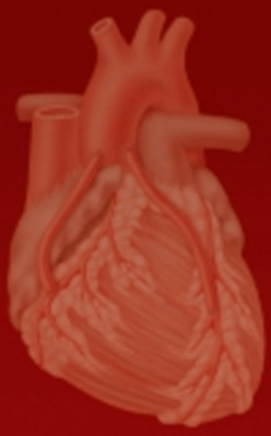
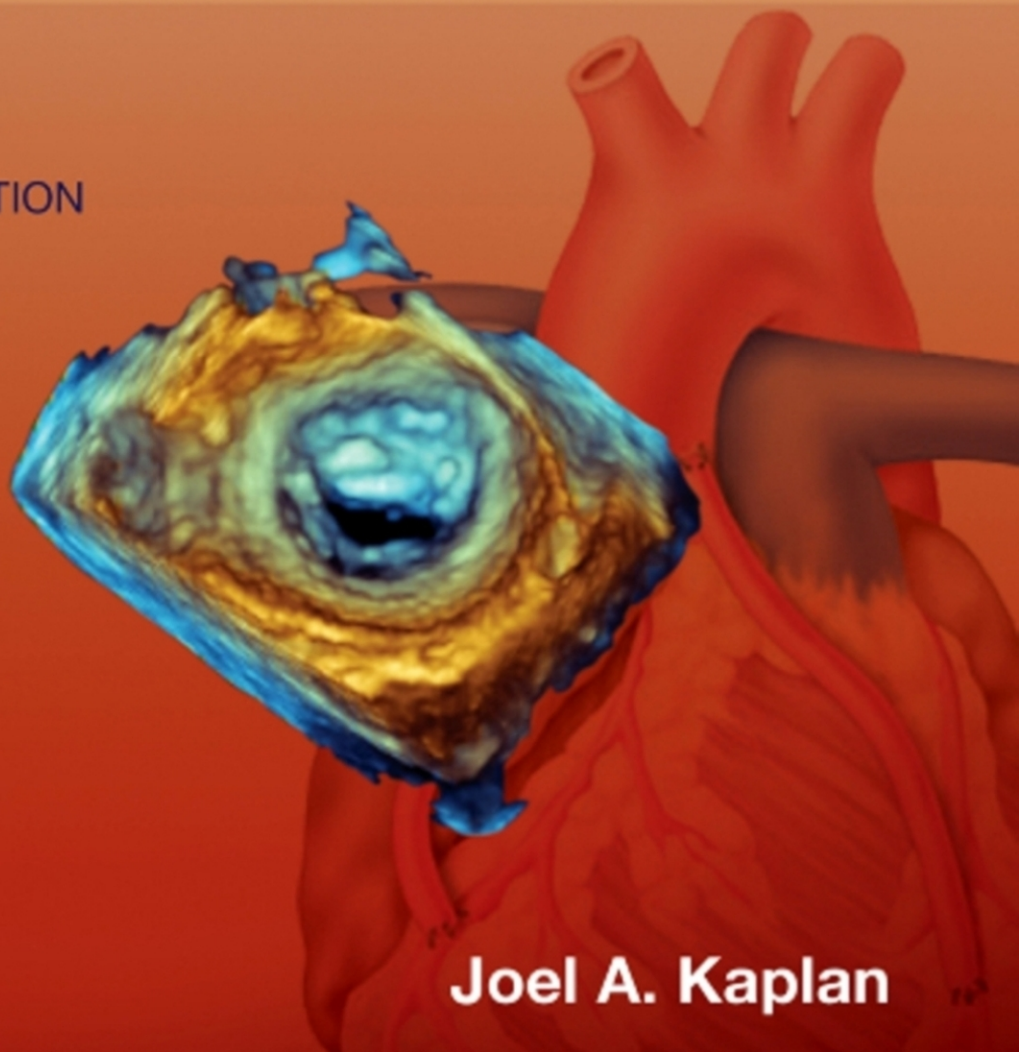


KAPLAN'S

CARDIAC ANESTHESIA

The Echo Era

SIXTH EDITION



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KAPLAN'S CARDIAC ANESTHESIA: THE ECHO ERA

Sixth Edition

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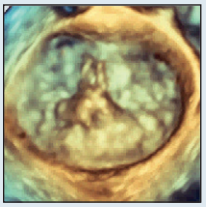

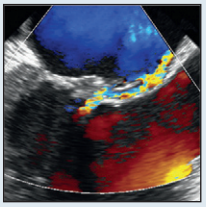

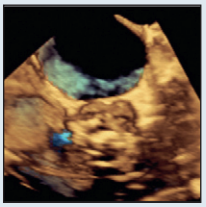

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FOREWORD

The Next Frontier in Cardiac Surgery and Interventions

Nothing endures but change.
Heraclitus

Medicine is in constant flux. Humans constantly are pushing the realm of scientific discovery into meaningful medical applications that ultimately alleviate suffering. The art and science of anesthesia care, as the practice of medicine, continues to progress significantly, especially in cardiac anesthesia. Our responsibilities have expanded beyond creating insensitivity to pain to the practice of sophisticated medical techniques based on fundamental scientific principles. As a specialty, we are much more involved in disease assessment and physiologic manipulation. The distinctions among anesthesiologist, diagnostician, and even interventionalist have blurred. The cardiac anesthesiologists' pivotal role constantly is growing in the successful outcome of a patient population that is becoming ever more complex.

These advances in our specialty come from our ever-expanding knowledge of cardiopulmonary physiology, biochemistry, pharmacology, and neuroscience. However, much of our deeper understanding has come from advancements in technology. This edition of *Kaplan's Cardiac Anesthesia* comes at a time that witnesses the practice of our subspecialty at a major crossroads. Cardiac surgery is undergoing a revolution in the way both simple and complex heart disease will be treated. Simultaneously, anesthesiology and cardiology are undergoing major advancements in imaging. Regional anesthesia now moves beyond the art of landmark assessment to the science of *looking* and *guiding*. In cardiology, it is fascinating to see that as new imaging or quantification technologies are brought online, new physiologic variables of the heart are discovered, rediscovered, or simply appreciated better. Moreover, newer imaging methodologies will serve as the eyes for catheter-guided hands in what can only be called a revolution in the development of new cardiac implantables and repair techniques that avoid sternotomy and cardiopulmonary bypass. Enter the "Echo Era."

We have moved away from an era of palpation of the post-mitral repair thrill to sophisticated techniques to quantify a myriad of cardiac physiologic parameters. We are also moving away from an era of opening the chest to operate on the still heart. Newer image-guided procedures ultimately will lead to less invasive incisions, less infection, and less end-organ insult from cardiopulmonary bypass. Cardiopulmonary bypass will still predominate over the next few years, but this decade will witness an explosion of newer catheter-based techniques that avoid reanimating the nonbeating heart. Imaging will be the cornerstone of these new minimally invasive procedures. Advances in materials science and microelectronics ultimately will put three-dimensional eyes onto the tips of catheters, and these procedures will be performed by physicians who now operate inside the beating heart. Valve surgery is changing in a major way with adult senile calcific stenosis. Progressive change is accelerating transcatheter aortic valve intervention (TAVI). More than 20,000 cases have been performed. These procedures already avoid sternotomy and cardiopulmonary bypass to the point at which some patients are treated without endotracheal intubation and general anesthesia. Time will tell whether this procedure can be done safely. Nonetheless, the course is set and clear; cardiopulmonary bypass has brought us into the 21st century and imaging will advance us in the decades to come. Cardiac anesthesiologists now face a career-changing decision: will they embrace being key members of the new interventional team, or will they be content to be sideline observers of these new procedures?

The pivotal role of echocardiography as both monitoring and diagnostic tool evidenced itself in the 1990s with mitral valve repair. The technology revolution is only going to accelerate. New advancements will include technologies that look at structures with more detail in space and time. Ultimately, newer parallel-processing algorithms in beamforming and automated machine analysis of cardiac images will allow assessment of 3D regurgitant volume, myocardial contraction, and full four-chamber and valvular quantification. Because computers have become more powerful, imaging will be embraced only as it progresses in simplicity.

This new echo era will advance both diagnostics and therapeutic guidance. I have been most privileged that my path from medical student to cardiac anesthesiologist has been mentored by Drs. Kaplan, Reich, and Savino. This edition's framework, penned by a world-renowned group of experts, not only is current and complete but also will equip its readers well for the dynamic ride to come.

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PREFACE

The sixth edition of *Kaplan's Cardiac Anesthesia* has been written to further improve the anesthetic management of the patient with cardiac disease undergoing both cardiac and noncardiac surgery. Since publication of the first edition in 1979, at the beginning of the modern era of cardiac surgery, continued advances in the field have made cardiac anesthesia the leading subspecialty of anesthesiology. To maintain its place as the standard reference textbook in the field, this edition has been completely revised, expanded, and updated throughout to reflect the ongoing changes in cardiovascular care, especially the rapid growth and use of ultrasound and other imaging technologies. Significant contributions to the text have been made by leading cardiologists and cardiac surgeons to fully cover the broader aspects of the total care of the cardiac patient.

