Easy ECG

Interpretation
Differential Diagnoses

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For Elena, Daniela,
and my parents

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Furthermore, I would like to thank all my colleagues in and around Dresden and Riesa, working in hospitals and private practices, who with their numerous and important suggestions have contributed to the success of this book.

Riesa, Hans-Holger Ebert
Summer 2004

Foreword

Why do we need another ECG book when so many good, and some not so good, ECG textbooks, atlases, manuals, and guides are available? My former colleague of many years, Dr. Hans-Holger Ebert, has written an ECG guide which bridges the gap between the ECG atlases and textbooks based on electrophysiology. It gives the reader a closer understanding of the subject, including valuable ECG conduction diagrams, which can be used for teaching purposes, and clear, standardized schematic diagrams. Corresponding characteristic ECGs and the basics of electrophysiology are explained in a manner which makes them easily comprehensible. Konrad Spang, one of the pioneers of cardiology in Germany, said in 1957: "The correct interpretation of rhythm disorders often demands great effort and detailed in-depth analysis. Conducting such analyses is of huge didactic value. This is a pathway for the development of the ability to make exact observations and also sharpens the senses in other areas…"

Easy ECG meets these demands. The author has linked electrocardiographic phenomena and the underlying electrophysiological principles with practical conclusions for clinical diagnosis and treatment. Many years of experience in cardiology in hospital and outpatient settings have been of value to the author, and the numerous internships he has supervised and lectures he has given have benefited his teaching skills.

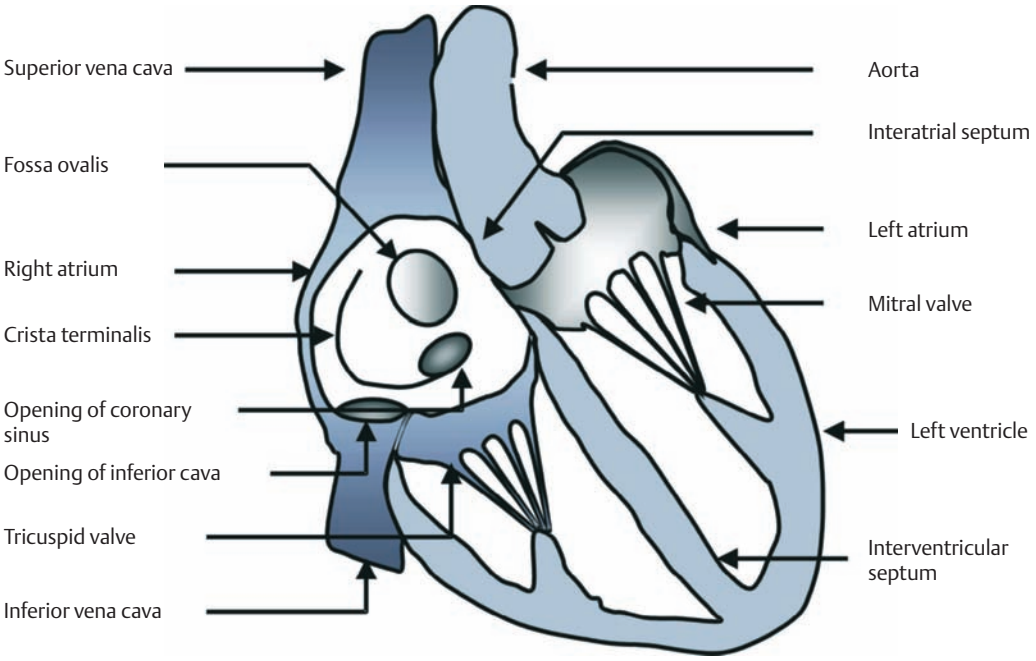
I hope *Easy ECG* will draw many interested readers and contribute to an in-depth understanding of current diagnostic and therapeutic options to be derived from ECG analysis, just as a pilot boat guides ships safely in and out the harbor.

Hans Volkmann
Professor and Senior Consultant
Internal Medicine
Erzgebirgsklinikum Annaberg

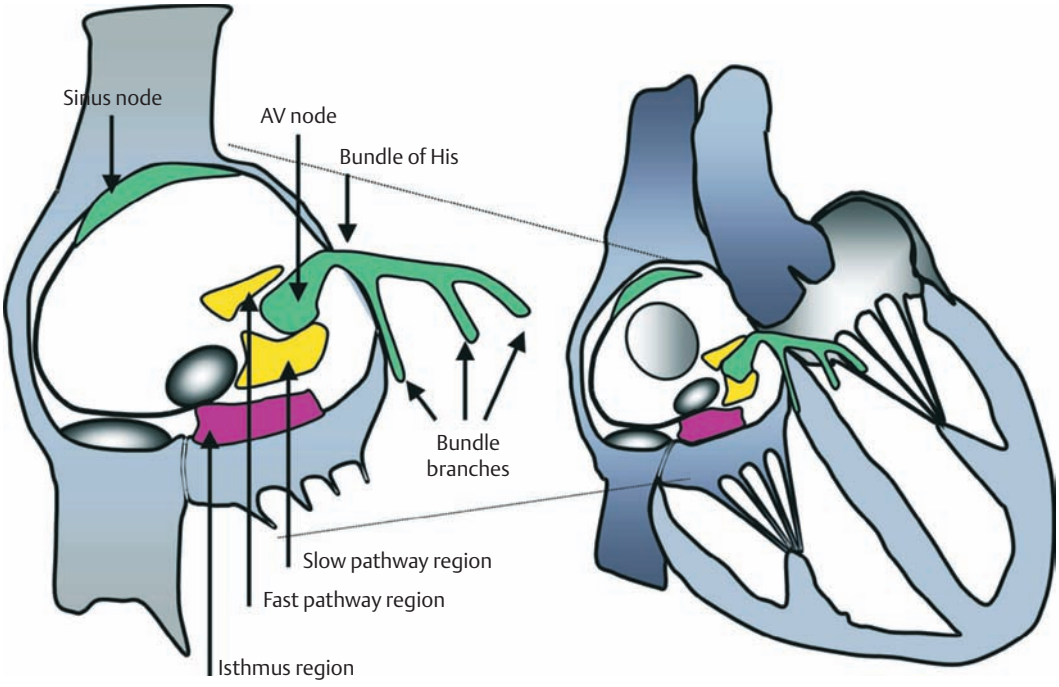
Contents

1 The Human Heart

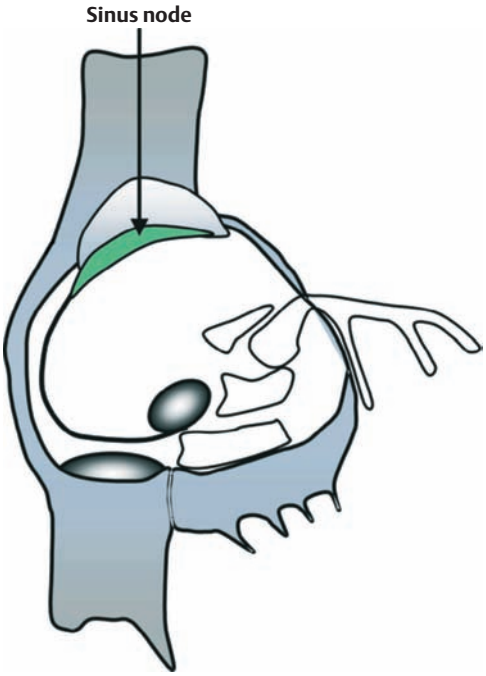
1.1 Basic Anatomy



1.2 Anatomy of the Conduction System



1.3 The Sinus Node



Size/form: 10–20 mm, spindle-shaped

Location:
In the first third of the sulcus terminalis along the axis between the opening of the superior vena cava and the right subepicardial auricle

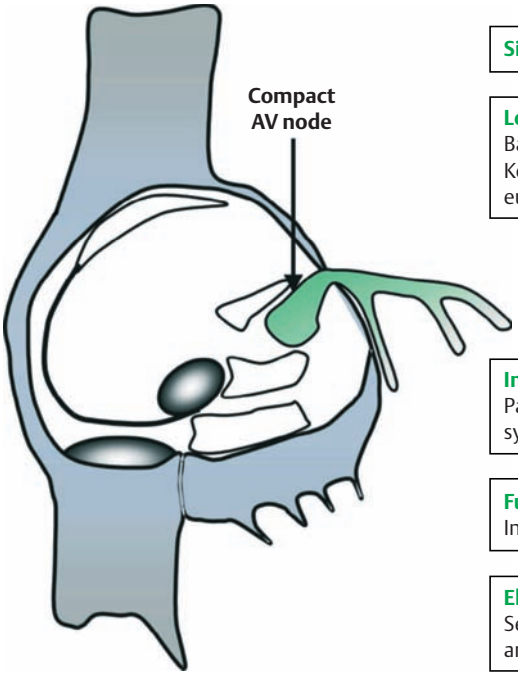
Blood supply:
Sinus node artery (arises from RCA in 50–59%, from RCX in 20–38%, dual blood supply in 3–30%)

Innervation:
Parasympathetic postganglionic and sympathetic fibers

Function: Physiological site of impulse formation

Electrophysiological significance:
Site of sinus node reentry

1.4 The Compact AV Node



Size/form: ca. 1 × 3 × 5 mm

Location:
Base of the right atrium at the point of the so-called Koch triangle (between the tricuspid annulus and the eustachian valve), subendocardial

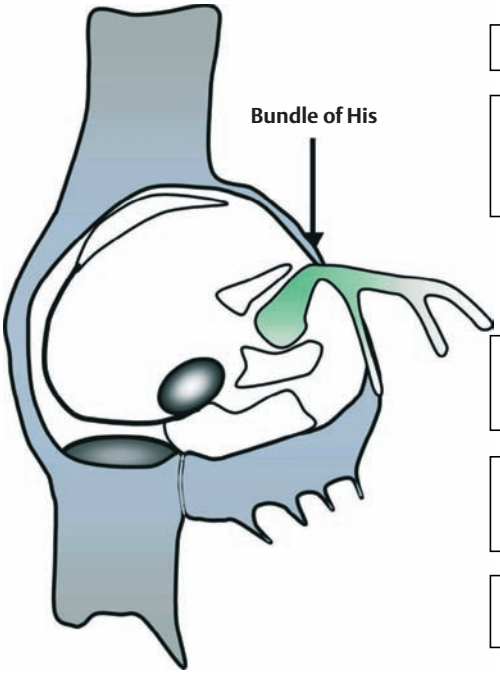
Blood supply:
AV node artery (arises from RCA in 90%, in 10% from RCX)

Innervation:
Parasympathetic postganglionic and sympathetic fibers

Function:
Impulse conduction from the atrium to the bundle of His

Electrophysiological significance:
Secondary site for impulse formation, area of slowest impulse conduction (0.2 m/s)

1.5 The Bundle of His



Size/form: 10 – 20 mm

Location:
Continuation of the compact AV node, penetrates the annulus fibrosus, subendocardial course in the pars membranacea of the interventricular septum

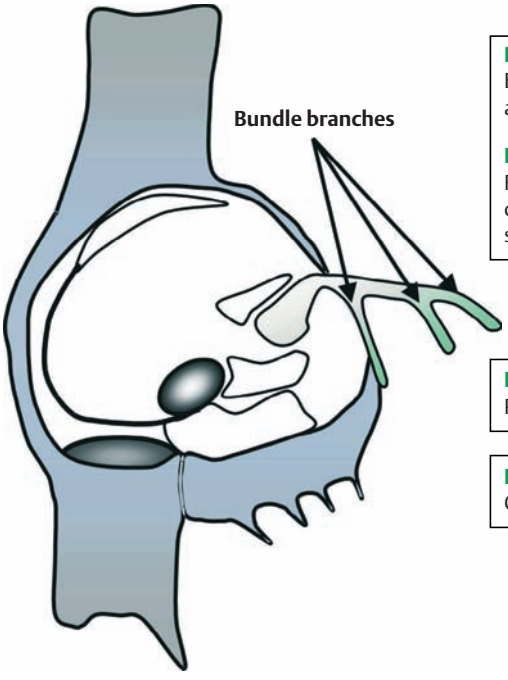
Blood supply:
AV node artery (90% from RCA, 10% from RCX)

Innervation:
Parasympathetic postganglionic and sympathetic fibers

Function:
Impulse conduction from the compact AV node to the bundle branches

Electrophysiological significance:
Possible blockade of AV conduction

1.6 The Bundle Branches



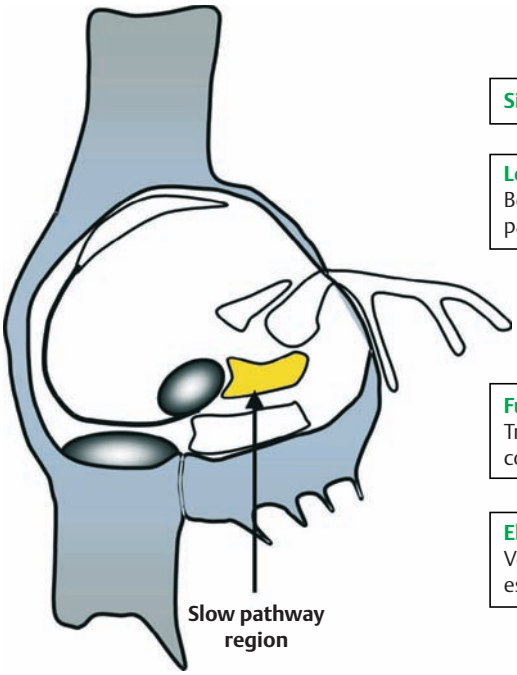
Right bundle branch:
Extension of the bundle of His coursing along the right aspect of interventricular septum

Left bundle branch:
Runs through the ventricular septum to the left and divides into two branches: the inferoposterior and the superoanterior

Innervation:
Parasympathetic and sympathetic

Function:
Conduction of the electrical impulse to the myocardium

1.7 The Slow Pathway Region



Size/form: Variable expansion of the fibers

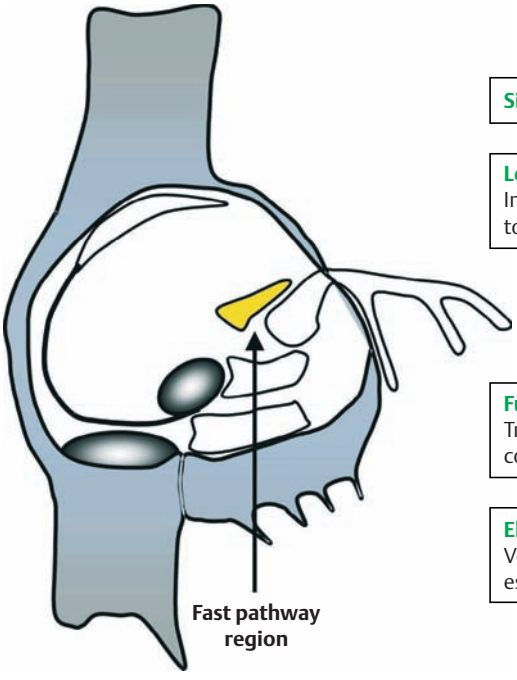
Location:
Between the opening of the coronary sinus and the compact AV node (posteroinferior to the compact AV node)

Innervation and blood supply:
No reliable data to date

Function:
Transition zone between the atrial myocardium and the compact AV node

Electrophysiological significance:
Very premature impulses are conducted with delay; essential component of AV node reentry tachycardia

1.8 The Fast Pathway Region



Size/form: Variable expansion of the fibers

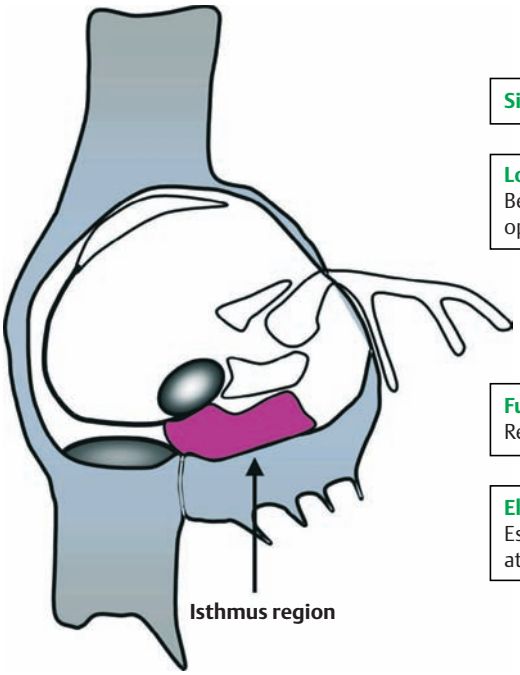
Location:
In the region of the intraatrial septum anterosuperior to the KOCH triangle

Innervation and blood supply:
No reliable data to date

Function:
Transition zone between the atrial myocardium and the compact AV node

Electrophysiological significance:
Very premature impulses are not conducted; essential component of AV node reentry tachycardia

1.9 The Isthmus Region



Size/form: Variable expansion of the area

Location:
Between the tricuspid valve, inferior vena cava, and the opening of the coronary sinus

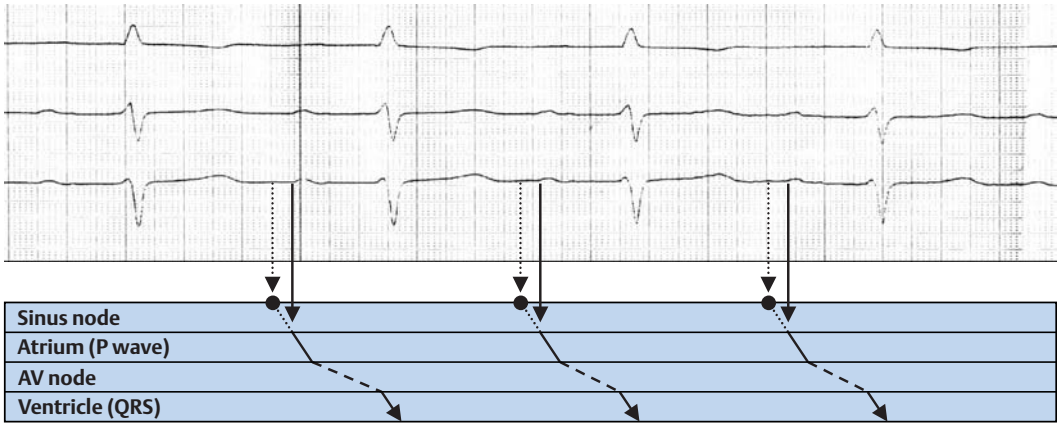
Innervation and blood supply:
No reliable data to date

Function:
Region of delayed impulse conduction

Electrophysiological significance:
Essential component of reentry circuit in typical atrial flutter

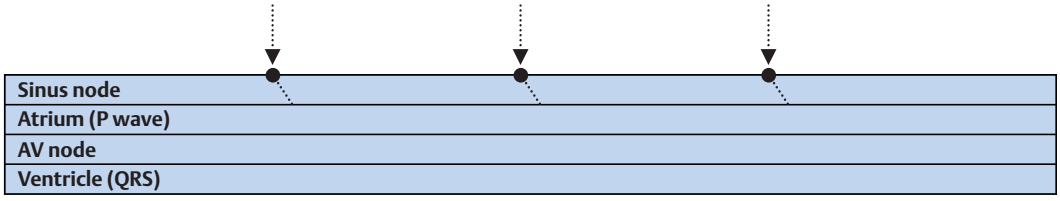
2 The Conduction Diagrams and Text Fields

2.1 Basic Concepts



The conduction diagram represents a simplified projection of impulse conduction at the various anatomical levels of the heart on a time axis. Using typical ECG recordings a 1:1 correlation of the impulse components is made with the anatomical levels. The arrows are designed to help with recognition of the start of sinus rhythm and of the P wave.

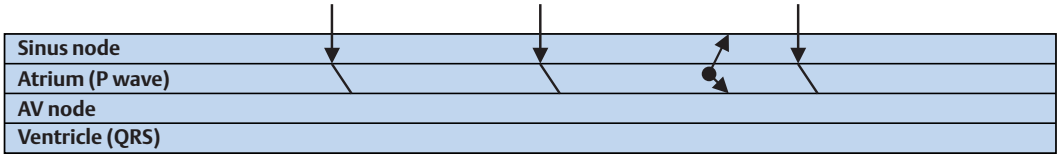
2.2 Impulse Formation in the Sinus Node



The formation of the electrical impulse in the sinus node is necessary to give rise to so-called sinus rhythm. Formation of an impulse in the sinus node is not conspicuous in the superficial ECG itself and can only be detected indirectly from atrial depolarization (P wave).

To better explain rhythm disorders in the region of the sinus node, impulse formation and conduction at the sinus node are represented schematically using a dotted arrow (in the ECG) and schematically in the diagrams by a dotted line ( ).

2.3 Depolarization of the Atria (P Wave)

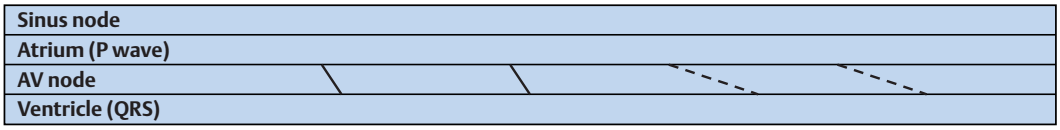


The first electrical activity conspicuous in sinus rhythm is depolarization of the atria; a P wave occurs. The beginning of the P wave is indicated by a solid arrow (↓) and in the diagrams by a solid line (\ ).

In sinus rhythm the P wave has the largest positive deflection in lead II (the atrial vector runs parallel to lead II in sinus rhythm).

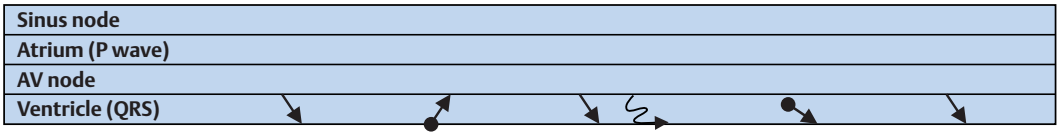
The occurrence of supraventricular extrasystoles (SVES) is indicated by a dot with two arrows (⤢) and always occurs midway at atrial level.

2.4 Conduction of the AV Node



Following successful atrial depolarization, the electrical impulse is further conducted via the AV node, the bundle of His and the bundle branches. The individual components of conduction, however, cannot be visualized in the superficial ECG. The time from the end of the P wave to formation of the QRS complex is equated with a simplified explanation of rhythm disorder in the AV node-His-bundle branch region. A dashed line (⤡) is always used in the diagrams to indicate pathological conduction, otherwise a solid line (\ ) is employed.

2.5 Depolarization of the Ventricles

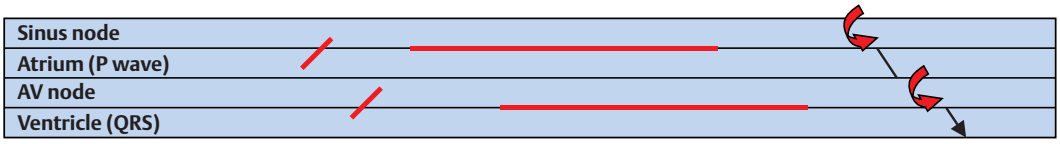


The depolarization of the ventricles is represented by a QRS complex and is indicated by a solid arrow ( ).

Ventricular ectopics are represented by a dot with a solid arrow ( ).

Asynchronous ventricular excitation, for example due to bundle branch block is indicated by a wavy line with an arrow ( ).

2.6 Further Explanations (I)

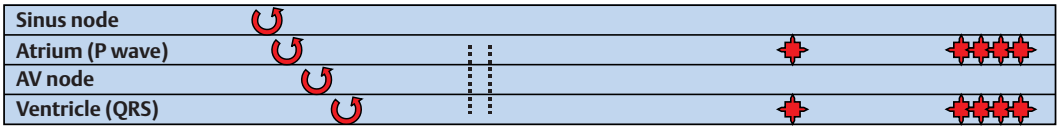


Blockade of conduction arises between the sinus node and the atrium or in the region of the AV node (between the atrium and the ventricle).

A complete conduction block is characterized by a red line; a single interruption of conduction is represented by a diagonal line ( ) and a longer block by a horizontal line ( ).

Pacemaker stimulation is indicated by a red arrow ( ).

Further Explanations (II)



Reentry mechanisms are indicated by ( ).

An accessory conduction pathway (e.g., a Kent fiber) is indicated by a dotted double line ( ).

The occurrence of supraventricular or ventricular ectopics is characterized by a star ( ).

With supraventricular non-reentry tachycardia (e.g., atrial fibrillation) and ventricular non-reentry tachycardia a group of stars are seen ( ).

Half-moon symbols indicate the direction of propagation of the impulse: ( ) or ( ).

Text Fields

ECG changes are explained under key headings with respect to mechanism, characteristic features in the superficial ECG, etiology, and corresponding treatment options.

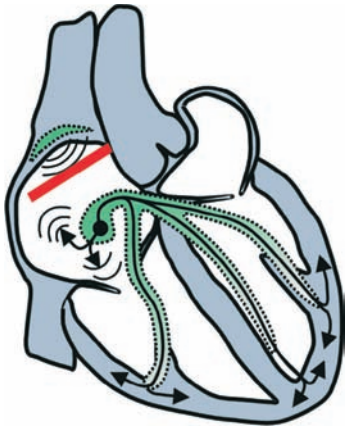
Mechanism:

ECG characteristics:

Etiology:

Treatment:

Text Fields

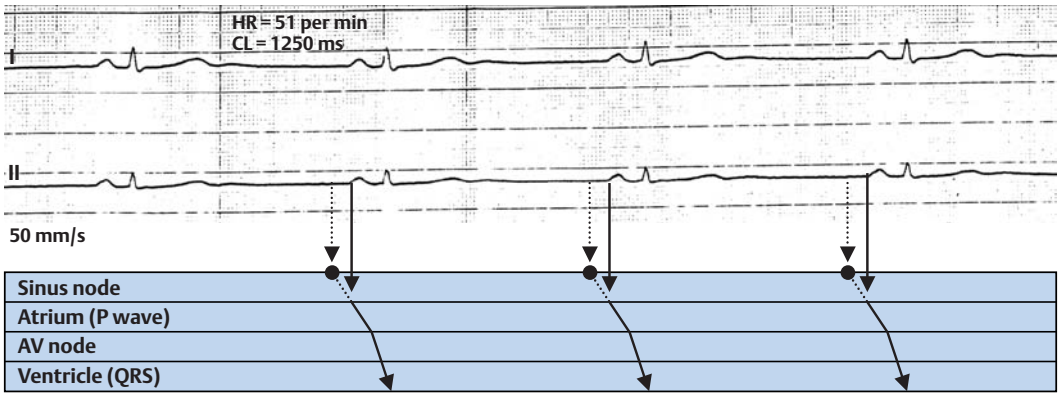


Differential diagnoses are also provided under a key heading in association with a general ECG diagram.

Differential diagnosis:

3 Cardiac Rhythm Disorders and Conduction Disorders

Sinus Bradycardia



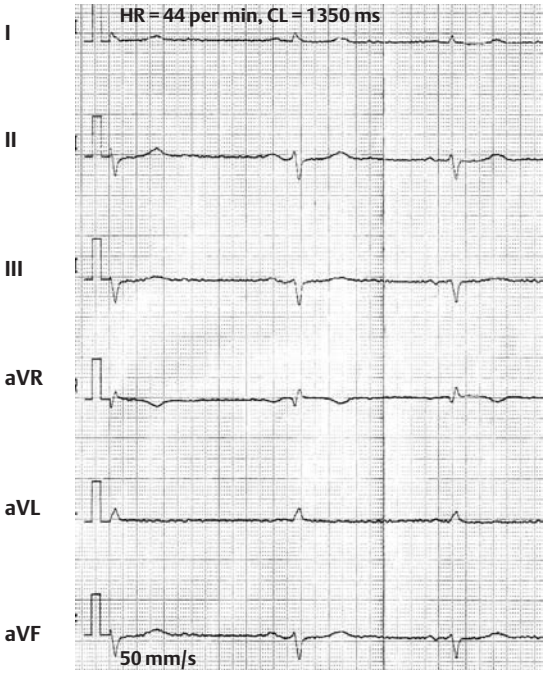
Mechanism:
- Slow diastolic depolarization of the sinus node

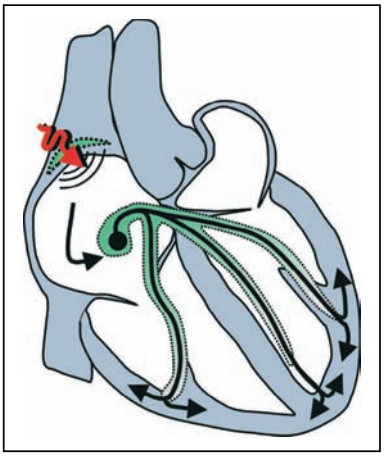
ECG characteristics:
- Sinus rhythm with a frequency of less than 60 per minute

Etiology:
- Sinus node syndrome, medication that may cause bradycardia, raised intracranial pressure, icterus, aortic stenosis, hypothyroidosis, acute myocardial infarction (posterior wall)
- Physiological in nature in cases of marked vagotonia

Treatment:
- If the patient is symptomatic, medications that increase cardiac frequency (e.g., atropine) or insertion of pacemaker
- Cessation of medication that may cause bradycardia (if possible)

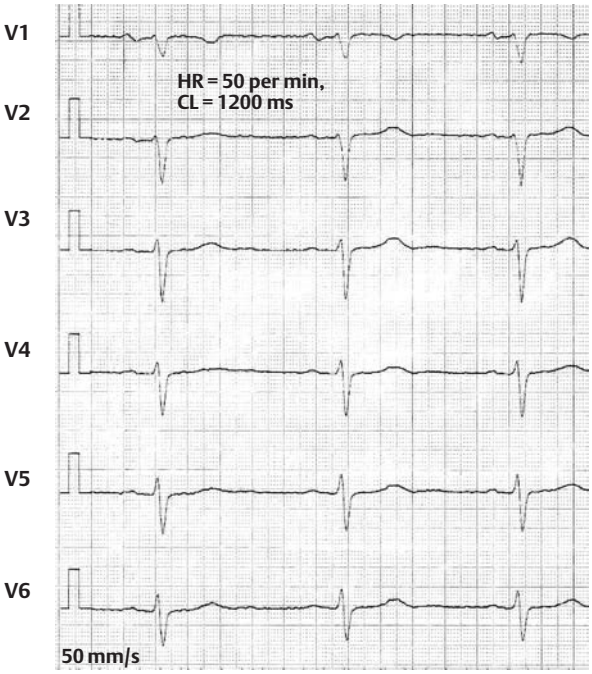
Sinus Bradycardia

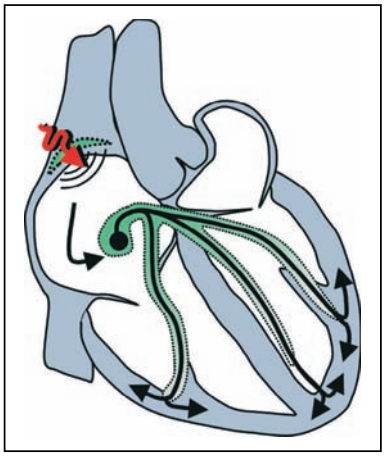




Differential diagnosis:
- Sinus arrhythmia
- 2nd degree AV block (Mobitz/Wenckebach)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude

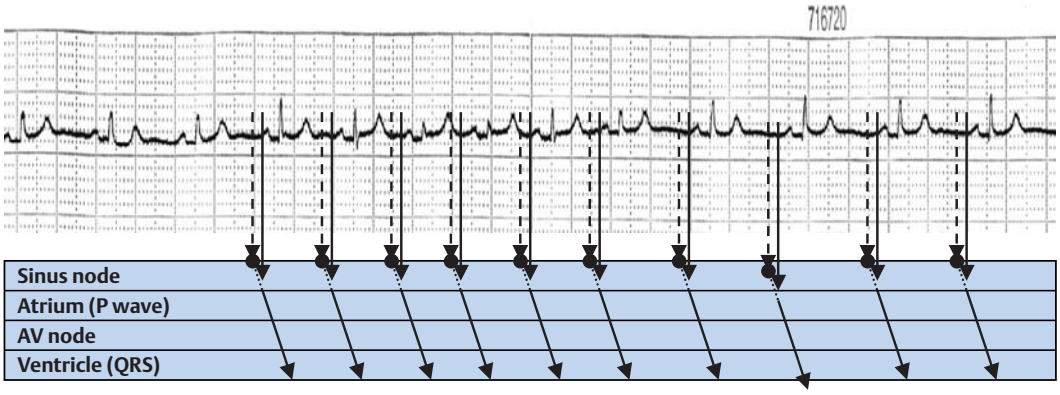
Sinus Bradycardia





Differential diagnosis:
- Sinus arrhythmia
- 2nd degree AV block with 2 : 1 block (Mobitz/Wenckebach)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude

Sinus Arrhythmia



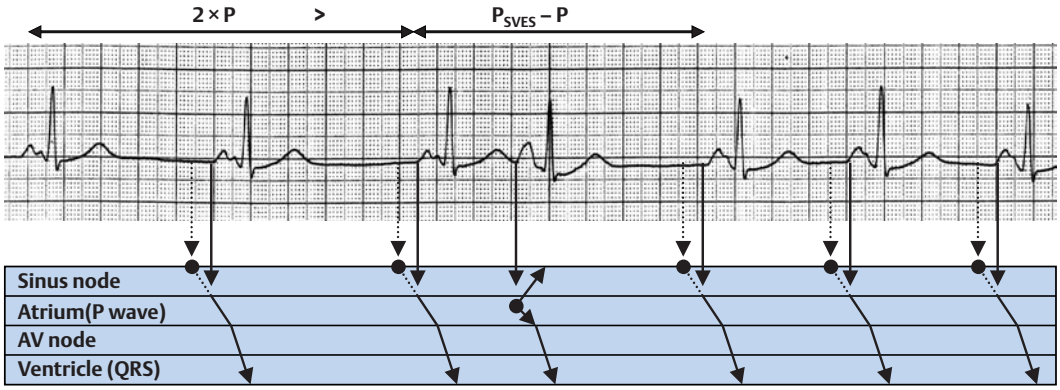
Mechanism:
- Physiological and respiration-dependent fluctuation of the frequency of the sinus node

ECG characteristics:
- Continuous transition between decrease and increase of the time interval between P waves; no pauses

Etiology:
- Physiological in nature

Treatment:
- none

Postextrasystolic Pause Following SVES



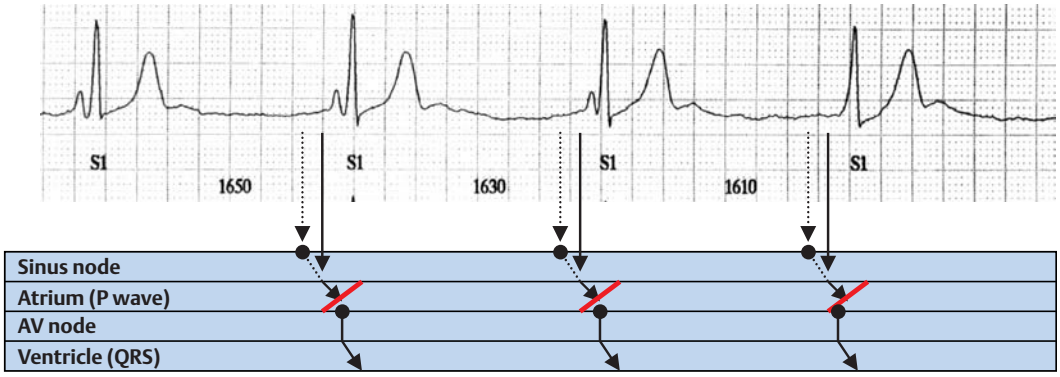
Mechanism:
- The basic rhythm is influenced by extrasystole; following an SVES, diastolic depolarization of the sinus node is extinguished (reset) and begins anew

ECG characteristics:
- Noncompensatory pause; sum of the PP intervals of two normal beats > sum of PP intervals before and after the pause

Etiology:
- Often physiological in nature, otherwise in degenerative and inflammatory heart disease; heart defects

Treatment:
- none

Sinus Bradycardia with AV Dissociation



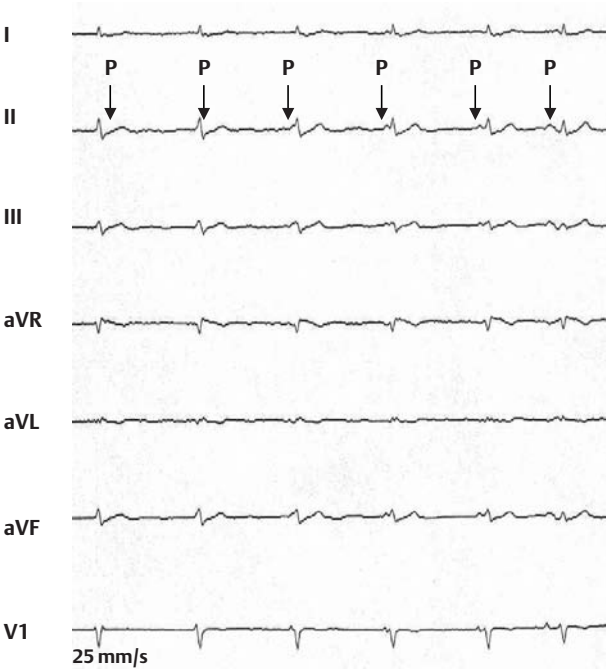
Mechanism:
- In sinus bradycardia the "too slow" sinus node can be "overtaken" by a faster focus

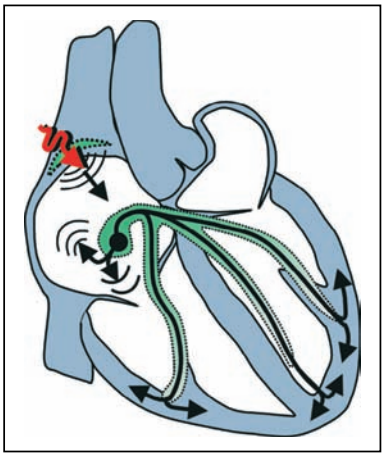
ECG characteristics:
- From "wandering" of the P wave into the QRS complex to complete amalgamation of the P wave and the QRS complex

Etiology:
- Sinus node syndrome
- Medication that may cause bradycardia
- In marked vagotonia

Treatment:
- As a rule not necessary
- Cessation of medicines that may cause bradycardia

