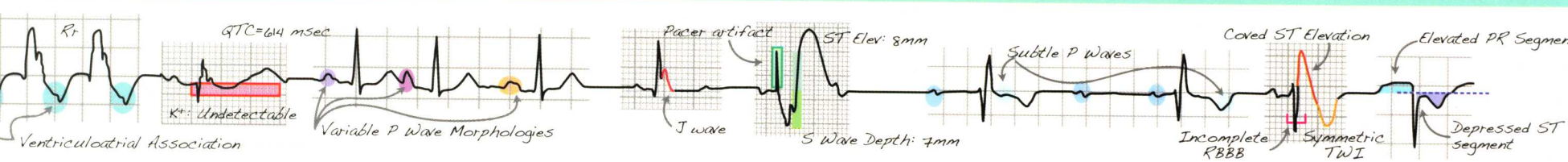

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Wolters Kluwer

A Visual Guide To ECG Interpretation

Second Edition

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A Visual Guide To ECG Interpretation

Second Edition

To the emergency medicine residents and faculty at SUNY Downstate/ Kings County Hospital.

– JLM

To the residents and graduates of the Harvard Affiliated Emergency Medicine Residency who continue to amaze and inspire me every day.

– DFMB

PREFACE

The main objective of this book is to provide a visual tool that will help physicians and other emergency care providers quickly recognize important ECG patterns. By the end of the book, the reader will have developed a mental repertoire of ECGs that represent medically significant conditions, including some that are potentially fatal. We hope that our illustrations and easy-to-follow explanations help to demystify ECG interpretation. This book is intentionally graphic and nontechnical. It is designed to help clinicians make visual diagnoses by pointing out abnormalities in a colorful and pictorial fashion.

The second edition of this book maintains the same format of the book. An ECG is first shown in its native state to give the reader a chance to recognize and interpret salient features. Abnormal patterns are enlarged, highlighted in color, and described in brief text on the following page. We have added ECGs that we have collected over recent years and chosen to include those that emphasized critical pathologies including hyperkalemia, coronary occlusion, and massive pulmonary embolism. In the ischemia chapter, we decided to include ECGs with more subtle signs of coronary occlusion. The second edition is accompanied by access to an online appendix that presents ECG abnormalities in random order. We hope this will allow our readers to practice and consolidate their learning.

We would like to thank everyone who contributed electrocardiograms to this collection.

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CHAPTER 1

Concept Review

Action Potential—Myocardial Cell

The different phases of the action potential relate directly to the waveforms, intervals, and segments that constitute a cardiac cycle on the ECG. Each phase is distinguished by an alteration in cell membrane permeability to sodium, potassium, and calcium ions. A basic understanding of these phases and their major ion currents will help in learning the ECG features associated with conduction abnormalities, drug toxicities, and electrolyte disturbances.

The action potential of the myocardial cell is divided into five phases (phases 0–4). The first phase, phase 0, represents ventricular depolarization. Rapid depolarization depends on initial sodium entry that triggers the explosive influx of more sodium through fast-gated sodium channels. This phase takes the myocardial cell from its resting potential of –90 mV to a positive potential of 20 mV. The summation of this phase across the ventricular myocardium is represented on the ECG as the QRS wave.

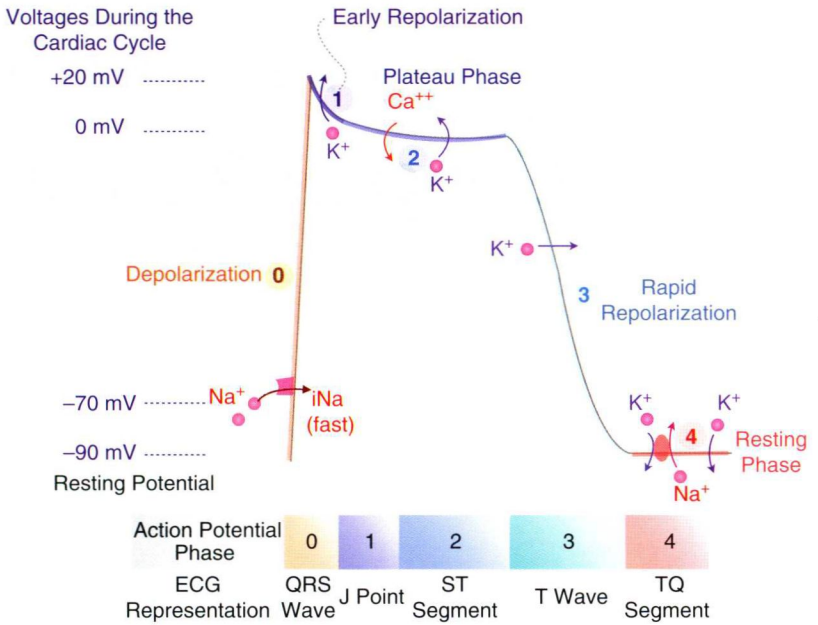


FIGURE 1.1 Action potential of a myocardial cell and phases of the action potential as they relate to ECG waves and segments.[1] The action potential also represents the cell membrane in this figure, with the area under the action potential representing intracellular space and the area above the action potential representing extracellular space.