This edition is subtitled *The Echo Era* to emphasize today's expanded role of transesophageal echocardiography (TEE) and other ultrasound techniques in the perioperative period. The developments leading to the clinical use of TEE are described, and many of the authors discuss the expanding applications in monitoring and diagnosis by the modern cardiac anesthesiologist. Specific clinical situations are described using the decision-making process highlighted by Weiss and Savino: (1) framing the question asked of the anesthesiologist/echocardiographer; (2) collecting echocardiographic and nonechocardiographic information; (3) making the clinical decision based on integration of knowledge, framing, and information; and (4) implementing the recommendations after a full discussion with the surgeon and other clinicians (e.g., cardiologists).

These case discussions dealing with clinical decision making are augmented by the full-color presentation of the text, multiple color echo and Doppler images, cine clips, and supplementary material on the Expert Consult premium website accompanying the print version of the text. The website also will be used to update the book as new material appears between editions. Some of the new information will be provided by integrating key clinical areas first described in the *Journal of Cardiothoracic and Vascular Anesthesia.* The reader will be able to move seamlessly from the text to the new electronic information technology available with the book.

The content of the sixth edition ranges from the basic sciences through translational medicine to the clinical care of the sickest and most complex cardiac patients. The final section of this edition is entitled "Education in Cardiac Anesthesia" and emphasizes reducing errors to further improve the quality of our patient care. Training and certification in cardiovascular anesthesia are discussed, as well as the educational process and certification available for TEE. Because of the success of the new teaching aides used in the last edition, the Key Points of each chapter appear at the start of the chapters, and Teaching Boxes appear with many of the important "take-home messages." The emphasis throughout the book is on using the latest scientific developments to guide proper therapeutic interventions in the perioperative period.

Kaplan's Cardiac Anesthesia: The Echo Era was written by acknowledged experts in each specific area or related specialties. It is the most authoritative and up-to-date collection of material in the field. Each chapter aims to provide the scientific foundation in the area as well as the clinical basis for practice, and outcome information is included when it is available. All of the chapters have been coordinated in an effort to maximize the clinical utility. Whenever possible, material has been integrated from the fields of anesthesiology, cardiology, cardiac surgery, physiology, and pharmacology to present a complete clinical picture. Thus, this edition should continue to serve as the definitive text for cardiac anesthesia residents, fellows, attendings, practitioners, cardiologists, cardiac surgeons, intensivists, and others interested in the management of the patient with cardiac disease for either cardiac or noncardiac surgery.

Cardiac anesthesia is a complex and comprehensive field of medicine, incorporating many aspects of the specialties of anesthesiology, cardiology, and cardiac surgery. Monitoring modalities always have been an integral part of the practice and have provided us with data to improve our therapeutic interventions. Over the past 30 years, these monitors have become progressively more sophisticated. Many of these monitoring techniques have been adapted from cardiologists and then applied to the cardiac surgical setting. This has been true of electrocardiographic monitoring, with the introduction of the V_5 lead for the intraoperative detection of myocardial ischemia modified from its use during exercise tolerance testing. The pulmonary artery catheter (PAC) was developed for use in the coronary care unit by Dr. Swan, but as he told me, the perioperative use of the PAC in high-risk patients with heart failure and cardiogenic shock was a better role for it, and this use would outlast its role for cardiologists; it turned out to be very true!

Now, we have arrived at the echo era in which TEE—adapted from transthoracic echocardiography use in cardiology—is used widely in cardiac anesthesia for monitoring, diagnosis, and helping to guide the surgery in procedures such as mitral valve repairs. This technique certainly has led to changes in the operative procedures, as well as improvements in our care and choices of pharmacologic treatments, as pointed out in this edition. However, the practice of cardiac anesthesia is and always has been more than the interpretation of any one monitor. Those who believe and emphasize that obtaining certification in TEE makes an anesthesiologist into a cardiac anesthesiologist are sadly mistaken. The practice of cardiac anesthesia includes the use and interpretation of TEE, as it does with other monitors, but it also includes much, much more, and explains the overall size and depth of this book, incorporating all of the areas involved in the complete care of a cardiac surgical patient. It was this overall care in the perioperative period that led J. Willis Hurst, MD, one of the world's leading cardiologists, to state, in his foreword to the first edition of *Kaplan's Cardiac Anesthesia,* that "This cardiologist views the modern cardiac anesthesiologist with awe."

The editors gratefully acknowledge the contributions made by the authors of each of the chapters. They are the dedicated experts who have made the field of cardiac anesthesia what it is today and are the teachers of our young colleagues practicing anesthesiology around the world. This book would not have been possible without their hard work and expertise.

Joel A. Kaplan, MD, CPE, FACC

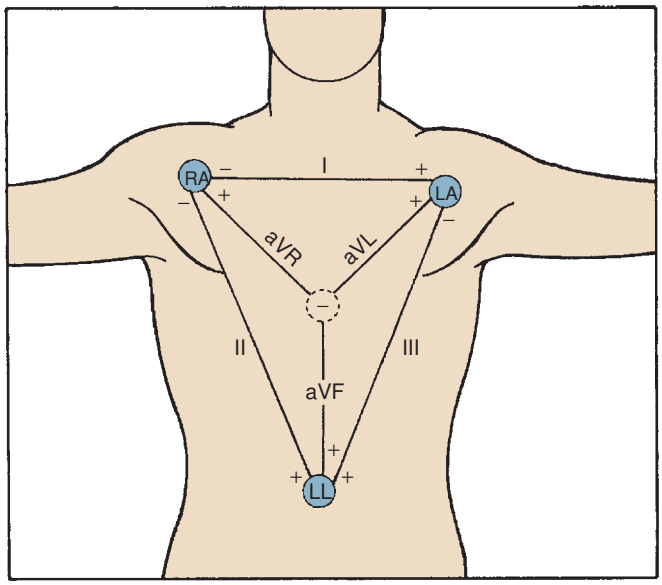
DEDICATION

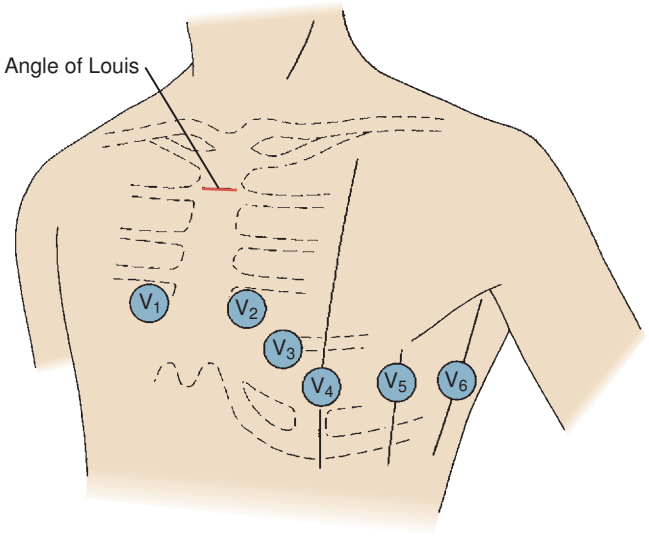
To the pioneers of cardiac surgery and anesthesia who have led us to this exciting era of techniques and technologies that continue to improve our patient care.

Joel A. Kaplan, MD, CPE, FACC

Electrocardiogram Atlas: A Summary of Important Changes on the Electrocardiogram

Lead Placement

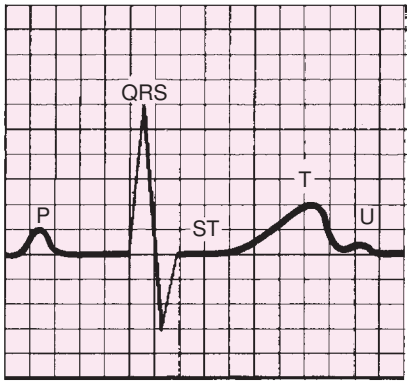


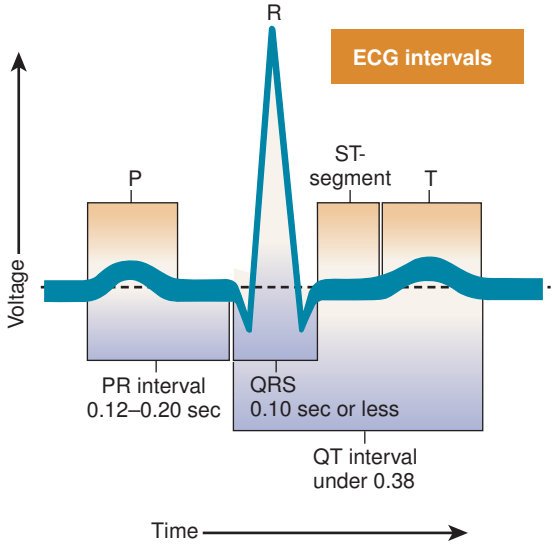


	ELECTRODE	
LEAD PLACEMENT	*POSITIVE*	*NEGATIVE*
BIPOLAR LEADS		
I	LA	RA
II	LL	RA
III	LL	LA
AUGMENTED UNIPOLAR		
aVR	RA	LA, LL
aVL	LA	RA, LL
aVF	LL	RA, LA
	PRECORDIAL	
V_1	4 ICS–RSB	
V_2	4 ICS–LSB	
V_3	Midway between V_2 and V_4	
V_4	5 ICS–MCL	
V_5	5 ICS–AAL	
V_6	5 ICS–MAL	

LA, left arm; LL, left leg; RA, right arm.