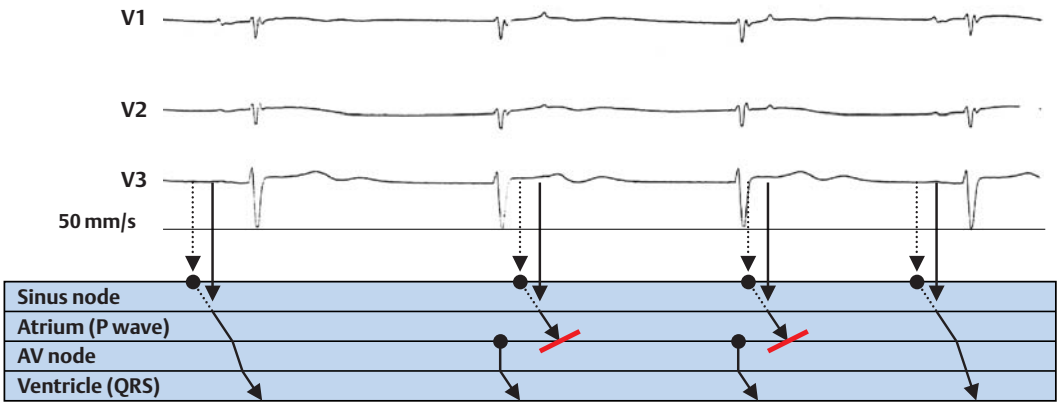
AV Dissociation with Transition to Sinus Rhythm





Differential diagnosis:
- Sinus bradycardia (P wave present)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block (P wave present)

Deceleration of Sinus Node Frequency with Nodal Escape Rhythm



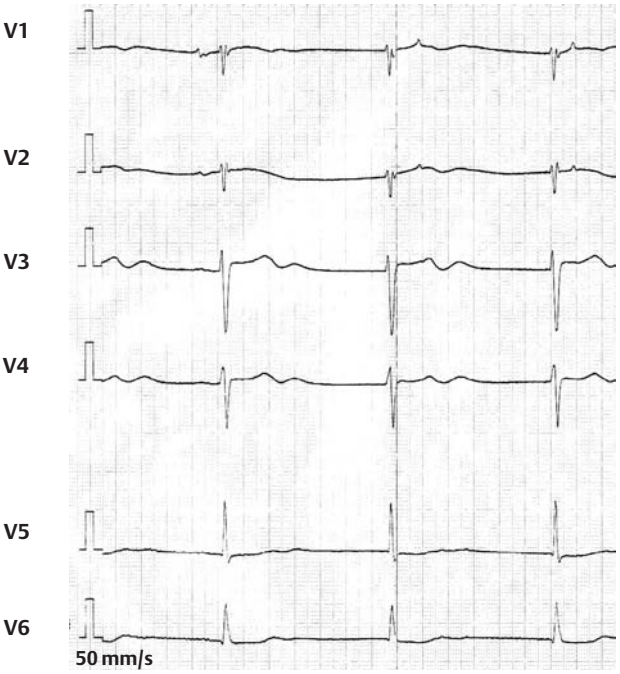
Mechanism:
- Intermittent sinus bradycardia with occurrence of escape rhythm, the atria are charged by the sinus node, the ventricles by the escape focus

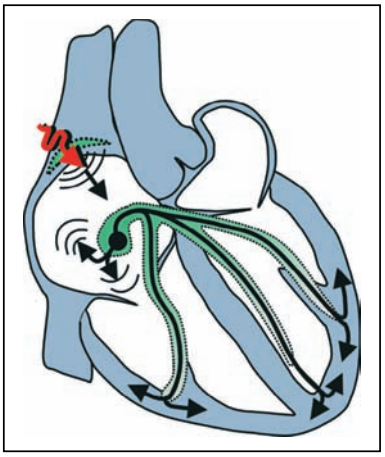
ECG characteristics:
- "Pause with no P wave" bridged by narrow QRS complex
- Projection of the P wave into the ST segment

Etiology:
- Almost exclusively sinus node syndrome
- Medication that may cause bradycardia

Treatment:
- Cessation of medicines that may cause bradycardia
- As a rule no indication for pacemaker

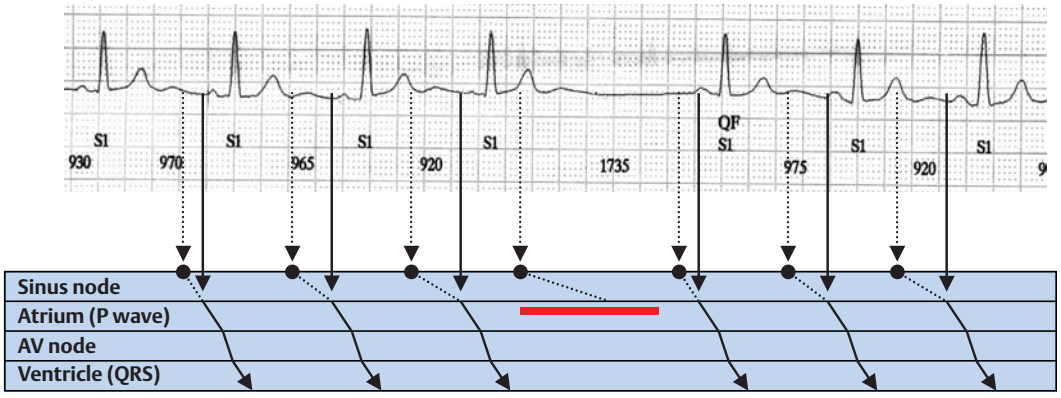
Deceleration of Sinus Node Frequency with Nodal Escape Rhythm





Differential diagnosis:
- Sinus bradycardia (P wave present), 2nd degree SA block (no P wave)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block

Sinoatrial 2nd Degree Block, Wenckebach Type



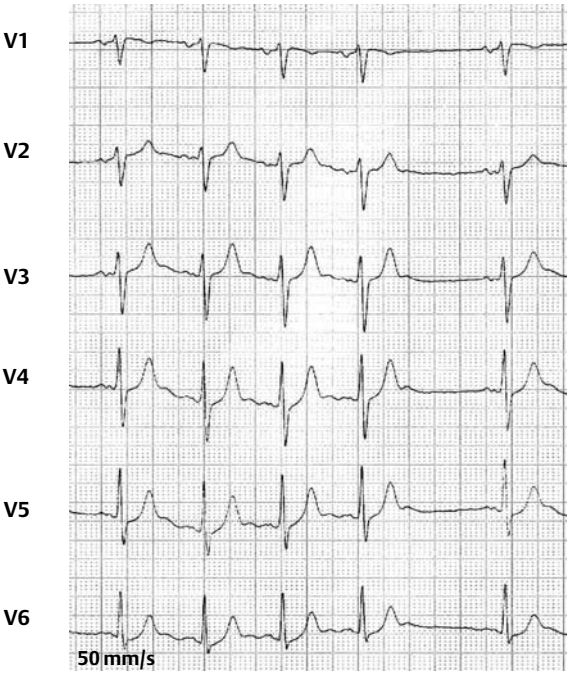
Mechanism:
- Single intermittent interruption of impulse formation in the sinus node

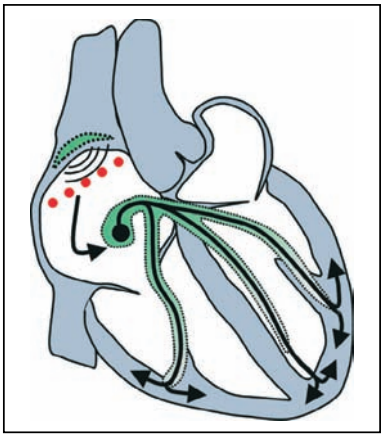
ECG characteristics:
- "Pause with no P wave"
- Absence of a sinus P wave with shorting of the PP intervals
- Length of pause < 2 × PP interval

Etiology:
- Often physiological, otherwise in the context of SSS, CHD, CMP, heart defects, hypertension, medication

Treatment:
- None in asymptomatic patients
- Cessation of medicines that may cause bradycardia in symptomatic patients and possibly insertion of a pacemaker in case of "severe" symptoms

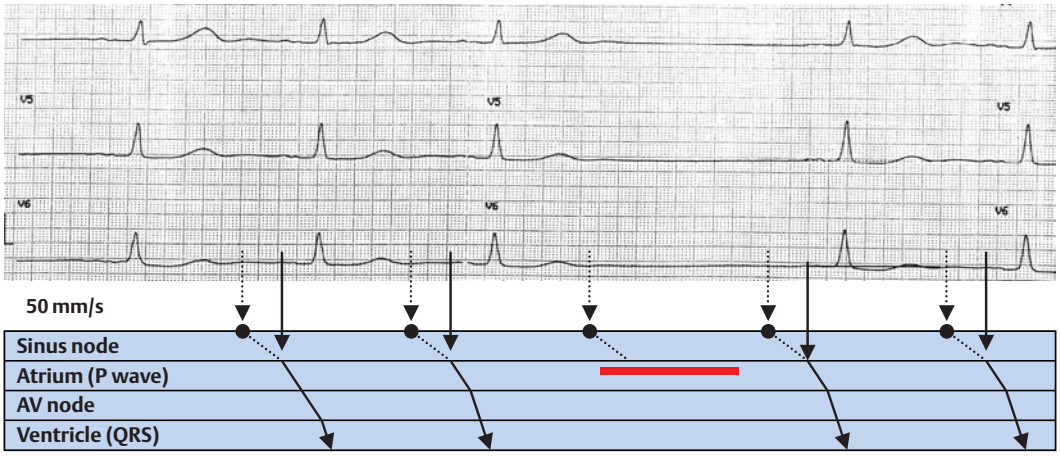
2nd Degree Sinoatrial Block, Wenckebach Type





Differential diagnosis:
- Sinus arrhythmia
- 2nd degree SA block (Mobitz), 3rd degree SA block
- 2nd degree AV block with 2:1 block (Mobitz/Wenckebach)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude

2nd Degree Sinoatrial Block, Mobitz Type

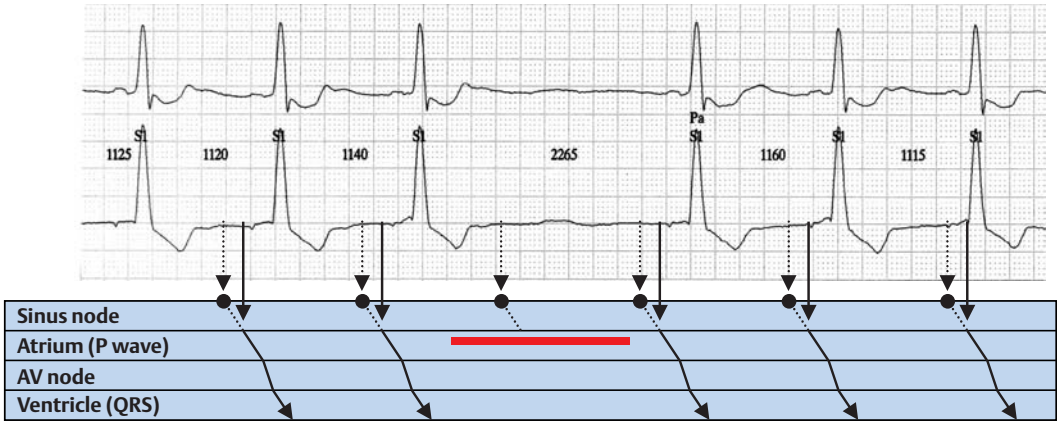


Mechanism:
- Single intermittent interruption of impulse formation in the sinus node

ECG characteristics:
- "Pause with no P wave"
- Absence of a sinus P wave with constant PP interval
- Length of pause = 2 × PP interval

Etiology:
- Often physiological
- Otherwise in:
  - Sinus node syndrome
  - CHD, heart defects, hypertension, etc.
- Medication-induced (see 5.11 Medication Related ECG Changes)

2nd Degree Sinoatrial Block, Mobitz Type

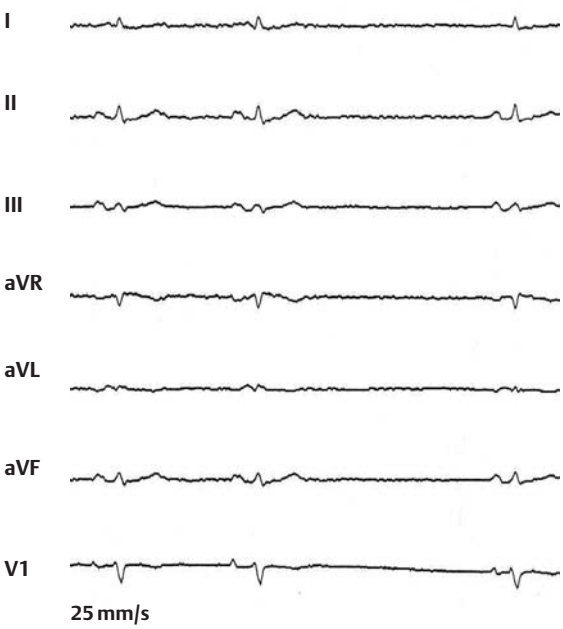


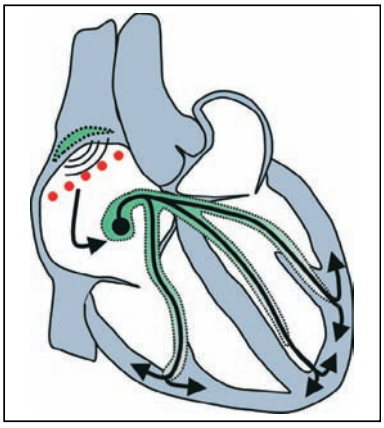
Mechanism:
- Single intermittent interruption of impulse formation in the sinus node

ECG characteristics:
- "Pause with no P wave"
- Absence of a sinus P wave with constant PP interval
- Length of pause = 2 × PP interval

Treatment:
- None in asymptomatic patients
- Cessation of medicines that may cause bradycardia in symptomatic patients
- In the case of severe symptoms, insertion of a pacemaker

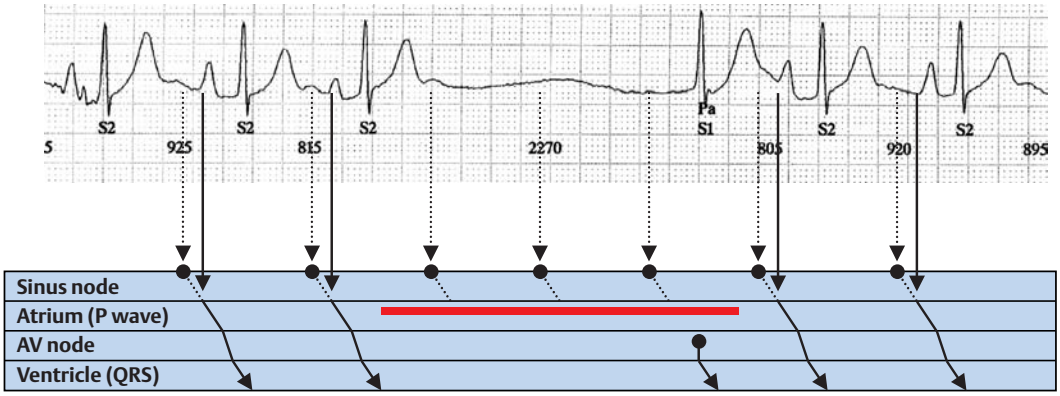
2nd Degree Sinoatrial Block, Mobitz Type





Differential diagnosis:
- Sinus arrhythmia
- 2nd degree SA block (Wenckebach), 3rd degree SA block
- 2nd degree AV block with 2 : 1 block (Mobitz/Wenckebach)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude

3rd Degree Sinoatrial Block



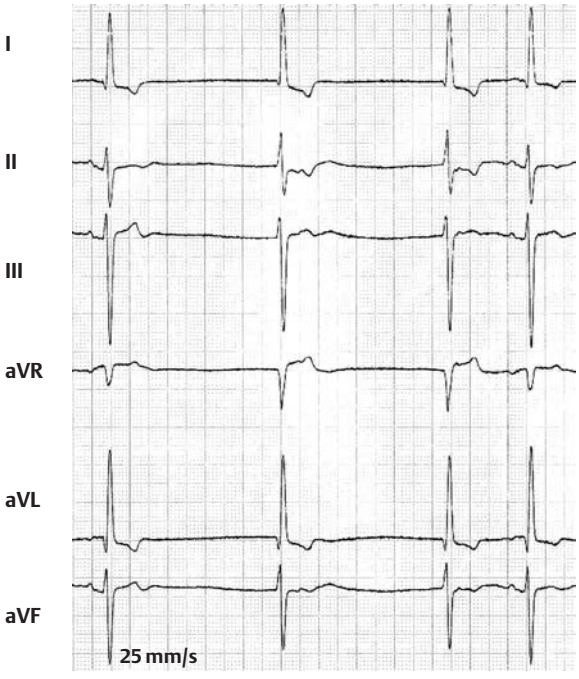
Mechanism:
- Temporary, complete interruption of impulse formation in the sinus node

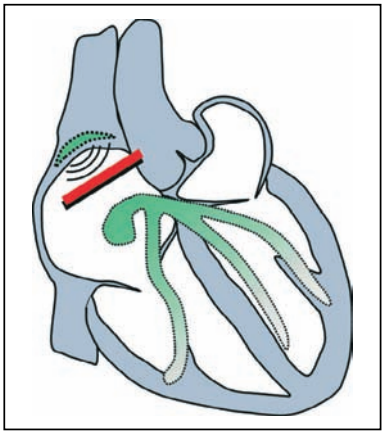
ECG characteristics:
- "Pause with no P wave"
- Absence of several sinus P waves
- Length of pause > 2 × PP interval

Etiology:
- Rarely physiological, often in the context of SSS, CHD, heart defects, CMP, hypertension, neurocardiogenic syncope

Treatment:
- None in asymptomatic patients
- Cessation of medicines that may cause bradycardia in symptomatic patients and in most cases insertion of a pacemaker

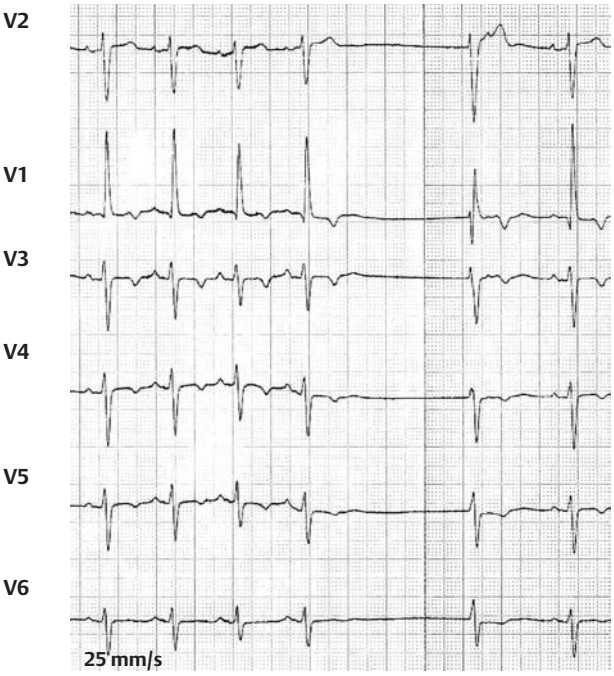
3rd Degree Sinoatrial Block with Junctional Escape Rhythm

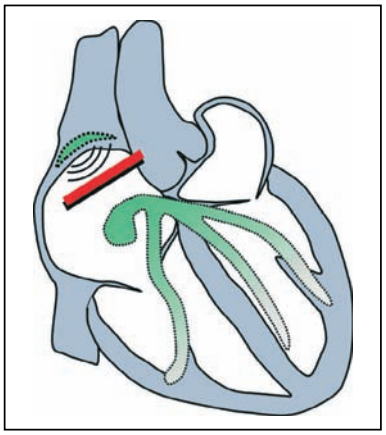




Differential diagnosis:
- Sinus arrhythmia
- 2nd degree SA block (Mobitz/Wenckebach)
- 2nd degree AV block with 2:1 block (Mobitz/Wenckebach)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude

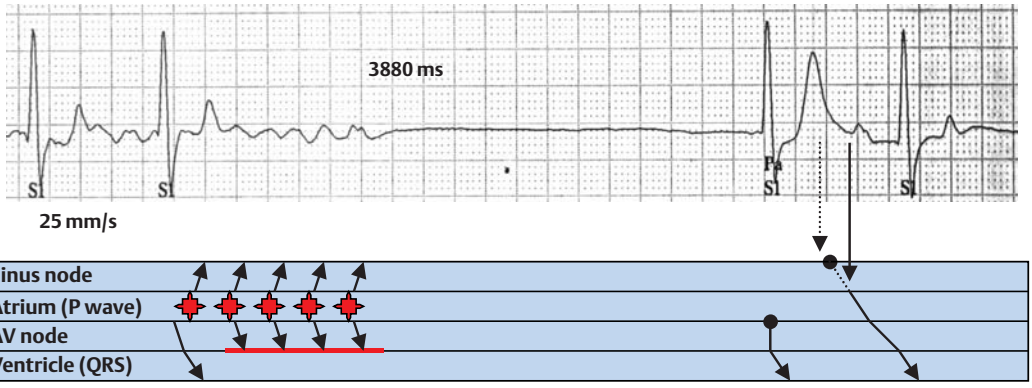
3rd Degree Sinoatrial Block with Junctional Escape Rhythm





Differential diagnosis:
- Sinus arrhythmia
- 2nd degree SA block (Mobitz/Wenckebach)
- 2nd degree AV block with 2:1 block (Mobitz/Wenckebach)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude

Sinus Node Recovery Period Following Spontaneous Cessation of Atrial Fibrillation



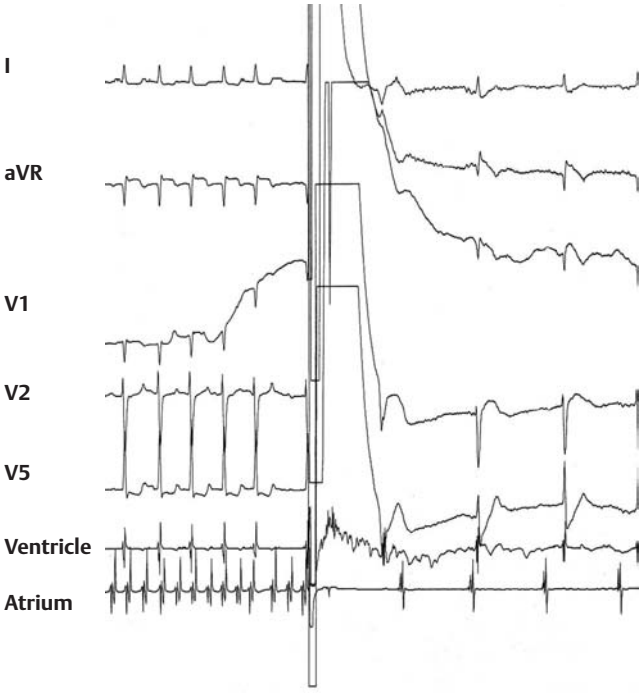
Mechanism:
- "Overstimulation" of the sinus node by atrial fibrillation, time between the end of arrhythmia and onset of sinus rhythm is called the sinus node recovery period (preautomatic pause)

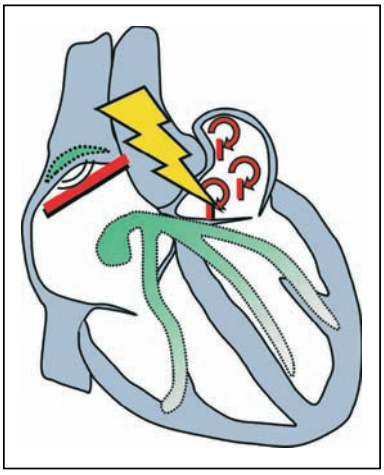
ECG characteristics:
- "Pause with no P wave" following cessation of atrial fibrillation, often nodal escape beats

Etiology:
- Often in the context of brady-tachycardia syndrome (variant of sinus node syndrome)
- In paroxysmal atrial fibrillation

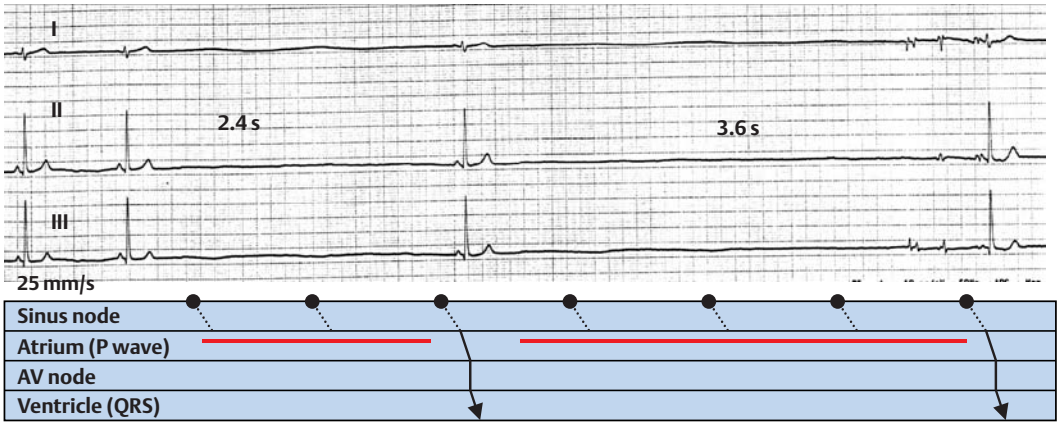
Treatment:
- Often symptomatic; therefore in most cases insertion of pacemaker is indicated

Electrical Cardioversion of Atrial Fibrillation





Sinus Cardiac Arrest in Neurocardiogenic Syncope



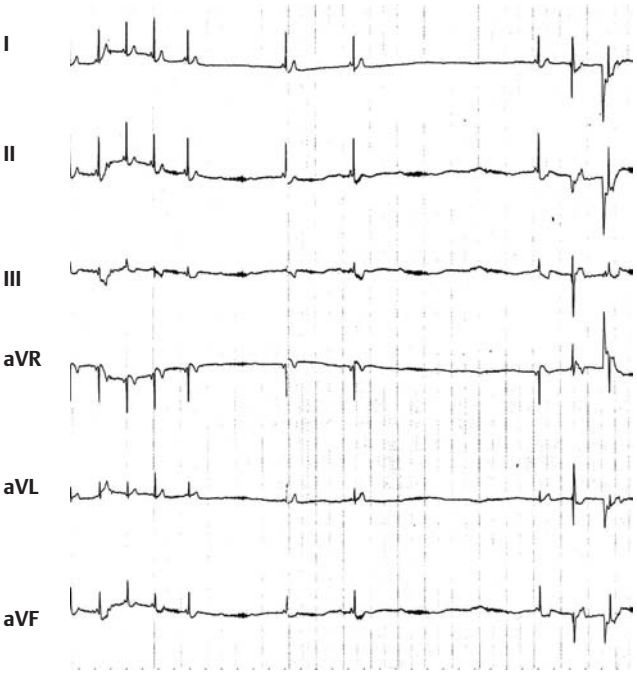
Mechanism:
- Sinus node arrest reflecting vagal tone
- Good prognosis

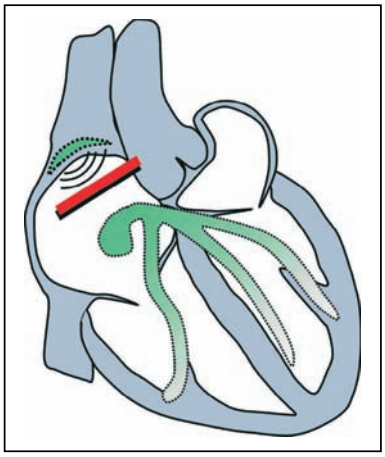
ECG characteristics:
- "Pause with no P wave"

Etiology:
- Almost only seen in younger people with no structural heart disease (variant of the so-called cardioinhibitory type of neurocardiogenic syncope)

Treatment:
- Conservative (to avoid trigger factors), no beta-blockade, "tilt training"
- Insertion of pacemaker only in "malignant" forms (no prodrome, serious injuries, etc.)

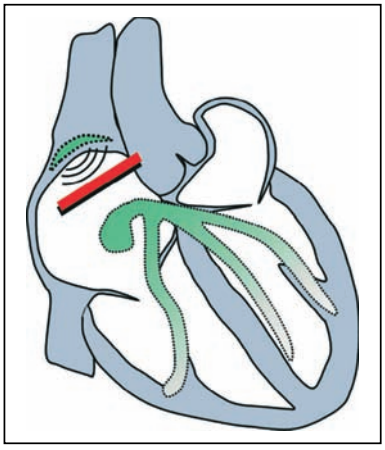
Sinus Cardiac Arrest in Neurocardiogenic Syncope



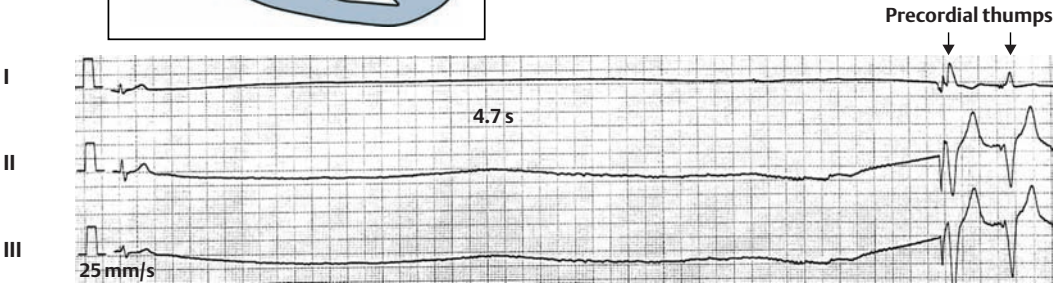


Differential diagnosis:
- Sinus node syndrome
- Other reflex-related types of syncope, including carotid sinus syndrome

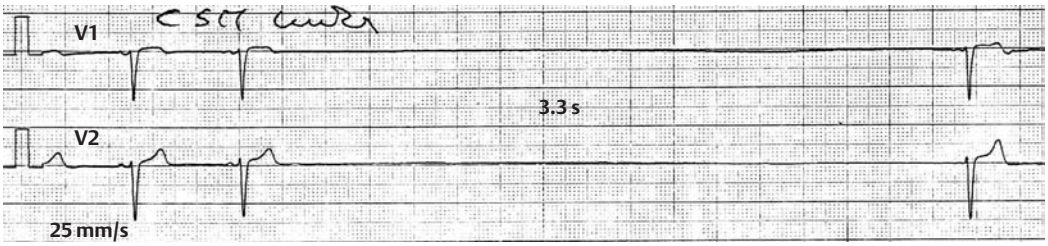
Sinus Cardiac Arrest in Neurocardiogenic Syncope

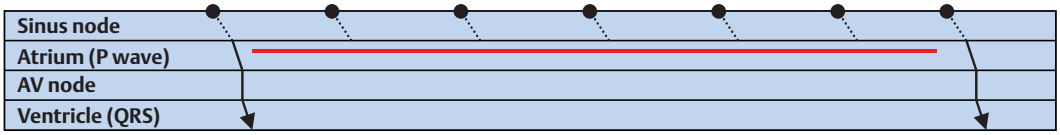


Differential diagnosis:
- Sinus node syndrome
- Other reflex-related types of syncope, including carotid sinus syndrome



Sinus Cardiac Arrest in Carotid Sinus Syndrome





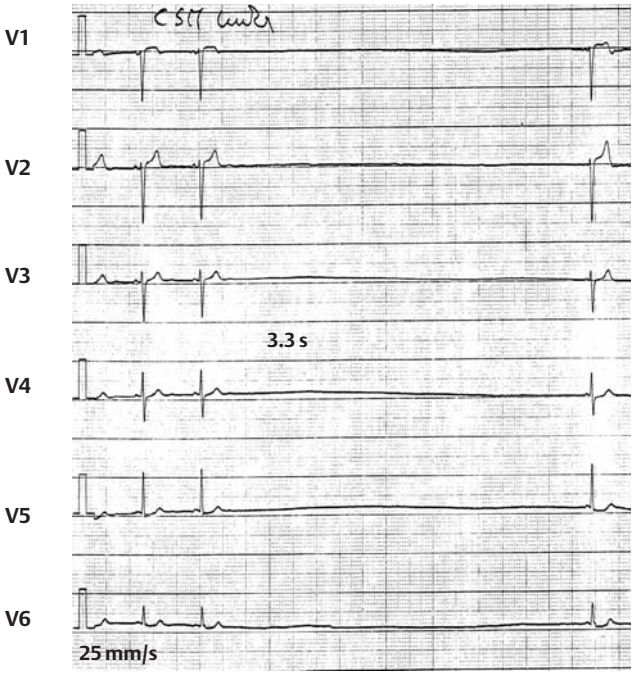
Mechanism:
- Reflex sinus node arrest

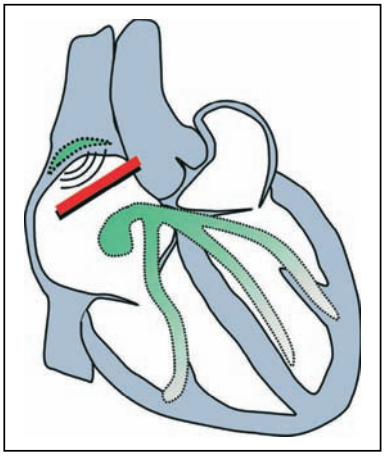
ECG characteristics:
- "Pause with no P wave"

Etiology:
- Almost only seen in older people (variant of the so-called cardioinhibitory type of carotid sinus syncope)

Treatment:
- Insertion of pacemaker only if symptomatic
- No insertion of pacemaker in so-called hypersensitive carotid sinus (not enough clinics!)