Ventricular repolarization occurs during phases 1, 2, and 3. By phase 1, the fast sodium channels are closed (and inactivated), and the cell returns to a neutral potential by the opening of potassium channels that allow for the outward movement of potassium.

During phase 2, the neutral potential is maintained by balancing potassium efflux with the sustained entry of calcium ions. Calcium entry during this phase initiates the release of intracellular calcium stores necessary for sarcomere shortening and ventricular contraction. The ST segment corresponds to phase 2.

Phase 3 represents rapid repolarization to the negative resting potential by potassium ion efflux and the closure of calcium channels on the cell membrane. This phase corresponds to the T wave.

After the membrane returns to the resting potential, this potential is maintained during phase 4 by continued potassium efflux and the Na^+/K^+ ATPase pump (red oval). The ventricular myocardium is at its resting potential (phase 4) between the end of the T wave and the beginning of the next QRS wave (T-Q segment).

Action Potential—Pacemaker Cell

Cardiac pacemaker cells are specialized to spontaneously depolarize and initiate action potentials. The action potential of pacemaker cells is divided into three phases (phases 0, 3, and 4). Spontaneous phase 4 depolarization (represented by an upsloping phase 4 in the action potential) distinguishes pacemaker cells from myocardial cells. During this phase, slow inward sodium current results in the gradual rise of the membrane potential toward its threshold potential. The current responsible for phase 4 depolarization is also known as the pacemaker current or funny current (I_f).

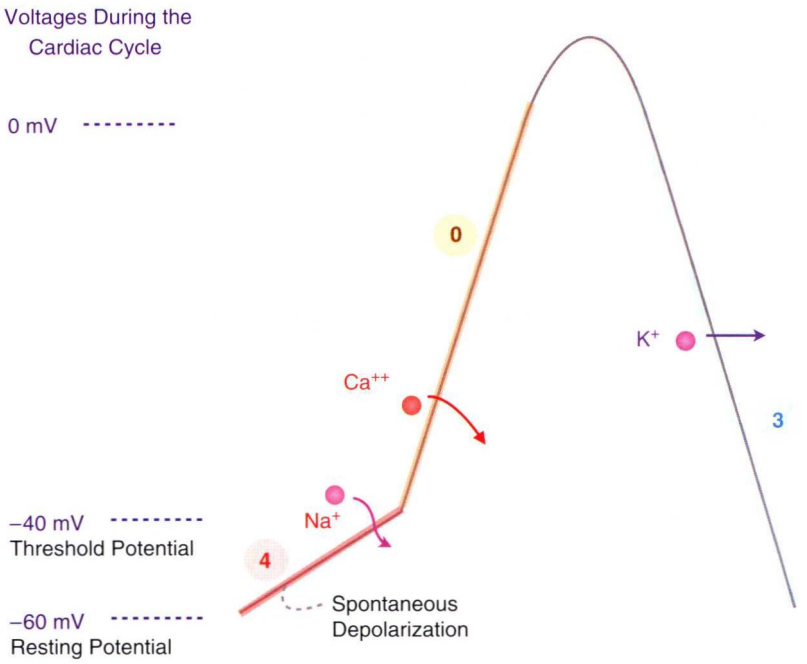


FIGURE 1.2 Action potential of a pacemaker cell. Lines of the action potential also represent the cell membrane of the pacemaker cell.

The slope of phase 4 is directly affected by sympathetic and parasympathetic tone. Sympathetic stimulation results in a steeper phase 4 and consequently a faster heart rate. Norepinephrine (NE) released from sympathetic nerves increases the membrane's permeability to sodium. Vagal tone decreases the slope of phase 4 and, consequently, the heart rate. Acetylcholine (ACh) released by the vagus nerve increases the cell membrane's permeability to potassium while decreasing its permeability to sodium.[2]

Once threshold potential is attained, calcium channels open to depolarize the cell during phase 0.

Repolarization occurs in phase 3 with the opening of potassium channels and closure of calcium channels. When the membrane potential returns to its resting potential, sodium channels immediately open again.

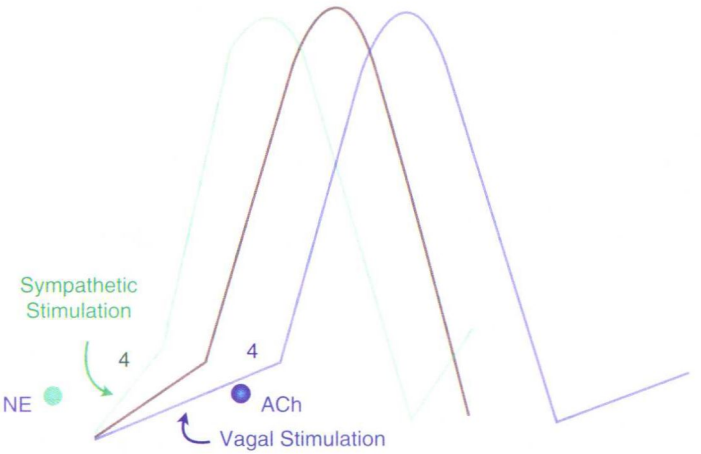


FIGURE 1.3 Effect of sympathetic and parasympathetic stimulation on phase 4 of the pacemaker action potential.