Normal Electrocardiogram—Cardiac Cycle

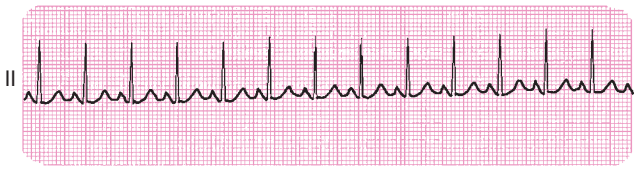




The normal electrocardiogram (ECG) is composed of waves (P, QRS, T, and U) and intervals (PR, QRS, ST, and QT).

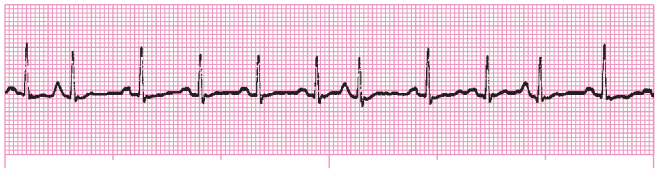
Arrhythmias

Sinus Tachycardia



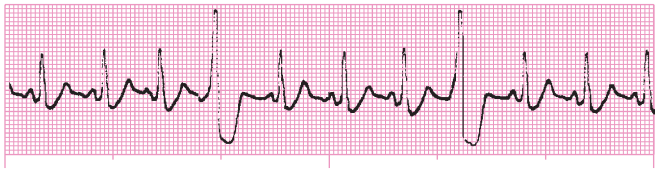
Rate: 100–160 beats/min
Rhythm: Regular
PR interval: Normal; P wave may be difficult to see
QT interval: Normal
Note: Should be differentiated from paroxysmal atrial tachycardia (PAT). With PAT, carotid massage terminates arrhythmia. Sinus tachycardia may respond to vagal maneuvers but reappears as soon as vagal stimulus is removed.

Premature Atrial Contraction



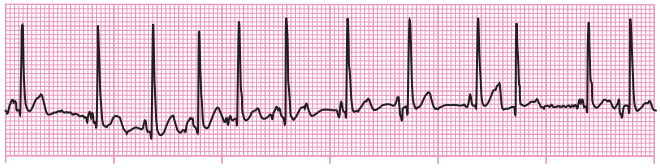
Rate: < 100 beats/min
Rhythm: Irregular
PR interval: P waves may be lost in preceding T waves; PR interval is variable
QT interval: QRS normal configuration; ST segment and T wave normal
Note: Nonconducted premature atrial contraction (PAC) appearance similar to that of sinus arrest; T waves with PAC may be distorted by inclusion of P wave in the T wave.

Premature Ventricular Contraction



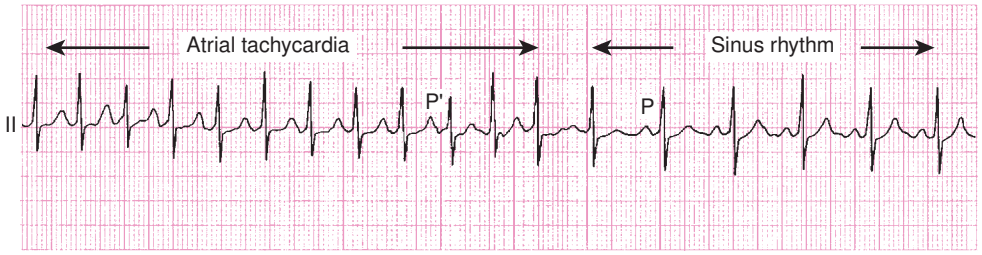
Rate: Usually < 100 beats/min
Rhythm: Irregular
PR interval: P wave and PR interval absent; retrograde conduction of P wave can be seen
QT interval: Wide QRS (> 0.12 sec); ST segment cannot be evaluated (e.g., ischemia); T wave opposite direction of QRS with compensatory pause; fourth and eighth beats are premature ventricular contractions

Multifocal Atrial Tachycardia



Rate: 100–200 beats/min
Rhythm: Irregular
PR interval: Consecutive P waves are of varying shape
QT interval: Normal
Note: Seen in patients with severe lung disease. Carotid massage has no effect. At heart rates < 100 beats/min, may appear as wandering atrial pacemaker. May be mistaken for atrial fibrillation.

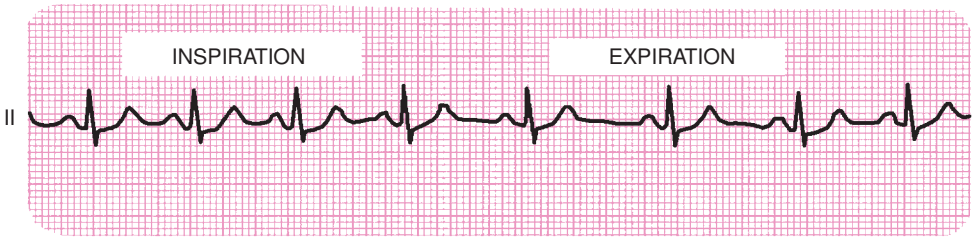
Paroxysmal Atrial Tachycardia



Rate: 150–250 beats/min
Rhythm: Regular
PR interval: Difficult to distinguish because of tachycardia obscuring P wave; P wave may precede, be included in, or follow QRS complex
QT interval: Normal, but ST segment and T wave may be difficult to distinguish

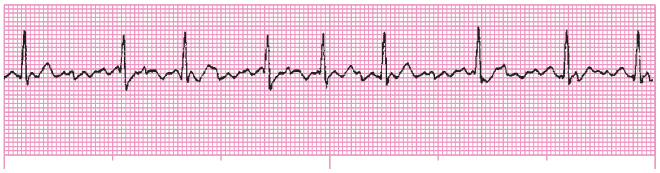
Note: Therapy depends on degree of hemodynamic compromise. Carotid sinus massage may terminate rhythm or decrease heart rate. In contrast with management of paroxysmal atrial tachycardia in awake patients, synchronized cardioversion rather than pharmacologic treatment is preferred in hemodynamically unstable anesthetized patients.

Sinus Arrhythmia



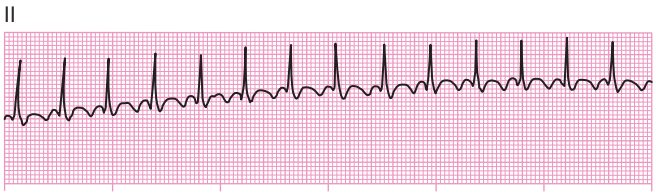
Rate: 60–100 beats/min
Rhythm: Sinus
PR interval: Normal
QT interval: R-R interval variable
Note: Heart rate increases with inhalation and decreases with exhalation ±10–20% (respiratory). Nonrespiratory sinus arrhythmia seen in elderly with heart disease. Also seen with increased intracranial pressure.

Atrial Fibrillation



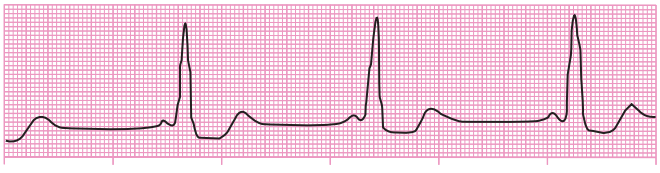
Rate: Variable (~150–200 beats/min)
Rhythm: Irregular
PR interval: No P wave; PR interval not discernible
QT interval: QRS normal
Note: Must be differentiated from atrial flutter: (1) absence of flutter waves and presence of fibrillatory line; (2) flutter usually associated with higher ventricular rates (> 150 beats/min). Loss of atrial contraction reduces cardiac output (10–20%). Mural atrial thrombi may develop. Considered controlled if ventricular rate < 100 beats/min.

Atrial Flutter



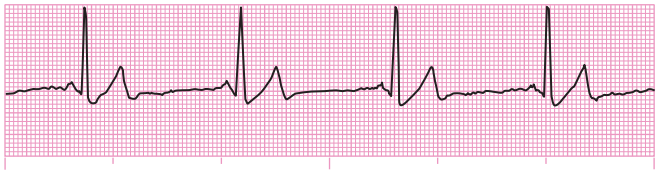
Rate: Rapid, atrial usually regular (250–350 beats/min); ventricular usually regular (<100 beats/min)
Rhythm: Atrial and ventricular regular
PR interval: Flutter (F) waves are saw-toothed; PR interval cannot be measured
QT interval: QRS usually normal; ST segment and T waves are not identifiable
Note: Carotid massage will slow ventricular response, simplifying recognition of the F waves.

Wolff-Parkinson-White Syndrome



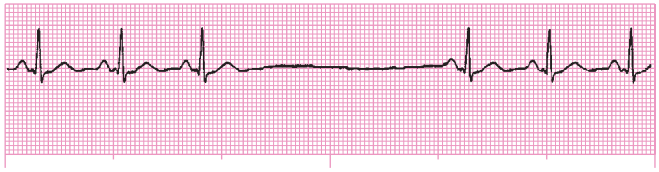
Rate: < 100 beats/min
Rhythm: Regular
PR interval: P wave normal; PR interval short (< 0.12 second)
QT interval: Duration (> 0.10 second) with slurred QRS complex; type A has delta wave, RBBB, with upright QRS complex V_1; type B has delta wave and downward QRS-V_1; ST segment and T wave usually normal
Note: Digoxin should be avoided in the presence of Wolff-Parkinson-White syndrome because it increases conduction through the accessory bypass tract (bundle of Kent) and decreases atrioventricular node conduction; consequently, ventricular fibrillation can occur.