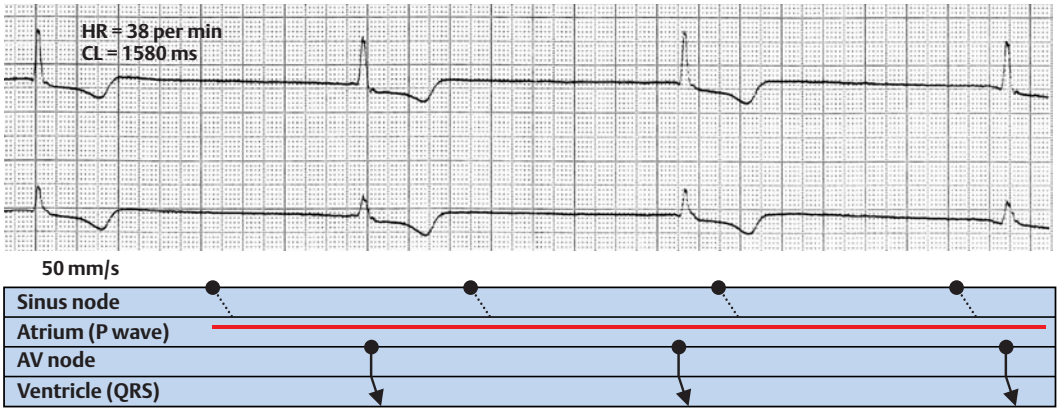
Sinus Cardiac Arrest in Carotid Sinus Syndrome





Differential diagnosis:
- Sinus node syndrome
- Other reflex-related causes (including neurocardiogenic syncope)

Sinus Cardiac Arrest with Junctional Escape Rhythm



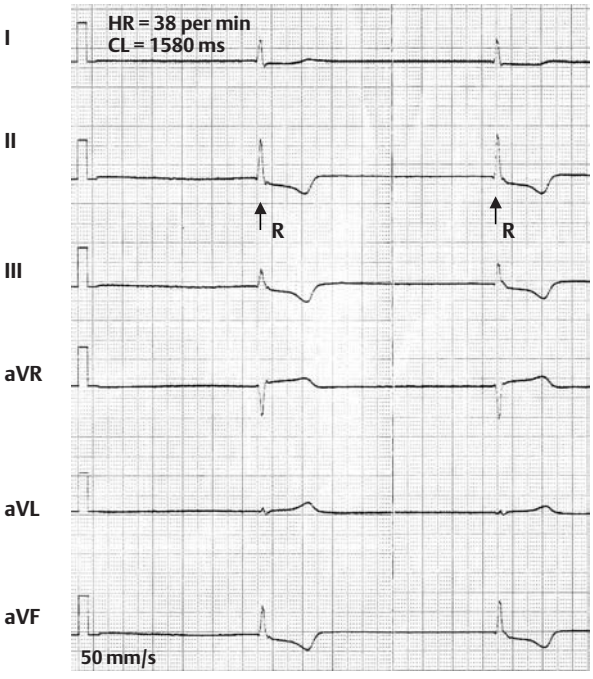
Mechanism:
- Complete electrical inactivity of the sinus node
- Occurrence of an escape rhythm in the region of the AV node; no retrograde atrial excitation

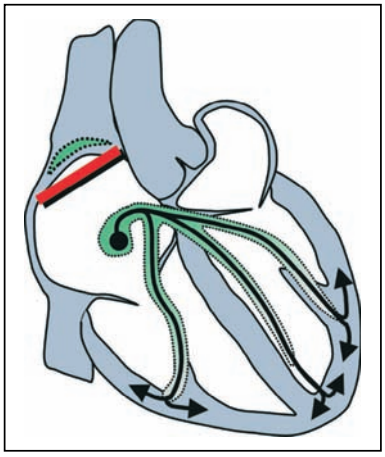
ECG characteristics:
- Rhythm with narrow ventricular complex with no P waves

Etiology:
- Almost only seen in the context of sinus node syndrome
- Medications that may cause bradycardia

Treatment:
- Cessation of medications that may cause bradycardia
- Insertion of pacemaker in symptomatic patients

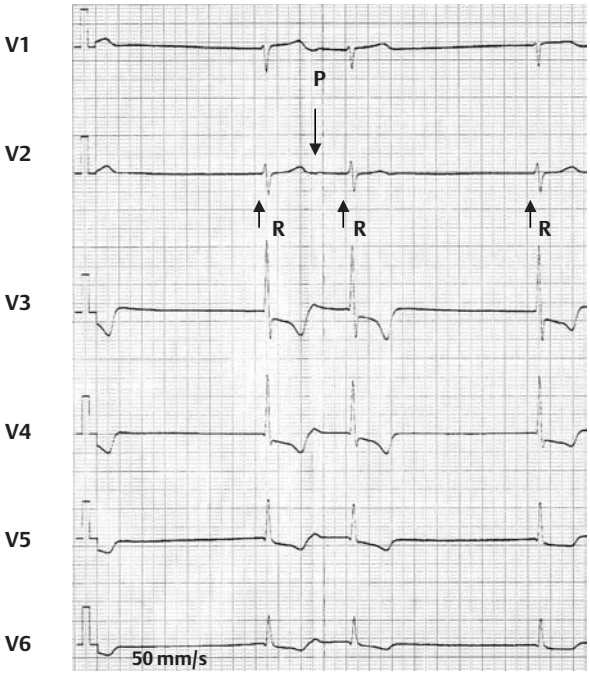
Cardiac Arrest with Junctional Escape Rhythm

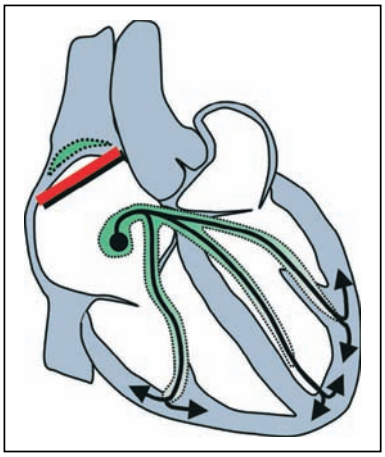




Differential diagnosis:
- Sinus bradycardia (P wave present), 2nd/3rd degree SA block
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block (P waves present)

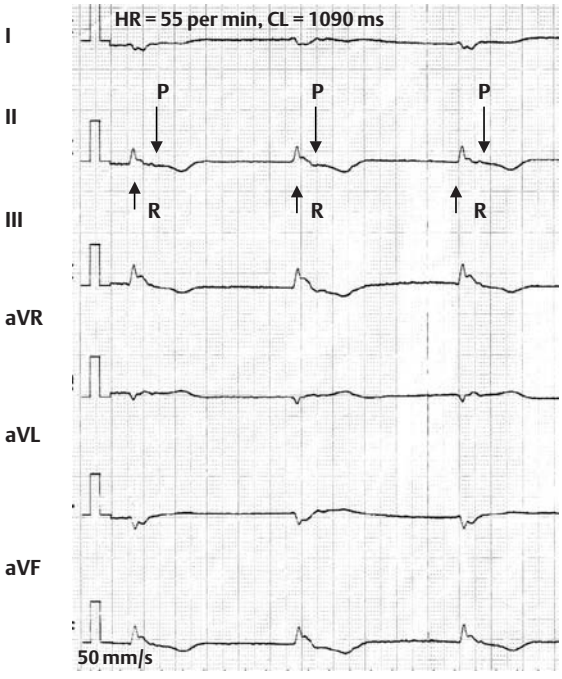
Cardiac Arrest with Junctional Escape Rhythm

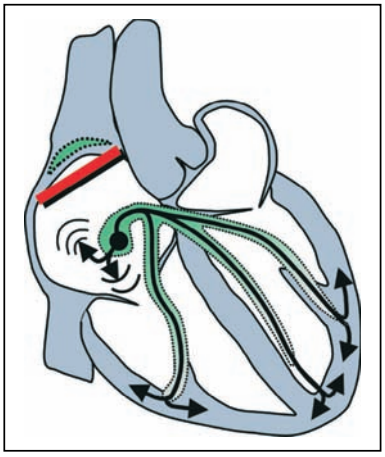




Differential diagnosis:
- Sinus bradycardia (P wave present), 2nd/3rd degree SA block
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block (P waves present)

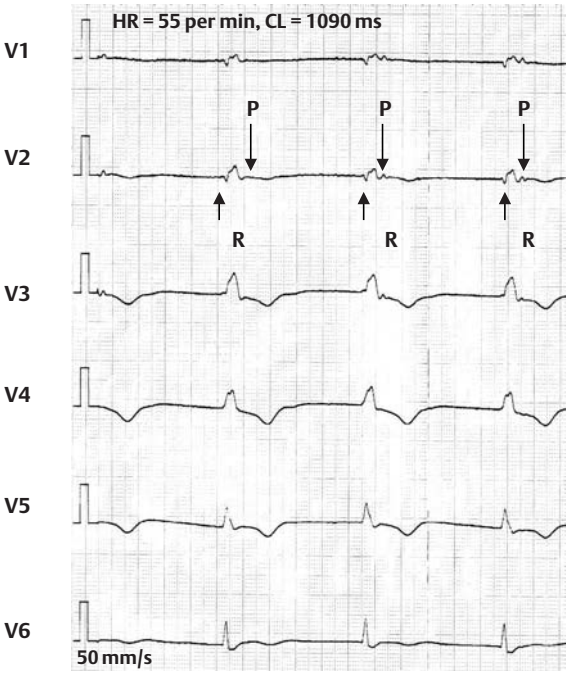
Junctional Rhythm with Retrograde Atrial Excitation

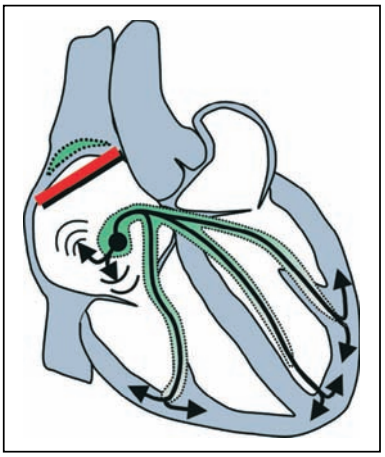




Differential diagnosis:
- Sinus bradycardia (P wave present), 2nd/3rd degree SA block
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block (P waves present)

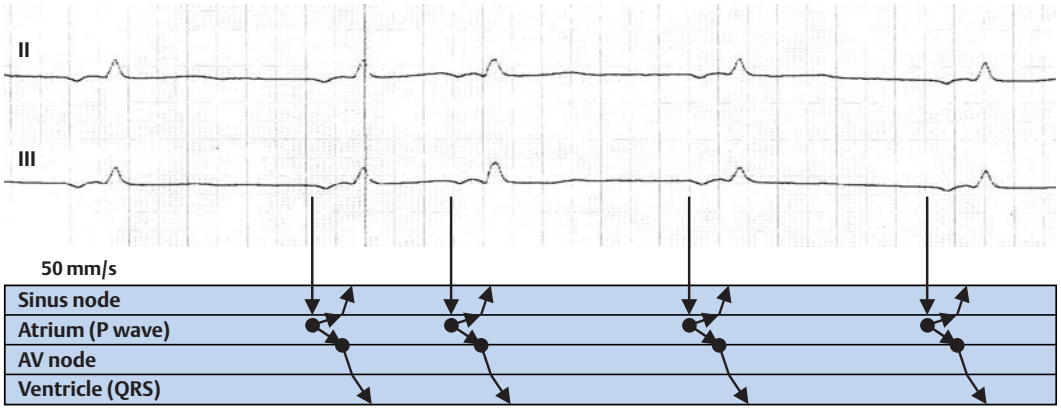
Junctional Rhythm with Retrograde Atrial Excitation





Differential diagnosis:
- Sinus bradycardia (P wave present), 2nd/3rd degree SA block
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block (P waves present)

Basal Atrial Rhythm



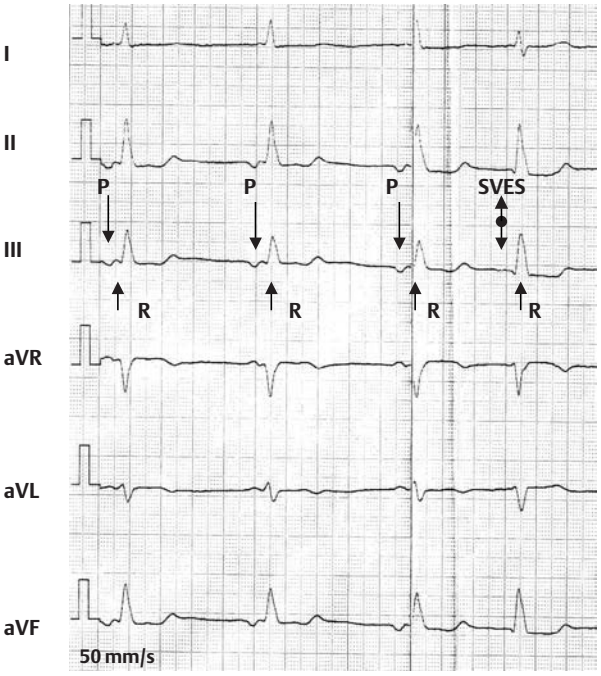
Mechanism:
- In sinus bradycardia occurrence of escape rhythms; with foci relatively close to the AV node there is a shortening of the PQ interval due to shorter conduction pathways

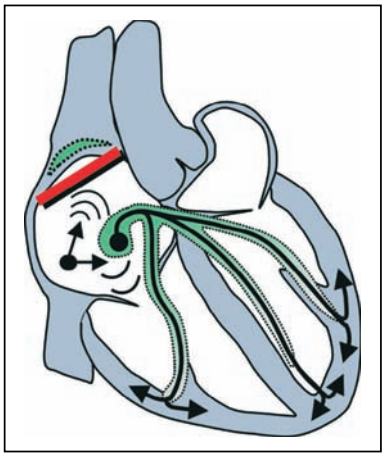
ECG characteristics:
- Rhythm with negative P waves in the limb leads II and III
- PQ interval often shorter

Etiology:
- Physiological in cases of increased vagal tone (sportspeople, young people)
- Sinus node syndrome, condition post carditis
- Medications that may cause bradycardia

Treatment:
- Cessation of medications that may cause bradycardia
- Insertion of pacemaker in symptoms of clinical significance (e.g., chronotropic incompetence)

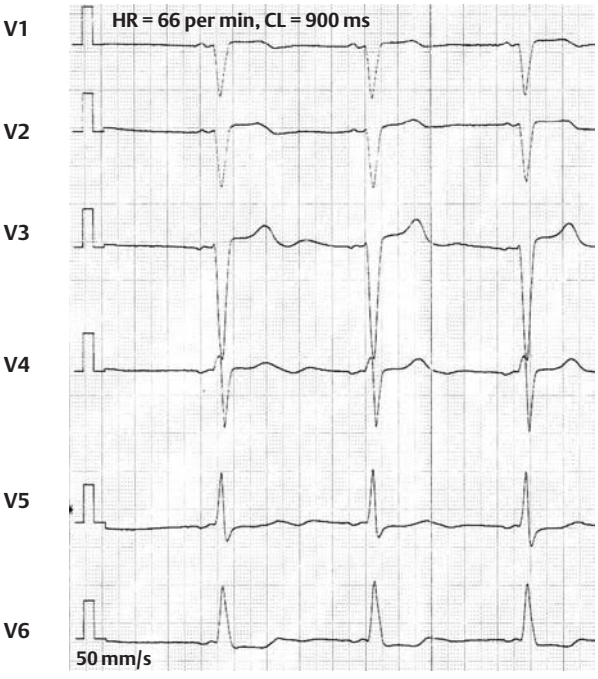
Basal Atrial Rhythm

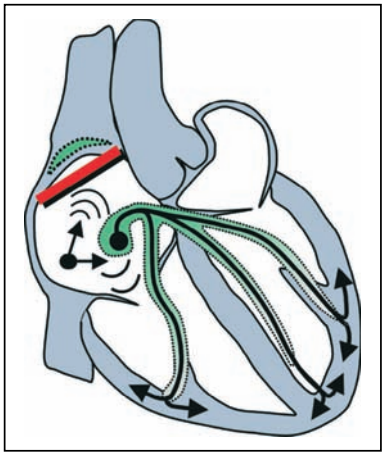




Differential diagnosis:
- Sinus bradycardia (positive P wave)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block (P waves present)
- Atrial tachycardia

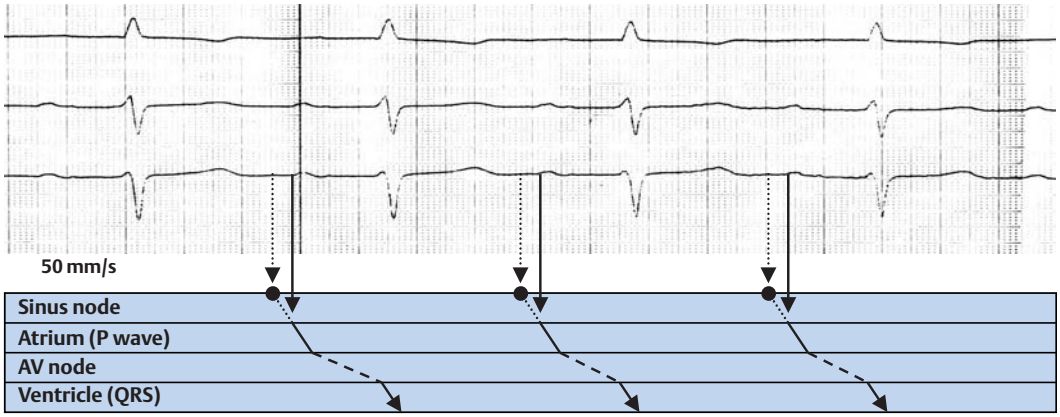
Basal Atrial Rhythm





Differential diagnosis:
- Sinus bradycardia (positive P wave)
- Bradycardic atrial fibrillation with fibrillation waves (f) of low amplitude
- Higher-grade AV block (P waves present)
- Atrial tachycardia

1st Degree AV Block



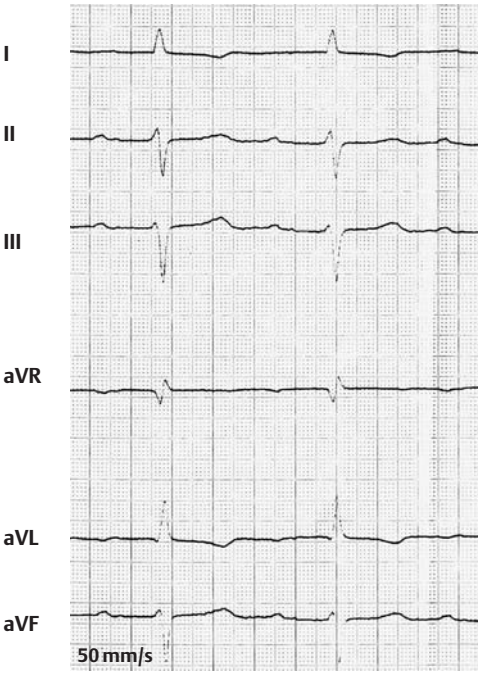
Mechanism:
- Delay in impulse conduction at the AV node without interruption of this impulse

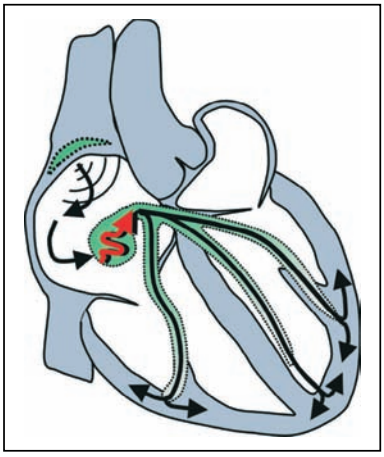
ECG characteristics:
- No pause
- Prolongation of the PQ interval > 200 ms

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, medications, CMP
- Increased vagal tone

Treatment:
- No treatment as a rule
- Cessation of medications that may cause delayed conduction if applicable

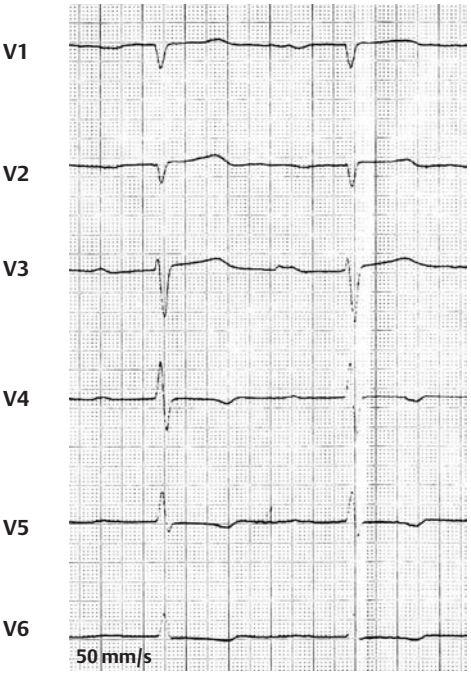
1st Degree AV Block

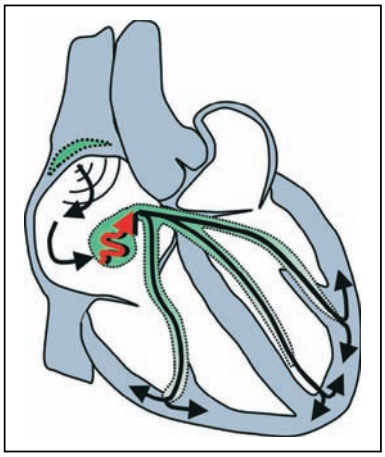




Differential diagnosis:
- Atrial flutter with 2 : 1 or 3 : 1 conduction
- AV conduction delay with SVES
- 2nd degree AV block, Mobitz type
- Prolonged P wave with interatrial block

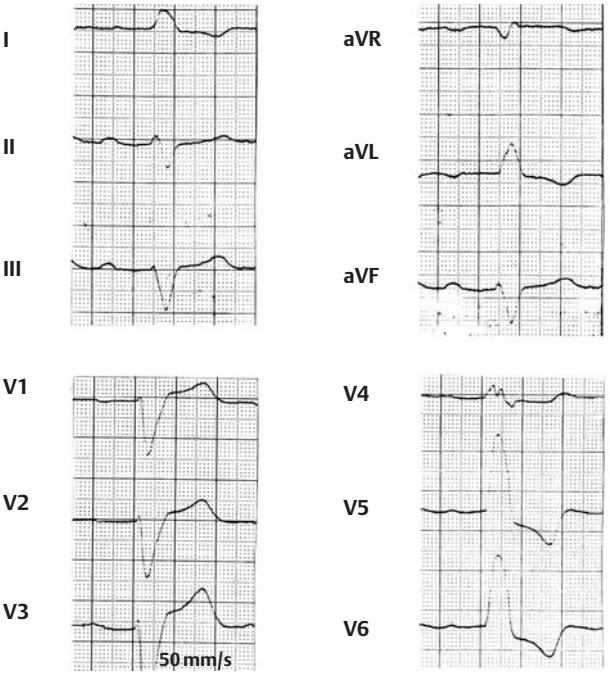
1st Degree AV Block

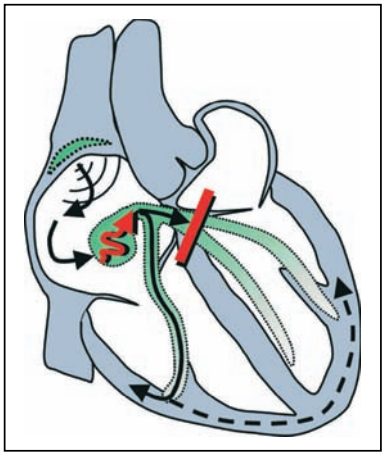




Differential diagnosis:
- AV conduction delay with SVES
- 2nd degree AV block, Mobitz type
- Prolonged P wave with interatrial block

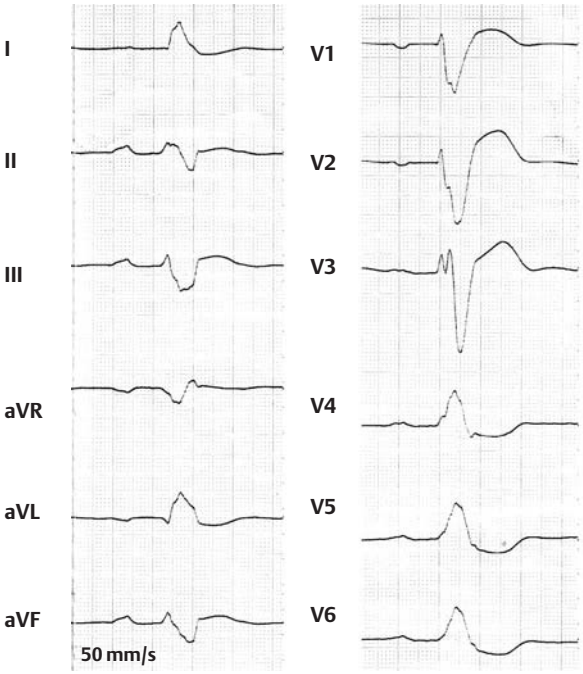
1st Degree AV Block with Complete Left Bundle Branch Block

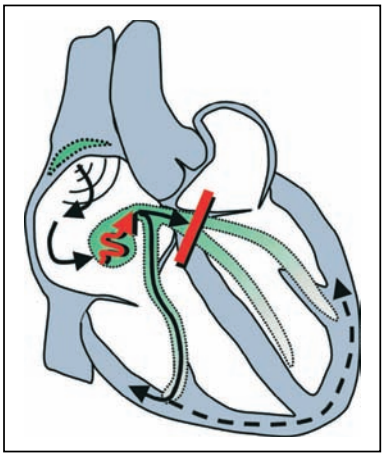




Differential diagnosis:
With preexisting bundle branch block:
- AV conduction delay with SVES
- 2nd or 3rd degree AV block
- Prolonged P wave with interatrial block

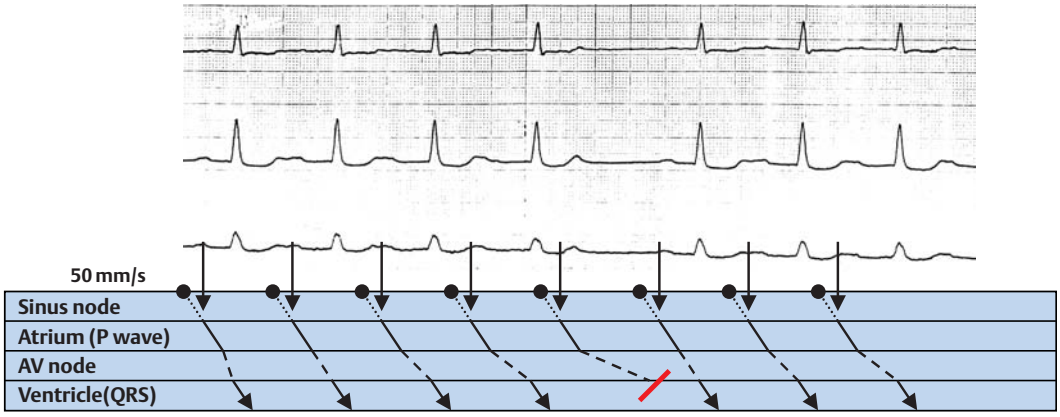
1st Degree AV Block with Complete Left Bundle Branch Block





Differential diagnosis:
With preexisting bundle branch block:
- AV conduction delay with SVES
- 2nd or 3rd degree AV block
- Prolonged P wave with interatrial block

2nd Degree AV Block, Wenckebach Type



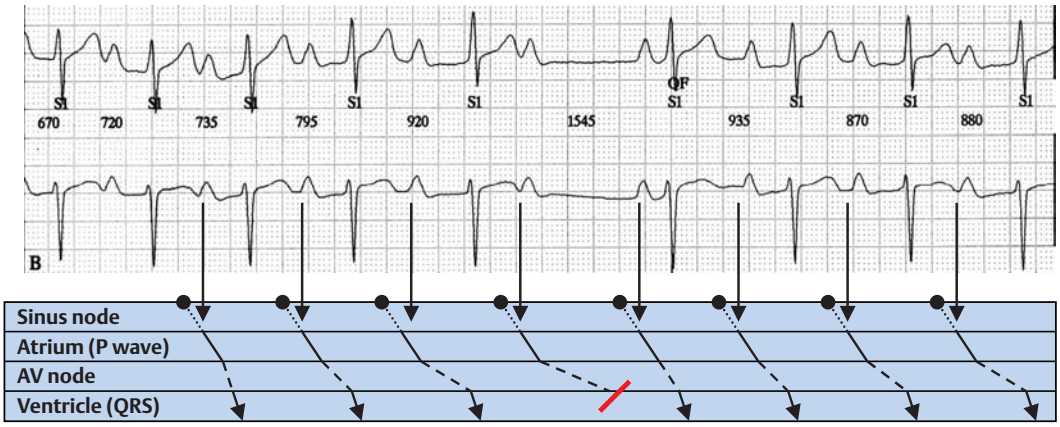
Mechanism:
- Single intermittent interruption of impulse conduction at the AV node
- Blockade above the level of the bundle of His

ECG characteristics:
- "Pause with P wave"
- Absence of QRS complex with prolongation of the PQ interval
- Length of pause < 2 × RR interval

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, medications, CMP
- Increased vagal tone

Treatment:
- Cessation of medications that may cause delayed conduction
- Insertion of pacemaker in symptomatic patients
- Conservative in the event of a lack of clinics

2nd Degree AV Block, Wenckebach Type



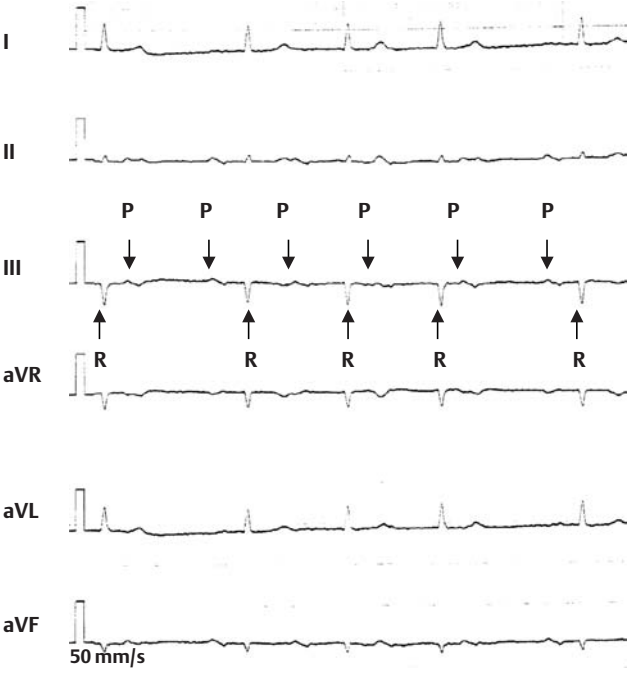
Mechanism:
- Single intermittent interruption of impulse conduction at the AV node
- Blockade above the level of the bundle of His

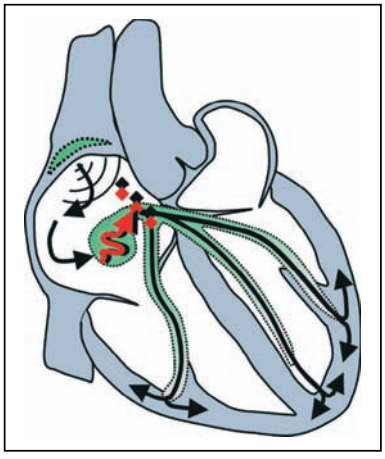
ECG characteristics:
- "Pause with P wave"
- Absence of QRS complex with prolongation of the PQ interval
- Length of pause < 2 × RR interval

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, medications, CMP
- Increased vagal tone

Treatment:
- Cessation of medications that may cause delayed conduction
- Insertion of pacemaker in symptomatic patients
- Conservative in the event of a lack of clinics

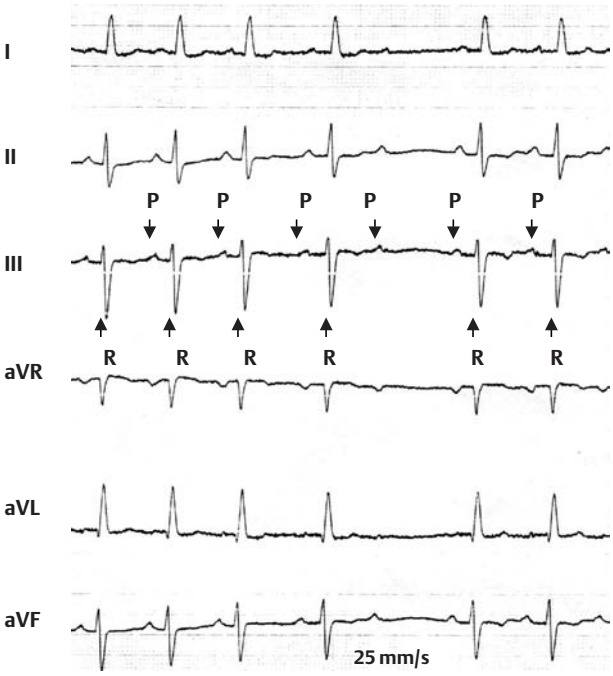
2nd Degree AV Block, Wenckebach Type

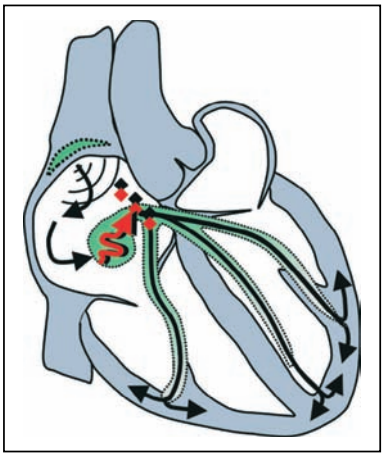




Differential diagnosis:
- AV blocked SVES
- 2nd degree AV block, Mobitz type
- 3rd degree AV block
- 2nd degree SA block
- Sinus arrhythmia

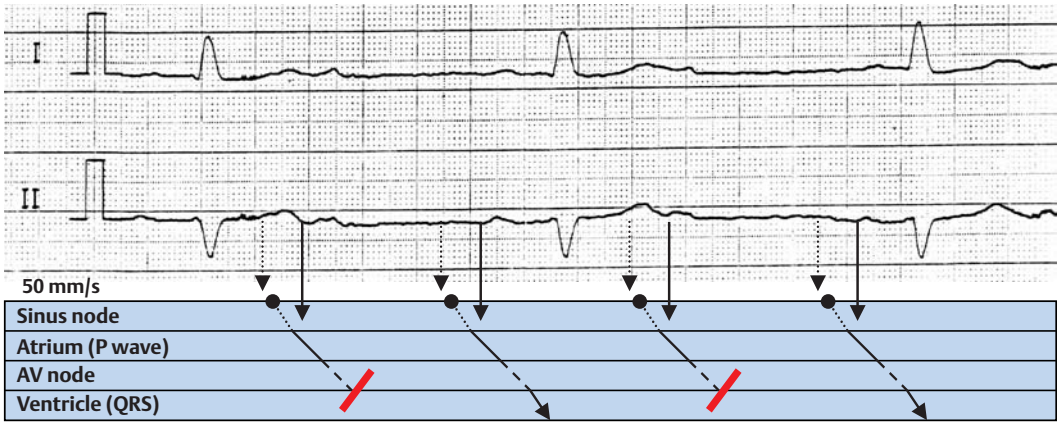
2nd Degree AV Block, Wenckebach Type





Differential diagnosis:
- AV blocked SVES
- 2nd degree AV block, Mobitz type
- 3rd degree AV block
- 2nd degree SA block
- Sinus arrhythmia

2nd Degree AV Block, 2:1 Block



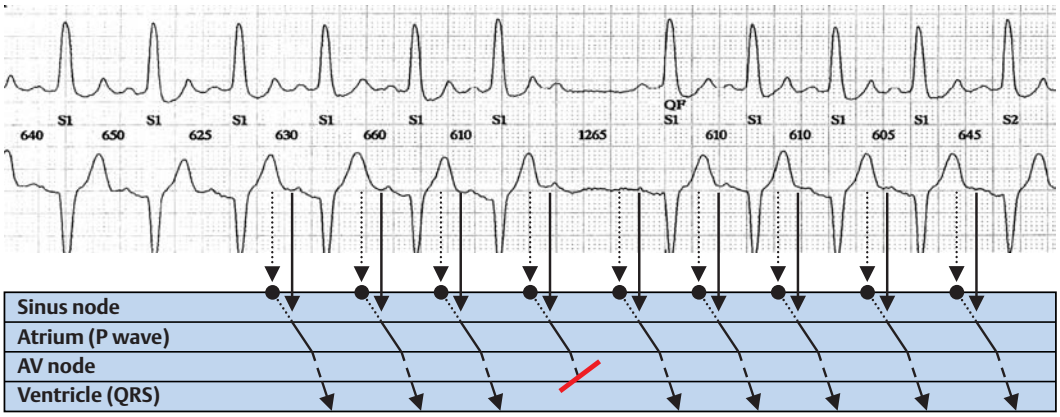
Mechanism:
- Intermittent interruption of impulse conduction at the AV node
- Both blockade above and below the level of the bundle of His

ECG characteristics:
- "Bradycardia with P waves"—only every second atrial beat is conducted to the ventricles with prolongation of the PQ interval

Etiology:
- Heart defects, hypertension, CMP, inflammatory
- Medications
- CHD, acute posterior myocardial infarction

Treatment:
- Insertion of pacemaker in the majority of patients (relative indication in asymptomatic patients)

2nd Degree AV Block, Mobitz Type



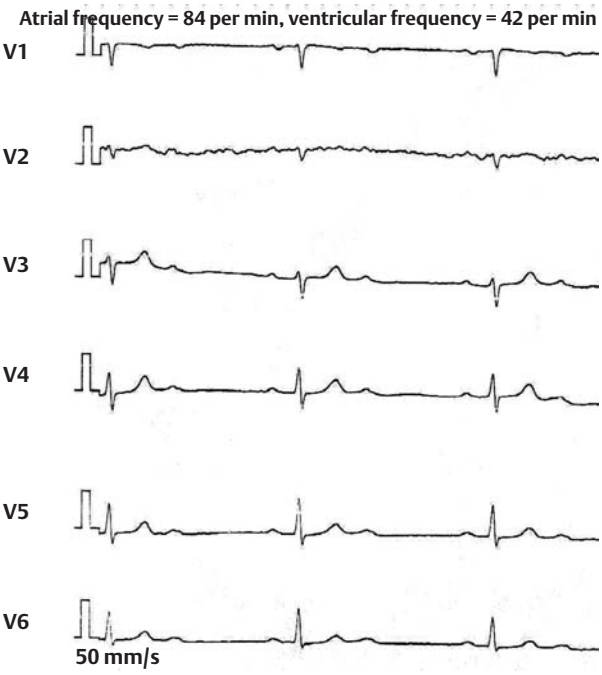
Mechanism:
- Intermittent interruption of impulse conduction at the AV node
- Blockade below the level of the bundle of His

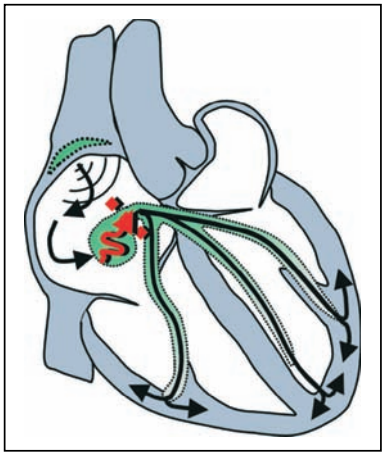
ECG characteristics:
- "Pause with P wave"
  Single failure of conduction via the AV node without prolongation of the PQ interval
  Pause = 2 × RR interval

Etiology:
- Heart defects, hypertension, CMP, inflammatory, medications
- CHD, acute posterior myocardial infarction

Treatment:
- Insertion of pacemaker in the majority of patients (relative indication in asymptomatic patients)

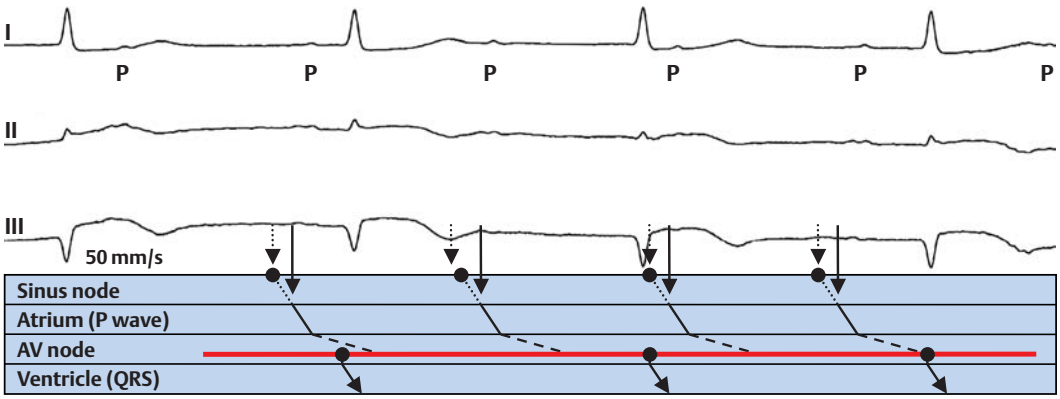
2nd Degree AV Block, 2:1 Block





Differential diagnosis:
- AV blocked SVES
- 3rd degree AV block
- 2nd degree SA block
- Sinus arrhythmia

Persistent 3rd Degree AV Block, Two Weeks after Posterior Myocardial Infarction



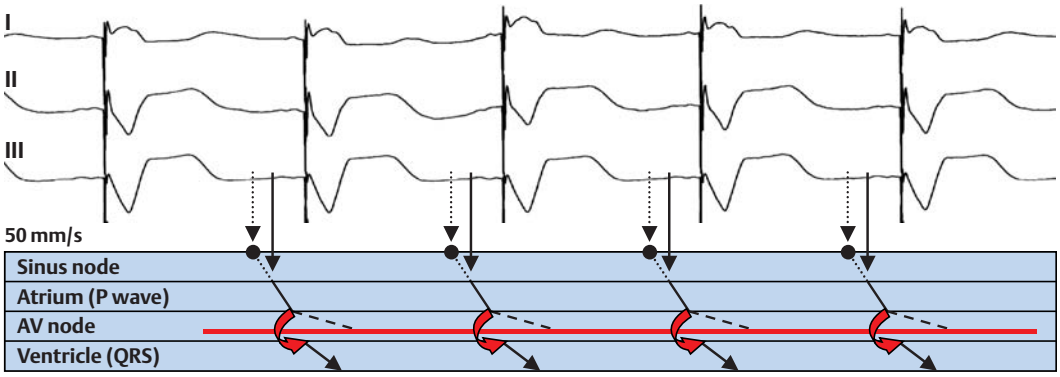
Mechanism:
- Complete interruption of impulse conduction at the AV node due to ischemia
- Blockade above the level of the bundle of His

ECG characteristics:
- Complete dissociation of atrial and ventricular excitation, no P wave can be matched with a QRS complex; with junctional escape rhythm two-week-old posterior infarction can be easily recognized

Etiology:
- Ischemia following acute posterior myocardial infarction, whereby AV blockade often completely regresses

Treatment:
- Initially, temporary insertion of pacemaker
- With persistent AV blockade more than two to four weeks post infarction, insertion of a permanent pacemaker is indicated

Persistent 3rd Degree AV Block Following Posterior Infarction and Insertion of Pacemaker



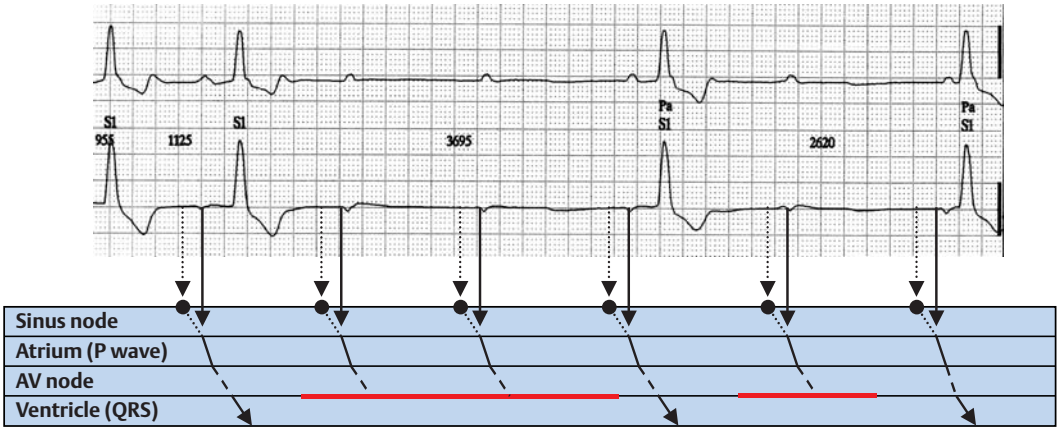
Mechanism:
- Complete interruption of impulse conduction at the AV node due to ischemia