Refractory Periods

Once myocardial cells have entered phase 0 depolarization, the cells become refractory to the conduction of incoming impulses. This allows cells to recover from depolarization. The relative differences in refractory states depend on the state of the fast sodium channels. Refractory states are defined by the strength of impulse required for a myocardial cell to generate and propagate an action potential. Figure 1.4 shows how these different refractory periods relate to phases of the action potential.[2] Refractory periods are especially relevant to ECG rhythms resulting from a reentry circuit and to antiarrhythmic drugs that prolong the duration of the action potential.

Effective Refractory Period — During phases 0 to 2 and part of phase 3, the myocardial cell is unable to propagate an action potential in response to a stimulus, regardless of the strength of that stimulus.

Relative Refractory Period — Before the cell has returned to its resting membrane potential of –90 mV, it may respond to a stronger than normal stimulus. This can occur during phase 3 of repolarization. The response to this stimulus is slower than normal.

Supranormal Period — A smaller than normal impulse can elicit a normal action potential.

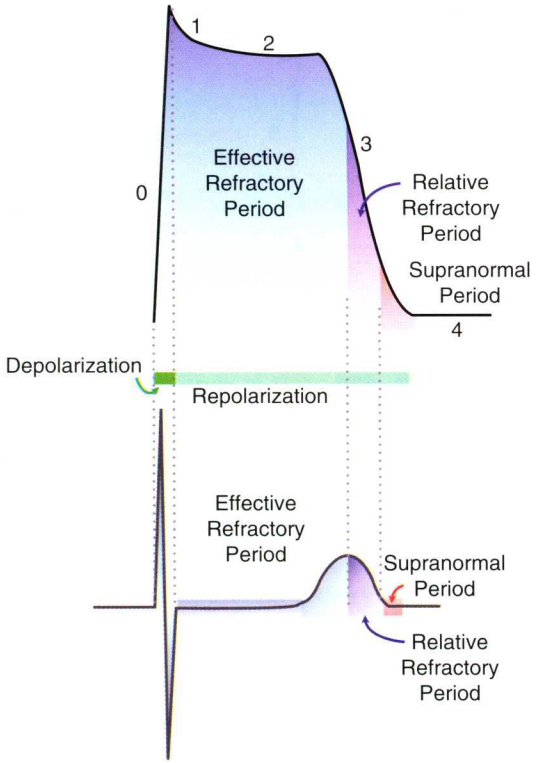


FIGURE 1.4 Refractory periods during different phases of the action potential and cardiac cycle on ECG.

Reentry Circuits

A reentrant circuit consists of two separate pathways that differ in refractory period duration. An impulse may simultaneously encounter a group of refractory cells and a group of cells ready to conduct. Conduction preferentially occurs down the pathway out of its refractory period.

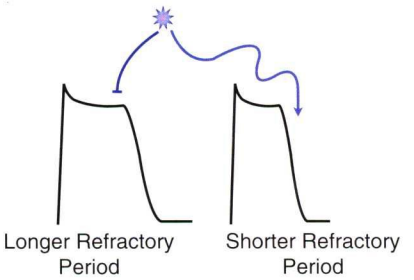


FIGURE 1.5 Variable refractory periods play a key role in reentry.

Drugs that prolong the action potential and effective refractory period are used to terminate reentrant circuits.

Class IA Antiarrhythmics

Quinidine, procainamide, and disopyramide prolong the effective refractory period by blocking the sodium channels responsible for rapid depolarization, thereby prolonging phase 0. This is represented on the ECG by QRS widening.

Class III Antiarrhythmics

Amiodarone, dofetilide, and ibutilide prolong the action potential duration by blocking the outward flow of potassium during phase 3. This increases the effective refractory period of cardiac myocytes. On the ECG, this is represented by QT prolongation.