Sinus Bradycardia



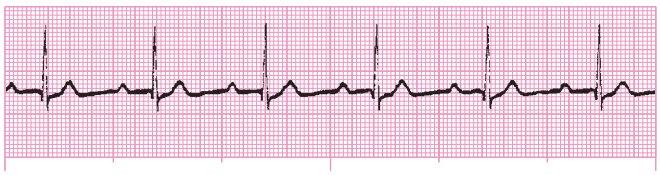
Rate: < 60 beats/min
Rhythm: Sinus
PR interval: Normal
QT interval: Normal
Note: Seen in trained athletes as normal variant.

Sinus Arrest



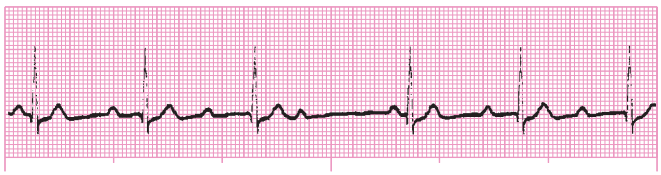
Rate: < 60 beats/min
Rhythm: Varies
PR interval: Variable
QT interval: Variable
Note: Rhythm depends on the cardiac pacemaker firing in the absence of sinoatrial stimulus (atrial pacemaker 60–75 beats/min; junctional 40–60 beats/min; ventricular 30–45 beats/min). Junctional rhythm most common. Occasional P waves may be seen (retrograde P wave).

Atrioventricular Block



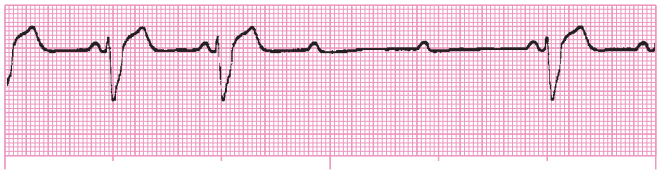
(First-Degree)
Rate: 60–100 beats/min
Rhythm: Regular
PR interval: Prolonged (> 0.20 second) and constant
QT interval: Normal
Note: Usually clinically insignificant; may be early harbinger of drug toxicity.

Atrioventricular Block



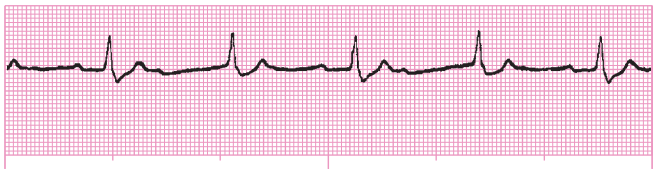
(Second-Degree) Mobitz Type I/ Wenckebach Block
Rate: 60–100 beats/min
Rhythm: Atrial regular; ventricular irregular
PR interval: P-wave normal; PR interval progressively lengthens with each cycle until QRS complex is dropped (dropped beat); PR interval following dropped beat is shorter than normal
QT interval: QRS complex normal but dropped periodically
Note: Commonly seen (1) in trained athletes and (2) with drug toxicity.

Atrioventricular Block



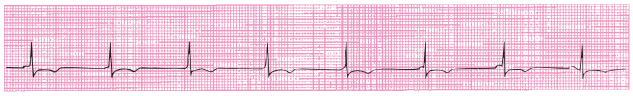
(Second-Degree) Mobitz Type II
Rate: < 100 beats/min
Rhythm: Atrial regular; ventricular regular or irregular
PR interval: P waves normal, but some are not followed by QRS complex
QT interval: Normal but may have widened QRS complex if block is at level of bundle branch. ST segment and T wave may be abnormal, depending on location of block
Note: In contrast with Mobitz type I block, the PR and RR intervals are constant and the dropped QRS occurs without warning. The wider the QRS complex (block lower in the conduction system), the greater the amount of myocardial damage.

Atrioventricular Block



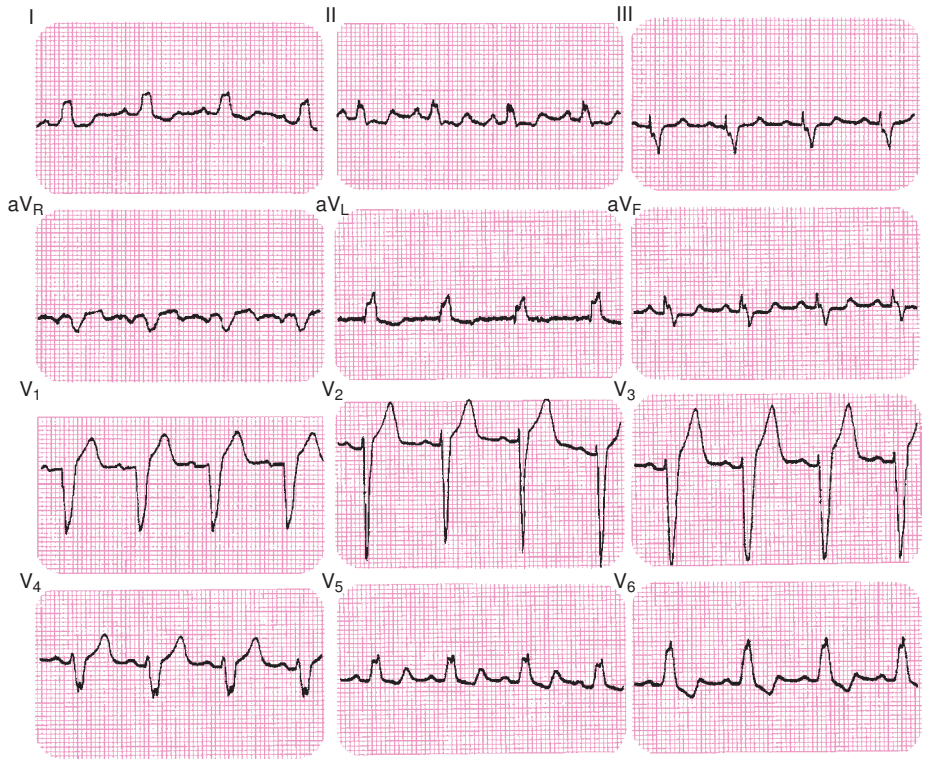
(Third-Degree) Complete Heart Block
Rate: <45 beats/min
Rhythm: Atrial regular; ventricular regular; no relation between P wave and QRS complex
PR interval: Variable because atria and ventricles beat independently
QT interval: QRS morphology variable, depending on the origin of the ventricular beat in the intrinsic pacemaker system (atrioventricular junctional vs. ventricular pacemaker); ST segment and T wave normal
Note: Atrioventricular block represents complete failure of conduction from atria to ventricles (no P wave is conducted to the ventricle). The atrial rate is faster than ventricular rate. P waves have no relation to QRS complexes, for example, they are electrically disconnected. In contrast, with atrioventricular dissociation, the P wave is conducted through the atrioventricular node, and the atrial and ventricular rates are similar. Immediate treatment with atropine or isoproterenol is required if cardiac output is reduced. Consideration should be given to insertion of a pacemaker. Seen as a complication of mitral valve replacement.

Atrioventricular Dissociation



Rate: Variable
Rhythm: Atrial regular; ventricular regular; ventricular rate faster than atrial rate; no relation between P wave and QRS complex
PR interval: Variable because atria and ventricles beat independently
QT interval: QRS morphology depends on location of ventricular pacemaker. ST segment and T wave abnormal
Note: In atrioventricular dissociation, the atria and ventricles beat independently. The P wave is conducted through the atrioventricular node, and the atrial and ventricular rate are similar. In contrast, atrioventricular block represents complete failure of conduction from atria to ventricles (no P wave is conducted to the ventricle). The atrial rate is faster than the ventricular rate. P waves have no relation to QRS complexes; for example, they are electrically disconnected. Digitalis toxicity can present as atrioventricular dissociation.