ECG characteristics:
- Resynchronization of atrial and ventricular activity, each P wave is followed by ventricular stimulation, signs of posterior infarction are no longer visible

Etiology:
- Ischemia following acute posterior myocardial infarction

Treatment:
- Successful insertion of pacemaker

Advanced AV Block (High-Grade AV Block)



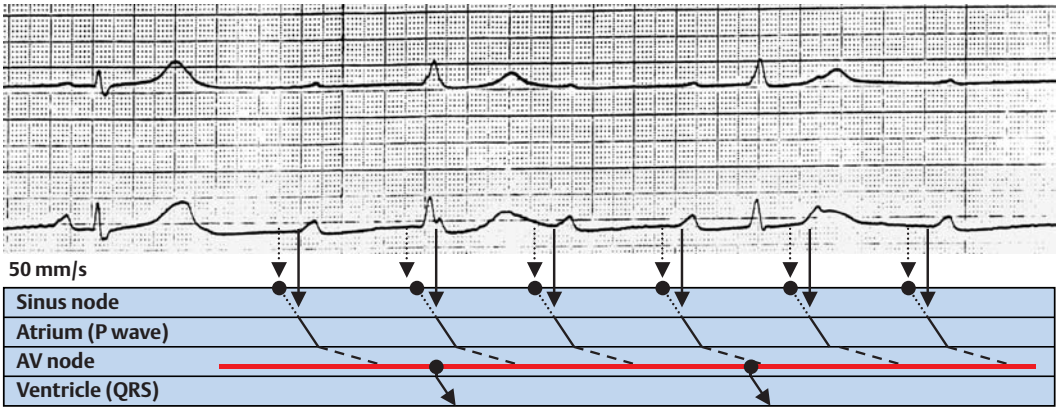
Mechanism:
- Intermittent, complete interruption of impulse conduction at the AV node

ECG characteristics:
- "Pause with P waves"
- Absence of several QRS complexes; in the example above additionally 2nd degree block, Mobitz type, followed by single normal AV conduction

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, CMP, inflammatory, medications

Treatment:
- Insertion of pacemaker in the majority of patients (relative indication in asymptomatic patients)
- Cessation of medications that may cause bradycardia

3rd Degree AV Block, Junctional Escape Rhythm



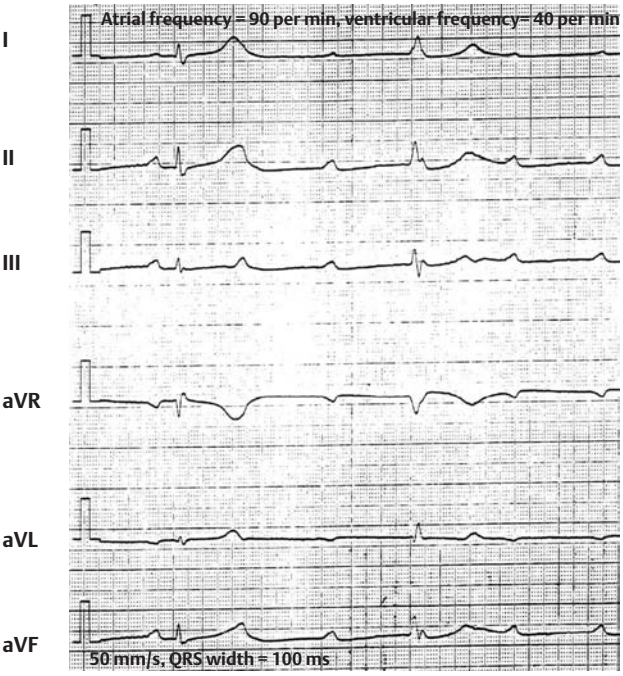
Mechanism:
- Complete interruption of impulse conduction at the AV node
- Blockade above the level of the bundle of His

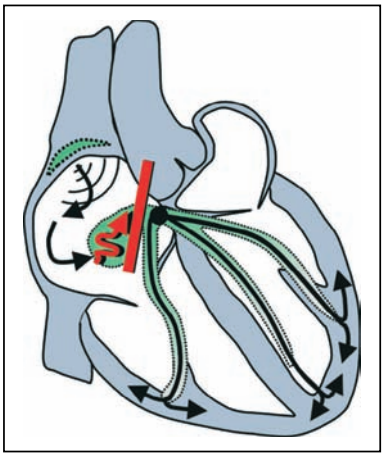
ECG characteristics:
- Complete dissociation of atrial and ventricular excitation, no P wave can be matched with a QRS complex, junctional escape rhythm can be easily recognized by the narrow QRS complexes

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, CMP, inflammatory, medications
- Congenital

Treatment:
- Insertion of pacemaker in the majority of patients (relative indication in asymptomatic patients)
- Cessation of medications that may cause bradycardia

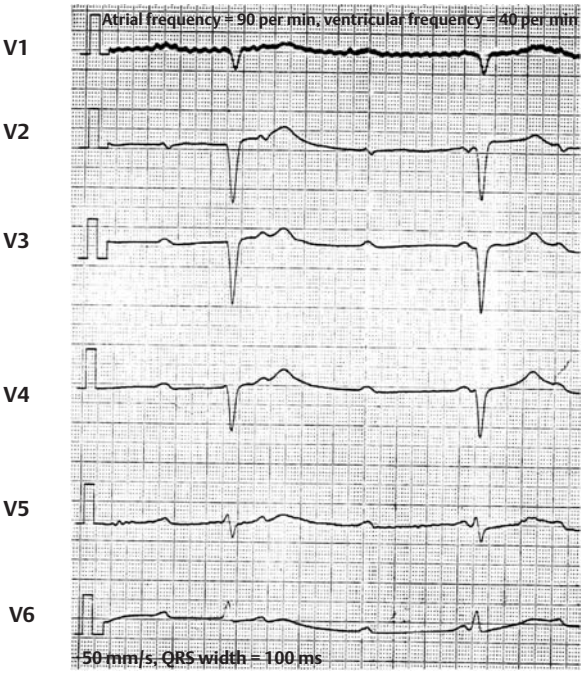
3rd Degree AV Block, Junctional Escape Rhythm

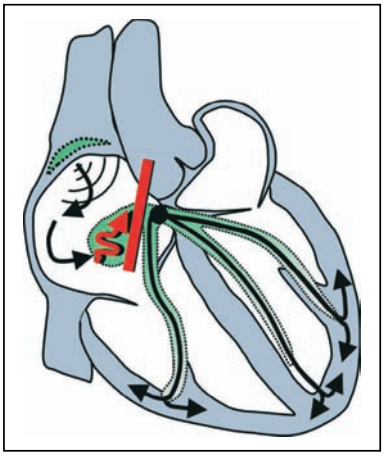




Differential diagnosis:
- AV-blocked SVES
- 2nd degree AV block, Mobitz/ Wenckebach type
- 2nd degree SA block
- Sinus arrhythmia

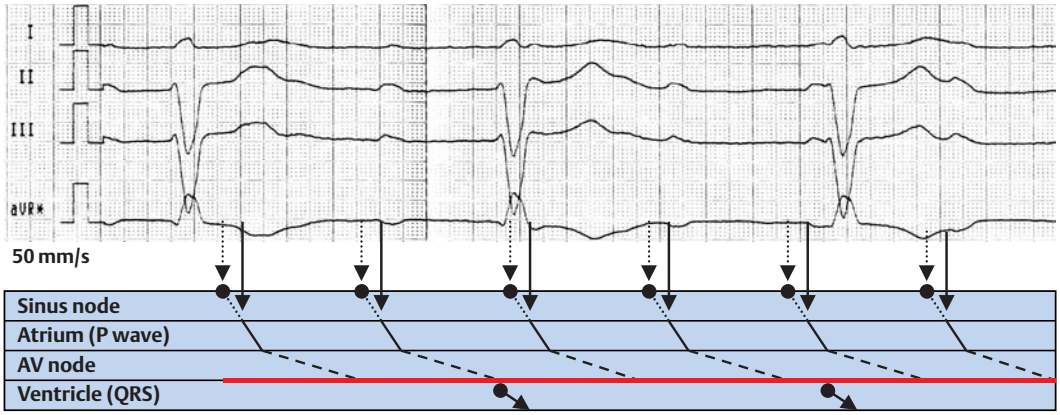
3rd Degree AV Block, Junctional Escape Rhythm





Differential diagnosis:
- AV blocked SVES
- 2nd degree AV block, Mobitz/ Wenckebach type
- 2nd degree SA block
- Sinus arrhythmia

3rd Degree AV Block, Ventricular Escape Rhythm



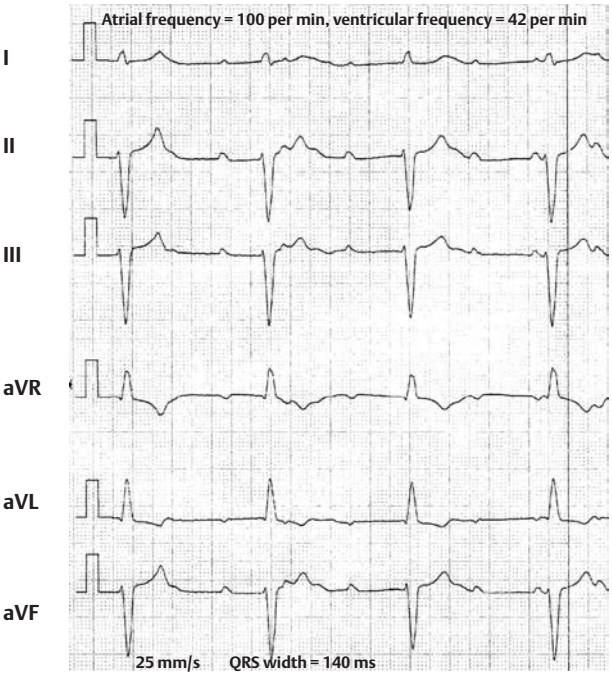
Mechanism:
- Complete interruption of impulse conduction at the AV node
- Blockade below the level of the bundle of His

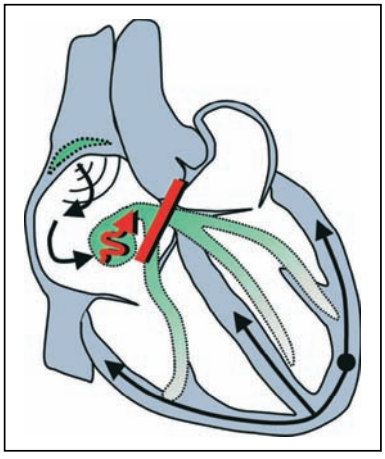
ECG characteristics:
- Complete dissociation of atrial and ventricular excitation, no P wave can be matched with a QRS complex, "distal-seated" escape rhythm easily recognized by the widened QRS complexes

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, CMP, inflammatory, medications

Treatment:
- Insertion of pacemaker
- Cessation of medications that may cause bradycardia

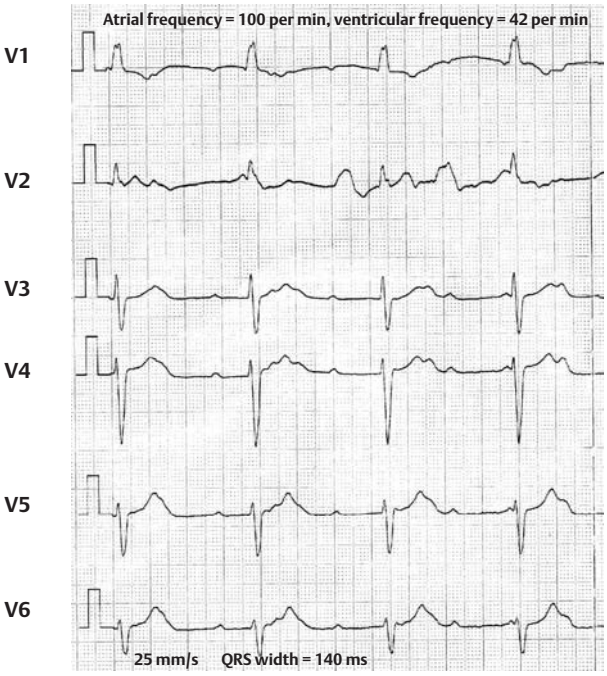
3rd Degree AV Block, Ventricular Escape Rhythm

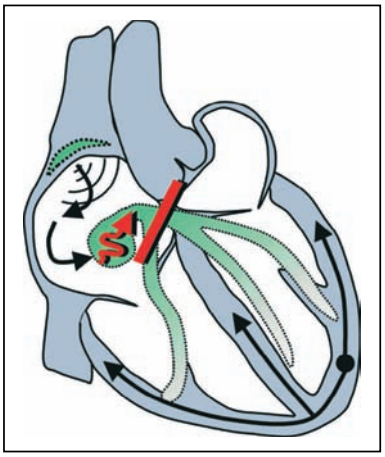




Differential diagnosis:
- 2nd degree AV block, Mobitz/Wenckebach type with bundle branch block
- 2nd degree SA block with bundle branch block
- Sinus bradycardia with bundle branch block

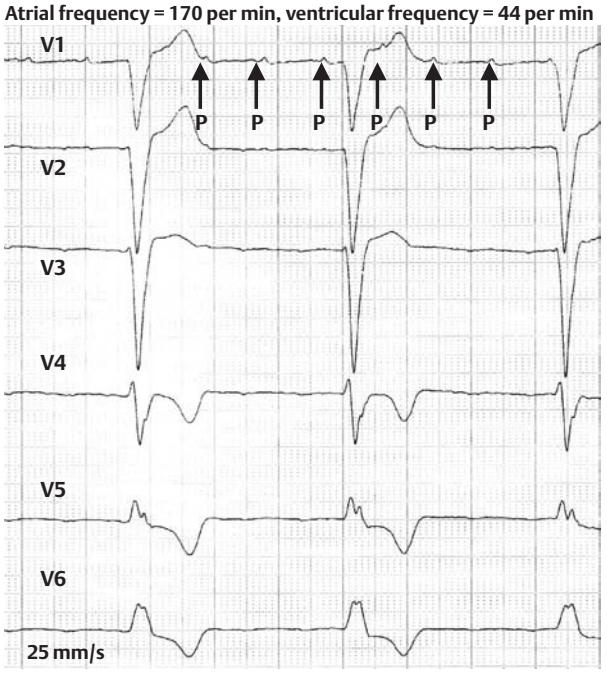
3rd Degree AV Block, Ventricular Escape Rhythm

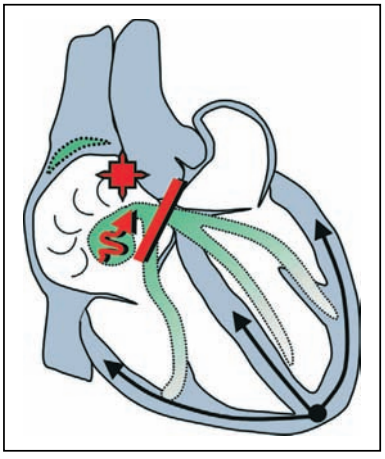




Differential diagnosis:
- 2nd degree AV block, Mobitz/Wenckebach type with bundle branch block
- 2nd degree SA block with bundle branch block
- Sinus bradycardia with bundle branch block

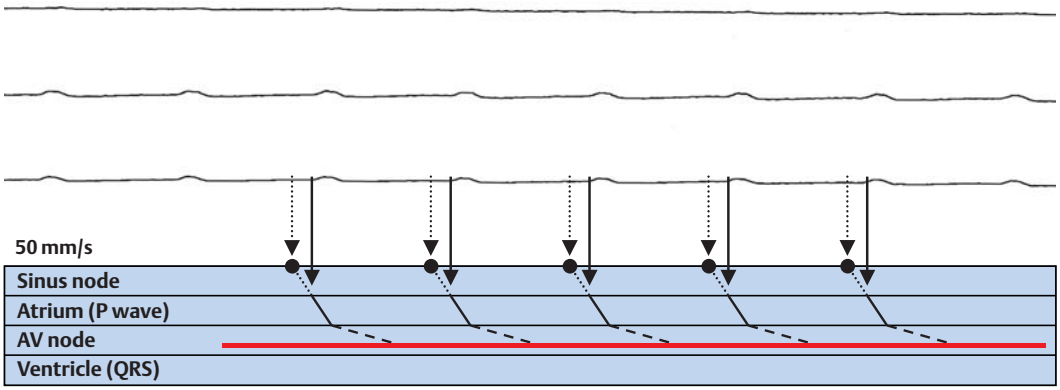
3rd Degree AV Block with Atrial Tachycardia, Ventricular Escape Rhythm





Differential diagnosis:
- 2nd degree AV block, Mobitz type with bundle branch block
- AV conduction disorders with other atrial arrhythmias (atrial fibrillation and flutter)

3rd Degree AV Block Without Escape Rhythm



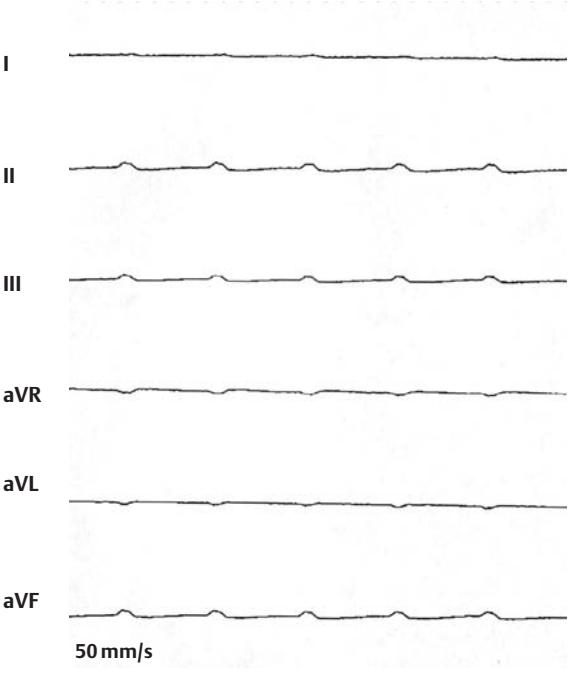
Mechanism:
- Complete interruption of impulse conduction at the AV node
- Blockade below the level of the bundle of His

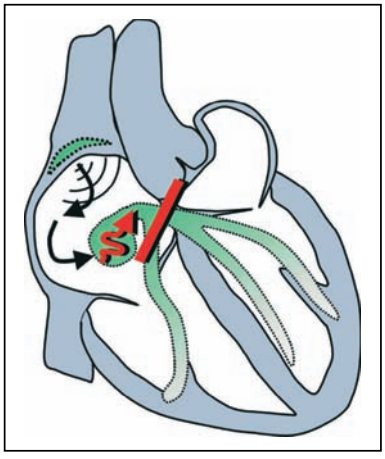
ECG characteristics:
- Complete dissociation of atria and ventricles,
- P waves can easily be recognized in the absence of an escape rhythm

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, CMP, inflammatory, medications

Treatment:
- Immediate insertion of pacemaker (in acute cases: temporary device)

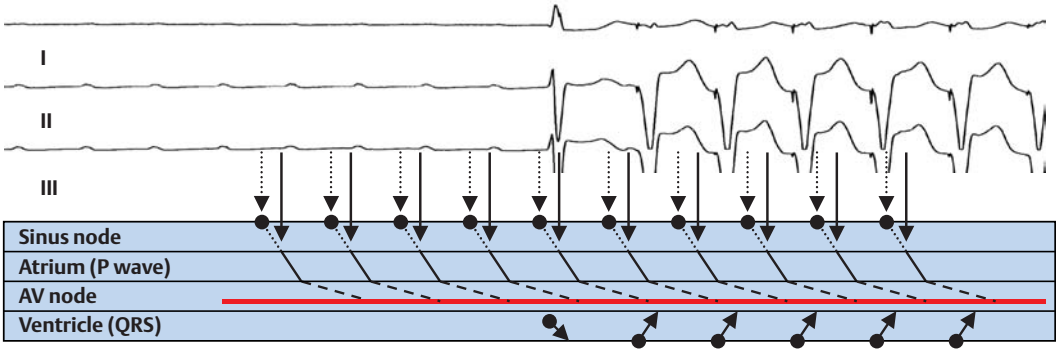
3rd Degree AV Block Without Escape Rhythm





Differential diagnosis:
- Sinus node arrest (no P waves)
- Artefact

3rd Degree AV Block Without Escape Rhythm Following Insertion of a Temporary Pacemaker



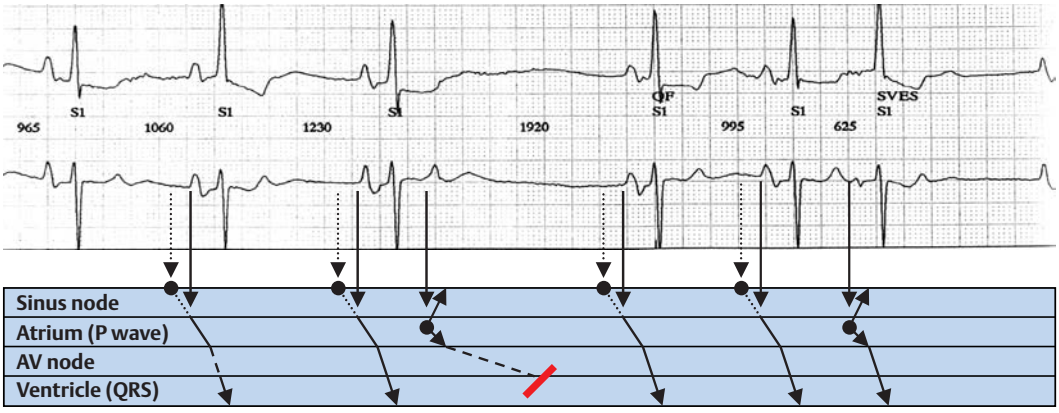
Mechanism:
- Complete interruption of impulse conduction at the AV node
- Insertion of a temporary ventricular pacing device

ECG characteristics:
- P waves can easily be recognized in the absence of an escape rhythm; prior to activation of the temporary ventricular pacemaker await an escape beat, then regular stimulation

Etiology:
- CHD, acute posterior myocardial infarction
- Heart defects, hypertension, CMP, inflammatory, medications

Treatment:
- Temporary insertion of pacemaker in acute cases; implantation of a permanent pacemaker is then required

SVES Blocked at the AV Node



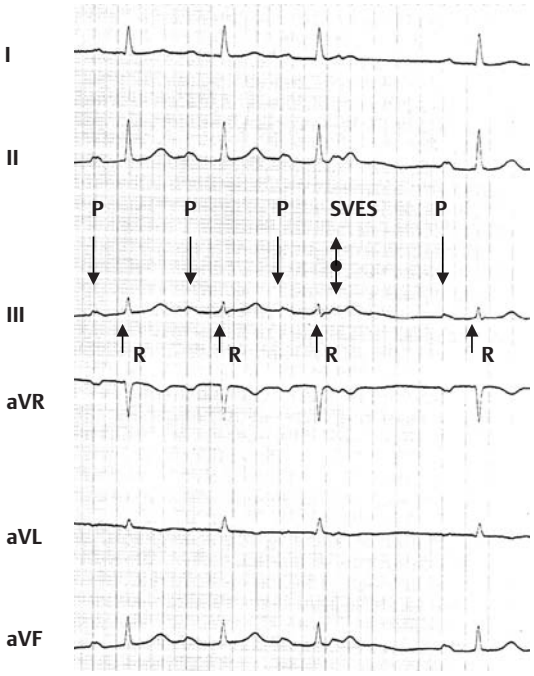
Mechanism:
- Single intermittent interruption of impulse conduction at the AV node following premature supraventricular contraction

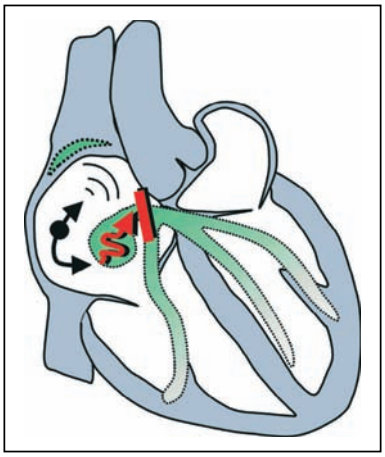
ECG characteristics:
- "Pause with premature P wave"
- Absence of a QRS complex
- Duration of pause < 2 × RR interval

Etiology:
- Medications that may cause conduction delay
- Degenerative disease

Treatment:
- Cessation of medications that may cause bradycardia
- Conservative, as long as no additional higher-grade AV blockades are present, otherwise insertion of pacemaker

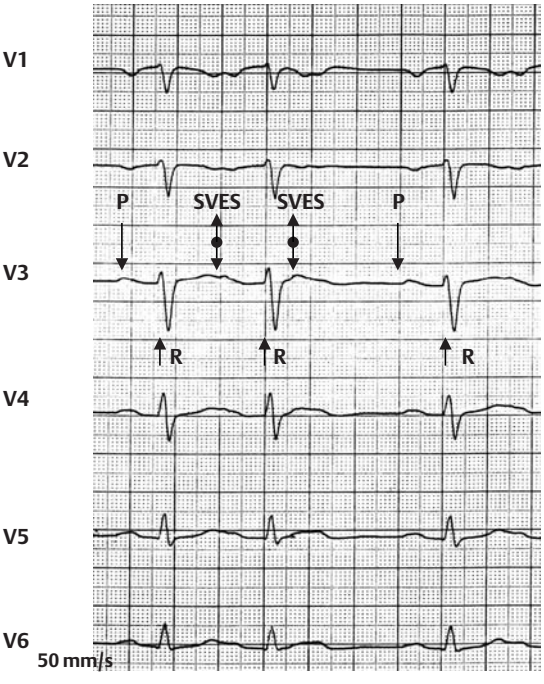
SVES Blocked at the AV Node, preexisting 1st Degree AV Block

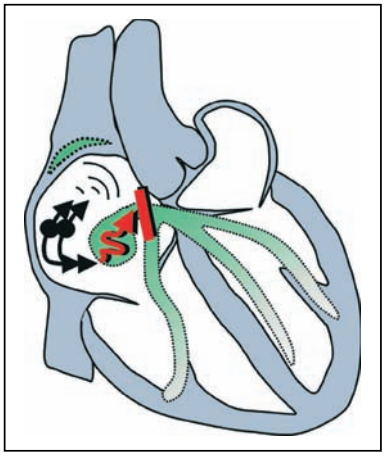




Differential diagnosis:
- 2nd degree AV block, Wenckebach and Mobitz
- 2nd degree SA block
- Sinus arrhythmia

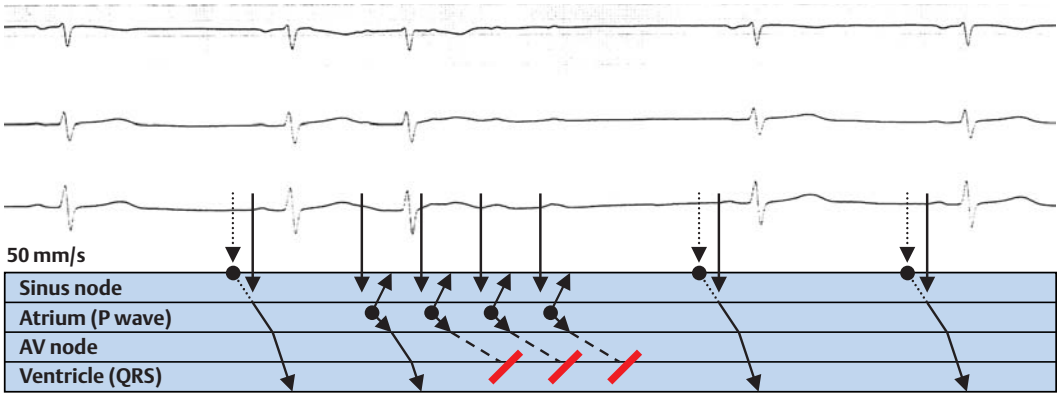
Supraventricular Couplet with Partial Block at the AV Node





Differential diagnosis:
- 2nd degree AV block, Wenckebach and Mobitz
- 2nd degree SA block
- Sinus arrhythmia

Supraventricular Salve with Partial Block at the AV Node



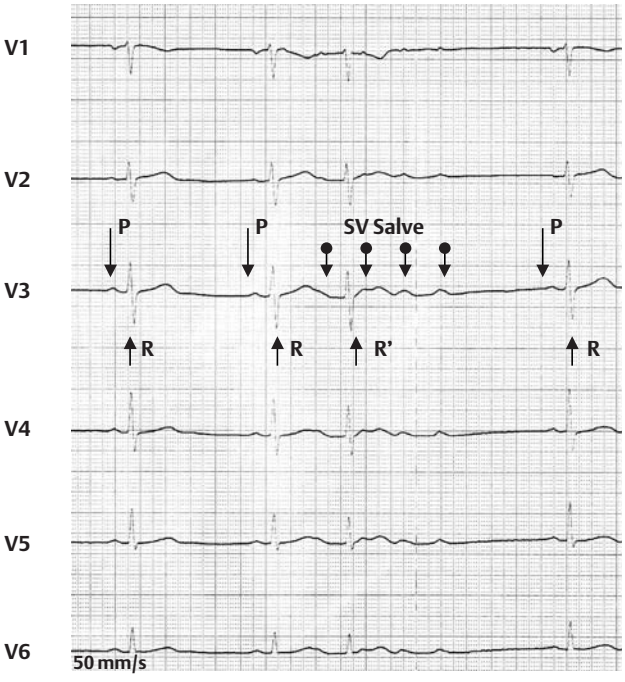
Mechanism:
- Intermittent interruption of impulse conduction at the AV node following premature supraventricular contraction

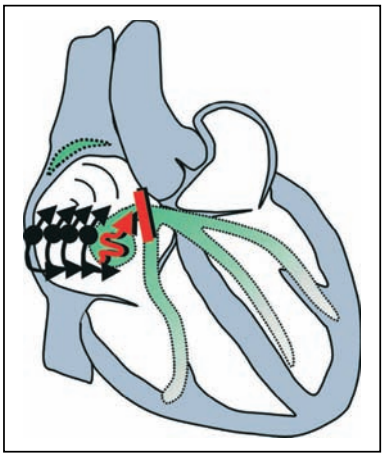
ECG characteristics:
- "Pause with premature P wave," whereby the first SVES at the AV node is conducted; no further conduction of successive contractions

Etiology:
- Medications causing conduction delay
- Degenerative disease

Treatment:
- Cessation of medications that may cause bradycardia
- Conservative, as long as no additional higher-grade AV blockades are present, otherwise insertion of pacemaker

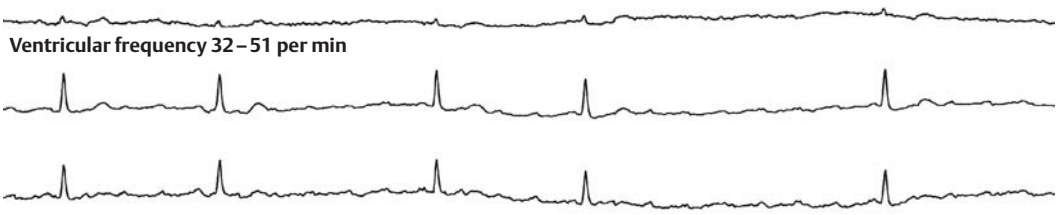
SV Salve with Partial Block at the AV Node

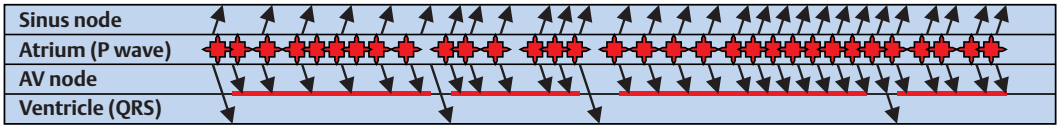




Differential diagnosis:
- SVES blocked at the AV node
- Episode of atrial flutter blocked at the AV node
- Intermittent 3rd degree AV block
- Sinus arrhythmia

Absolute Bradyarrhythmia with Atrial Fibrillation





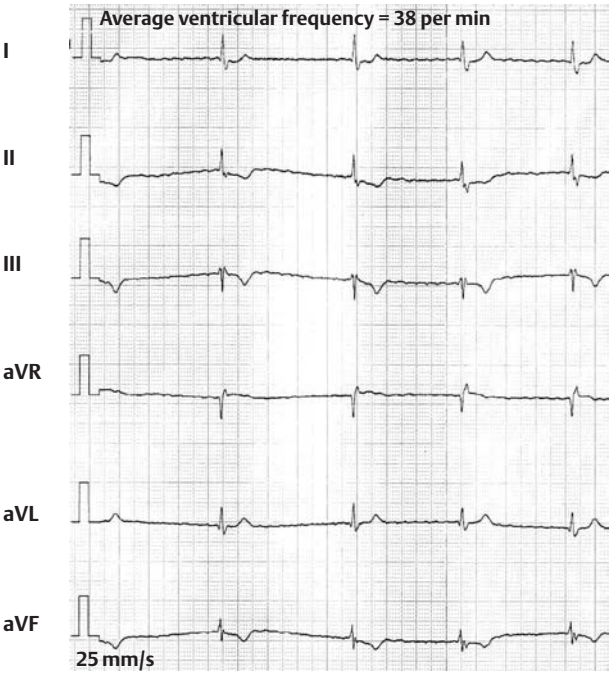
Mechanism:
- Intermittent interruption of impulse conduction at the AV node

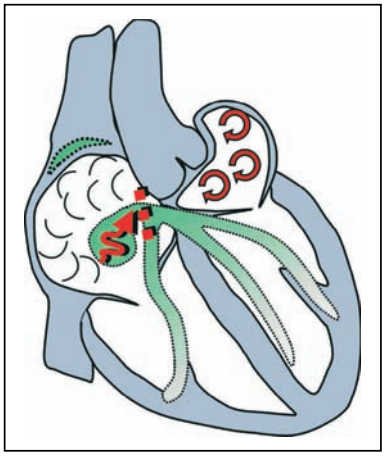
ECG characteristics:
- "Pause with fibrillation waves (f)"

Etiology:
- Medications that may cause conduction delay
- Other causes (see AV block)

Treatment:
- Cessation of medications that may cause bradycardia
- In persistent symptomatic bradycardia: insertion of pacemaker

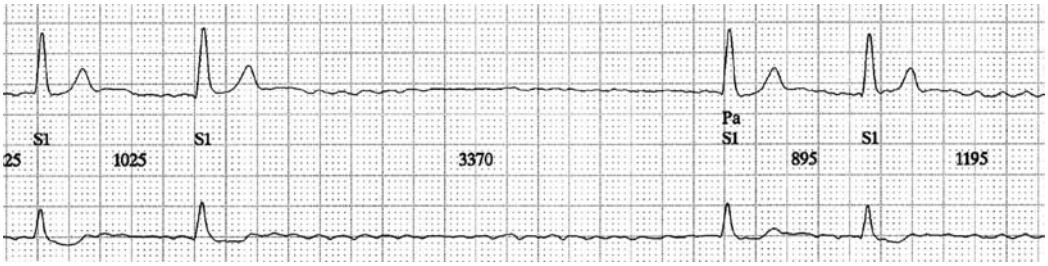
Absolute Bradyarrhythmia with Atrial Fibrillation

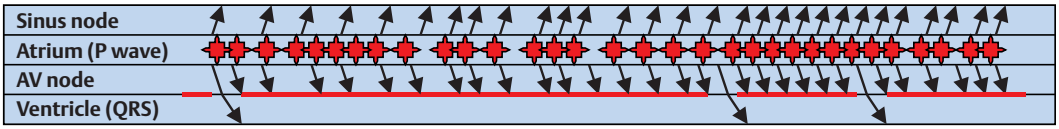




Differential diagnosis:
- SV salve blocked at the AV node
- Atrial flutter blocked at the AV node
- Intermittent 3rd degree AV block with sinus tachycardia

Intermittent AV Conduction Disorders with Atrial Fibrillation





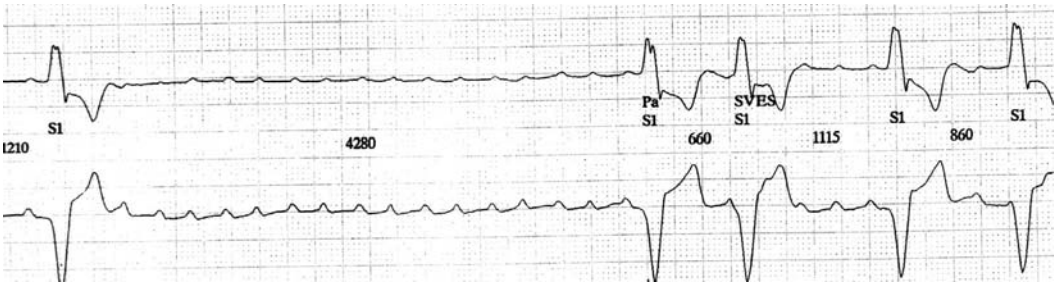
Mechanism:
- Intermittent interruption of impulse conduction at the AV node

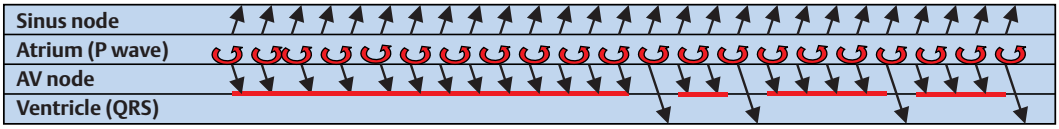
ECG characteristics:
- Bradycardic atrial fibrillation
- In addition, a "pause with fibrillation waves (f)"

Etiology:
- Medications that may cause conduction delay
- Other causes (see AV block)

Treatment:
- Cessation of medications that may cause bradycardia
- In persistent symptomatic bradycardia: insertion of pacemaker