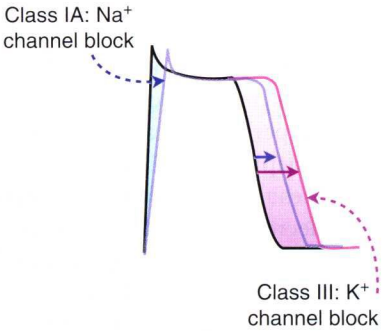
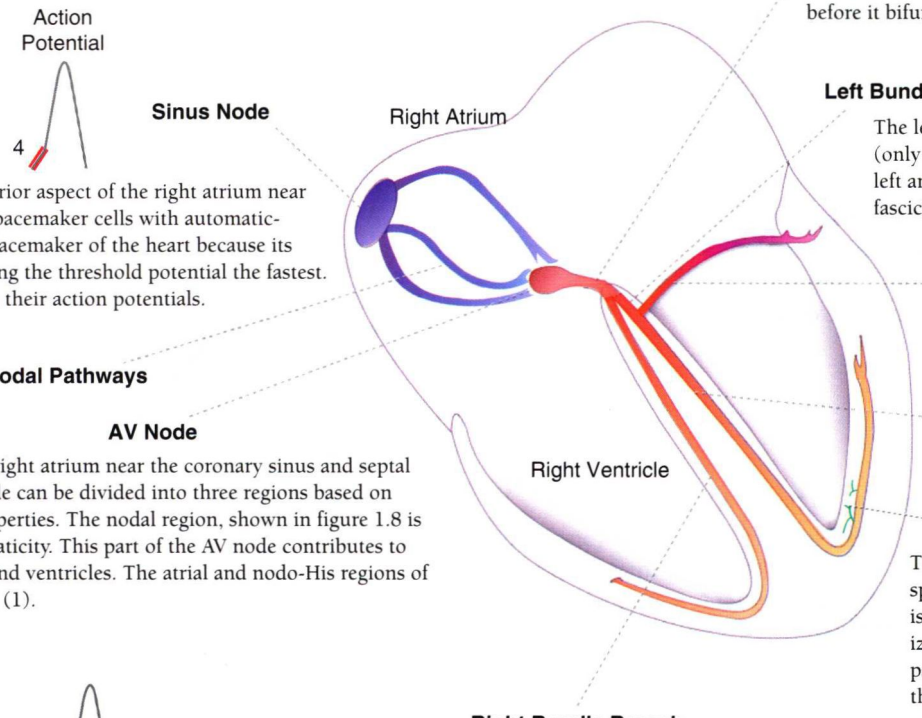


FIGURE 1.6 Effect of Na^+ channel blockade and K^+ channel blockade on action potential duration.

Conduction Anatomy

Normal conduction is essential to coordinated and efficient ventricular contraction. Impulses from the sinus node activate the atrial myocardium and travel down internodal tracts to the AV node. After a brief delay at the AV node, simultaneous conduction down the right and left bundle branches allows for synchronous contraction of the right and left ventricles. Simultaneous conduction down the left anterior and posterior fascicles of the left bundle branch allows for coordinated contraction of the left ventricular free wall.



Bundle of His

The Bundle of His, or common bundle, stems from the AV node before it bifurcates into the right and left bundle branches.

Left Bundle Branch

The left bundle branch is short and separates into three fascicles (only two are depicted here and throughout this book): the left anterior fascicle, the septal fascicle, and the left posterior fascicle. The role of the septal fascicle is less understood.

Left Posterior Fascicle

The left posterior fascicle activates the posterior and inferior segments of the left ventricle. This fascicle has a dual blood supply.

Left Anterior Fascicle

The left anterior fascicle activates the anterior and superior segments of the left ventricle.

Purkinje Fibers

The Purkinje fibers arise from the bundle branches and spread throughout the subendocardium. The endocardium is the first part of the cardiac muscle to become depolarized. Purkinje fibers also have automaticity. When other pacemakers fail to take over, the Purkinje fibers may fulfill the pacemaker role.

The sinus node is located in the superior aspect of the right atrium near the superior vena cava and contains pacemaker cells with automaticity. The sinus node is the dominant pacemaker of the heart because its pacemaker cells are capable of reaching the threshold potential the fastest. This is reflected by a steep phase 4 in their action potentials.

The AV node is located in the lower right atrium near the coronary sinus and septal leaflet of the tricuspid valve. The node can be divided into three regions based on their different electrophysiologic properties. The nodal region, shown in figure 1.8 is the middle segment and lacks automaticity. This part of the AV node contributes to conduction delay between the atria and ventricles. The atrial and nodo-His regions of the AV node contain pacemaker cells (1).

Right Bundle Branch

The right bundle branch travels alongside the left anterior fascicle down the interventricular septum.

FIGURE 1.7 Anatomy of the conduction system.

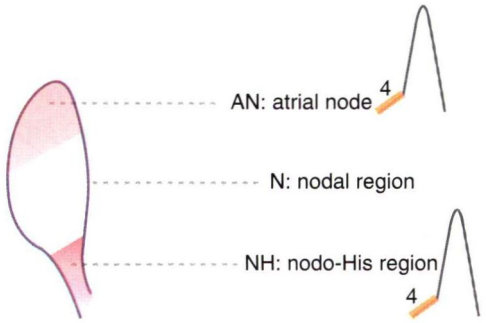


FIGURE 1.8 Properties of the AV node.

Retrograde Conduction Patterns

Basic teaching of conduction anatomy addresses anterograde conduction down normal pathways from the sinus node to the His-Purkinje system. Conduction can also occur in retrograde fashion (in a ventricular to atrial direction) through normal and bypass conduction pathways.

From Atrium to Sinus Node

A premature atrial impulse travels to both the AV node and sinus node (A). This impulse can depolarize the sinus node and effectively reset the pacemaker while it travels through the AV node and His-Purkinje system.