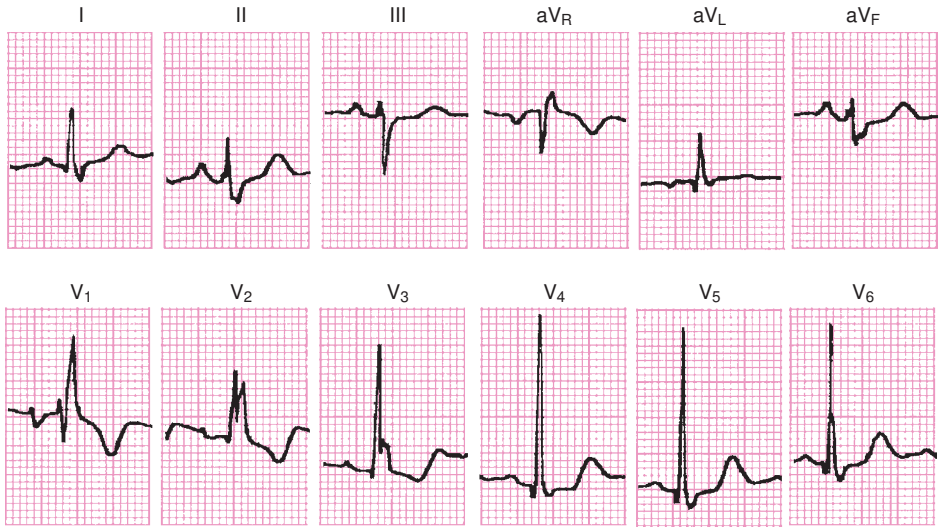
Left Bundle Branch Block



Rate: <100 beats/min
Rhythm: Regular
PR interval: Normal
QT interval: Complete LBBB (QRS > 0.12 second); incomplete LBBB (QRS = 0.10–0.12 second); lead V_1 negative RS complex; I, aVL, V_6 wide R wave without Q or S component; ST-segment and T-wave defection opposite direction of the R wave

Note: Left bundle branch block (LBBB) does not occur in healthy patients and usually indicates serious heart disease with a poorer prognosis. In patients with LBBB, insertion of a pulmonary artery catheter may lead to complete heart block.

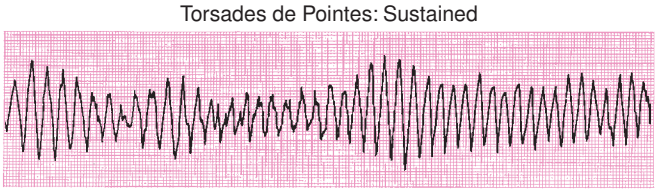
Right Bundle Branch Block



Rate: < 100 beats/min
Rhythm: Regular
PR interval: Normal
QT interval: Complete right bundle branch block (RBBB; QRS > 0.12 second); incomplete RBBB (QRS = 0.10–0.12 second); varying patterns of QRS complex; rSR (V_1); RS, wide R with M pattern; ST-segment and T-wave opposite direction of the R wave

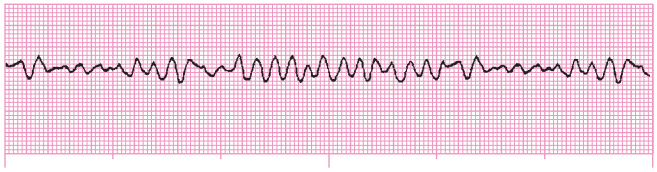
Note: In the presence of RBBB, Q waves may be seen with a myocardial infarction.

Torsades de Pointes

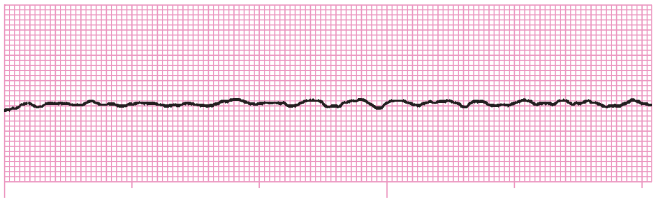


Rate: 150–250 beats/min
Rhythm: No atrial component seen; ventricular rhythm regular or irregular
PR interval: P wave buried in QRS complex
QT interval: QRS complexes usually wide and with phasic variation twisting around a central axis (a few complexes point upward, then a few point downward); ST segments and T waves difficult to discern
Note: Type of ventricular tachycardia associated with prolonged QT interval. Seen with electrolyte disturbances (e.g., hypokalemia, hypocalcemia, and hypomagnesemia) and bradycardia. Administering standard antiarrhythmics (lidocaine, procainamide, etc.) may worsen torsades de pointes. Treatment includes increasing heart rate pharmacologically or by pacing.

Coarse Ventricular Fibrillation



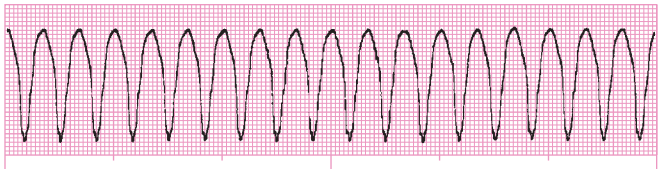
Fine Ventricular Fibrillation



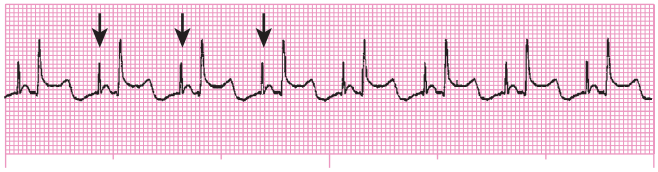
Ventricular Fibrillation

Rate: Absent
Rhythm: None
PR interval: Absent
QT interval: Absent
Note: "Pseudoventricular fibrillation" may be the result of a monitor malfunction (e.g., ECG lead disconnect). Always check for carotid pulse before instituting therapy.

Ventricular Tachycardia



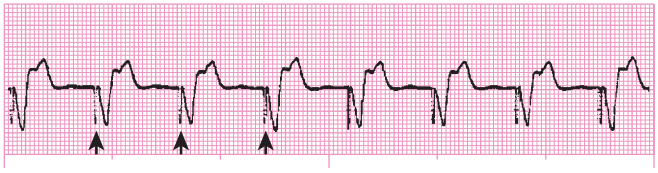
Rate: 100–250 beats/min
Rhythm: No atrial component seen; ventricular rhythm irregular or regular
PR interval: Absent; retrograde P wave may be seen in QRS complex
QT interval: Wide, bizarre QRS complex; ST segment and T wave difficult to determine
Note: In the presence of hemodynamic compromise, immediate direct current (DC) synchronized cardioversion is required. If the patient is stable, with short bursts of ventricular tachycardia, pharmacologic management is preferred. Should be differentiated from supraventricular tachycardia with aberrancy (SVT-A). Compensatory pause and atrioventricular dissociation suggest a PVC. P waves and SR′ (V_1) and slowing to vagal stimulus also suggest SVT-A.



Atrial pacing

Pacemaker Tracings

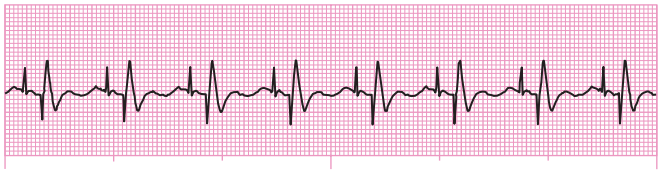
Atrial pacing as demonstrated in this figure is used when the atrial impulse can proceed through the atrioventricular node. Examples are sinus bradycardia and junctional rhythms associated with clinically significant decreases in blood pressure. (Arrows are pacemaker spike.)



Ventricular Pacing

In this tracing, ventricular pacing is evident by absence of atrial wave (P wave) and pacemaker spike preceding QRS complex. Ventricular pacing is used in the presence of bradycardia secondary to atrioventricular block or atrial fibrillation. (Arrows are pacemaker spike.)

DDD Pacing



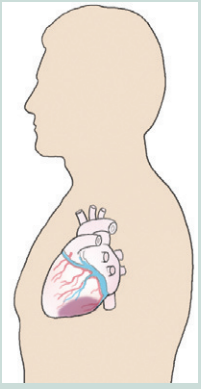
The DDD pacemaker (generator), one of the most commonly used, paces and senses both atrium and ventricle. Each atrial and ventricular complex are preceded by a pacemaker spike.

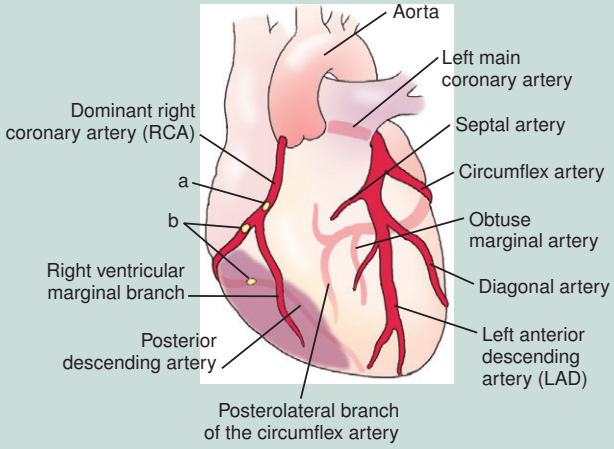
Coronary Artery Disease

Transmural Myocardial Infarction

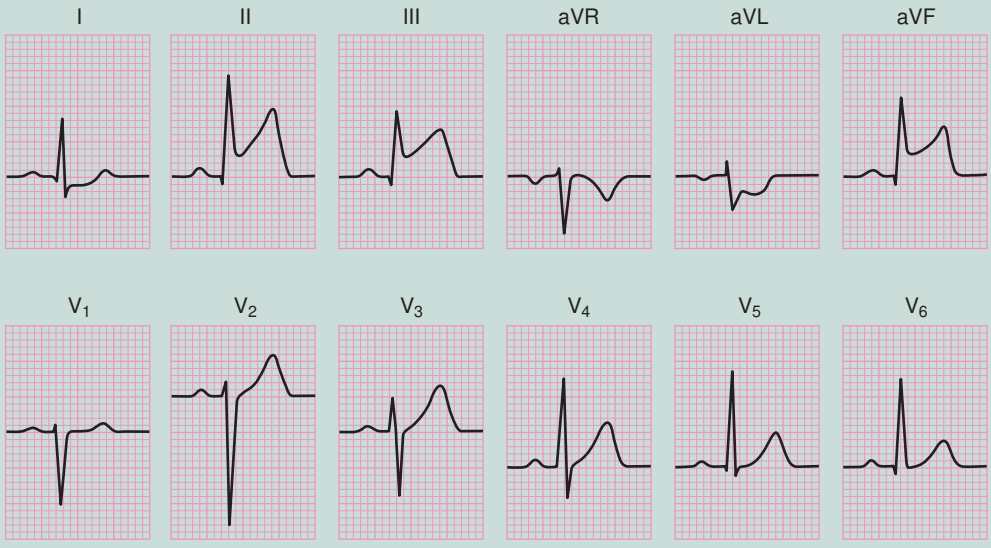
Q waves seen on ECG, useful in confirming diagnosis, are associated with poorer prognosis and more significant hemodynamic impairment. Arrhythmias frequently complicate course. Small Q waves may be normal variant. For myocardial infarction (MI), Q waves > 0.04 second and depth exceeds one third of R wave (inferior wall MI). For inferior wall MI, differentiate from right ventricular hypertrophy by axis deviation.