Intermittent AV Conduction Disorders with Atrial Fibrillation





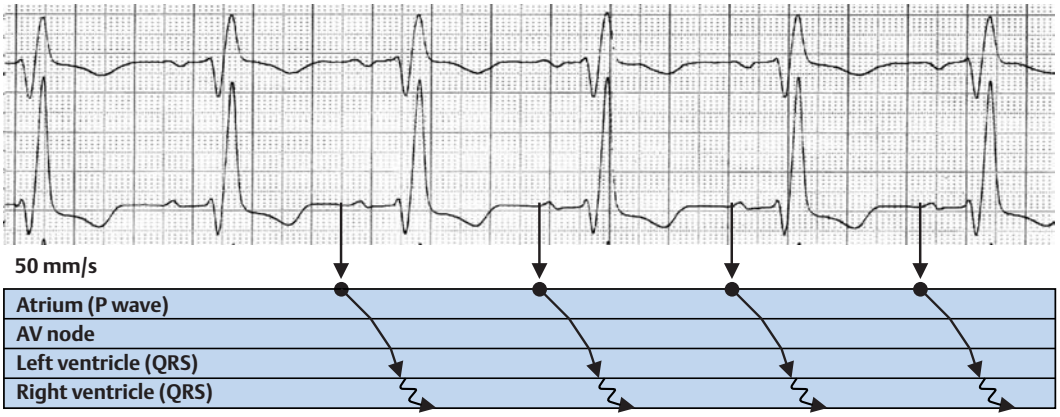
Mechanism:
- Intermittent interruption of impulse conduction at the AV node

ECG characteristics:
- "Pause with flutter waves"

Etiology:
- Medications that may cause conduction delay
- Other causes (see AV block)

Treatment:
- Cessation of medications that may cause bradycardia
- In persistent symptomatic bradycardia: insertion of pacemaker

Isolated Complete Right Bundle Branch Block



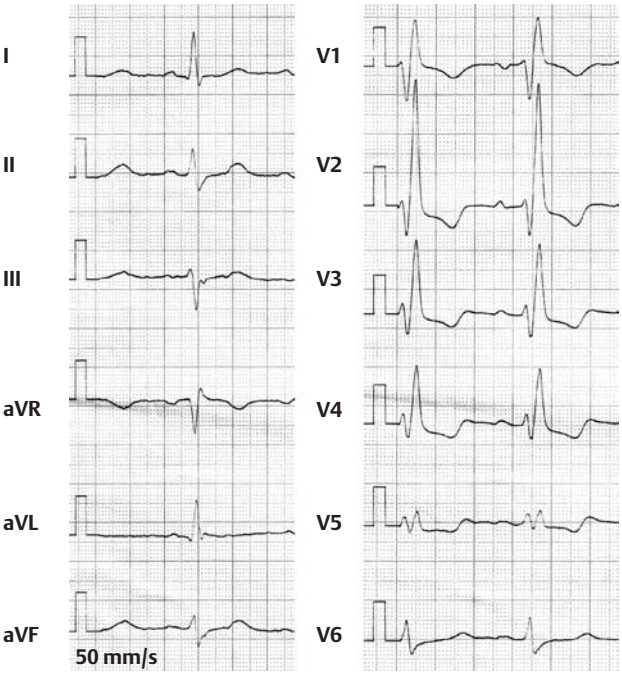
Mechanism:
- Delayed conduction in the right bundle branch, secondary (delayed) excitation of the right ventricle via the left ventricular myocardium

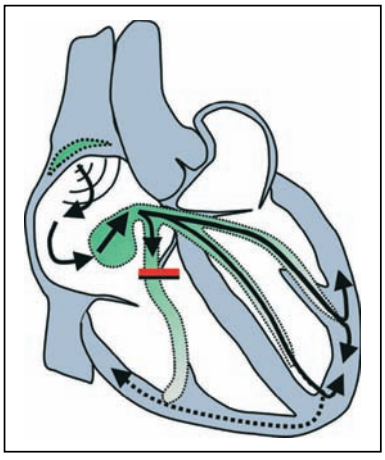
ECG characteristics:
- QRS ≥ 0.12 s; discordance of the ventricular repolarization (ST segment and T wave)
- All axes
- QRS configuration: V1/2 – "rSR," V5/6 – "qRS'"
- Turning point in V1: > 0.08 s

Etiology:
- Congenital, functional
- Right heart load (acute: pulmonary embolization; chronic: lung disease, shunt defects)

Treatment:
- Treatment of the underlying disease; no specific treatment of the rhythm

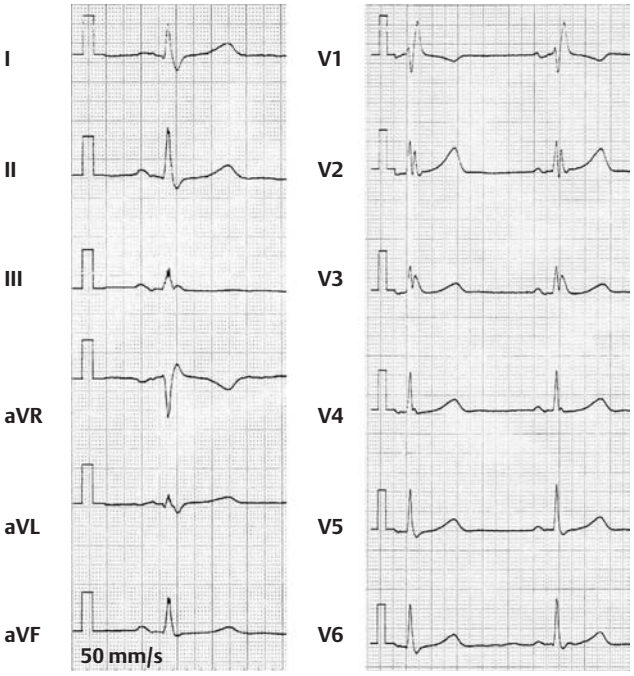
Isolated Complete Right Bundle Branch Block (I)

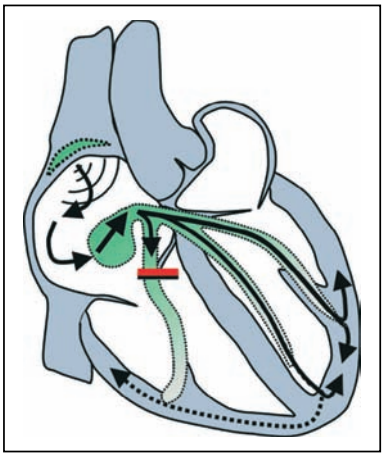




Differential diagnosis:
- Incomplete right bundle branch block
- Bifascicular block
- WPW syndrome

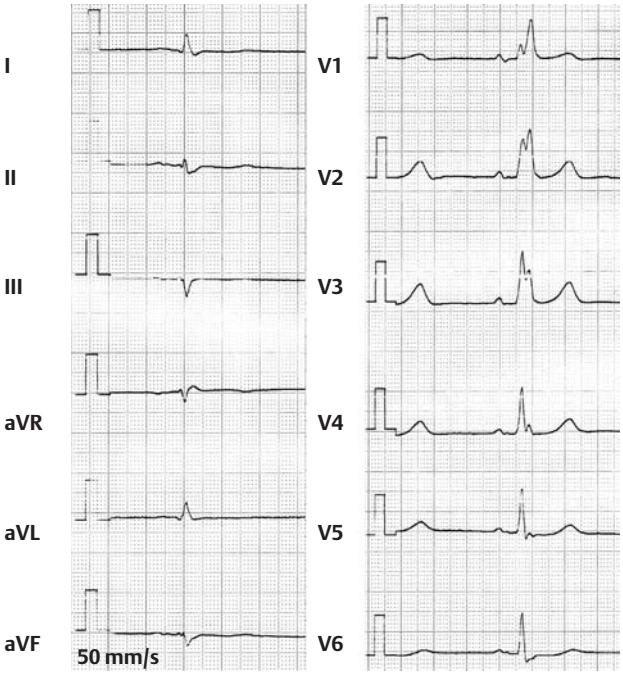
Isolated Complete Right Bundle Branch Block (II)

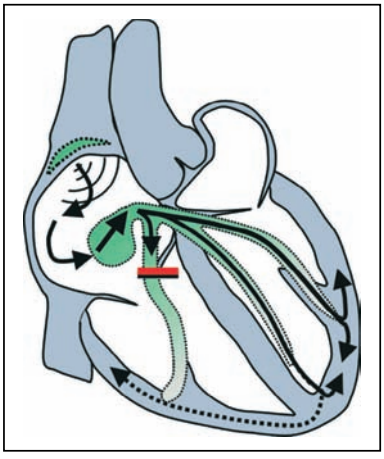




Differential diagnosis:
- Incomplete right bundle branch block
- Bifascicular block
- WPW syndrome

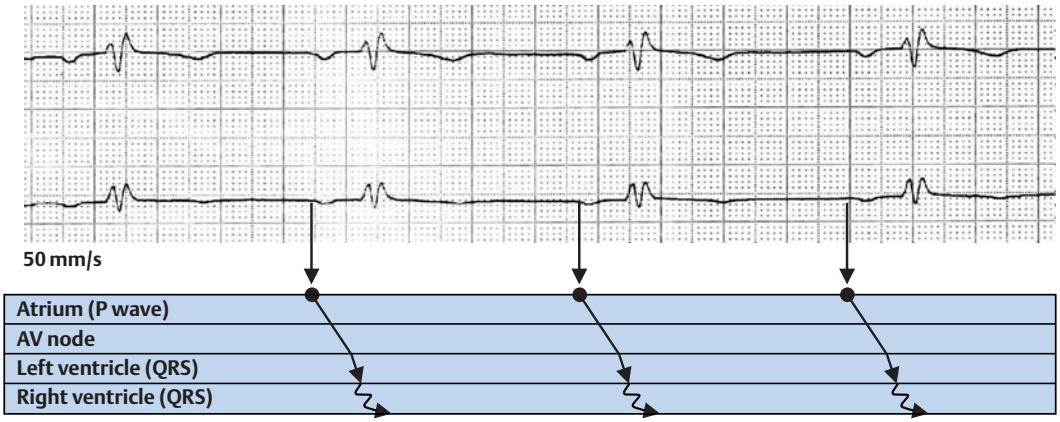
Isolated Complete Right Bundle Branch Block (III)





Differential diagnosis:
- Incomplete right bundle branch block
- Bifascicular block
- WPW syndrome

Isolated Incomplete Right Bundle Branch Block



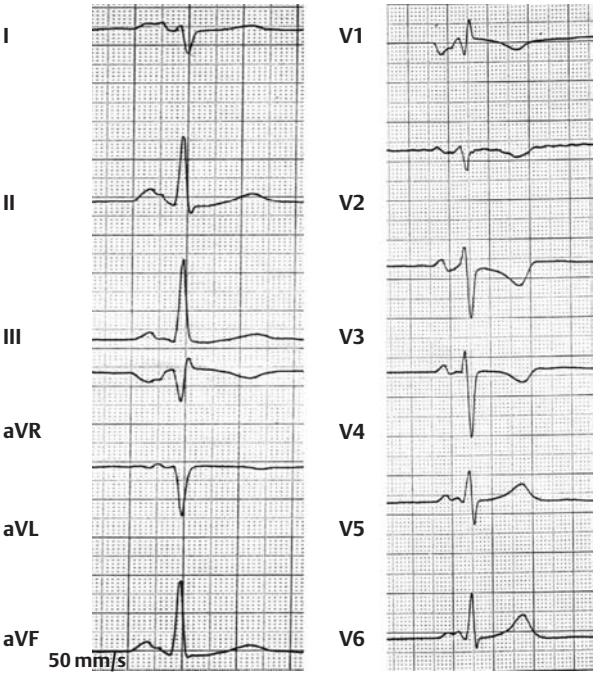
Mechanism:
- Delayed conduction in the right bundle branch, secondary (delayed) excitation of the right ventricle via the left ventricular myocardium

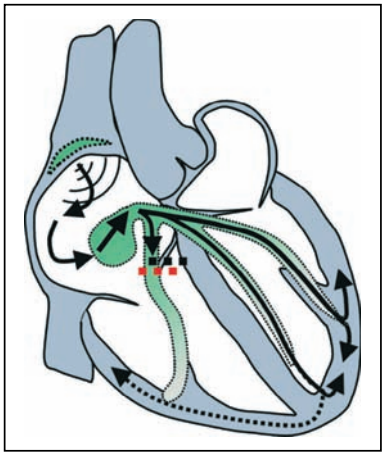
ECG characteristics:
- QRS 0.10–0.11 s; discordance of the ventricular repolarization (ST segment and T wave)
- All axes
- QRS configuration: V1/2 – "rSR," V5/6 – "qRS'"
- Turning point in V1: > 0.08 s

Etiology:
- Congenital, functional
- Right heart load (acute: pulmonary embolization; chronic: lung disease, shunt defects)

Treatment:
- Treatment of the underlying disease; no specific treatment of the rhythm

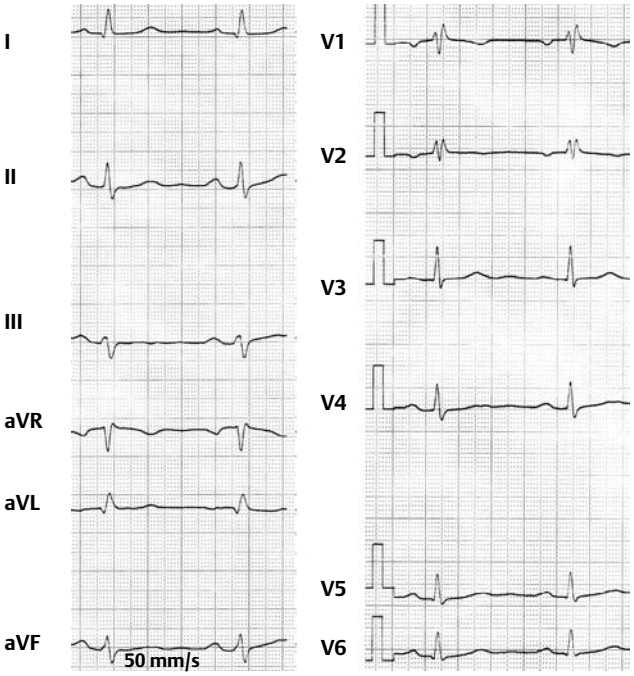
Isolated Incomplete Right Bundle Branch Block

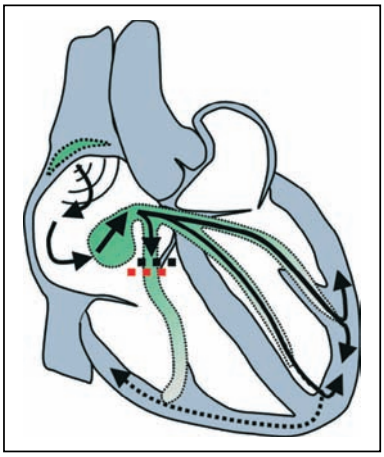




Differential diagnosis:
- Complete right bundle branch block
- Bifascicular block
- WPW syndrome

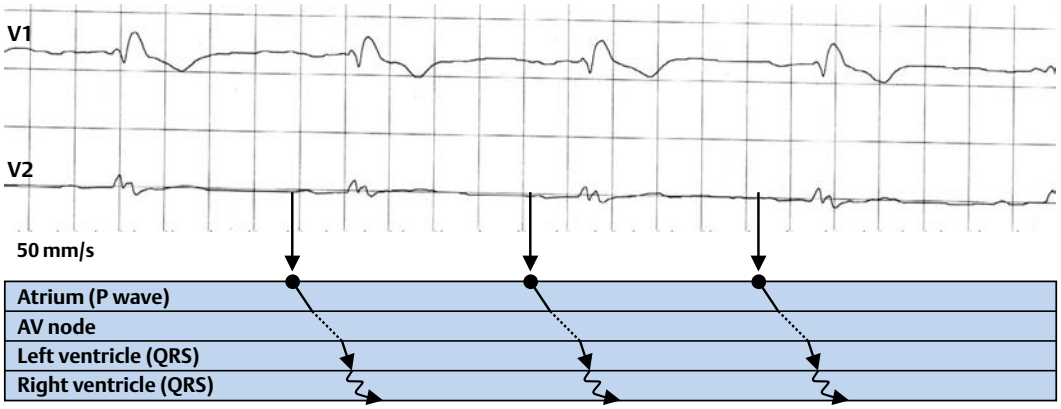
Isolated Incomplete Right Bundle Branch Block





Differential diagnosis:
- Complete right bundle branch block
- Bifascicular block
- WPW syndrome

Complete Right Bundle Branch Block With 1st Degree AV Block



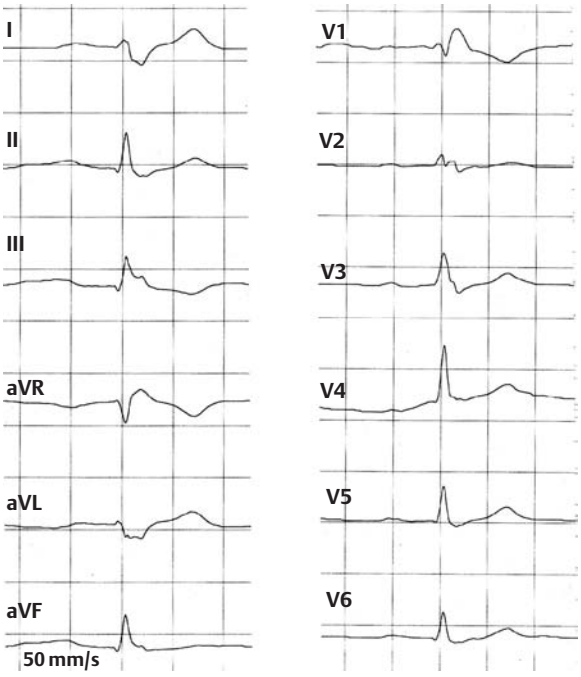
Mechanism:
- Delayed conduction in the right bundle branch, secondary (delayed) excitation of the right ventricle via the left ventricular myocardium

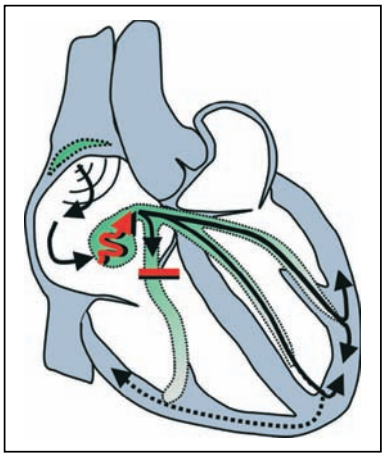
ECG characteristics:
- Complete right bundle branch block (see p. 47) with prolonged PQ interval (> 0.20 s)

Etiology:
- Congenital, functional
- Right heart load (acute: pulmonary embolization; chronic: lung disease, shunt defects, other forms of right heart load)

Treatment:
- Treatment of the underlying disease; no specific treatment of the rhythm

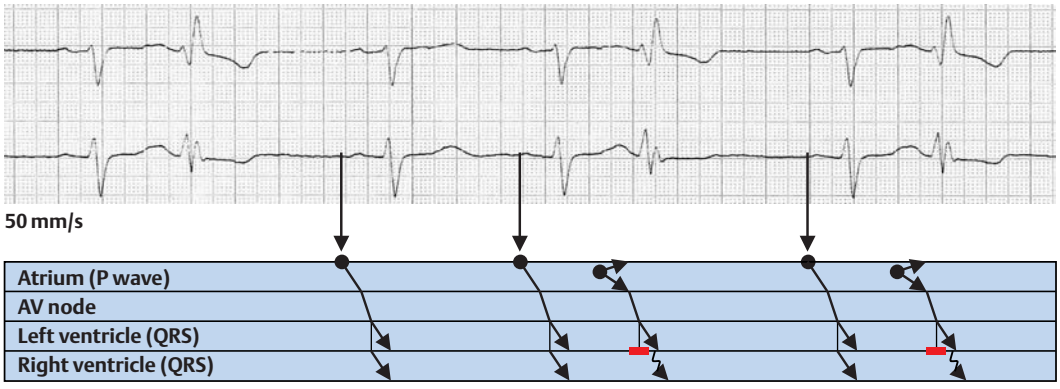
Complete Right Bundle Branch Block With 1st Degree AV Block





Differential diagnosis:
- Incomplete right bundle branch block
- Bifascicular block
- WPW syndrome

Functional Right Bundle Branch Block With SVES



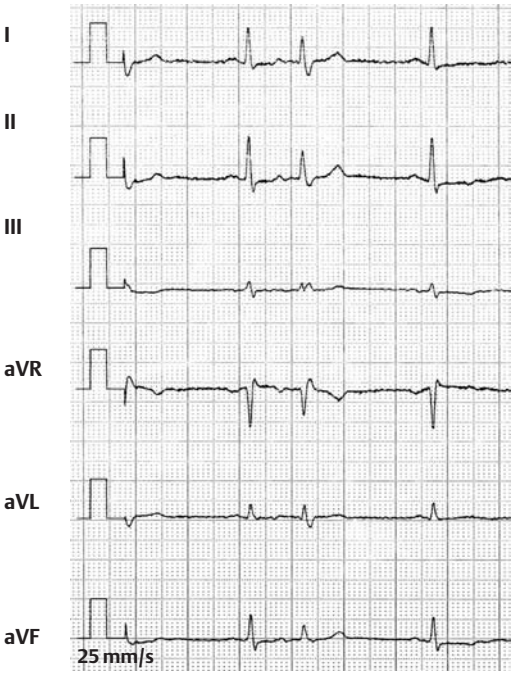
Mechanism:
- Single intermittent right bundle branch block as a result of premature supraventricular contraction

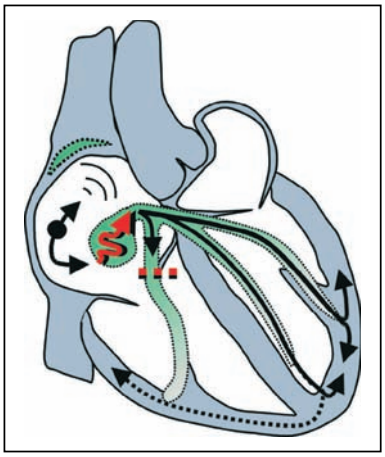
ECG characteristics:
- Premature P wave with right bundle branch block

Etiology:
- Functional, not organic
- Also with right heart load (lung disease, shunt defects)

Treatment:
- Treatment of the underlying disease; no specific treatment of the rhythm

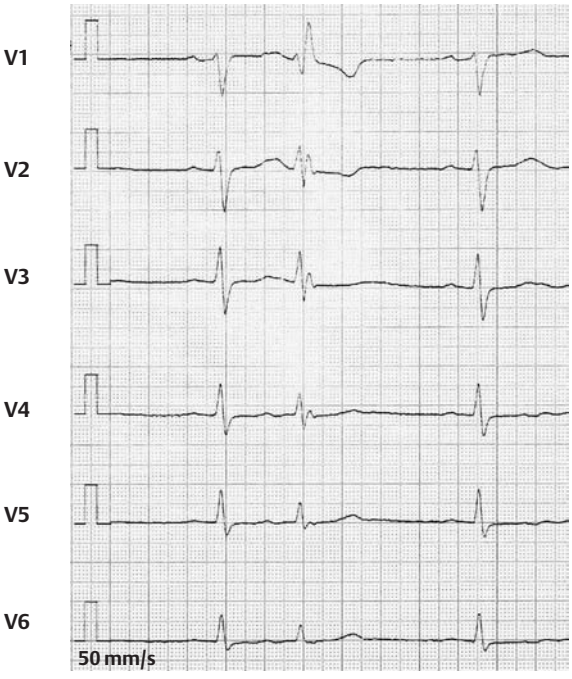
Functional Incomplete Right Bundle Branch Block with SVES

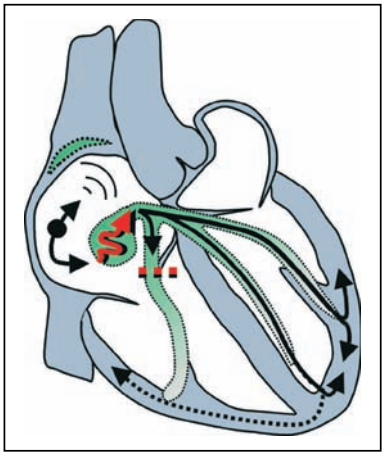




Differential diagnosis:
– Ventricular extrasystole with right bundle branch block configuration

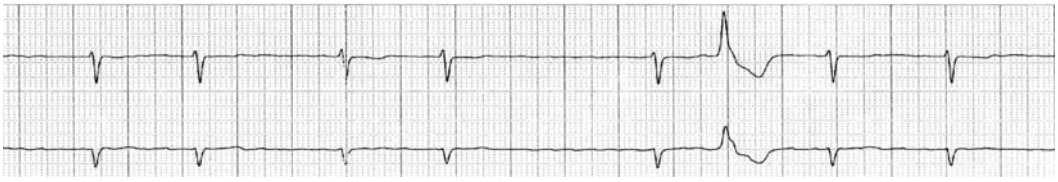
Functional Incomplete Right Bundle Branch Block with SVES



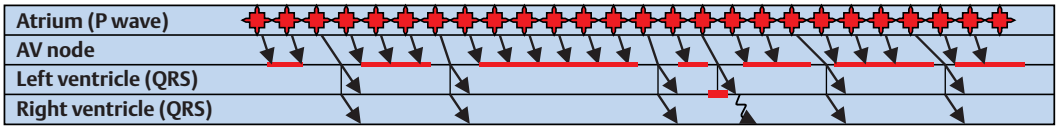


Differential diagnosis:
– Ventricular extrasystole with right bundle branch block configuration

Intermittent Functional Right Bundle Branch Block With Atrial Fibrillation (Ashmann Phenomenon)



25 mm/s



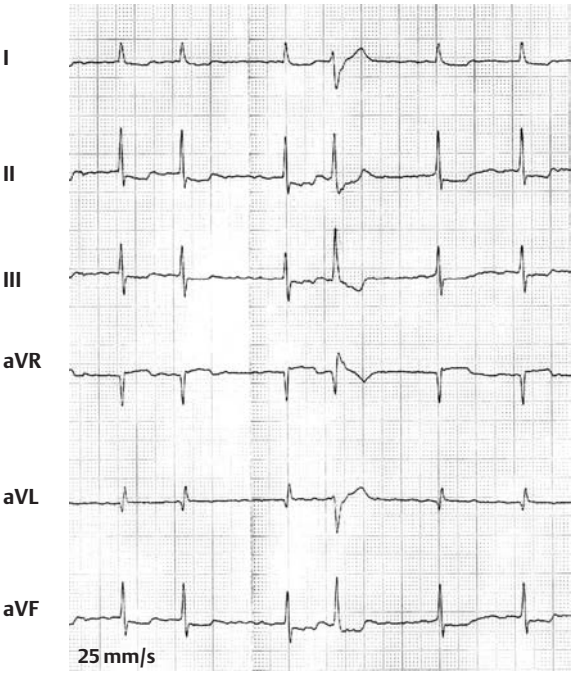
Mechanism:
- Intermittent right bundle branch block as a result of rapid conduction of atrial fibrillation

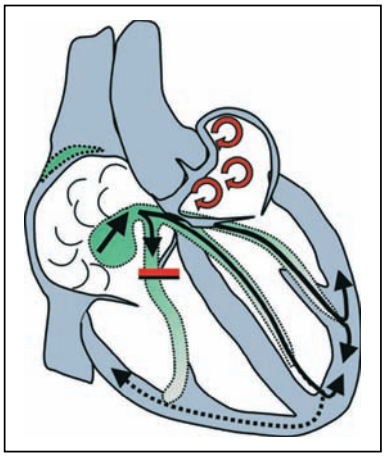
ECG characteristics:
- Slow contraction followed by a fast contraction (short-long-short sequence), then occurrence of faster contractions with right bundle branch block

Etiology:
- Functional, not organic
- Also with right heart load (lung disease, shunt defects)

Treatment:
- Treatment of the underlying disease; no specific treatment of the rhythm

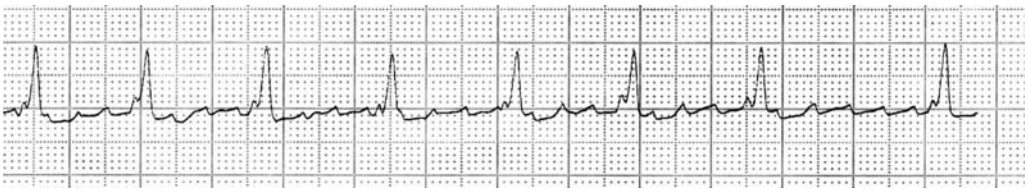
Intermittent Functional Right Bundle Branch Block With Atrial Fibrillation (Ashmann Phenomenon)

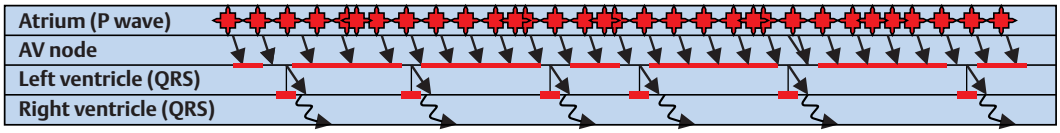




Differential diagnosis:
- Ventricular extrasystole with right bundle branch block configuration or salve

Complete Right Bundle Branch Block with Atrial Fibrillation





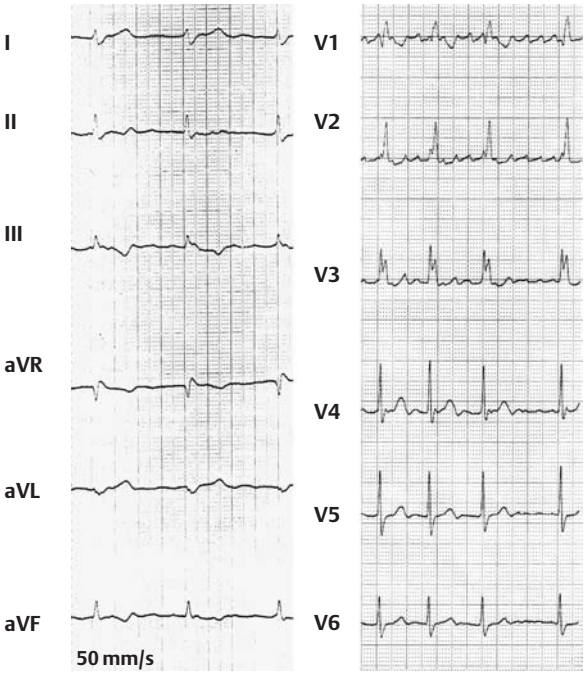
Mechanism:
- Right bundle branch block and thereby secondary (delayed) excitation of the right ventricle via the left ventricular myocardium

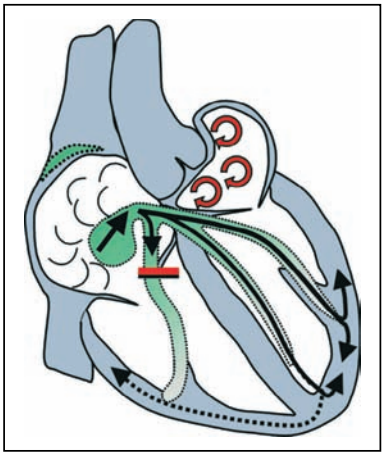
ECG characteristics:
- Complete right bundle branch block/absolute arrhythmia with atrial fibrillation
- The conduction capacity of the AV node determines the ventricular frequency

Etiology:
- Congenital, functional
- Right heart load (acute: pulmonary embolization; chronic: lung disease, shunt defects)

Treatment:
- Insertion of a pacemaker in patients with symptoms due to bradycardic atrial fibrillation
- Conservative in the event of a lack of clinics

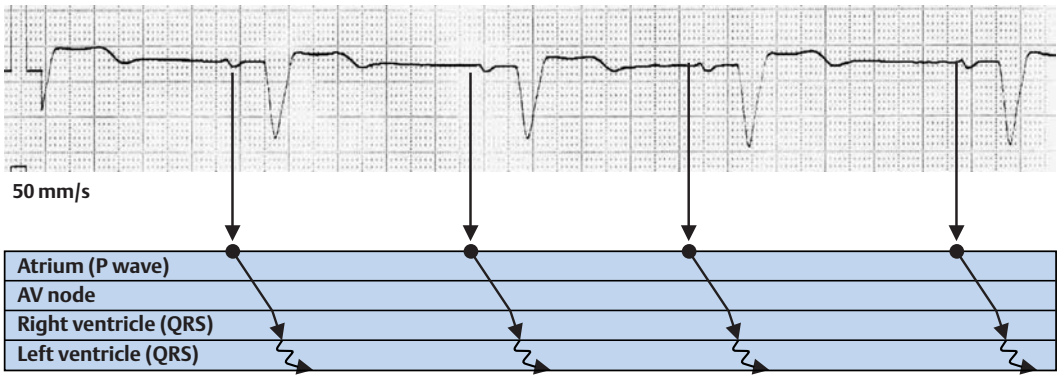
Complete Right Bundle Branch Block with Atrial Fibrillation





Differential diagnosis:
- Ventricular salve with right bundle branch block configuration

Isolated Complete Left Bundle Branch Block



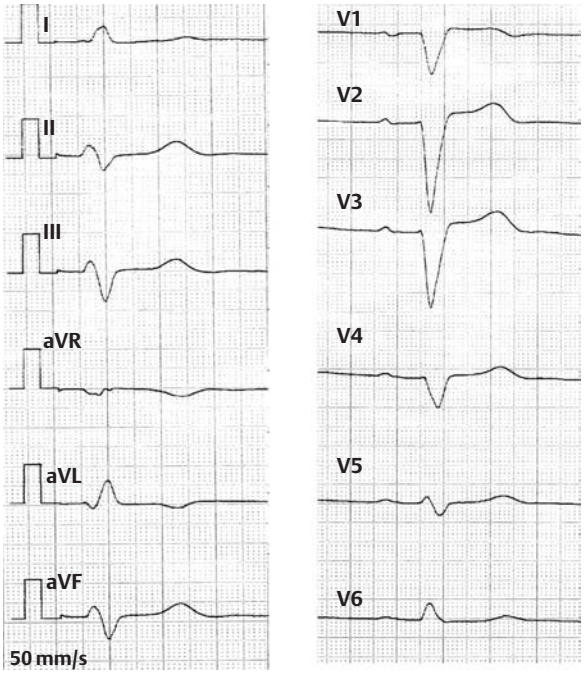
Mechanism:
- Block in the left bundle branch resulting in secondary (delayed) excitation of the left ventricle via the right ventricular myocardium

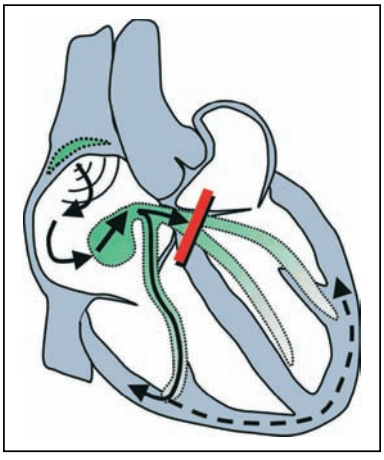
ECG characteristics:
- QRS ≥ 0.12 s; discordance of the ventricular repolarization (ST segment and T wave)
- Ranging from left axis to marked left axis deviation
- QRS configuration : V1/2 – "QS," V5/6 – "R'"
- Turning point in V6: > 0.05 s

Etiology:
- CHD, acute myocardial infarction (poor prognosis), heart defects, hypertension, medications that may cause conduction delay, carditis, DCM

Treatment:
- Cessation of medications that may cause conduction delay
- In symptomatic patients (syncope) bradycardia: insertion of pacemaker (or in cases of an unknown cause of syncope: electrophysiological study to exclude ventricular arrhythmias)
- Conservative in the event of a lack of clinics

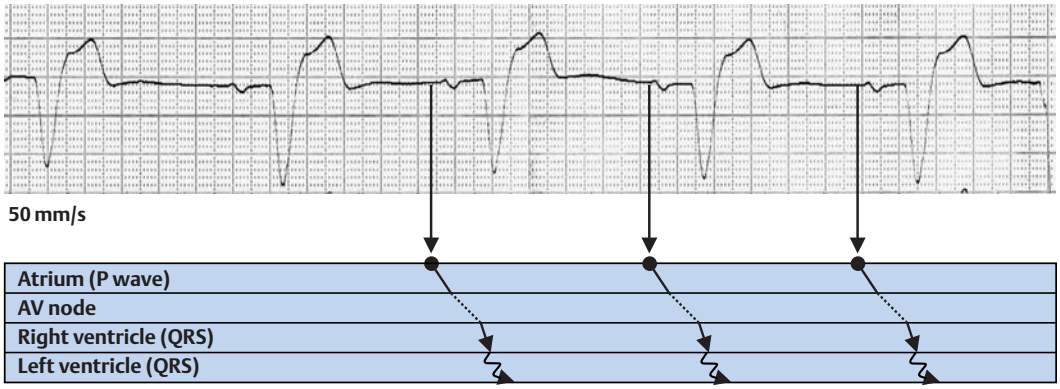
Isolated Complete Left Bundle Branch Block





Differential diagnosis:
In preexisting bundle branch block:
- Atrial flutter with 3 : 1 conduction
- AV conduction delay with SVES
- 2nd or 3rd degree AV block
- Prolonged P wave duration with interatrial block

Complete Left Bundle Branch Block With 1st Degree AV Block



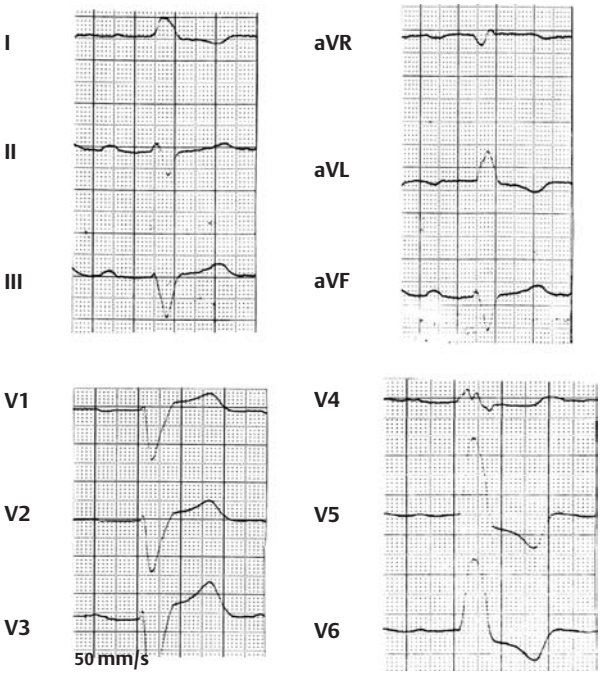
Mechanism:
- Delayed conduction at the AV node with additional block in the left bundle branch resulting in secondary (delayed) excitation of the left ventricle via the right ventricular myocardium

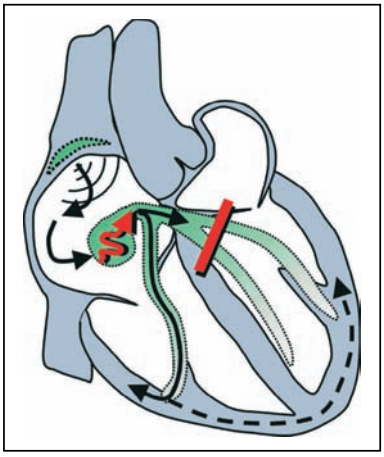
ECG characteristics:
- Complete left bundle branch block with delayed PQ interval (> 0.20 s)