From AV Node to Atrium

Retrograde conduction can also occur from a focus or reentrant circuit within the AV node (B). Retrograde conduction from the AV node to atria can result in atrial depolarization. When not buried by a QRS wave, this can result in the appearance of a retrograde P wave on the ECG.

Concealed Retrograde Conduction

Retrograde conduction may reach the AV node but fail to penetrate and depolarize atrial tissue (C). By reaching the AV node, however, a retrograde impulse can render the AV node refractory to the next incoming atrial impulse. The next sinus P wave will be blocked and appear without an associated QRS complex.

From Ventricle to Atrium

Ventriculoatrial conduction can occur by retrograde conduction through normal conduction pathways or through a bypass tract (C and D).

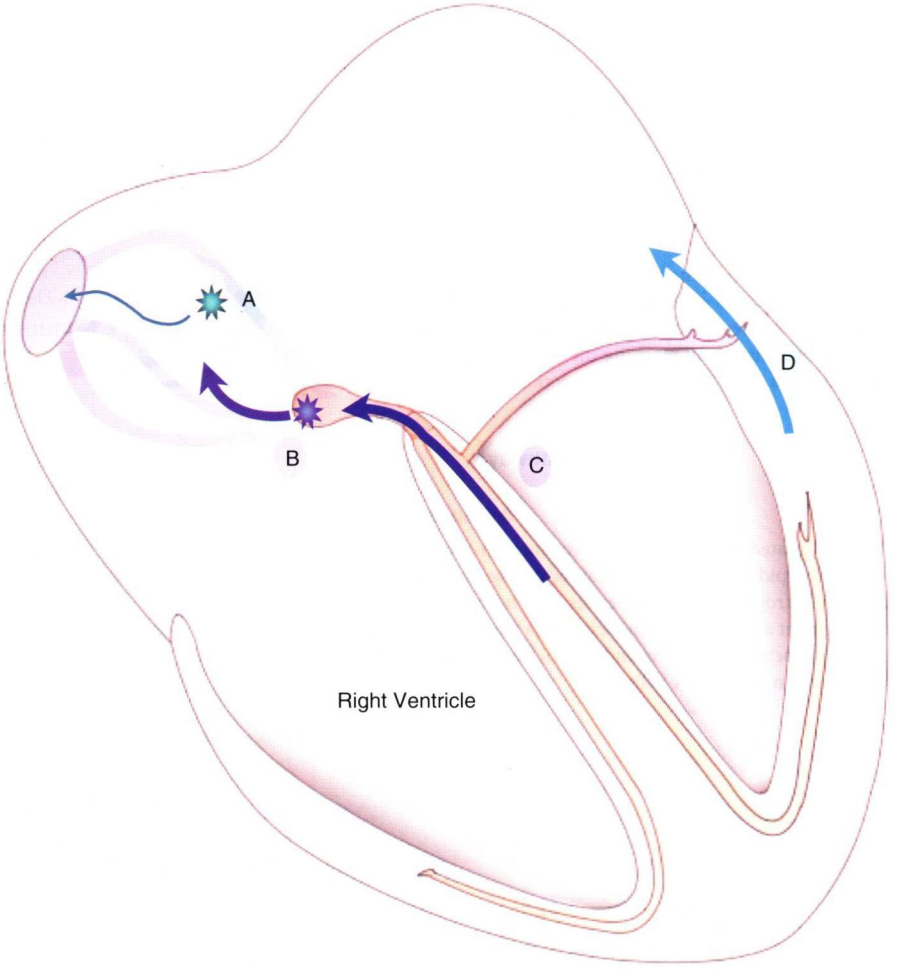


FIGURE 1.9 Retrograde conduction patterns.

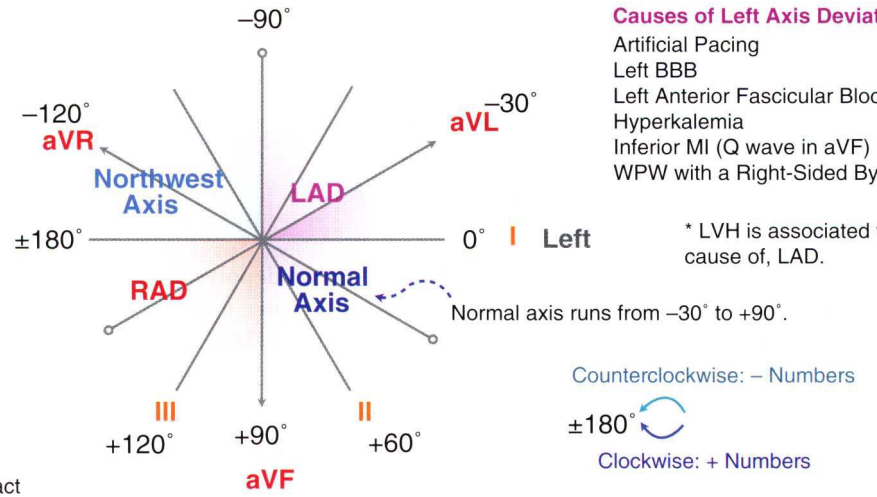
Axis and the Frontal Leads

The ECG axis is a vector representation of the direction of current. Axis can be described for P, QRS, and T waves, but when unspecified, the term usually refers to the QRS axis (representing the direction of ventricular current and depolarization). There are several approaches for determining the QRS axis.