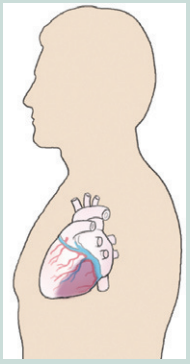
MYOCARDIAL INFARCTIONS

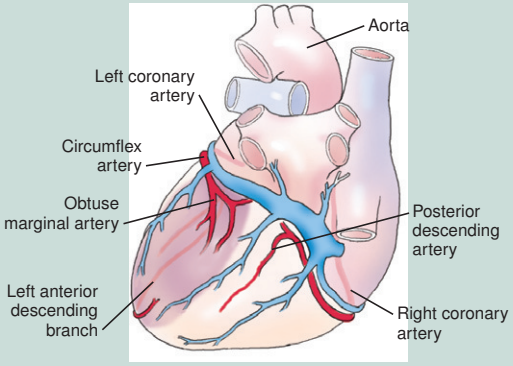




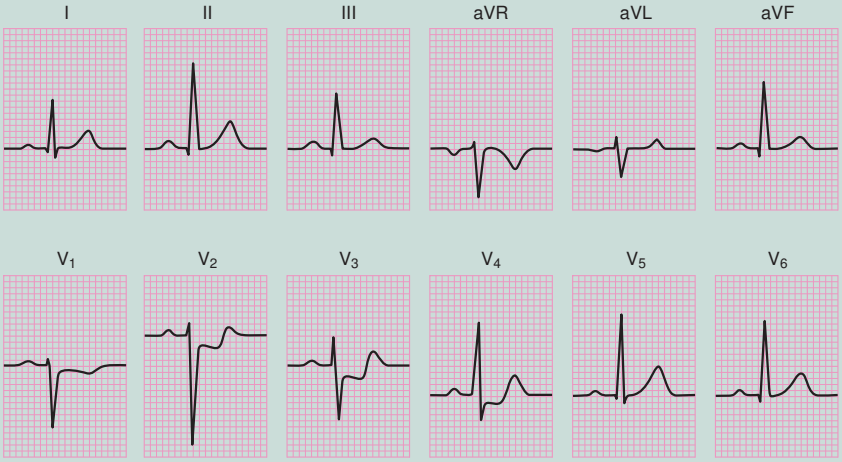
ANATOMIC SITE	*LEADS*	*ECG CHANGES*	*CORONARY ARTERY*
Inferior	II, III, aVF	Q, ↑ST, ↑T	Right

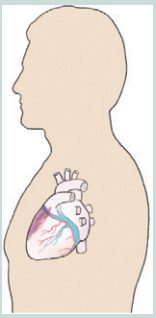


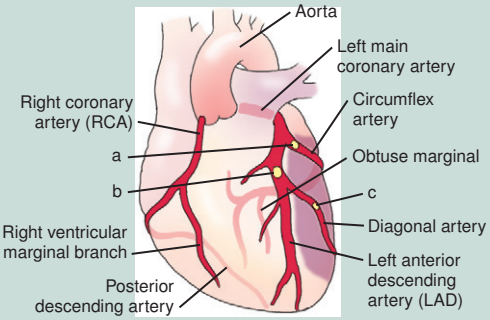




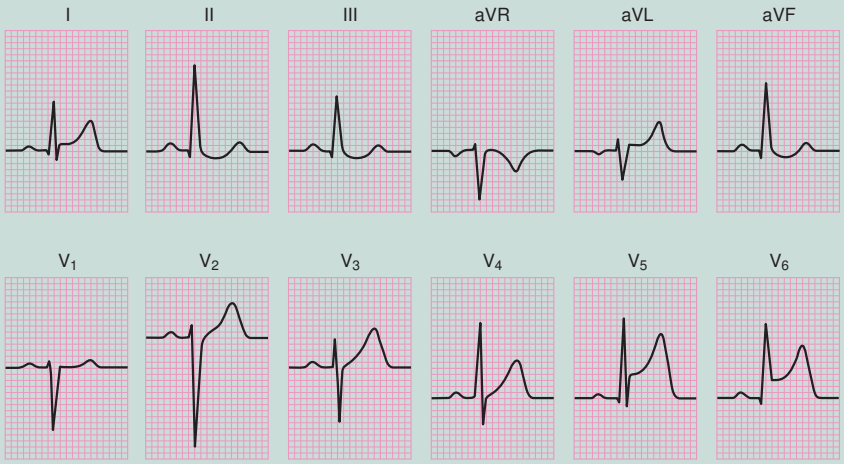
ANATOMIC SITE	*LEADS*	*ECG CHANGES*	*CORONARY ARTERY*
Posterior	V_{1-2}	↑R, ↓ST, ↓T	Left circumflex

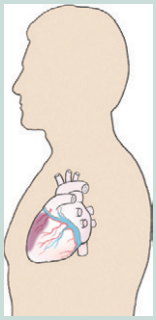


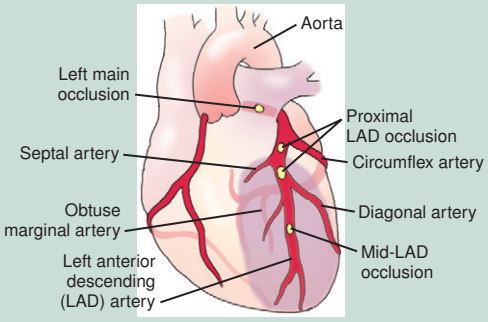




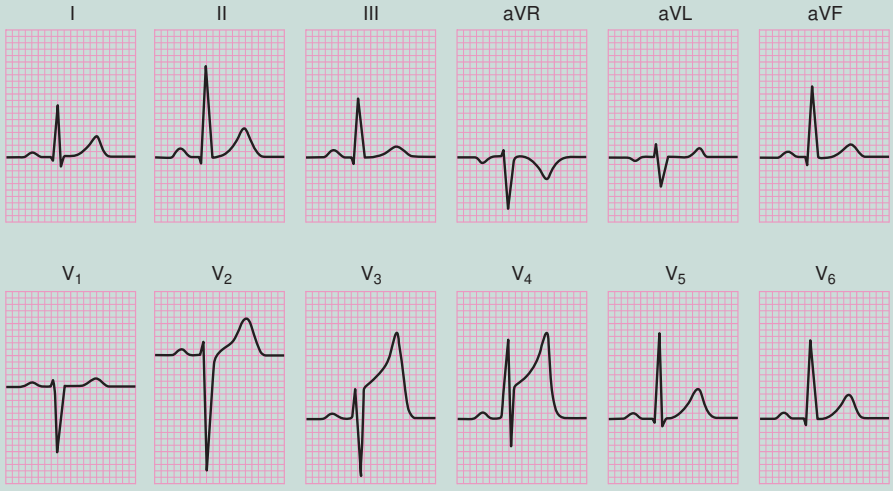
ANATOMIC SITE	*LEADS*	*ECG CHANGES*	*CORONARY ARTERY*
Lateral	I, aVL, V_5-V_6	Q, ↑ST, ↑T	Left circumflex