Etiology:
- CHD, acute myocardial infarction (poor prognosis), cardiac defects, hypertension, medications that may cause conduction delay, carditis, DCM

Treatment:
- Cessation of medications that may cause conduction delay
- In symptomatic patients (syncope) bradycardia: insertion of pacemaker (or in cases of an unknown cause of syncope: electrophysiological study to exclude ventricular arrhythmias)
- Conservative in the event of a lack of clinics

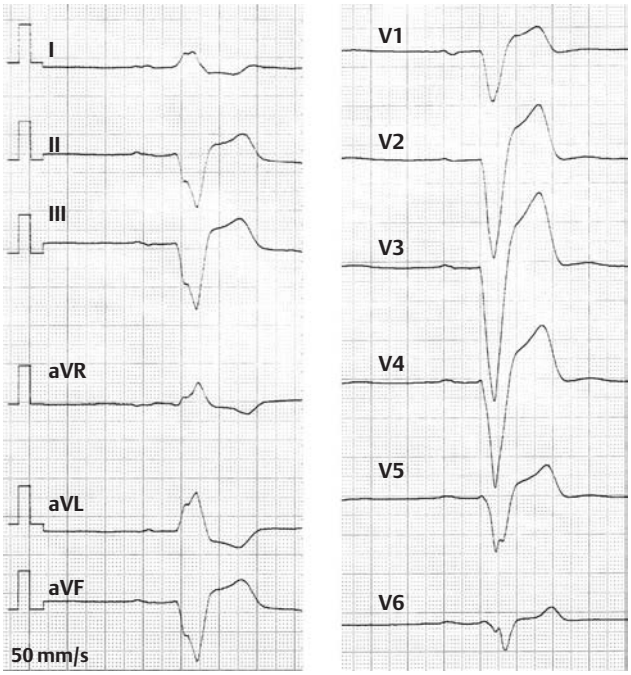
Complete Left Bundle Branch Block With 1st Degree AV Block

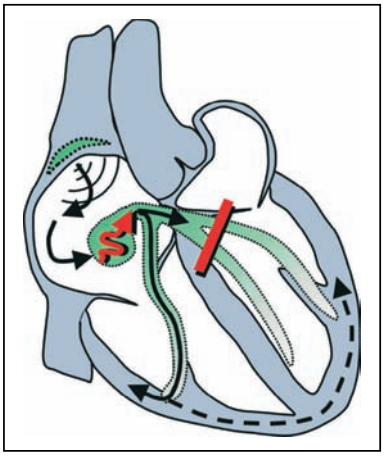




Differential diagnosis:
In preexisting bundle branch block
- Atrial flutter with 3 : 1 conduction
- AV conduction delay with SVES
- 2nd or 3rd degree AV block
- Prolonged P wave duration with interatrial block

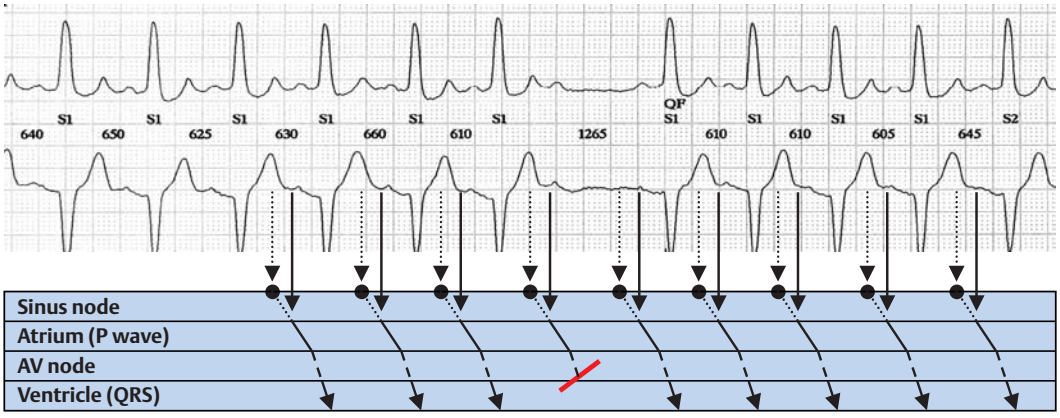
Complete Left Bundle Branch Block With 1st Degree AV Block





Differential diagnosis:
In preexisting bundle branch block
- Atrial flutter with 3 : 1 conduction
- AV conduction delay with SVES
- 2nd or 3rd degree AV block
- Prolonged P wave duration with interatrial block

Complete Left Bundle Branch Block With 2nd Degree AV Block, Mobitz Type



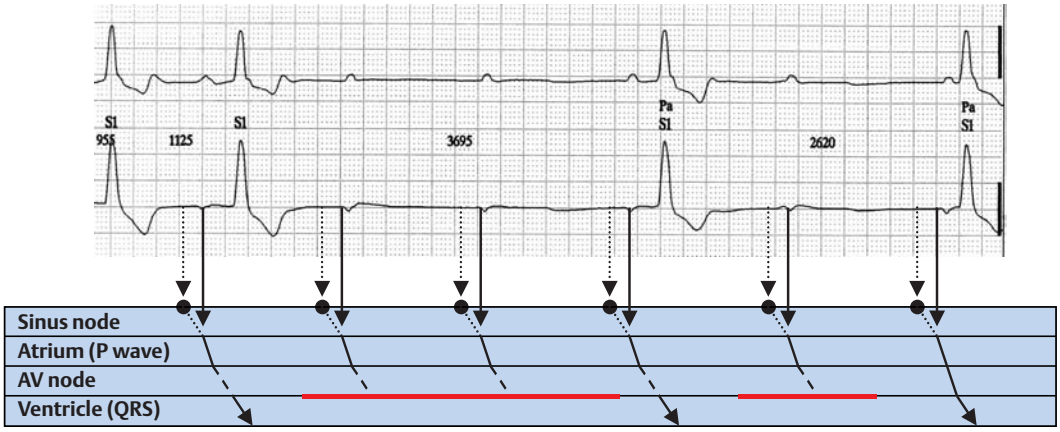
Mechanism:
- Intermittent interruption of impulse conduction at the AV node with complete left bundle branch block
- inferior to the bundle of His

ECG characteristics:
- "Pause with P wave" and bundle branch pattern
- Single interruption of impulse conduction at the AV node with no prolongation of the PQ interval
- Pause = 2 × RR interval

Etiology:
- Heart defects, hypertension, medications
- CHD, acute posterior myocardial infarction

Treatment:
- In the majority of cases, insertion of pacemaker (relative indication in asymptomatic patients)

Complete Left Bundle Branch Block With Intermittent High-Grade (Advanced) AV Block



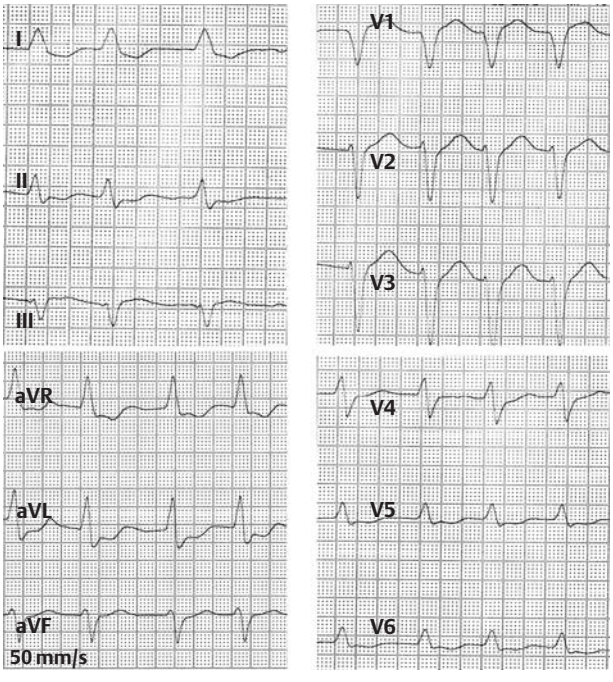
Mechanism:
- Intermittent, complete interruption of impulse conduction at the AV node

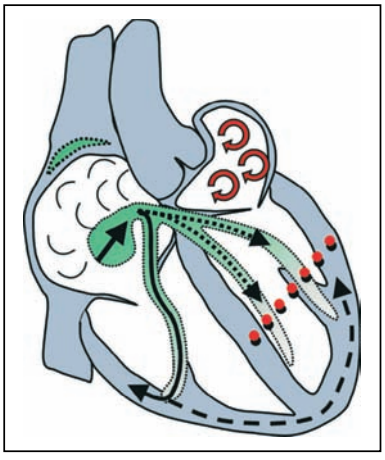
ECG characteristics:
- "Pause with P wave"
- Absence of several QRS complexes; in the above example, additional 2nd degree AV block, Mobitz type, then one episode of normal AV conduction

Etiology:
- Heart defects, hypertension, medications
- CHD, acute posterior myocardial infarction

Treatment:
- In the majority of cases, insertion of a pacemaker (relative indication in asymptomatic patients)
- Cessation of medications that may cause bradycardia

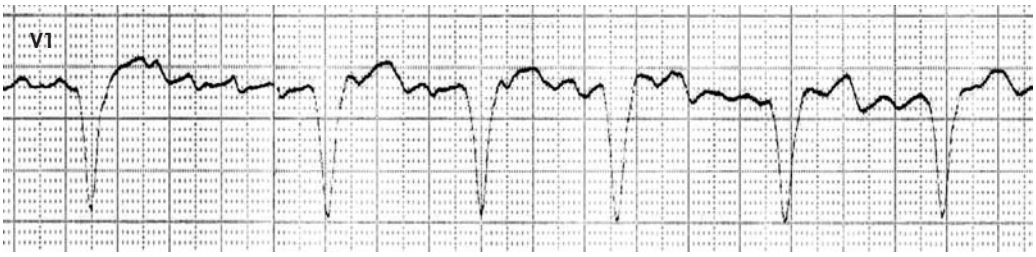
Incomplete Left Bundle Branch Block (With Tachycardial Atrial Fibrillation)

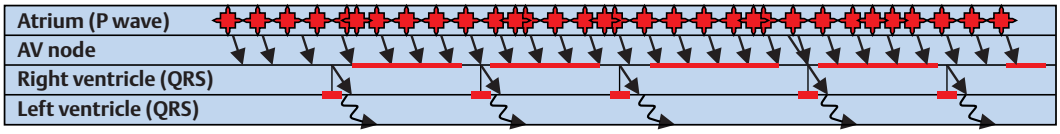




Differential diagnosis:
In preexisting bundle branch block
- Atrial flutter with tachycardic conduction
- AV conduction delay with SVES
- Other supraventricular tachycardias, ventricular tachycardia

Complete Left Bundle Branch Block With Atrial Fibrillation





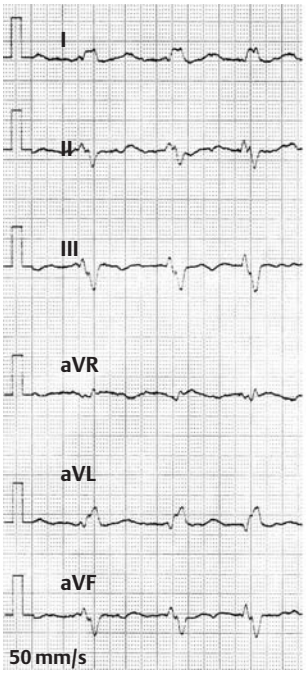
Mechanism:
- Block in the left bundle branch resulting in secondary (delayed) excitation of the left ventricle via the right ventricular myocardium

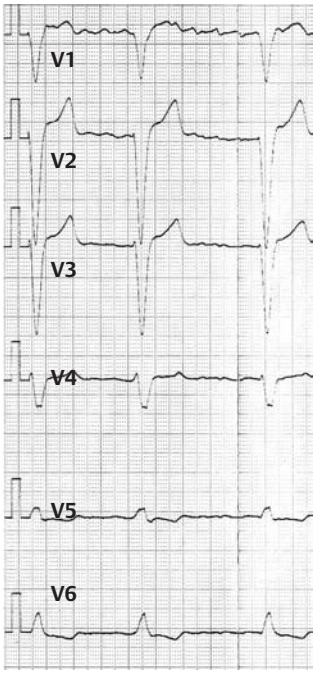
ECG characteristics:
- Complete left bundle branch block with absolute arrhythmia with atrial fibrillation
- The conduction capacity of the AV node determines the ventricular frequency

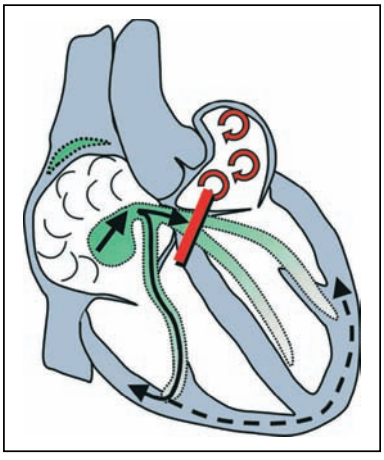
Etiology:
- CHD, acute myocardial infarction (poor prognosis), heart defects, hypertension, medications that may cause conduction delay, carditis, DCM

Treatment:
- Cessation of medications that may cause conduction delay
- In symptomatic patients (syncope) bradycardia: insertion of pacemaker (or in cases of unknown cause of syncope: electrophysiological study to exclude ventricular arrhythmias)

Complete Left Bundle Branch Block With Atrial Fibrillation

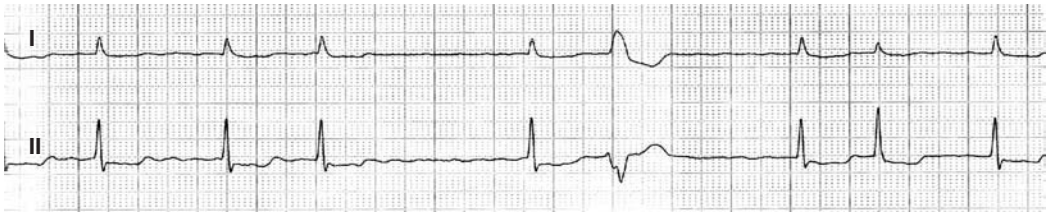


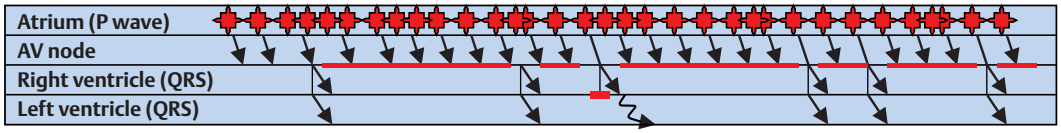




Differential diagnosis:
In preexisting bundle branch block
- Atrial flutter, with 3:1 conduction
- Ventricular tachycardia with left bundle branch configuration

Intermittent Functional Left Bundle Branch Block With Atrial Fibrillation (Ashmann Phenomenon)





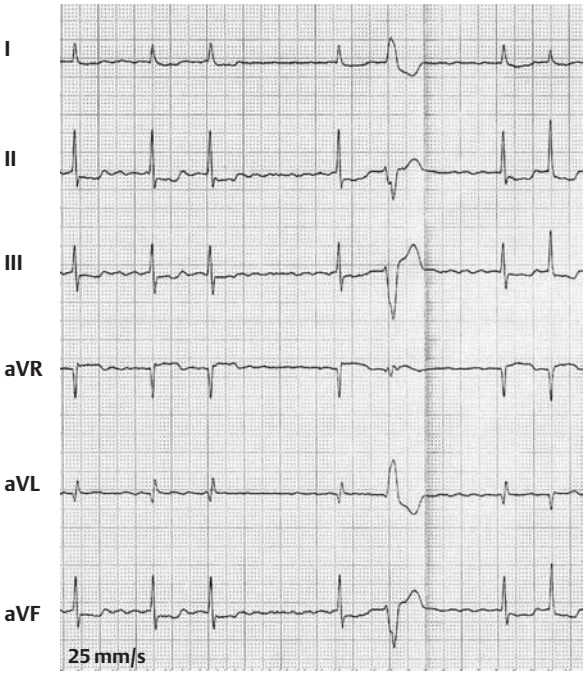
Mechanism:
- Intermittent left bundle branch block as a result of rapidly conducted atrial fibrillation

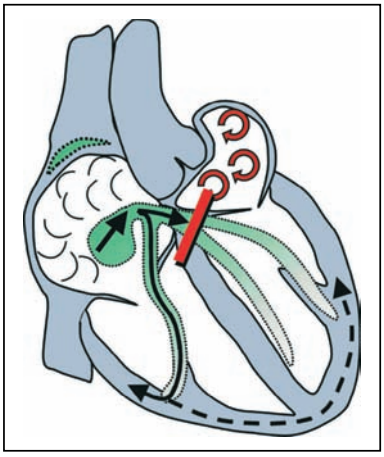
ECG characteristics:
- Slow contraction followed by a fast contraction (long-short sequence), then occurrence of faster contractions with left bundle branch block

Etiology:
- Functional, not organic
- Possible in all types of cardiac disease

Treatment:
- Treatment of the underlying disease, no specific treatment of the rhythm

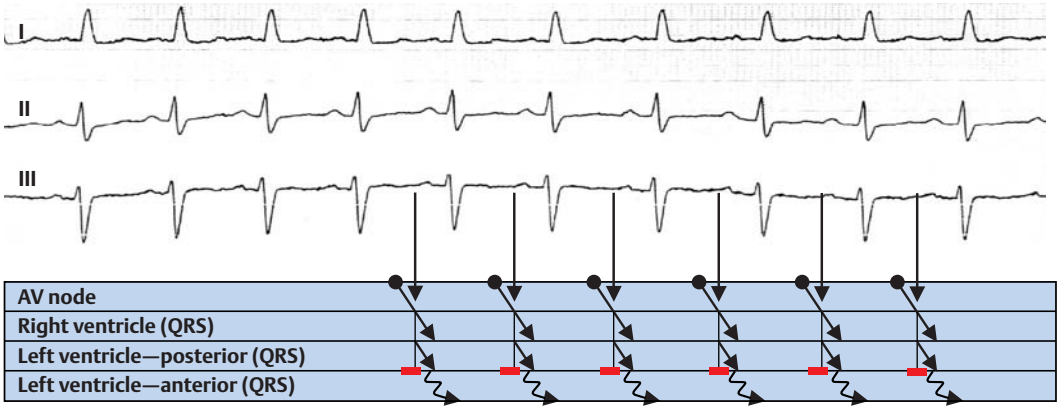
Intermittent Functional Left Bundle Branch Block With Atrial Fibrillation (Ashmann Phenomenon)





Differential diagnosis:
- Ventricular extrasystole with left bundle branch block configuration or salve

Isolated Left Anterior Hemiblock



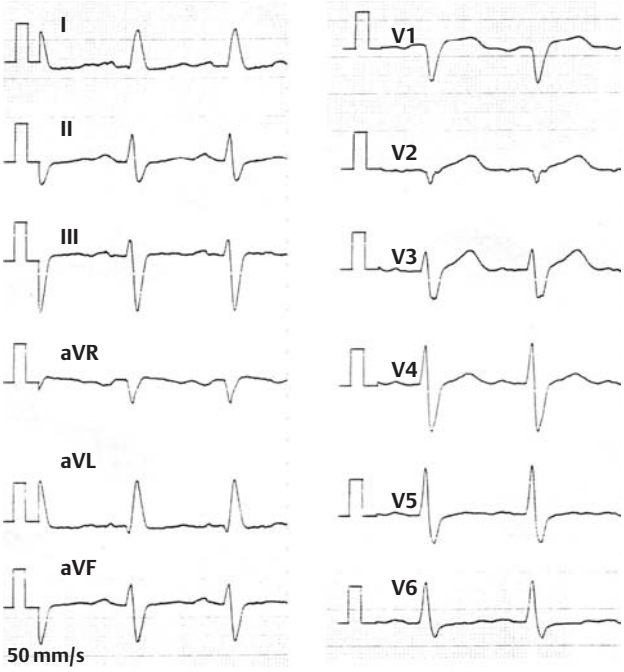
Mechanism:
- Block in the left anterior bundle branch resulting in secondary (delayed) excitation of the anterosuperior regions of the left ventricle

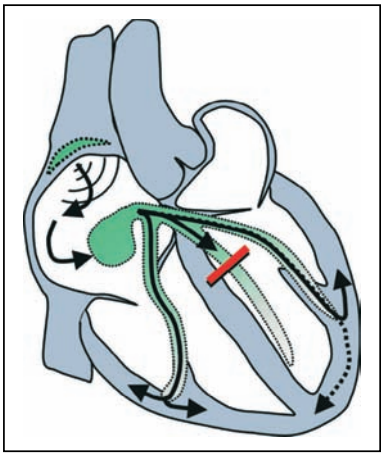
ECG characteristics:
- QRS > 0.10 s; concordance of the ventricular repolarization (ST segment and T wave)
- Marked left axis deviation
- QRS configuration: "deep S" to V6, "R" in I, aVL

Etiology:
- CHD, acute myocardial infarction, heart defects, hypertension, carditis, DCM, thorax deformities, lung disease

Treatment:
- Treatment of the underlying disease; no specific treatment of the rhythm

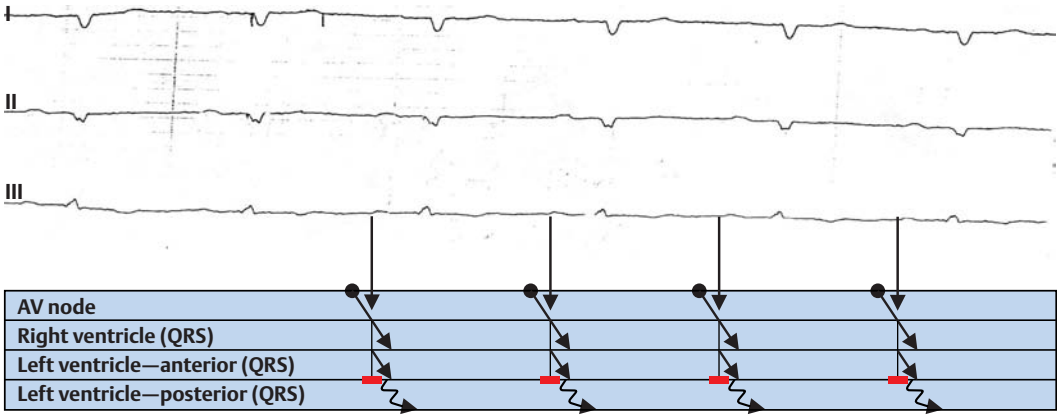
Isolated Left Anterior Hemiblock





Differential diagnosis:
- Right bundle branch block
- Left bundle branch block
- Left posterior hemiblock
- Bifascicular block
- WPW syndrome

Isolated Left Posterior Hemiblock



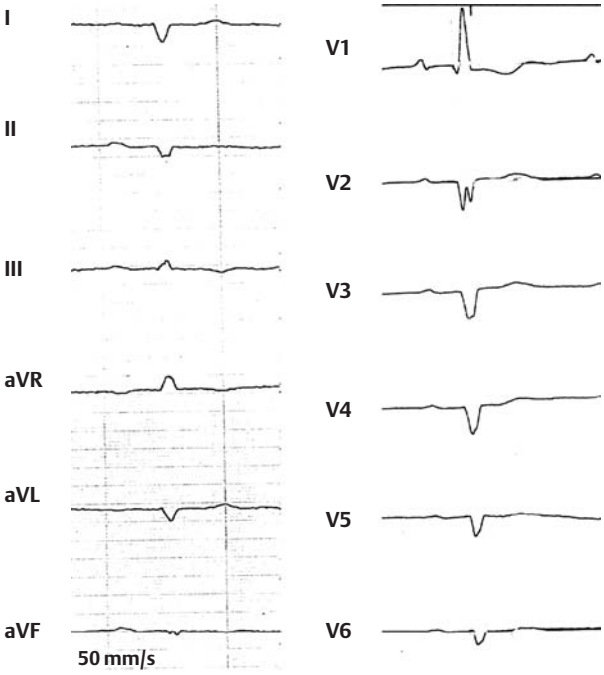
Mechanism:
- Block in the left posterior bundle branch resulting in secondary (delayed) excitation of the postero-inferior regions of the left ventricle

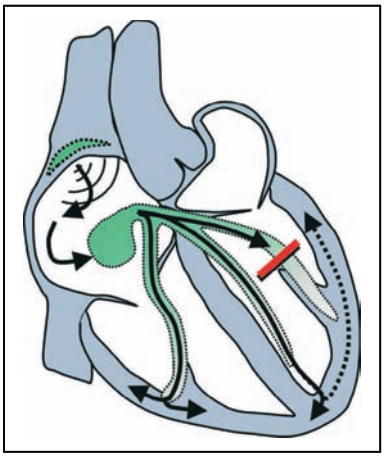
ECG characteristics:
- QRS > 0.10 s; concordance of the ventricular repolarization (ST segment and T wave)
- Marked right axis deviation
- QRS configuration: "absent Q" in V5/6, "rs or rS" in I, II

Etiology:
- CHD, acute myocardial infarction, heart defects, hypertension, carditis, DCM, thorax deformities, lung disease

Treatment:
- Treatment of the underlying disease; no specific treatment of the rhythm

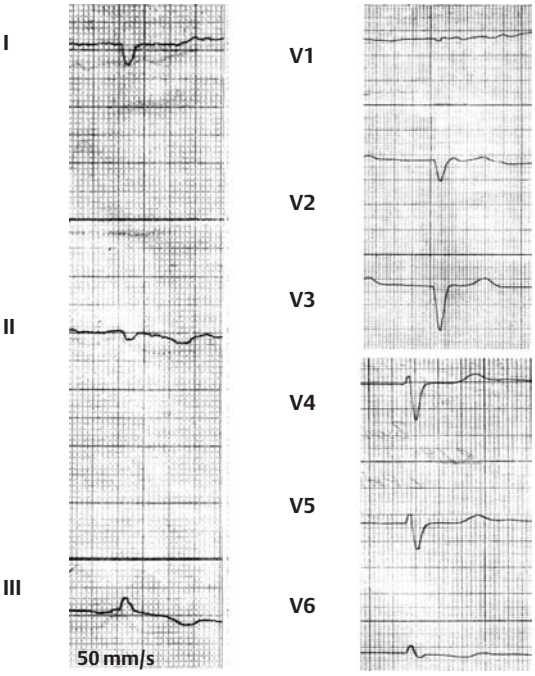
Left Posterior Hemiblock

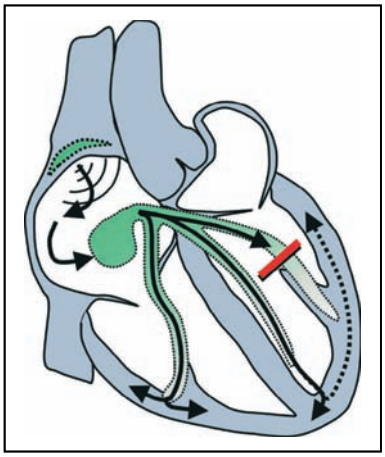




Differential diagnosis:
- Right bundle branch block
- Left bundle branch block
- Left anterior hemiblock
- Bifascicular block
- WPW syndrome

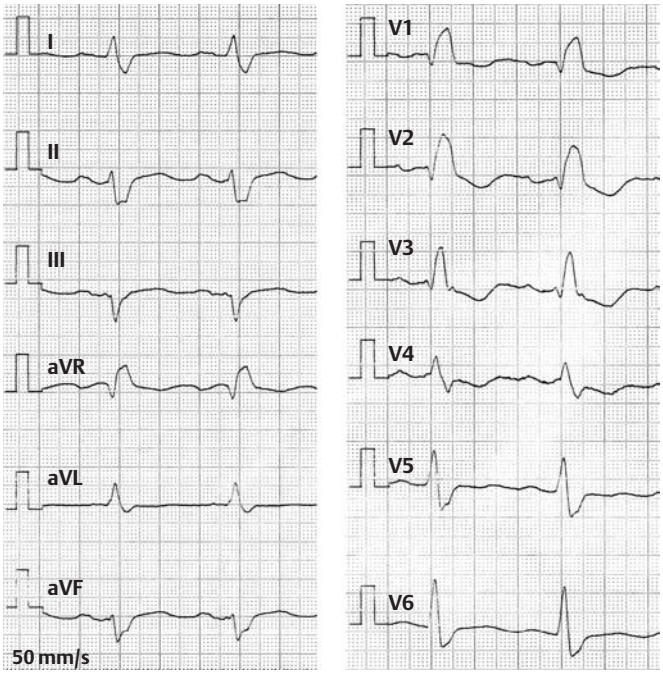
Left Posterior Hemiblock

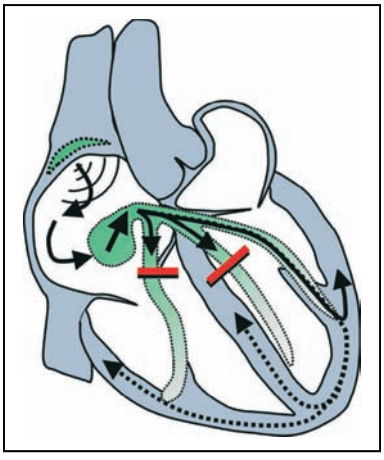




Differential diagnosis:
- Right bundle branch block
- Left bundle branch block
- Left anterior hemiblock
- Bifascicular block
- WPW syndrome

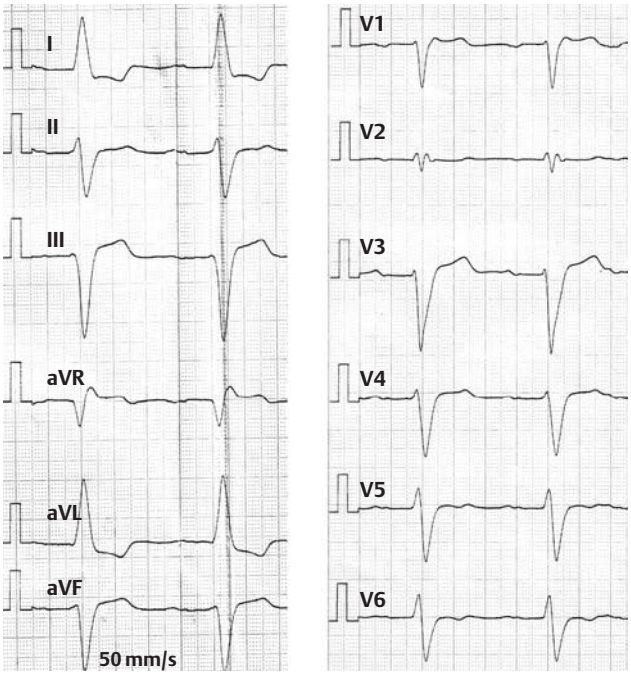
Bifascicular Block—Complete Right Bundle Branch Block and Left Anterior Hemiblock

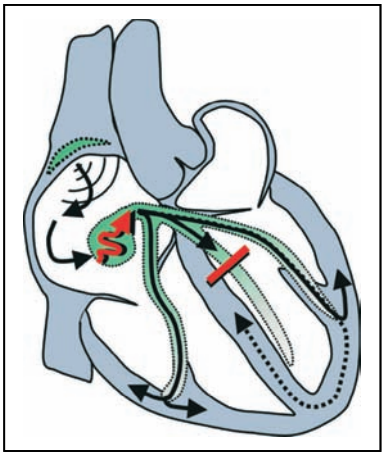




Differential diagnosis:
- Complete left bundle branch block
- Complete right bundle branch block and left posterior hemiblock

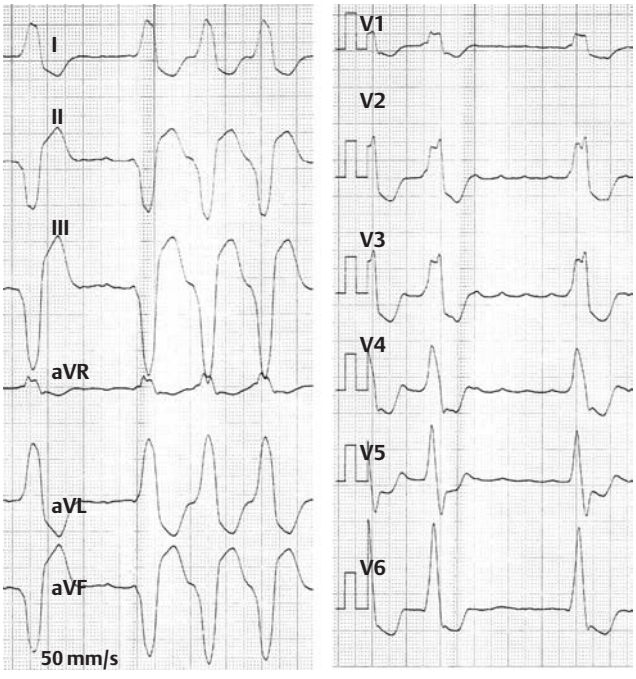
Left Anterior Hemiblock With 1st Degree AV Block

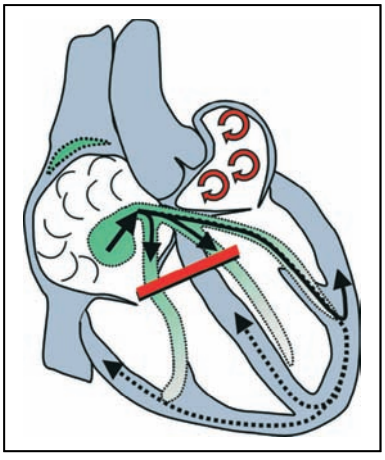




Differential diagnosis:
- 1st degree AV block with complete left bundle branch block
- 1st degree AV block with complete right bundle branch block and left posterior hemiblock

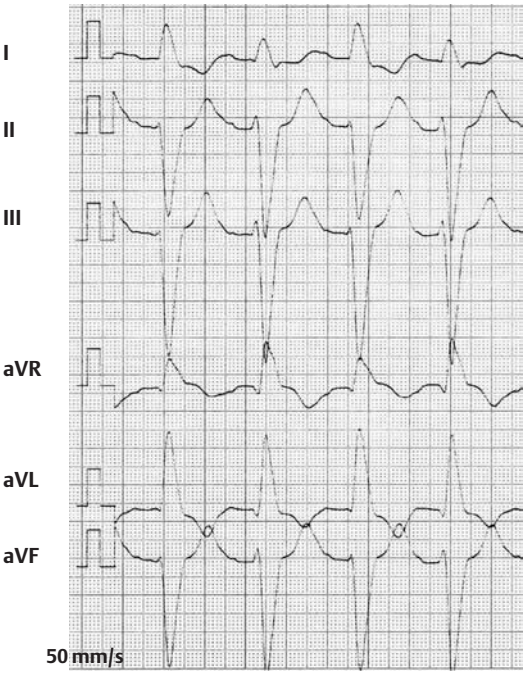
Bifascicular Block—Complete Right Bundle Branch Block and Left Anterior Hemiblock With Atrial Fibrillation

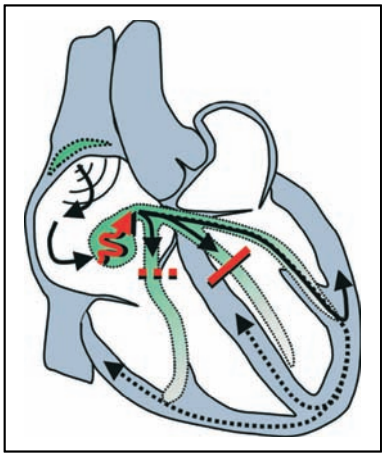




Differential diagnosis:
- Atrial fibrillation with complete left bundle branch block
- Atrial fibrillation with complete right bundle branch block and left posterior hemiblock

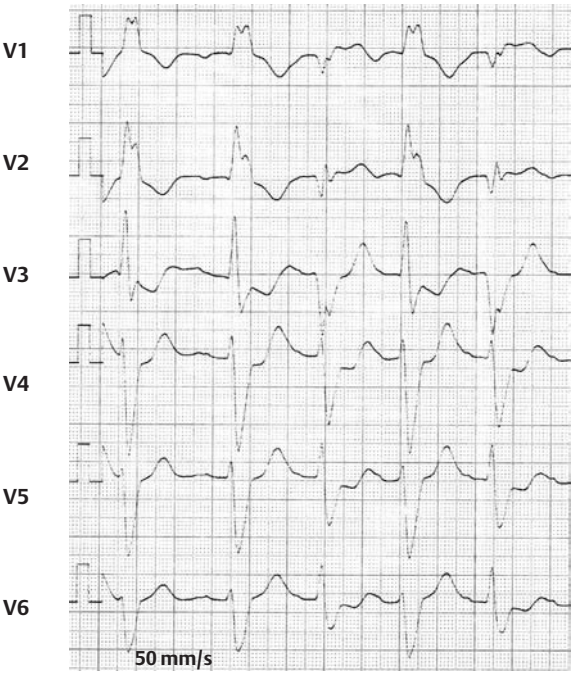
Intermittent Complete Right Bundle Branch Block With Preexisting Left Anterior Hemiblock and 1st Degree AV, Status Post Anterior Infarct

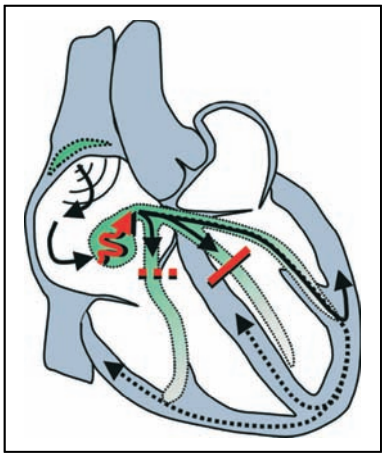




Differential diagnosis:
- Bigeminal form of ventricular extrasystole with left anterior hemiblock and 1st degree AV block

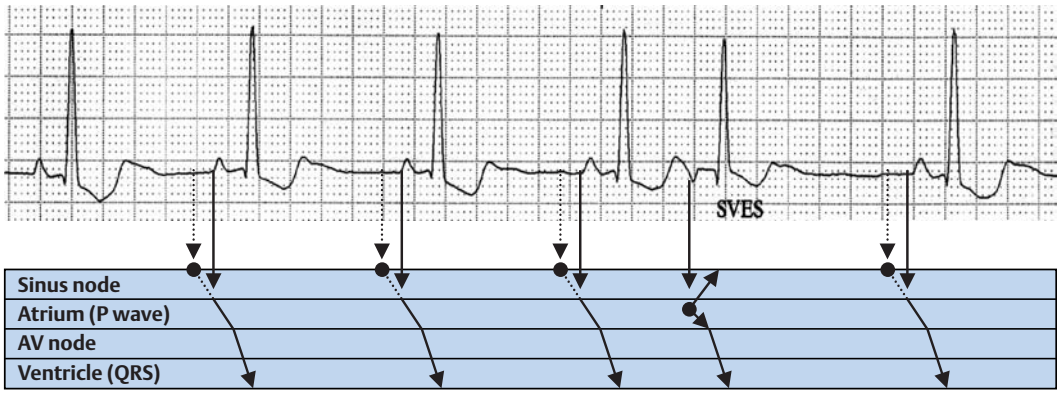
Intermittent Complete Right Bundle Branch Block With Preexisting Left Anterior Hemiblock and 1st Degree AV, Status Post Anterior Infarct