Causes of Northwest Axis
Ventricular Tachycardia
Hyperkalemia
Paced Rhythms
COPD

Causes of Left Axis Deviation
Artificial Pacing
Left BBB
Left Anterior Fascicular Block
Hyperkalemia
Inferior MI (Q wave in aVF)
WPW with a Right-Sided Bypass Tract

Causes of Right Axis Deviation
Right Ventricular Hypertrophy
Right BBB
Vertical Heart/COPD
Pulmonary Embolism
Left Posterior Fascicular Block
Dextrocardia
Lateral MI (Q wave in lead I)
WPW with a Left-Sided Bypass Tract
Normal Variant
Ventricular or Atrial Septal Defect



* LVH is associated with, but not a cause of, LAD.

FIGURE 1.10 Frontal plane leads and the hexaxial reference system.

The Hexaxial Method

One approach to determining the QRS axis is to make use of isoelectric (equiphasic) QRS waves in limb leads. A lead with an isoelectric QRS wave is likely to be perpendicular to the axis, as the vector sum of such a wave (zero) reflects the absence of any current moving toward or away from that lead. The axis is then 90° clockwise or counterclockwise from the isoelectric lead (Fig. 1.11). The perpendicular lead with the positive QRS complex is the QRS axis (current is moving toward that lead). Each limb lead is perpendicular to another limb lead on the ECG.

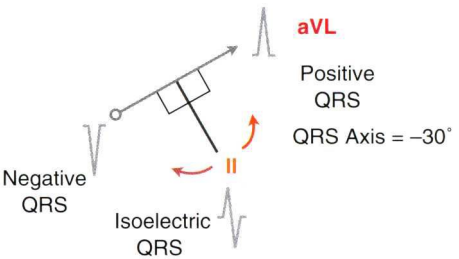


FIGURE 1.11 Perpendicular relationship between two leads used to determine axis.

The Quadrant Method

To get a quick sense of whether there is axis deviation, look at the QRS complexes in leads I and aVF, and simplify the hexaxial system into quadrants.

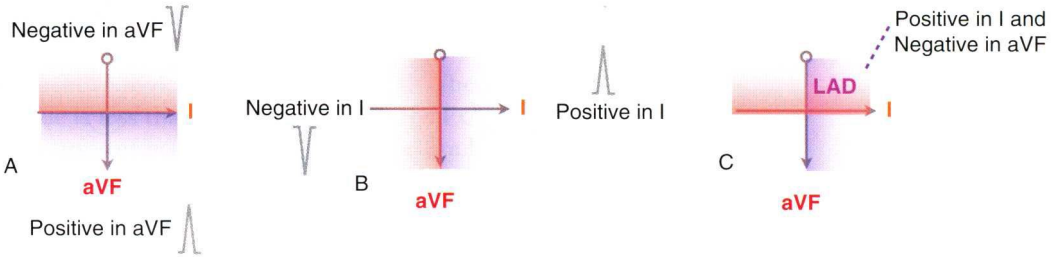


FIGURE 1.12 Use of I and aVF for axis determination. **A.** When the QRS wave in aVF is positive, the axis will be classified as either normal axis or right axis deviation. **B.** When the QRS wave is positive in I, the axis will be normal or deviated to the left. **C.** The combination of a negative QRS wave in aVF and a positive QRS wave in I indicates left axis deviation.

The QRS Wave

The morphology of the QRS wave is governed by the sequence of ventricular activation and the dominant vector force associated with each step. Normal ventricular depolarization can be simplified into two steps: depolarization of the septum followed by depolarization of the ventricular free walls. Because the Purkinje fibers are located just beneath the endocardium, activation of the ventricular walls spreads from the endocardium to the epicardium.

Septal Depolarization

The left aspect of the septum is the first part of the ventricles to depolarize. Normal septal depolarization occurs in a left-to-right direction. This results in the small septal R wave in the right precordial lead V1 and the small septal Q wave in V6.

Ventricular Depolarization

Depolarization of left and right ventricular free walls normally occurs simultaneously. The right-to-left depolarization in the larger and thicker left ventricle comprises the dominating vector force. The ECG interprets this depolarization as a right-to-left force even though depolarization in the right ventricle slightly opposes this force. This dominant vector accounts for the large S wave in V1 that transitions in V3/V4 to become the large R wave in the left precordial leads (V6).