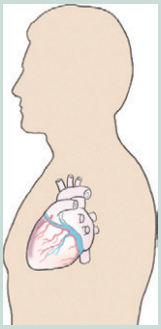


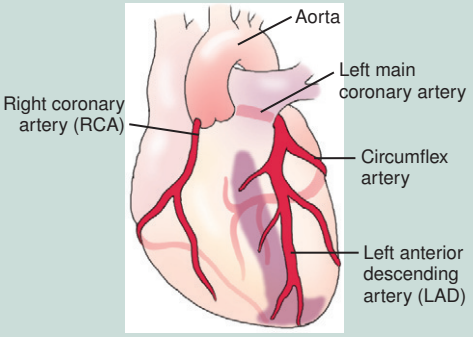




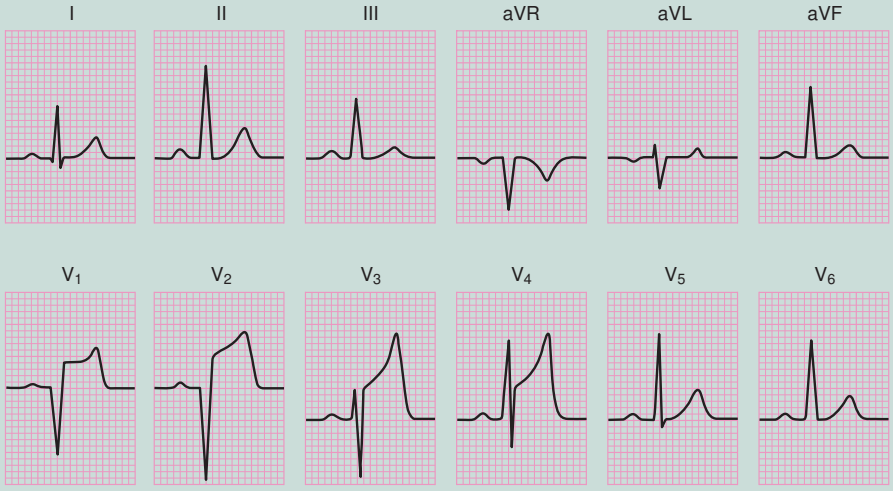
ANATOMIC SITE	*LEADS*	*ECG CHANGES*	*CORONARY ARTERY*
Anterior	I, aVL, V_1-V_4	Q, ↑ST, ↑T	Left



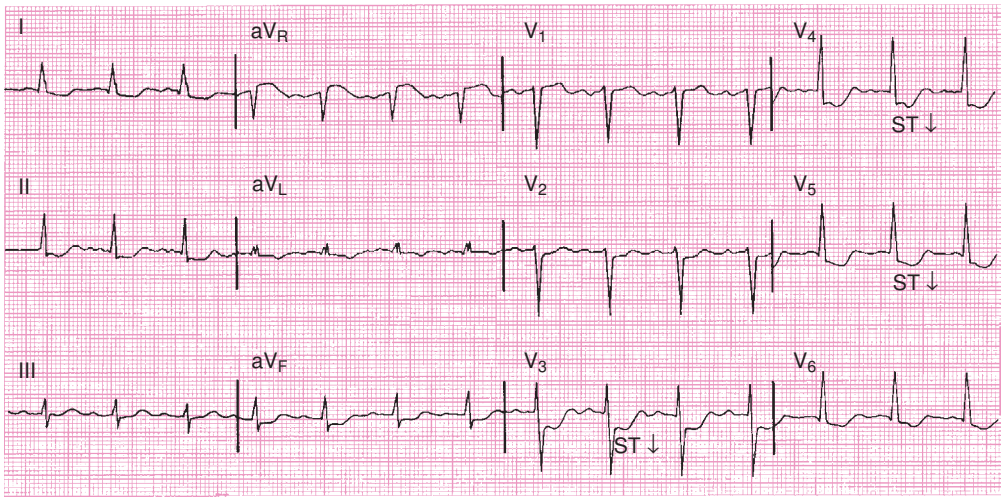




ANATOMIC SITE	*LEADS*	*ECG CHANGES*	*CORONARY ARTERY*
Anteroseptal	V_1-V_4	Q, ST, T	Left anterior descending

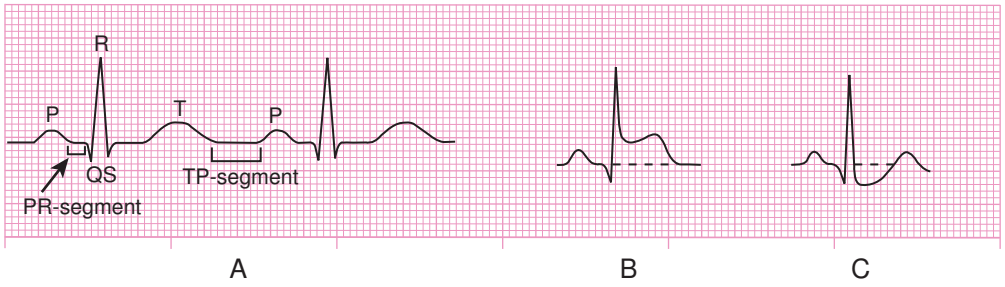


Subendocardial myocardial infarction



Persistent ST-segment depression and/or T-wave inversion in the absence of Q wave. Usually requires additional laboratory data (e.g., isoenzymes) to confirm diagnosis. Anatomic site of coronary lesion is similar to that of transmural myocardial infarction electrocardiographically.

Myocardial Ischemia



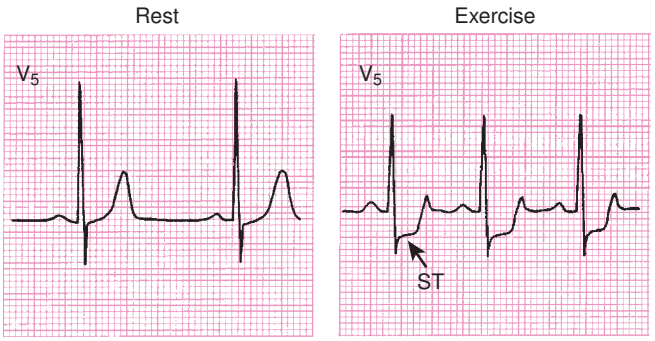
Rate: Variable
Rhythm: Usually regular, but may show atrial and/or ventricular arrhythmias
PR interval: Normal
QT interval: ST segment depressed; J-point depression; T-wave inversion; conduction disturbances; coronary vasospasm (Prinzmetal)

ST segment elevation; (A) TP and PR intervals are baseline for ST-segment deviation, (B) ST-segment elevation, (C) ST-segment depression

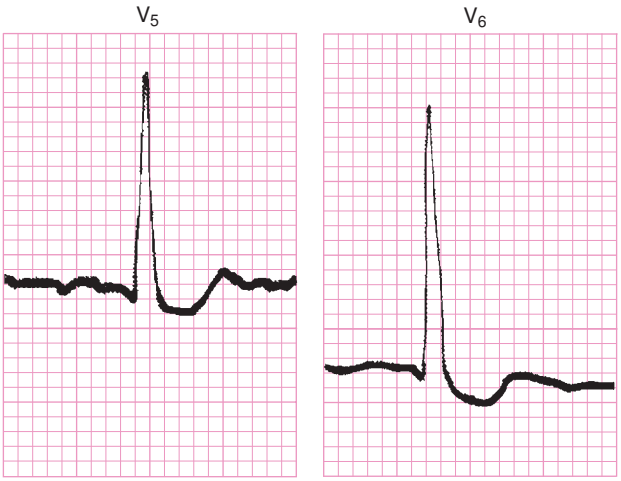
Note: Intraoperative ischemia usually is seen in the presence of "normal" vital signs (e.g., ±20% of preinduction values).

Other Important ECG Changes

Digitalis Effect



Rate: < 100 beats/min
Rhythm: Regular
PR interval: Normal or prolonged
QT interval: ST-segment sloping ("digitalis effect")
Note: Digitalis toxicity can be the cause of many common arrhythmias (e.g., premature ventricular contractions, second-degree heart block). Verapamil, quinidine, and amiodarone cause an increase in serum digitalis concentration.

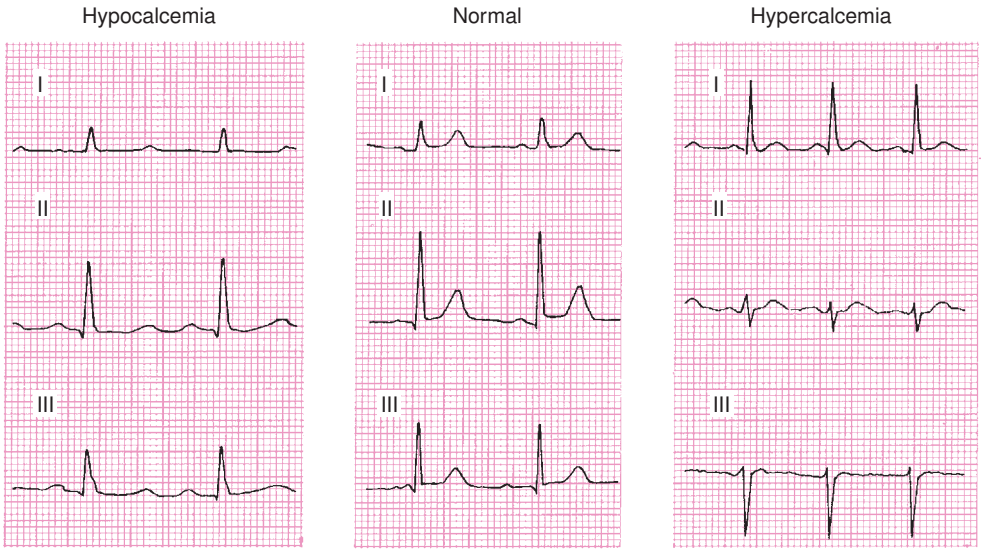


Electrolyte Disturbances

	↓ Ca^{2}	↑ Ca^{2+}	↓ K^{+}	↑ K^{+}
Rate	< 100 beats/min	< 100 beats/min	< 100 beats/min	< 100 beats/min
Rhythm	Regular	Regular	Regular	Regular
PR interval	Normal	Normal/increased	Normal	Normal
QT interval	Increased	Decreased	T wave flat U wave	T wave peaked QT increased

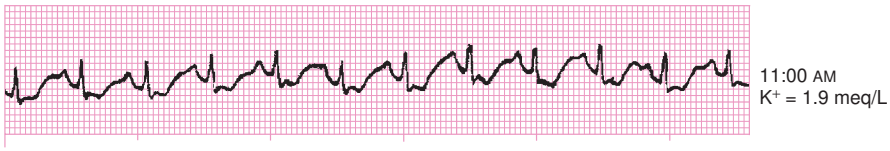
Note: ECG changes usually do not correlate with serum calcium. Hypocalcemia rarely causes arrhythmias in the absence of hypokalemia. In contrast, abnormalities in serum potassium concentration can be diagnosed by ECG. Similarly, in the clinical range, magnesium concentrations rarely are associated with unique ECG patterns. The presence of a "u" wave (> 1.5 mm in height) also is seen in left main coronary artery disease, certain medications, and long QT syndrome.