Differential diagnosis:
- Complete left bundle branch block
- Complete right bundle branch block and left posterior hemiblock

SVES, Singular



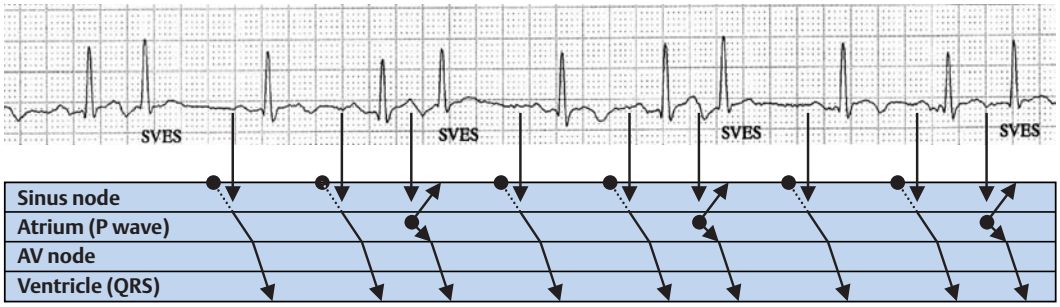
Mechanism:
- Focal atrial activity or intraatrial reentry
- AV node conduction capacity determines conduction to the ventricles (QRS complex)

ECG characteristics:
- Premature P wave (often other configuration)
- With impulse conduction at the AV node a "premature" QRS complex occurs

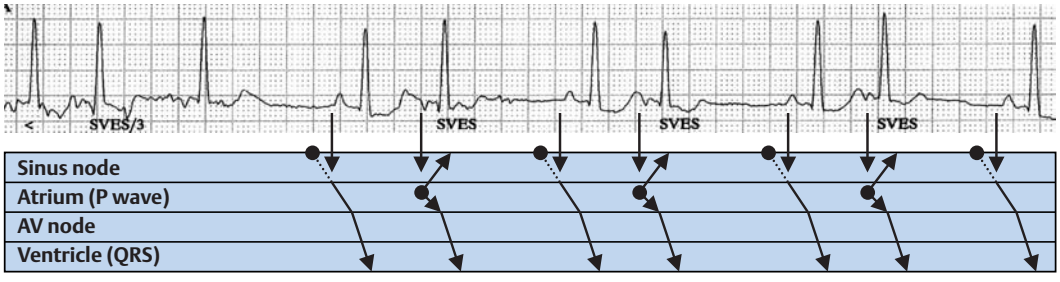
Etiology:
- Cardiac: hypertensive/coronary heart disease, heart defects, carditis
- Noncardiac: hyperthyroidosis

Treatment:
- No specific treatment, treatment of the underlying disease
- Only with marked symptoms and proven singular focus: possibly catheter ablation

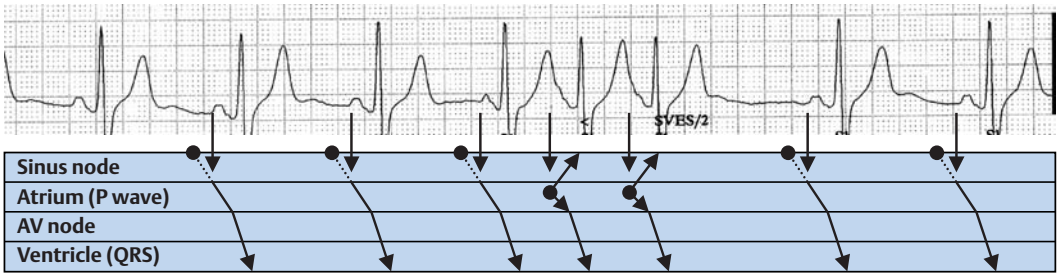
Supraventricular Trigeminy



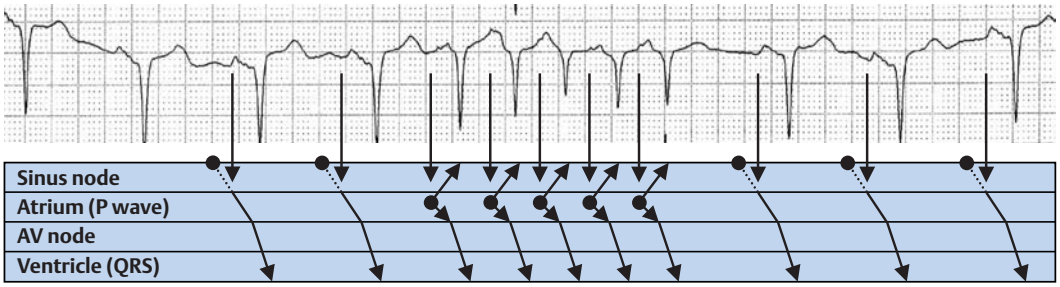
Supraventricular Bigeminy



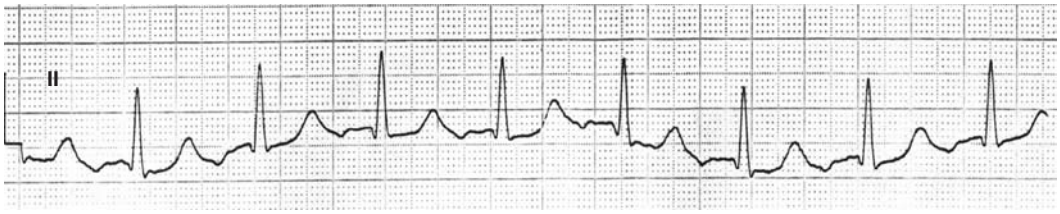
Supraventricular Couplet

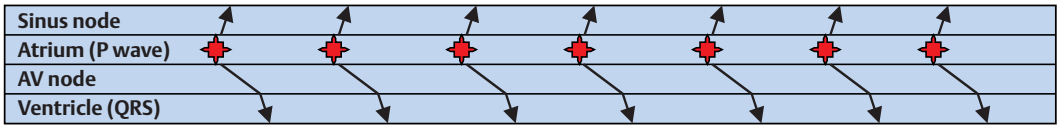


Supraventricular Salve



Focal Atrial Tachycardia



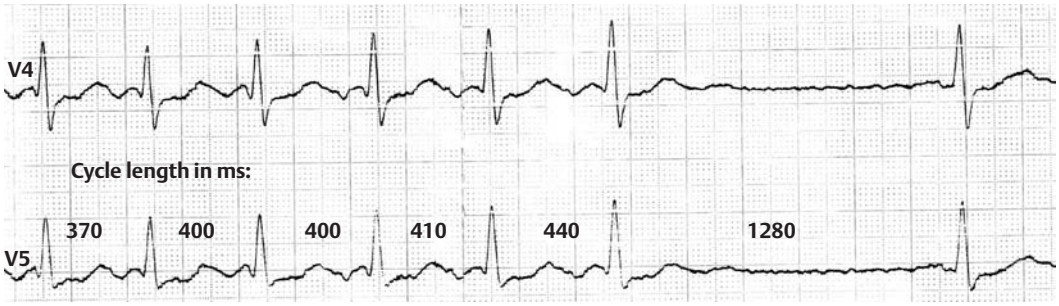


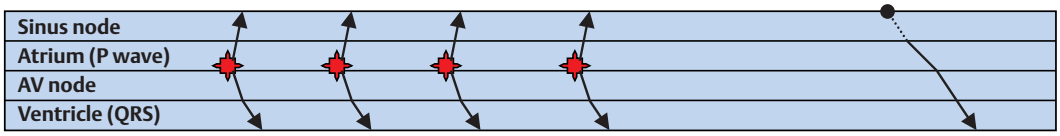
Mechanism:
- Increased automaticity of a left atrial focus
- Predisposition for the origin:
  - In the region of the opening of the coronary sinus
  - In the region of the opening of the pulmonary veins
  - Crista terminalis
  - Operative scarring

Etiology:
- Hypertensive/coronary heart disease, heart defects, carditis

Treatment:
- Transseptal catheter ablation possible (success rate ca. 70%; rate of recurrence ca. 30%)
- Antiarrhythmics (class Ic or III)
- Rate control (beta-blockers, digitalis, verapamil)

Focal Atrial Tachycardia

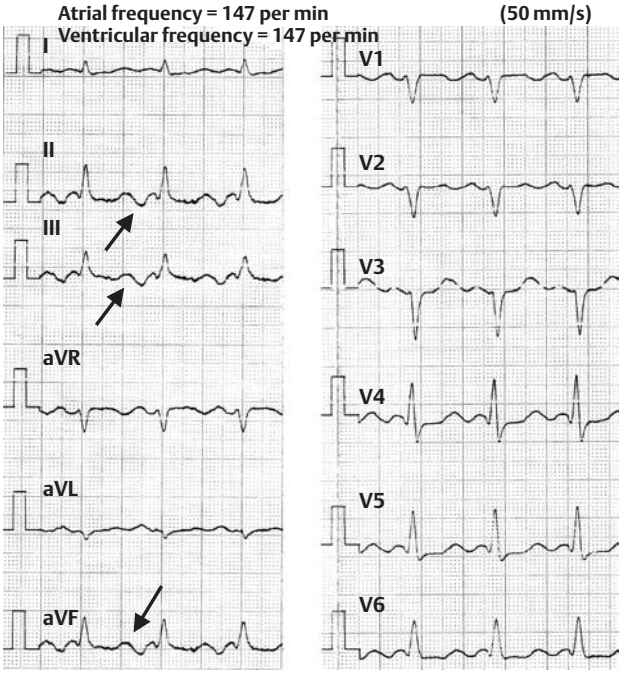


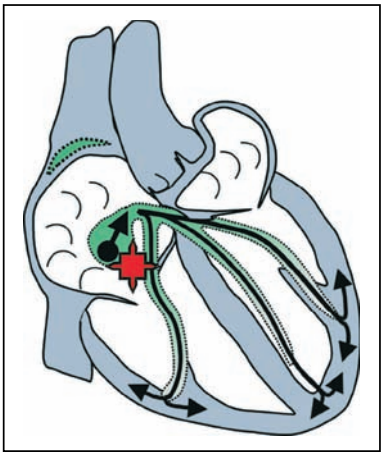


ECG characteristics:
- Arrhythmia commences with increasing shortening of the cycle length (increase in frequency, so-called warming up)
- Arrhythmia end: increasing lengthening of the cycle length (decrease in the frequency, so-called cooling down), see above
- Mostly shortened PQ interval

ECG characteristics:
- Atrial frequency between 100–250 per minute
- Often 1:1 conduction, resulting in tachycardia with narrow ventricular complexes
- Negative P waves in II, III, and aVF indicate a focus near to the opening of the coronary sinus (right atrium), whilst negative P waves in I and aVL represent a left atrial origin

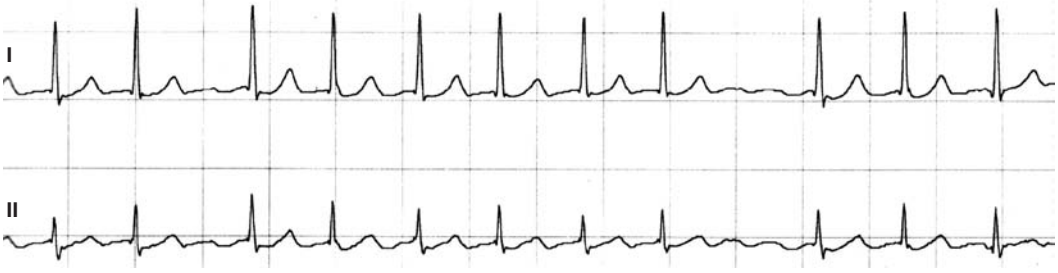
Focal Atrial Tachycardia (Basal Right Atrium)

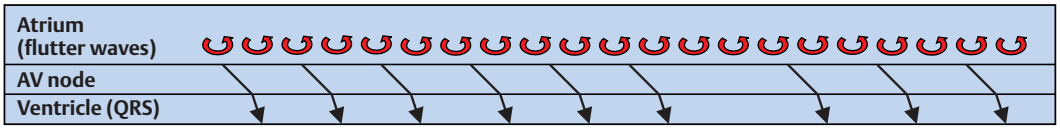




Differential diagnosis:
- Atrial flutter
- AV nodal reentry tachycardia
- Orthodromic WPW tachycardia
- Sinus tachycardia

Nonisthmus-Dependent "Atypical" Atrial Flutter





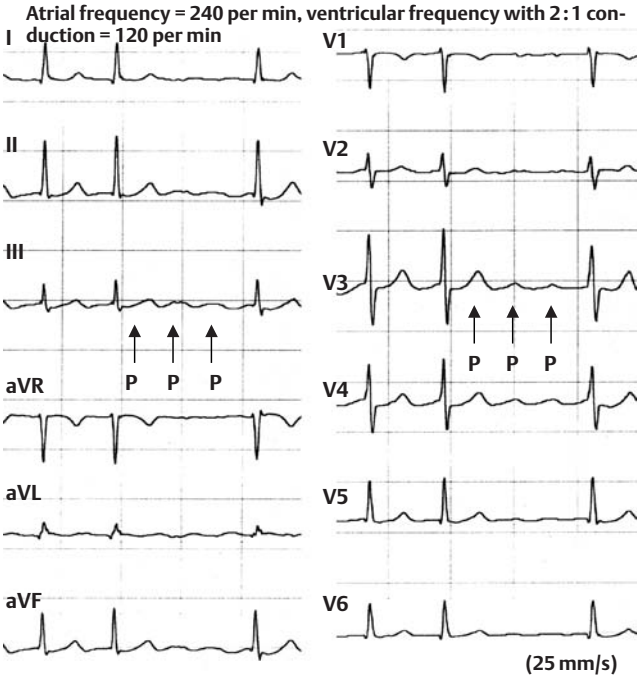
Mechanism:
- Reentry into the right or left atrium around the anatomical barriers (e.g., pulmonary veins, septal defects, operation scars, patches)
- No involvement of the isthmus

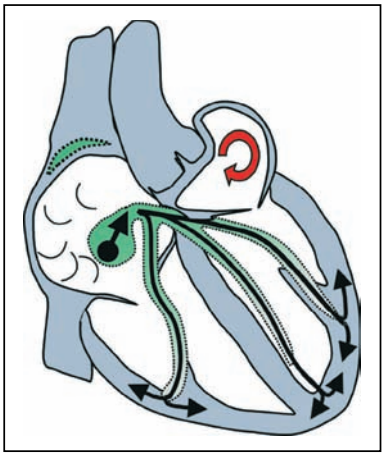
ECG characteristics:
- P wave morphology: no saw-tooth pattern
- Atrial frequency mostly between 200 and 300 per minute

Etiology:
- Cardiac: hypertensive/coronary heart disease, heart defects, carditis, cardiac operations
- Rarely noncardiac: hyperthyroidosis

Treatment:
- Cardioversion, or
- Rate control with embolic prophylaxis
- Curative catheter ablation only in exceptional cases
- Treatment of the underlying disease

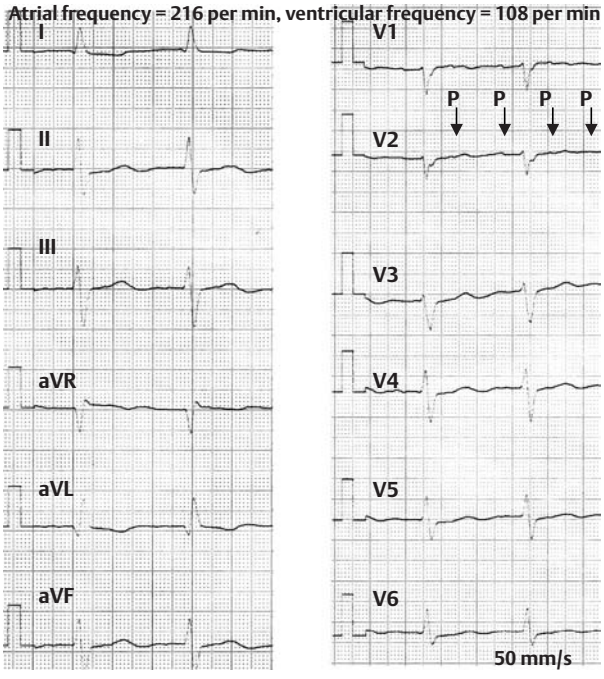
Nonisthmus-Dependent "Atypical" Atrial Flutter (Left Atrial)

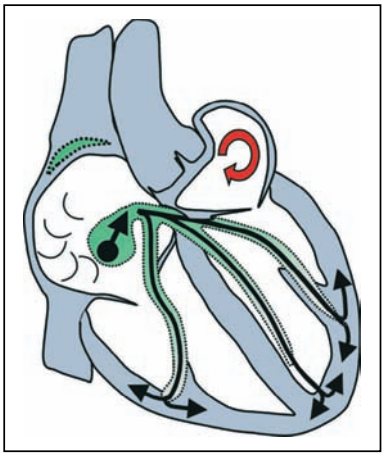




Differential diagnosis:
- Typical atrial flutter
- Atrial ectopic tachycardia
- AV nodal reentry tachycardia
- Orthodromic WPW tachycardia
- Sinus tachycardia
- Atrial fibrillation

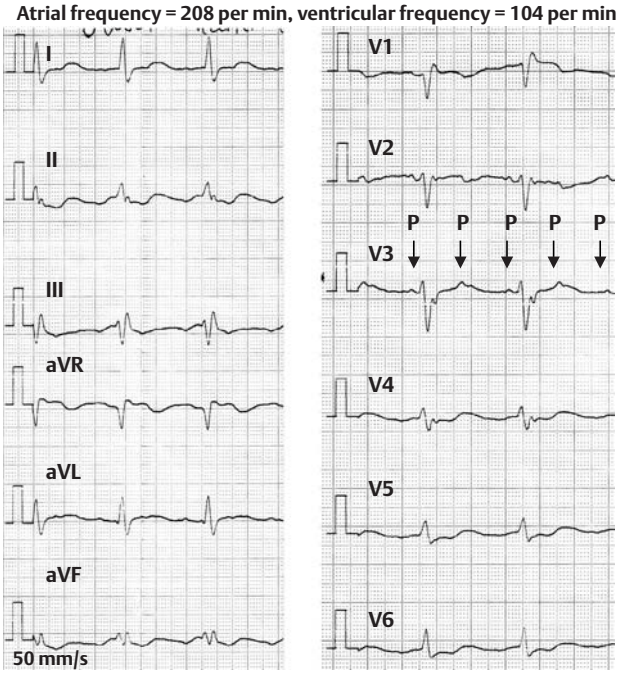
Nonisthmus-Dependent "Atypical" Atrial Flutter (Left Atrial)

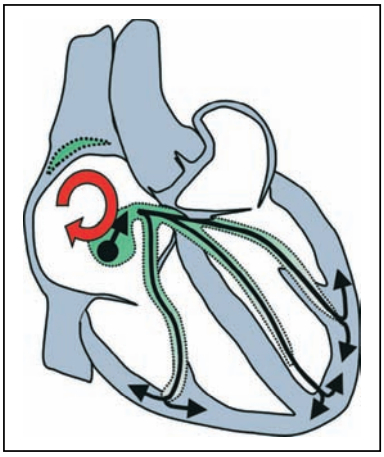




Differential diagnosis:
- Typical atrial flutter
- Atrial ectopic tachycardia
- AV nodal reentry tachycardia
- Orthodromic WPW tachycardia
- Sinus tachycardia
- Atrial fibrillation

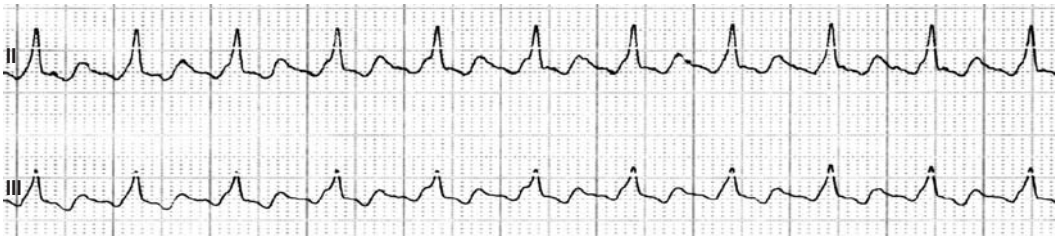
Nonisthmus-Dependent “Atypical” Atrial Flutter (Right Atrial With Atrial Septal Defect)

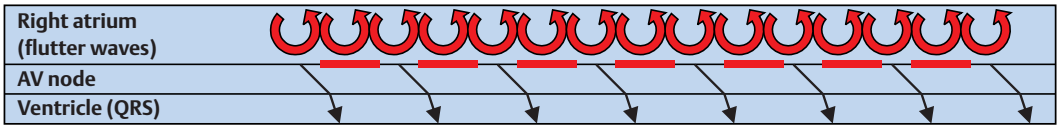




Differential diagnosis:
- Typical atrial flutter
- Atrial ectopic tachycardia
- AV nodal reentry tachycardia
- Orthodromic WPW tachycardia
- Sinus tachycardia
- Atrial fibrillation

Isthmus-Dependent “Typical” Atrial Flutter With 2:1 Conduction (Counter-Clockwise Orientation)





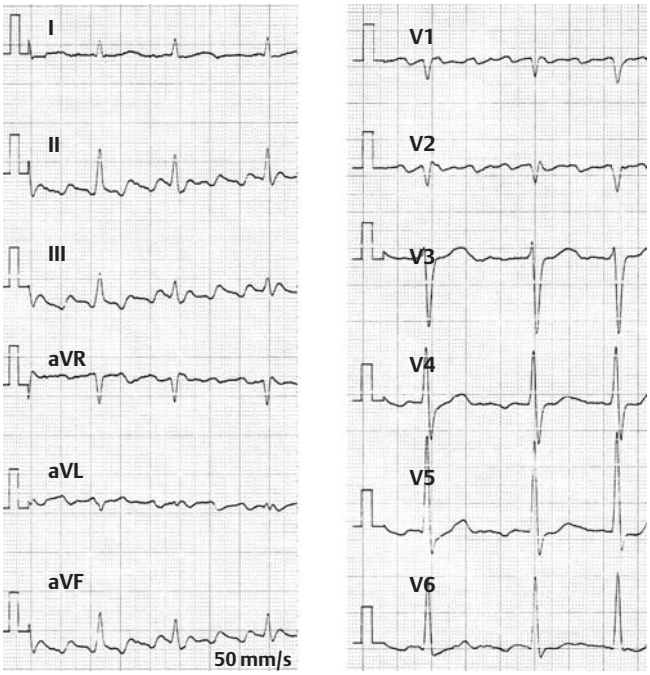
Mechanism:
- Macro-reentry into the right atrium with involvement of the isthmus; direction of excitation counter-clockwise

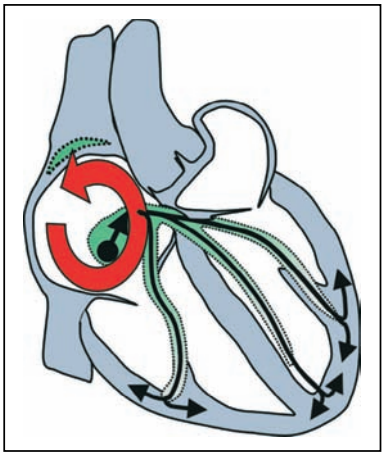
ECG characteristics:
- P wave morphology: typical “saw-tooth pattern” in leads II and III
- Tachycardia with narrow ventricular complex
- Atrial frequency: mostly between 250 and 350 per minute

Etiology:
- Cardiac: hypertensive/coronary heart disease, heart defects, carditis
- Rarely noncardiac: hyperthyroidosis

Treatment:
- Cardioversion/curative catheter ablation at the isthmus, or
- Rate control with embolic prophylaxis
- Treatment of the underlying disease

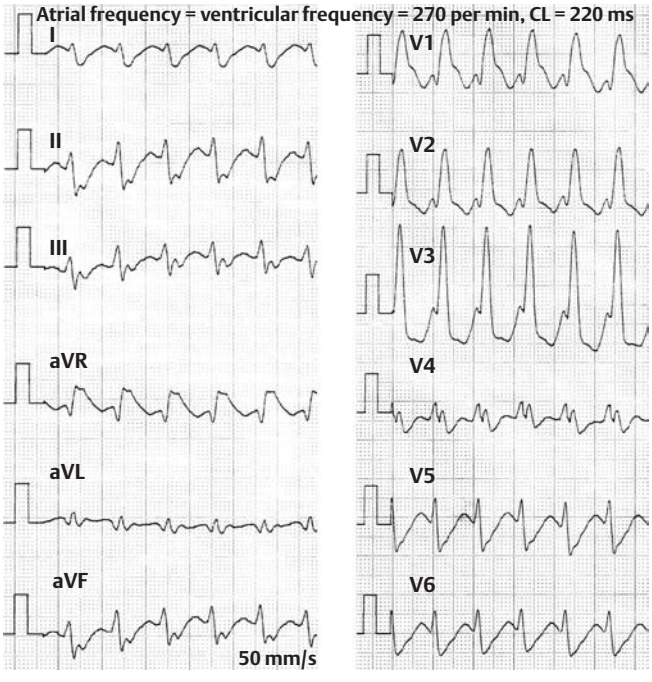
Isthmus-Dependent "Typical" Atrial Flutter (Counter-Clockwise Orientation)

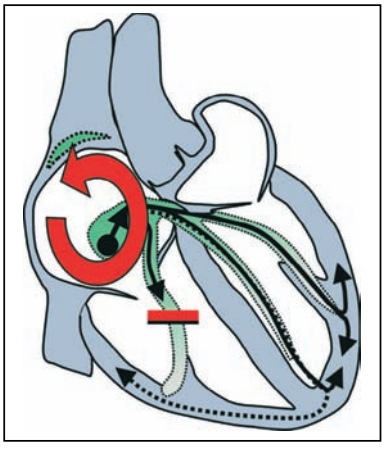




Differential diagnosis:
- Atypical atrial flutter
- Atrial ectopic tachycardia
- AV nodal reentry tachycardia
- Orthodromic WPW tachycardia
- Atrial fibrillation
- Sinus tachycardia/reentry

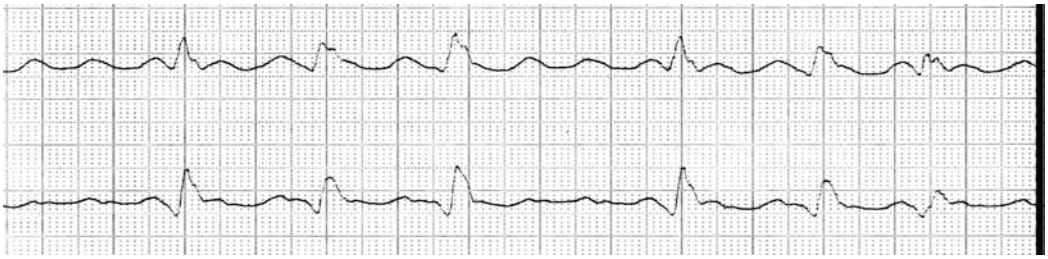
Isthmus-Dependent "Typical" Atrial Flutter With 1 : 1 Conduction, Functional Right Bundle Branch Block

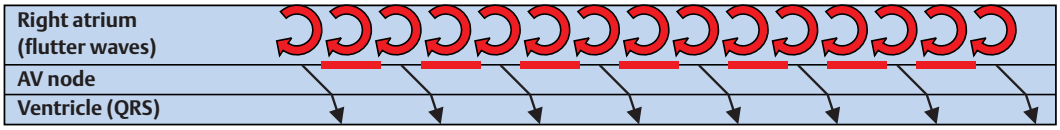




Differential diagnosis:
- Ventricular tachycardia
- Supraventricular tachycardia with functional block
- Antidromic WPW tachycardia
- Antegrade conduction in noncon-cealed WPW syndrome and supraven-tricular tachycardia/atrial fibrillation

Isthmus-Dependent "Typical" Atrial Flutter (Clockwise Orientation)





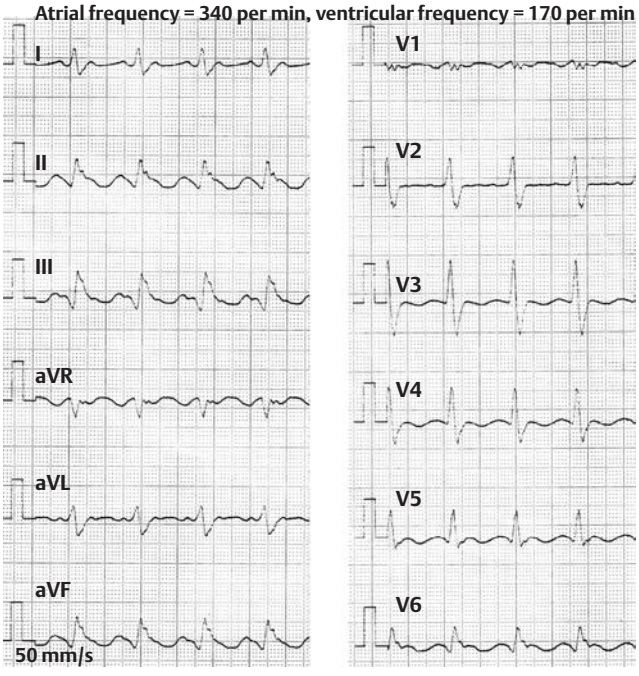
Mechanism:
- Macro-reentry into the right atrium with involvement of the isthmus; direction of excitation clockwise

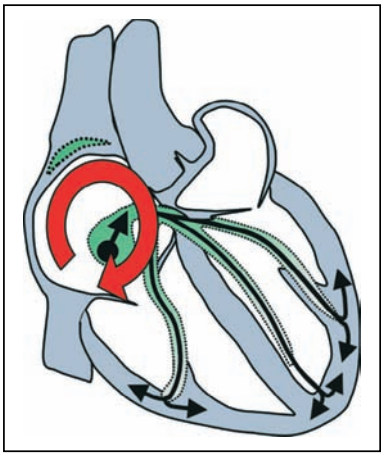
ECG characteristics:
- P wave morphology: wave form (no typical "saw-tooth pattern") in leads II and III
- Atrial frequency: mostly between 250 and 350 per minute

Etiology:
- Cardiac: hypertensive/coronary heart disease, heart defects, carditis
- Rarely noncardiac: hyperthyroidosis

Treatment:
- Cardioversion/curative catheter ablation at the isthmus, or
- Rate control with embolic prophylaxis
- Treatment of the underlying disease

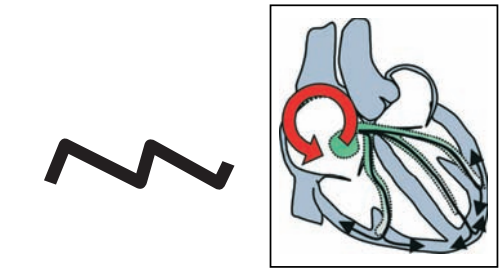
Isthmus-Dependent "Typical" Atrial Flutter (Clockwise Orientation)

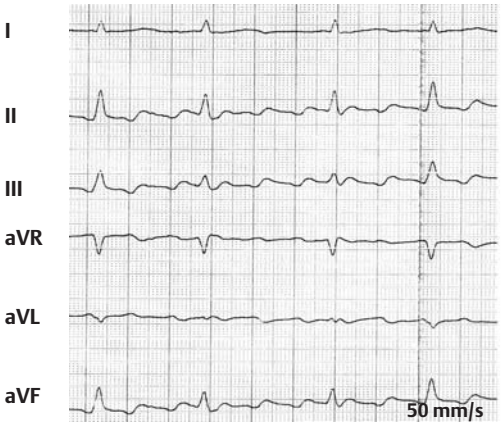


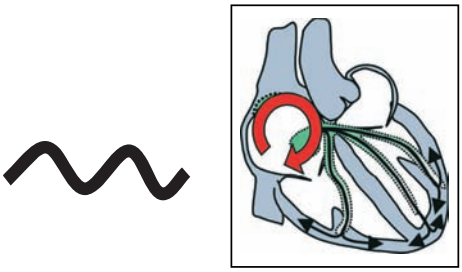


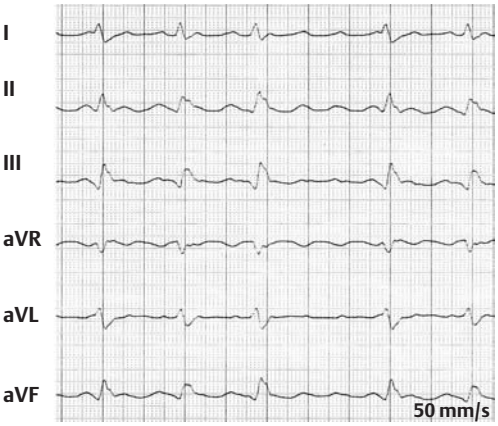
Differential diagnosis:
- Typical atrial flutter
- Atrial ectopic tachycardia
- Atrial fibrillation
- AV nodal reentry tachycardia
- Orthodromic WPW tachycardia
- Sinus tachycardia/reentry

Comparison of Isthmus-Dependent "Typical" Atrial Flutter (Counter-Clockwise vs. Clockwise)









Atrial Fibrillation—In General

Mechanism:
- Mostly left atrial micro-reentry with changing circuits of excitation
- Left atrial dilatation has a favorable effect
- Irregular conduction of fibrillation to the AV node

ECG characteristics:
- Irregular fibrillation waves (f) of variable amplitude, configuration, and cycle length
- Tachycardia, bradycardia, or normal frequency absolute arrhythmia (irregular intervals between the respective RR intervals) occur dependent of AV conduction capacity

Etiology:
- Cardiac: Hypertensive and coronary heart disease, heart defects (in particular mitral valve defects), carditis, degenerative disease of the impulse conduction system (sinus node syndrome), cardiac surgery
- Extracardiac: Hyperthyroidosis, noncardiac surgery, electrolyte shifts, alcohol abuse, infections, pulmonary disease, cerebrovascular event
- Primary: No underlying disease demonstrable (lone atrial fibrillation)

Forms:
- Paroxysmal: Always spontaneous conversion to sinus rhythm
- Persistent: No spontaneous conversion; however, sinus rhythm can be achieved with medication or electrical conversion
- Permanent: Sinus rhythm can be achieved neither with medication nor with electrical conversion

Atrial Fibrillation—Treatment, Rhythm Control vs. Rate Control, Prevention of Thrombembolism

Conversion:

High success rate: LA < 50 mm, duration of AF < one year (highest < seven days)

Electrical: External or internal (high success rates)

Medication: Antiarrhythmics of class IA, IC, and III

Alternative: Atrial defibrillator, MAZE operation, catheter ablation

Rhythm Control (medication of first choice):

- Class IC (in combination with beta-blocker): good LV pump function with no CHD
- Sotalol: good LV pump function and CHD without myocardial infarction
- Amiodarone: impaired LV pump function Class II (beta-blocker)

Rate control:

Medication:

- Beta-blockers (most effective).
- Calcium channel blockers (Verapamil, Diltiazem)
- Digitalis (Digitoxin, Digoxin)
- In rare cases also amiodarone

Alternative:

- AV node ablation with insertion of a pacemaker
- AV node modulation with no need for insertion of pacemaker

Prevention of thrombembolism before and after cardioversion:

Acute: Possible without oral anticoagulation up to 48 hours following onset of arrhythmia

Elective: Three weeks of effective oral anticoagulation (INR 2.0–3.0, target 2.5) beforehand: as an exception transesophageal echocardiography is possible to exclude thrombus in LA

After each cardioversion: oral anticoagulation (INR 2.0–3.0, target 2.5) for a minimum of one month – but so longer so better!