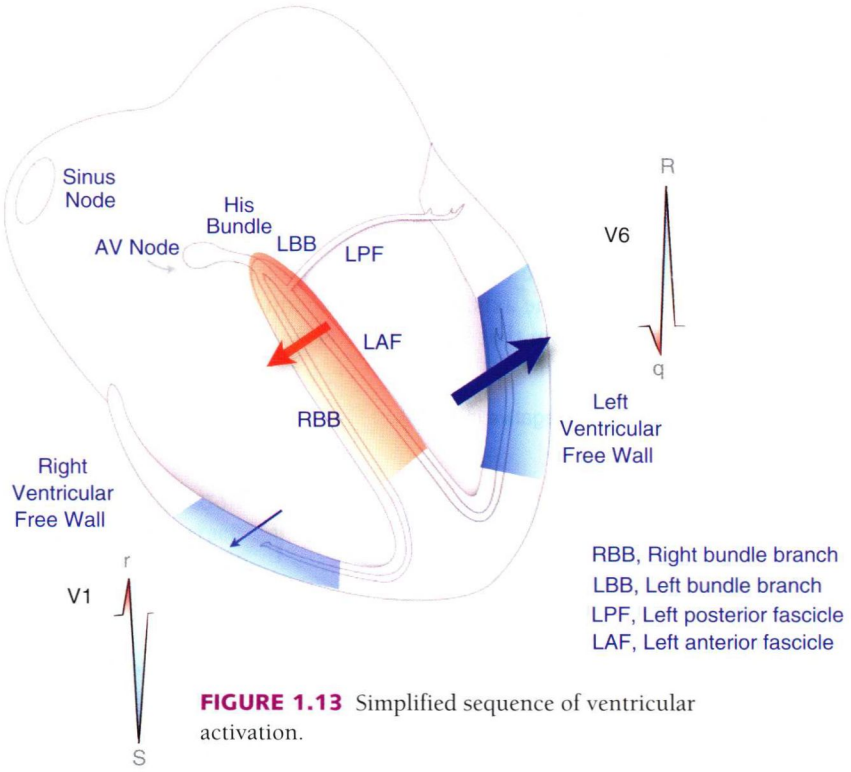


FIGURE 1.13 Simplified sequence of ventricular activation.

The Precordial Leads

Whereas the limb leads view the heart in the frontal plane, the precordial leads view it in the horizontal or transverse plane. Lead V1 is considered to be the window into the right ventricle. In general, leads V1 and V2 reflect the right ventricle and leads V5 and V6 reflect the left ventricle. The QRS complex becomes more positive progressing from V1 to V6.

Normally, R waves are small in lead V1. Causes of tall R waves in this lead are listed below.[3]

Causes of a Tall R Wave in V1 (R > S)

Right Bundle-Branch Block
Right Ventricular Hypertrophy
Hypertrophic Cardiomyopathy
Posterior MI
Paced Rhythm
Wolff-Parkinson-White Syndrome
Normal Variant (children/adolescents)
Duchenne Muscular Dystrophy
Dextrocardia

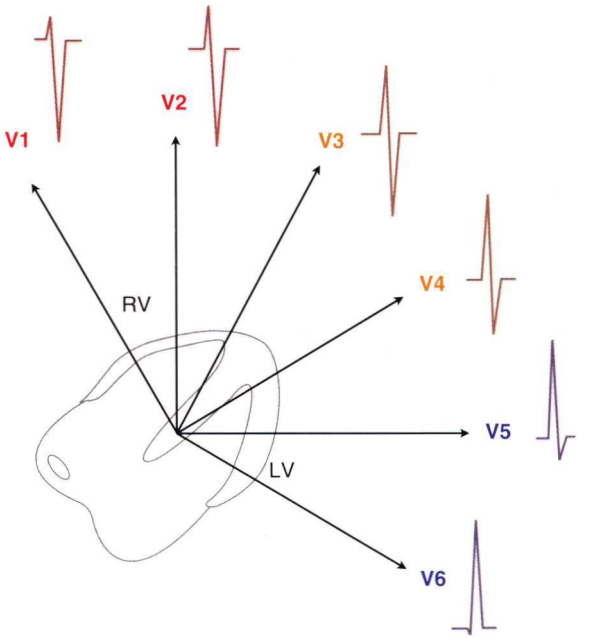


FIGURE 1.14 Normal relationship between precordial leads and the heart.

Lead Misplacement: An obvious aberration in the sequence of wave progression from V1 to V6 is commonly due to incorrect lead placement.