Calcium

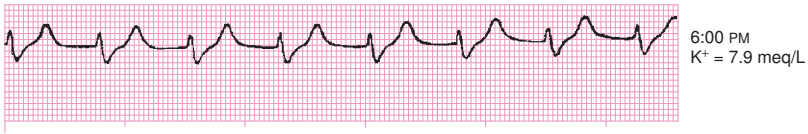


Potassium

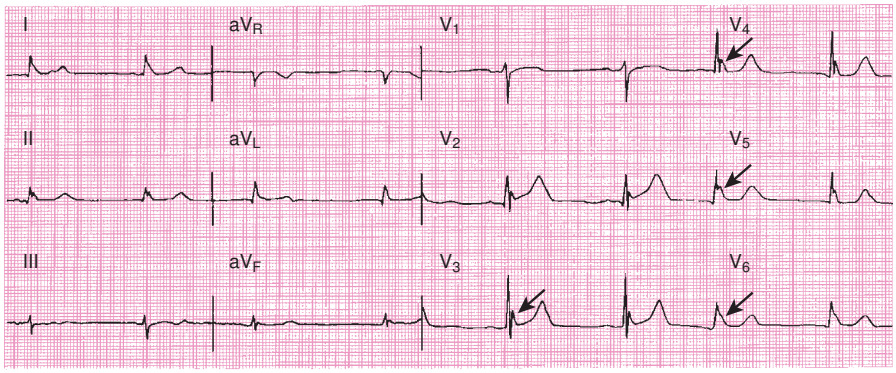
Hypokalemia (K^+ = 1.9 mEq/L)



Hyperkalemia (K^+ = 7.9 mEq/L)

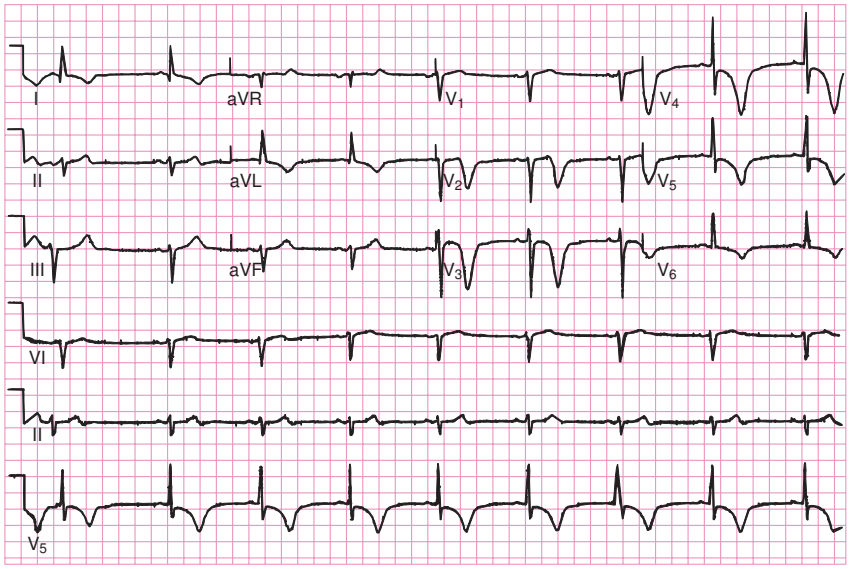


Hypothermia



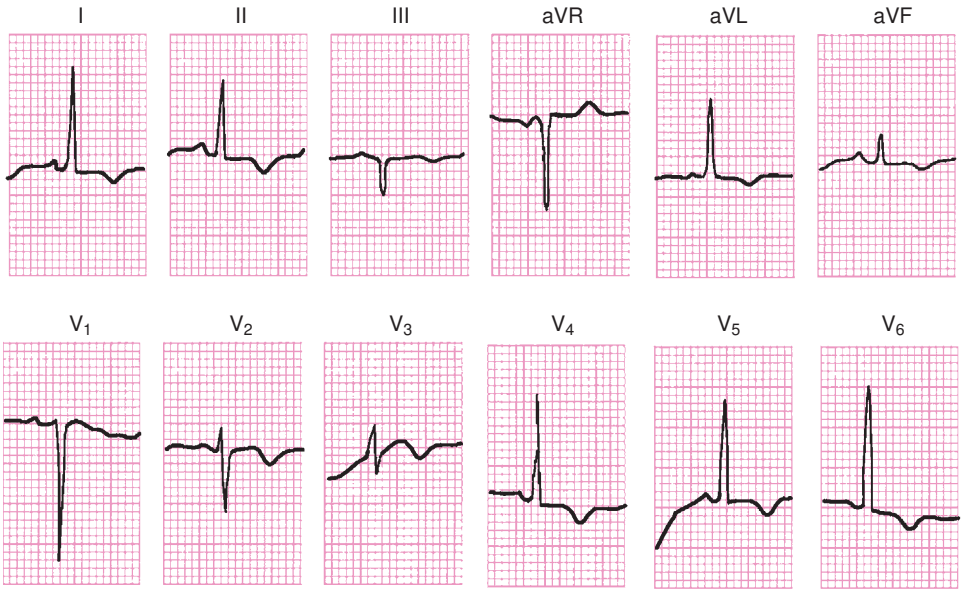
Rate: < 60 beats/min
Rhythm: Sinus
PR interval: Prolonged
QT interval: Prolonged
Note: Seen at temperatures less than 33° C with ST-segment elevation (J point or Osborn wave). Tremor caused by shivering or Parkinson disease may interfere with ECG interpretation and may be confused with atrial flutter. May represent normal variant of early ventricular repolarization. (Arrow indicates J point or Osborn waves.)

Subarachnoid Hemorrhage



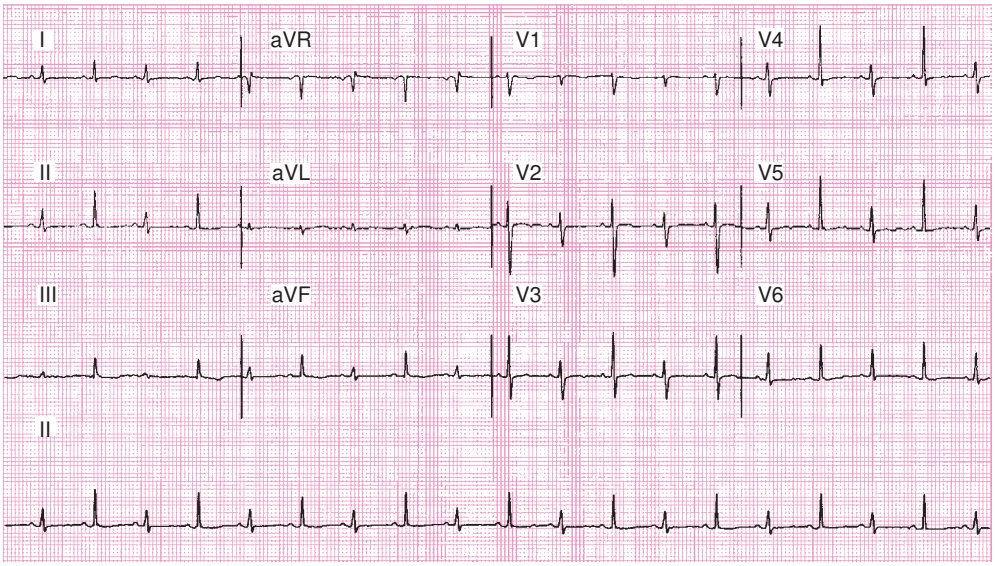
Rate: < 60 beats/min
Rhythm: Sinus
PR interval: Normal
QT interval: T-wave inversion is deep and wide. Prominent U waves are seen. Sinus arrhythmias are observed. Q waves may be seen and may mimic acute coronary syndrome.

Pericarditis



Rate: Variable
Rhythm: Variable
PR interval: Normal
QT interval: Diffuse ST- and T-wave changes with no Q wave and seen in more leads than a myocardial infarction.

Pericardial Tamponade



Rate: Variable
Rhythm: Variable
PR interval: Low-voltage P wave
QT interval: Seen as electrical alternans with low-voltage complexes and varying amplitude of P, QRS, and T waves with each heartbeat