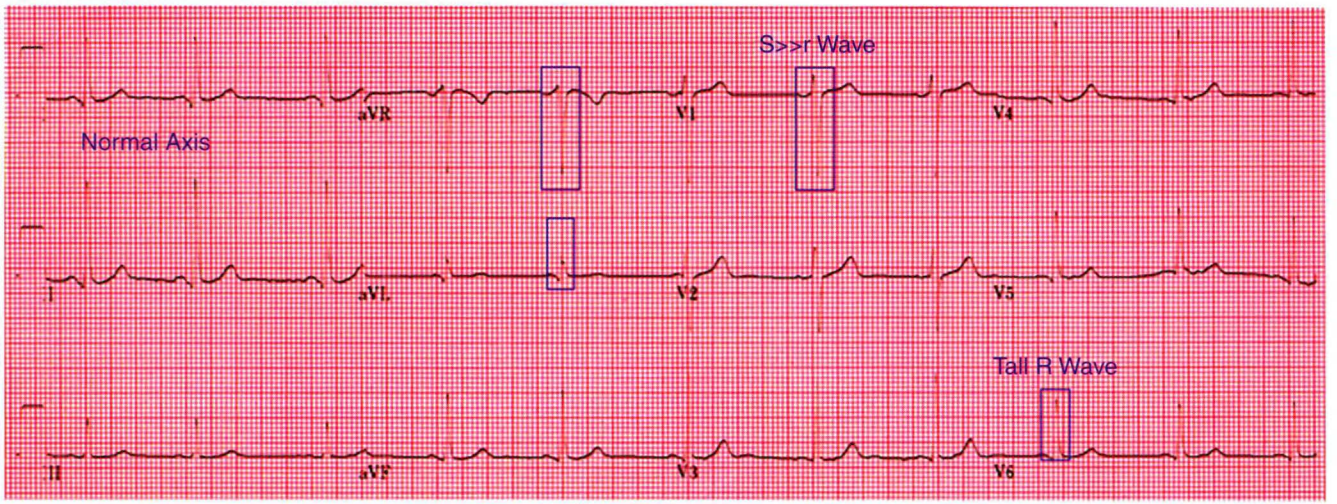
Normally, the QRS complexes transition from being predominately negative to positive in leads V2-V4. When transition with isoelectric complexes occurs in V5 or V6 or the R-wave amplitude is less than 3 mm in lead V3, the ECG is said to demonstrate "poor R-wave progression" or "loss of anterior forces." Causes of poor R-wave progression are listed below.[4]

Causes of Poor R-Wave Progression

Left Bundle-Branch Block
Left Ventricular Hypertrophy
Anterior MI
Right Ventricular Hypertrophy/COPD

R-Wave Progression

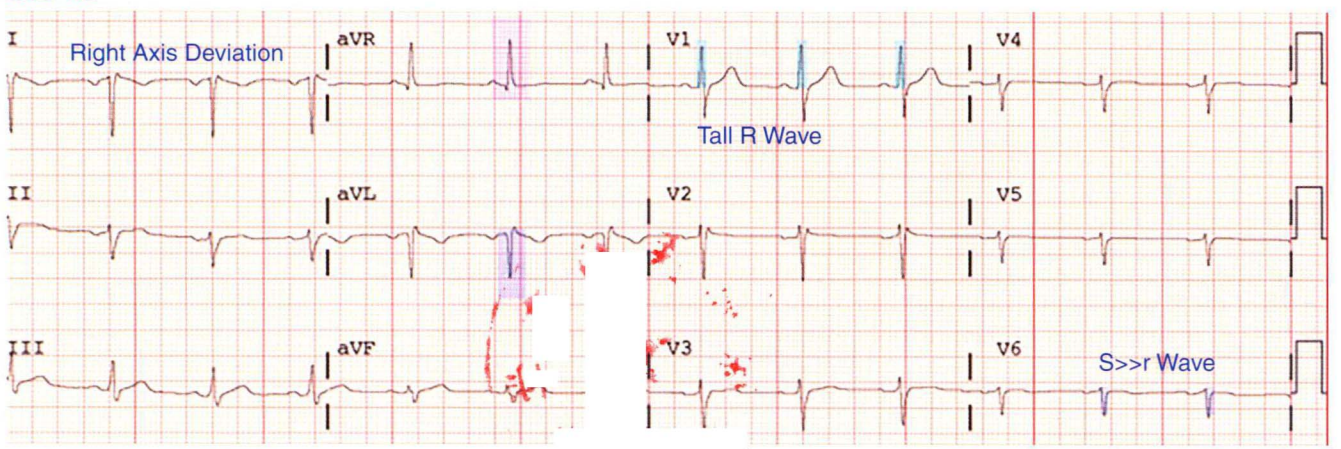
ECG 1.1



Normal ECG

The QRS complexes become more positive going from right-sided precordial leads (V1-V2) to left-sided precordial leads (V5-V6).

ECG 1.2



Dextrocardia

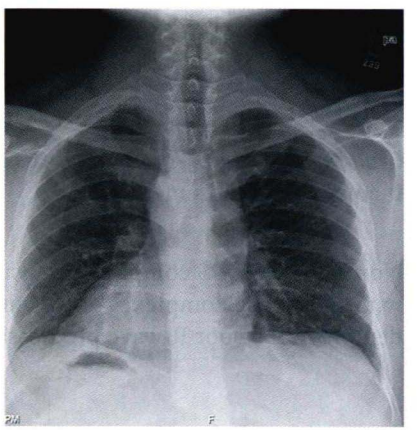


FIGURE 1.15 This patient's chest x-ray, demonstrating situs inversus.

This ECG is from a 39-year-old man with situs inversus. The pattern of R-wave progression is the reverse of what you would expect to see in a patient with normal anatomy. QRS complexes become more negative going from right to left across precordial leads.