Prevention of thrombembolism:

Age:	*Risk factors*:*	*Recommendation:*
< 60 y	Absent	Aspirin (325 mg)
	Present	oral anticoagulation (INR 2.0–3.0)
60–75 y	Absent	Aspirin (325 mg) or oral anticoagulation (INR 2.0–3.0)
	Present	oral anticoagulation (INR 2.0–3.0)
> 75 y	all patients	oral anticoagulation (INR 2.0–3.0)

* *Prior TIA, systemic embolus or stroke, hypertension, poor LV function, diabetes mellitus, CHD, thrombus in LA, rheumatic mitral-valve disease, prosthetic heart valve – INR 2.0–3.5!*

"Idiopathic" Atrial Fibrillation

Mechanism:

- Mostly left atrial micro-reentry with changing circuits of excitation; focal triggering also possible
- Irregular conduction of fibrillation to the AV node

ECG characteristics:

- Tachycardia with narrow ventricular complex with irregular RR intervals

Etiology:

- No cardiac or extracardiac underlying disease demonstrable

Treatment:

- Initially rate control, embolus prophylaxis to age 60 necessary
- Attempt conversion followed by arrhythmia prophylaxis
- Ablation as curative therapy (elimination of pulmonary vein focus or application of linear lesion)

Focal Atrial Fibrillation

Mechanism:

- see idiopathic atrial fibrillation

ECG characteristics:

- tachycardia with narrow ventricular complex with irregular RR intervals

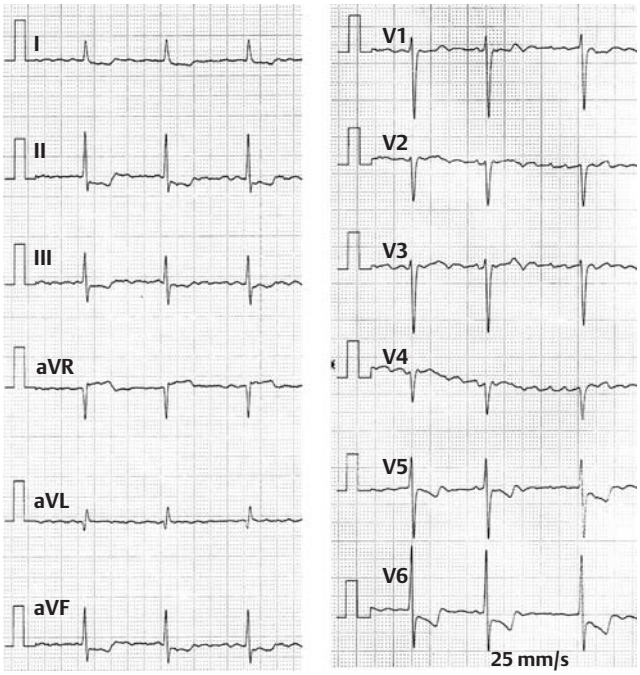
Etiology:

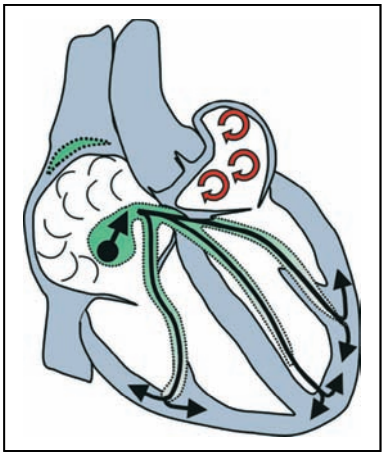
- cardiac: hypertensive/coronary HD, heart defects, carditis, brady-tachycardia syndrome
- extracardiac: hyperthyroidosis

Treatment:

- acute conversion up to 48 h post onset of arrhythmia possible without embolus prophylaxis
- antiarrhythmics (class Ic and III), anticoagulation with ineffective arrhythmia prophylaxis

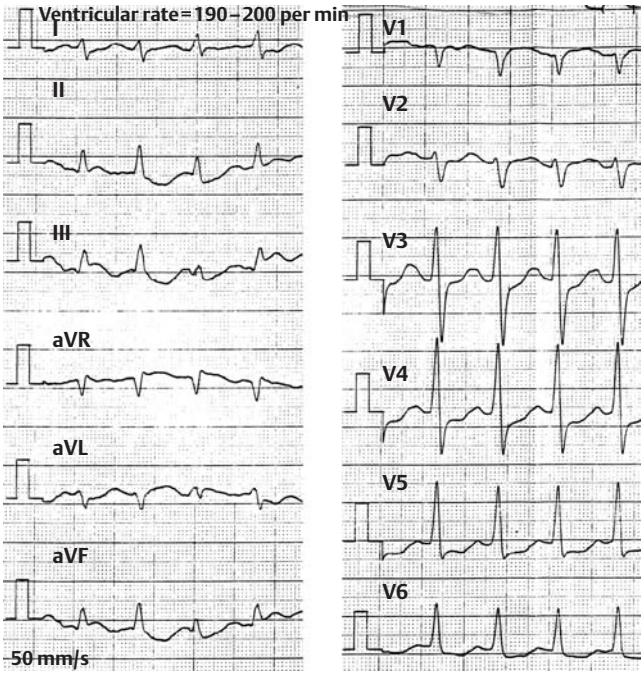
Normal Frequency Atrial Fibrillation

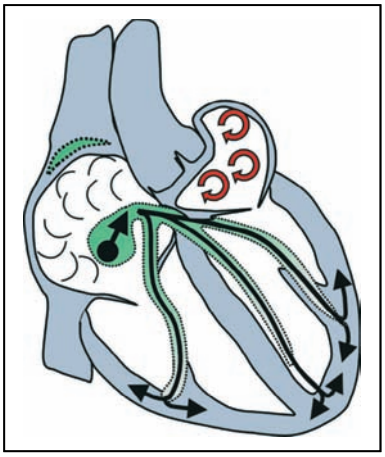




Differential diagnosis:
- Atrial flutter
- Atrial ectopic tachycardia
- AV nodal reentry tachycardia
- Sinus rhythm with SVES

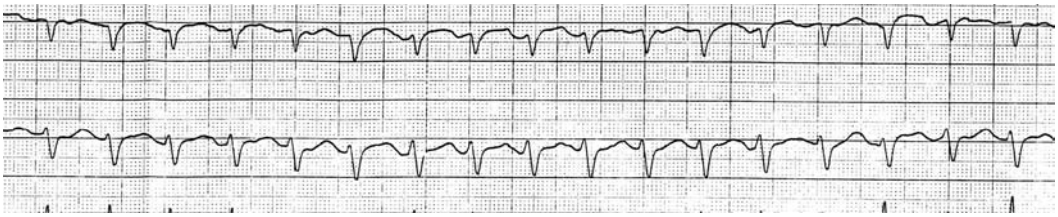
Tachycardic Atrial Fibrillation





Differential diagnosis:
- Atrial flutter
- Atrial ectopic tachycardia
- AV nodal reentry tachycardia
- Orthodromic WPW tachycardia
- Sinus tachycardia

Tachycardic Atrial Fibrillation



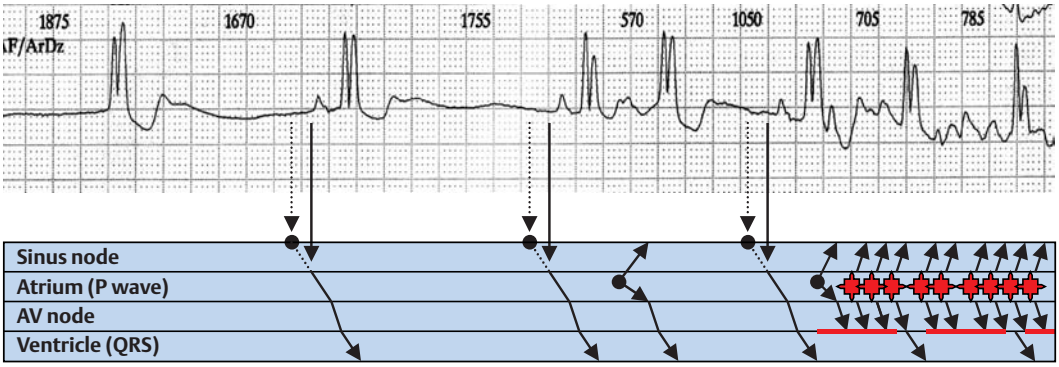
Mechanism:
- Mostly left atrial micro-reentry with changing circuits of excitation
- Accelerated impulse conduction at the AV node

ECG characteristics:
- Tachycardia with narrow ventricular complex with irregular RR intervals
- Fibrillation waves (f) not visible

Etiology:
- Idiopathic
- Cardiac: hypertensive and coronary heart disease, heart defects, carditis
- Noncardiac: hyperthyroidosis

Treatment:
- Decision on acute transesophageal echocardiography guided cardioversion or intravenous pharmacological rate control; embolus prophylaxis
- Treatment of underlying disease

Atrial Fibrillation Triggered by Bradycardia



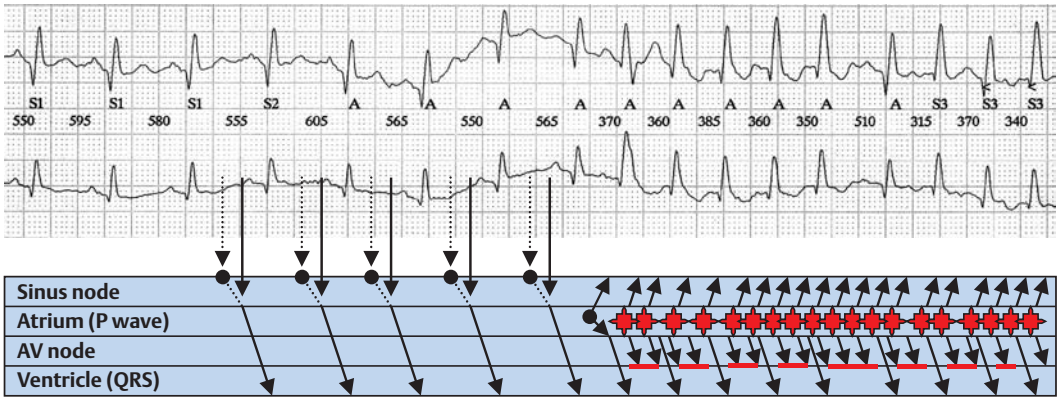
Mechanism:
- Triggered by bradycardia
- Arrhythmia commences from sinus bradycardia or SA blockade
- Variable impulse conduction at the AV node

ECG characteristics:
- Pause-dependent onset of atrial fibrillation
- Often well-demarcated fibrillation waves
- Irregular RR intervals

Etiology:
- Vagotonia (sportspeople)
- Cardiac: sinus node syndrome
- Medication: beta-blockers, antiarrhythmics

Treatment:
- Treatment of underlying disease
- Increase the heart rate: pacemaker, cessation of medications that may cause bradycardia
- Embolus prophylaxis if necessary (mostly)

Sympathetic-Induced Atrial Fibrillation



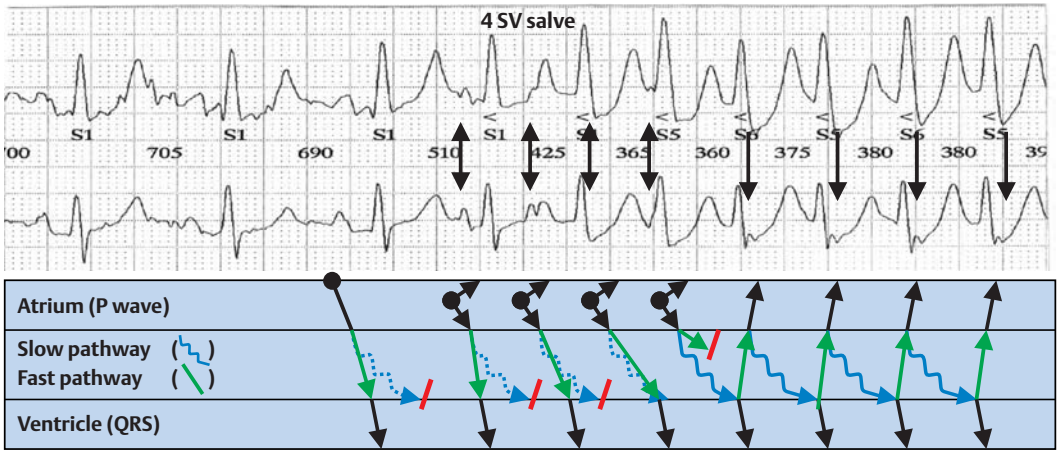
Mechanism:
- Triggered by sympathetic nervous system
- Arrhythmia arises from sinus tachycardia
- Accelerated impulse conduction at the AV node

ECG characteristics:
- Sinus tachycardia changes in a tachycardia with narrow ventricular complex with irregular RR intervals (beginning with SVES)

Etiology:
- Increased sympathetic tone
- Hyperthyroidosis

Treatment:
- Treatment of underlying disease
- Selective beta–1 blockade
- Embolus prophylaxis if necessary

AV Nodal Reentry Tachycardia (Slow–Fast Type)

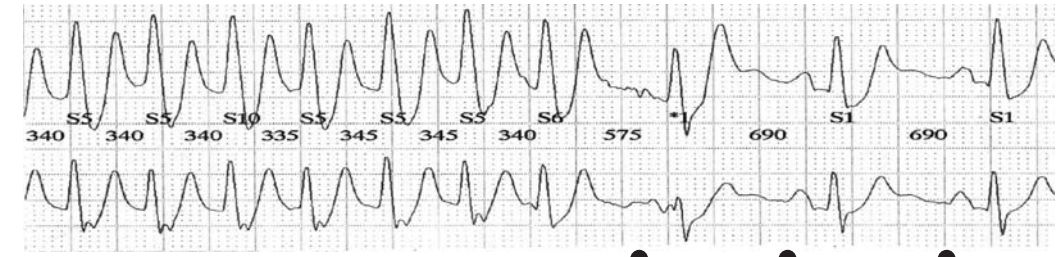


Induction mechanism:
In sinus rhythm conduction via the fast pathway ( ), four supraventricular salve events with prolongation of conduction along the "fast pathway" with Wenckebach pattern; after reaching a critical conduction time (with four SVES) block of the "fast pathway" and impulse conduction to the ventricle via the "slow pathway ( )," then retrograde conduction to the atrium via the "fast pathway" → reentry begins.

Etiology:
- Existence of two different approaches to the AV node with different electrophysiological properties ("slow" and "fast" pathway region)

Treatment:
- Curative: ablation of the slow pathway (risk of complete AV block → pacemaker)
- Prophylaxis of attacks: antiarrhythmic (Ic, III, IV)
- In acute cases: 12 – 18 mg adenosine i.v.

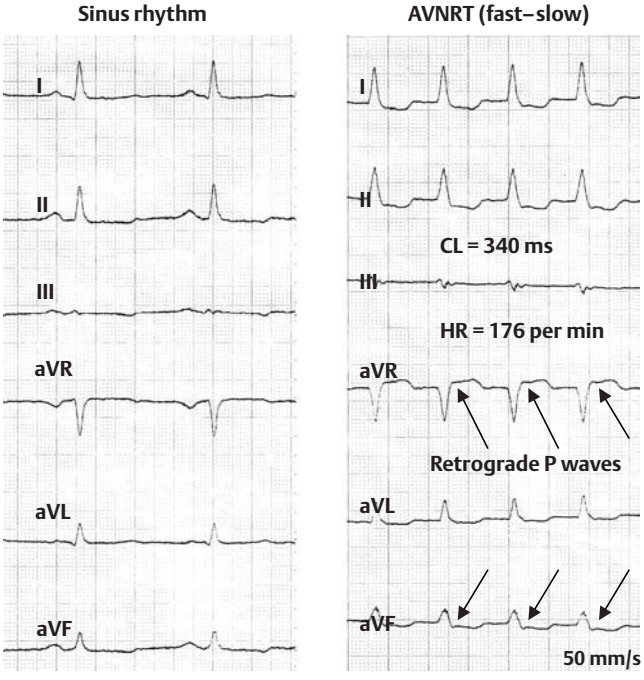
AV Nodal Reentry Tachycardia (Slow–Fast Type)

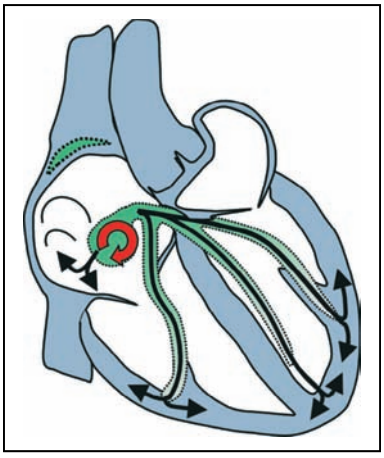


Termination mechanism:
- After establishing the reentry circuit, sudden blockade of retrograde conduction in the fast pathway, hence no excitation of the atria (absent P wave at the end of the QRS complex); termination of the reentry circuit
- Renewed sinus rhythm

ECG characteristics:
- Regular tachycardia with narrow ventricular complex; retrograde P wave at the end of the QRS ("rSr configuration in V1")

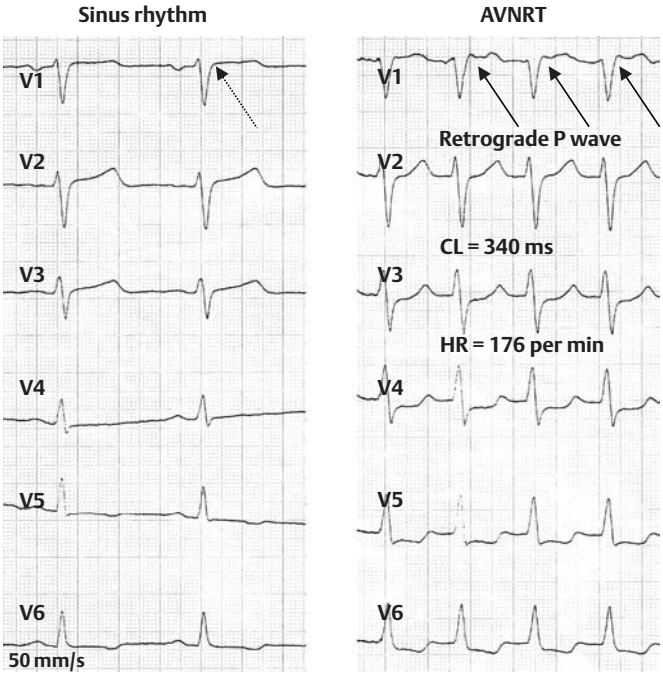
AV Nodal Reentry Tachycardia (Slow–Fast Type)

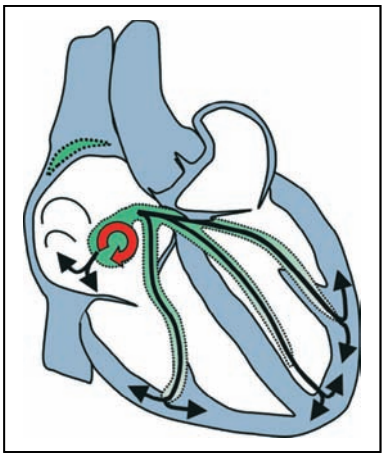




Differential diagnosis:
- Orthodromic WPW reentry tachycardia
- AV nodal reentry tachycardia (fast–slow type)
- Atrial flutter (2:1; 1:1 conduction)
- Atrial ectopic tachycardia
- Sinus tachycardia/reentry

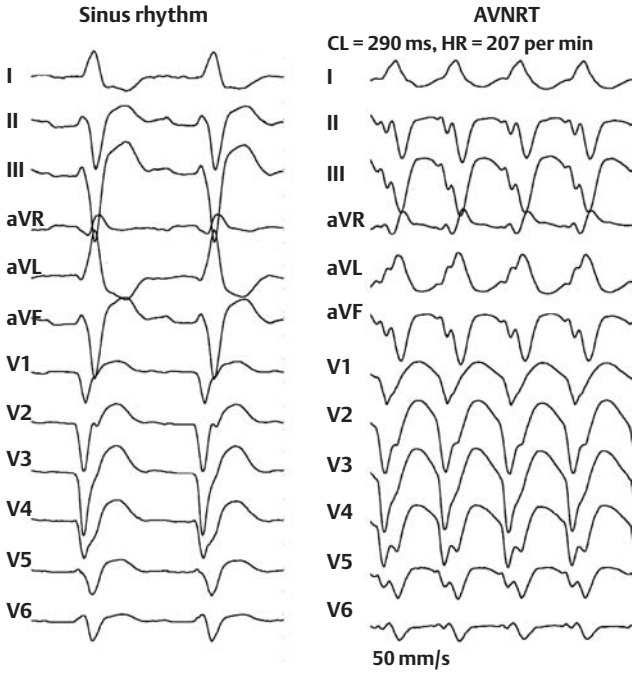
AV Nodal Reentry Tachycardia (Slow–Fast Type)

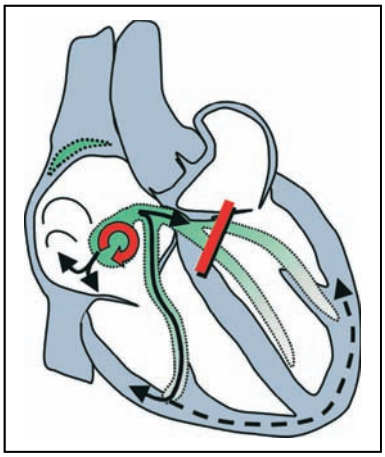




Differential diagnosis:
- Orthodromic WPW reentry tachycardia
- AV nodal reentry tachycardia (fast–slow type)
- Atrial flutter (2 : 1; 1 : 1 conduction)
- Atrial ectopic tachycardia
- Sinus tachycardia/reentry

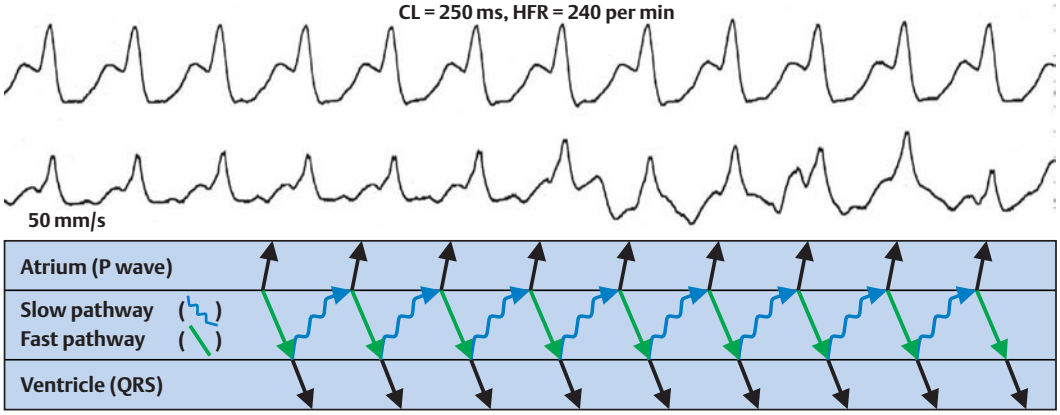
AV Nodal Reentry Tachycardia (Slow–Fast Type) With Left Bundle Branch Block





Differential diagnosis:
- Ventricular tachycardia
- Antidromic Mahaim tachycardia
- Supraventricular tachycardia with left bundle branch block
- Antidromic WPW tachycardia

AV Nodal Reentry Tachycardia (Fast–Slow Type)



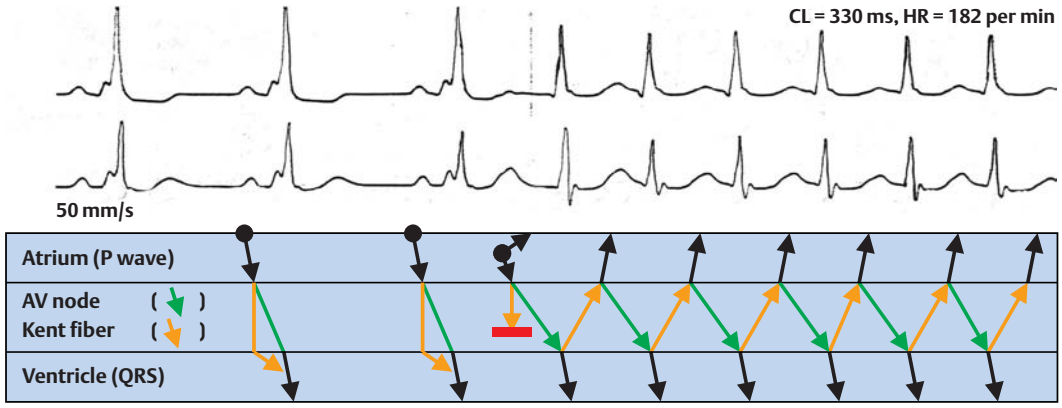
Mechanism:
- Rare form of AV nodal reentry tachycardia in which the antegrade conduction (from atrium to ventricle) occurs via the "fast pathway" and the retrograde excitation of the atria via the "slow pathway"

ECG characteristics:
- Regular tachycardia with narrow ventricular complex; retrograde (negative) P wave in front of QRS (interval "PR" < "RP")

Etiology:
- Existence of two different approaches to the AV node with different electrophysiological properties ("slow" and "fast" pathway region)

Treatment:
- Ablation of the slow pathway (risk of complete block)
- Antiarrhythmics (class Ic, III, IV)
- In acute cases: 12–18 mg adenosine i.v.

Orthodromic WPW Reentry Tachycardia With Nonconcealed WPW Syndrome



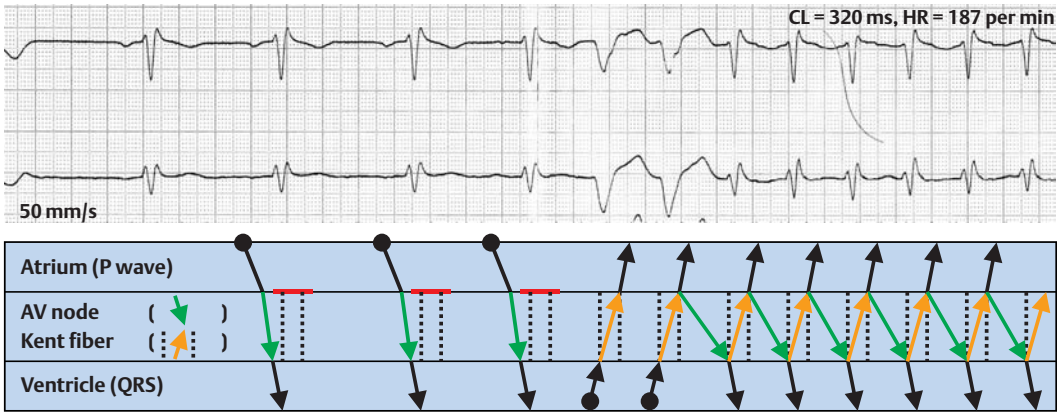
Induction mechanism:
- With SVES antegrade blockade of the Kent fiber, impulse conduction via the AV node, excitation of the ventricles, retrograde impulse conduction via Kent, and commencement of reentry

ECG characteristics:
- Tachycardia with narrow ventricular complex
- Retrograde P wave in front of QRS (interval "PR" > "RP")

Etiology:
- Congenital accessory pathways between the atrium and ventricle, so-called Kent fiber

Treatment:
- Curative: ablation of Kent fiber
- Antiarrhythmics (see WPW) as an alternative to ablation
- In acute cases: 12–18 mg adenosine i.v.

Orthodromic WPW Reentry Tachycardia With Concealed WPW Syndrome



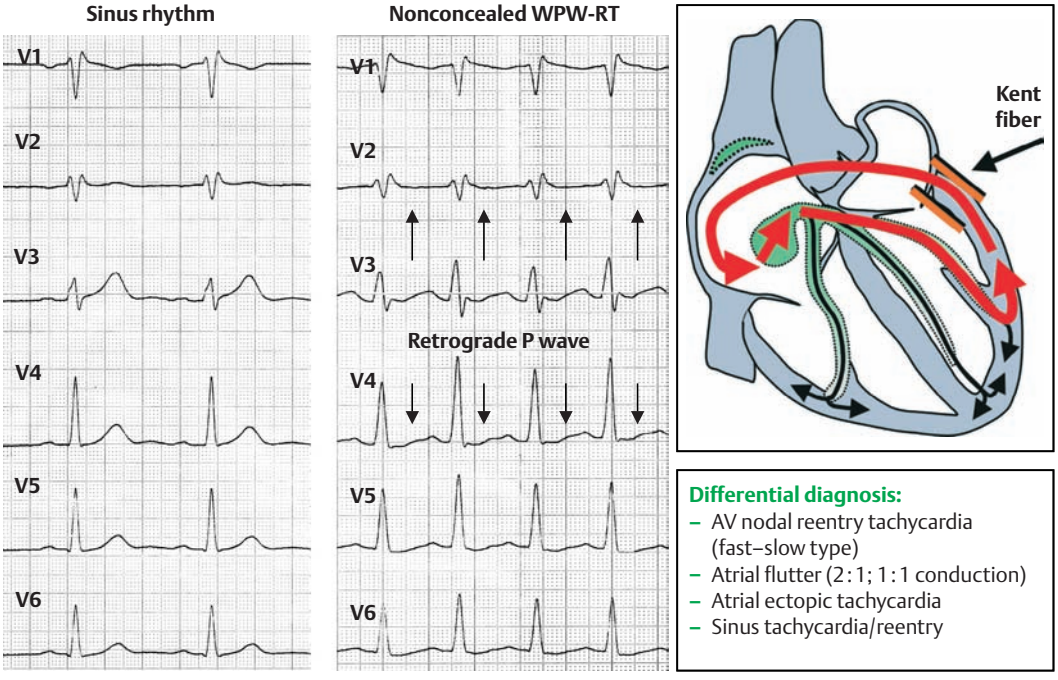
Induction mechanism:
- Concealed WPW syndrome = Kent fiber with solely retrograde conduction
- During sinus rhythm, complete antegrade blockade of the Kent fiber (no delta wave); conduction via the AV node
- With ventricular extrasystole couplet retrograde conduction via Kent fiber, renewed antegrade conduction via the AV node; start of reentry

ECG characteristics:
- Tachycardia with narrow ventricular complex
- Retrograde (negative) P wave in front of QRS (interval "PR" > "RP")

Etiology:
see above

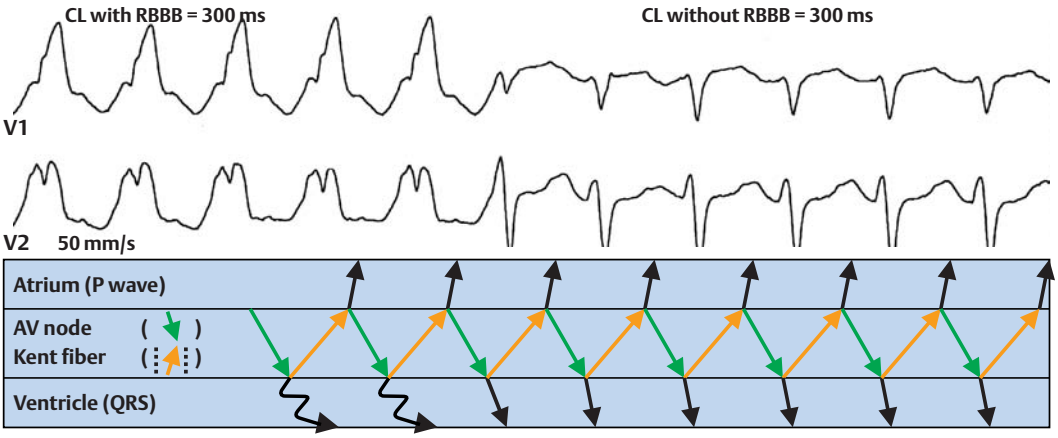
Treatment:
see above

Orthodromic WPW Reentry Tachycardia



Differential diagnosis:
- AV nodal reentry tachycardia (fast–slow type)
- Atrial flutter (2 : 1; 1 : 1 conduction)
- Atrial ectopic tachycardia
- Sinus tachycardia/reentry

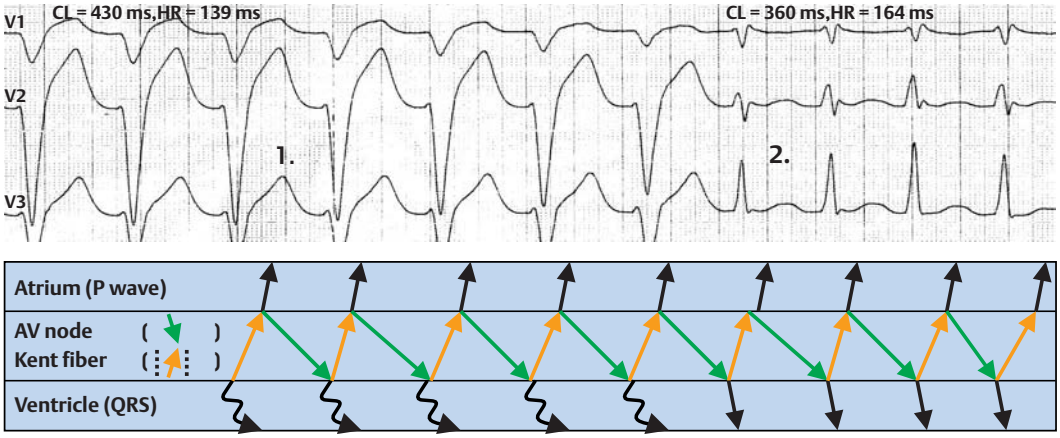
Functional Right Bundle Branch Block With Orthodromic WPW Reentry Tachycardia



Mechanism:
- Initial fatigue of the right bundle branch due to tachycardia resulting in tachycardia with a wide QRS complex; P waves from retrograde conduction easily recognized
- Following recovery of the right bundle branch, transition to a tachycardia with narrow ventricular complex with no change in tachycardia frequency

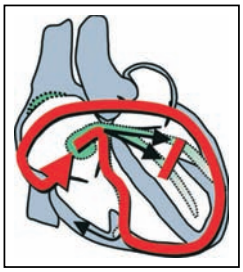
ECG characteristics:
- Tachycardia with wide ventricular complex (right bundle branch deformation)
- Transition to a tachycardia with narrow ventricular complex

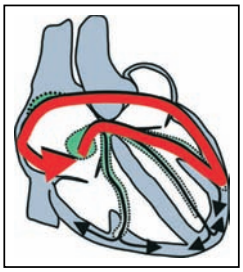
Left-Sided Kent Bundle With Orthodromic WPW Reentry Tachycardia, Intermittent Left Bundle Branch Block

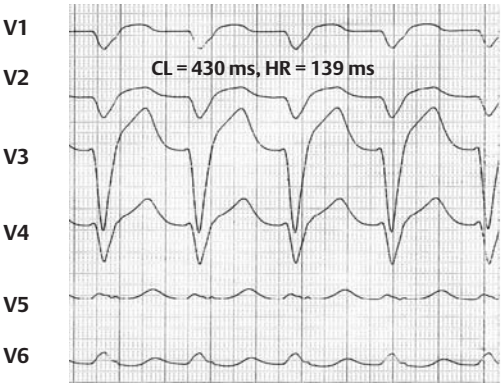


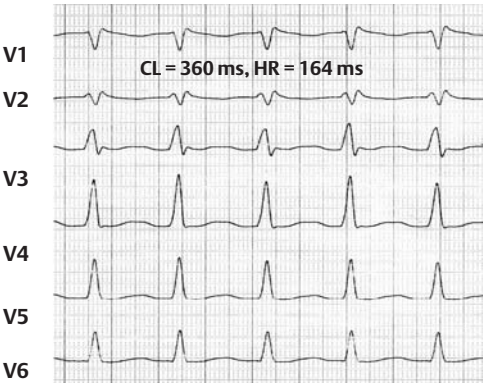
Mechanism:
1. Fatigue of the left bundle branch due to tachycardia resulting in tachycardia with a wide QRS complex. Due to the left bundle branch block the impulse can only be conducted via the right bundle branch and with a left-sided Kent bundle needs longer to reach the left ventricle, the reentry circuit expands, the length of the tachycardia cycle (CL) increases, the tachycardia decelerates.
2. Following recovery of the left bundle branch, this is then available for reentry; the circuit becomes smaller, the length of the tachycardia cycle (CL) decreases, the tachycardia accelerates (occurrence of tachycardia with narrow ventricular complex and increase in rate [HR]).

Comparison of Orthodromic WPW Reentry Tachycardia With and Without Complete Left Bundle Branch Block With Left-Sided Kent Bundle

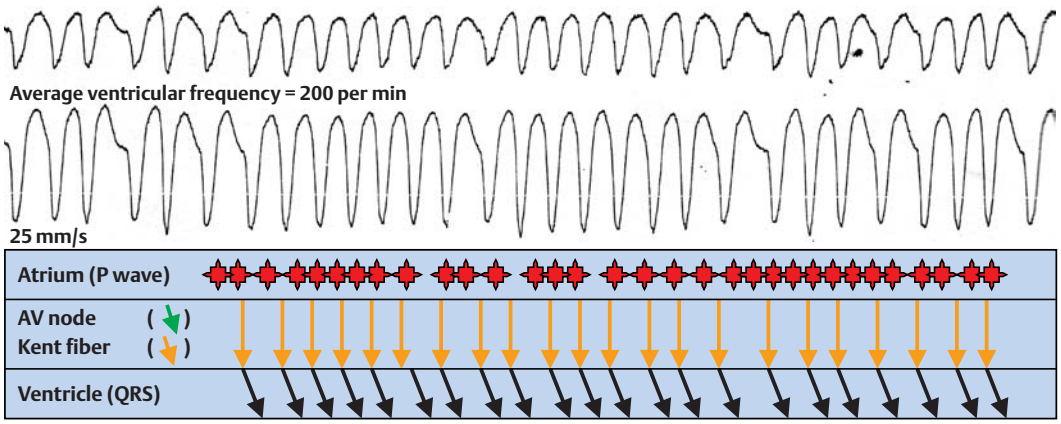








Tachycardic Conduction of Atrial Fibrillation With WPW Syndrome



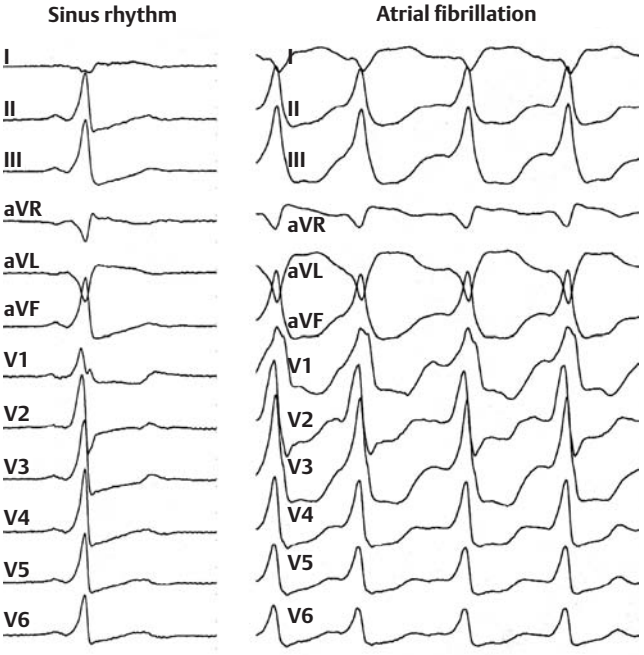
Mechanism:
- As a result of extremely good antegrade conduction properties of the Kent fiber, tachycardic conduction of atrial fibrillation occurs; transition to ventricular fibrillation is possible!

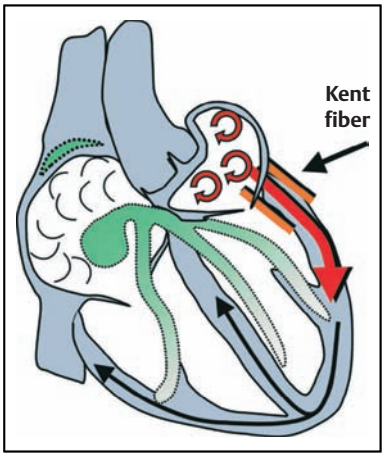
ECG characteristics:
- Tachycardia with wide ventricular complex, often high ventricular frequency; irregular QRS complexes follow

Etiology:
- Atrial fibrillation with Kent fiber present

Treatment:
- Curative: ablation of Kent fiber
- Antiarrhythmics (class Ic, III, ajmaline)
- In acute cases: cardioversion or antiarrhythmic (see above)

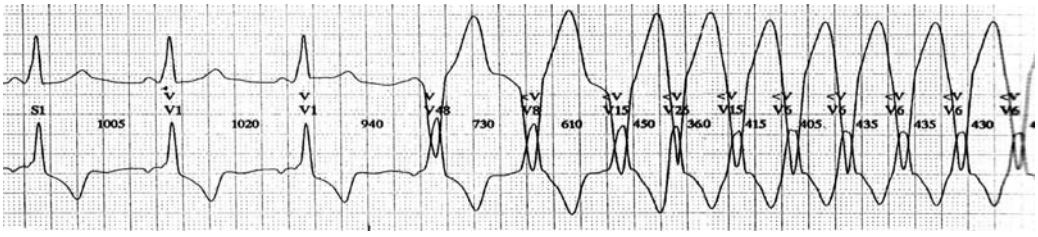
Tachycardic Conduction of Atrial Fibrillation in WPW Syndrome





Differential diagnosis:
- Torsade de pointes
- Ventricular fibrillation
- Ventricular flutter
- (Ventricular tachycardia)
- (Supraventricular tachycardia with functional bundle branch block)

Antidromic WPW Reentry Tachycardia



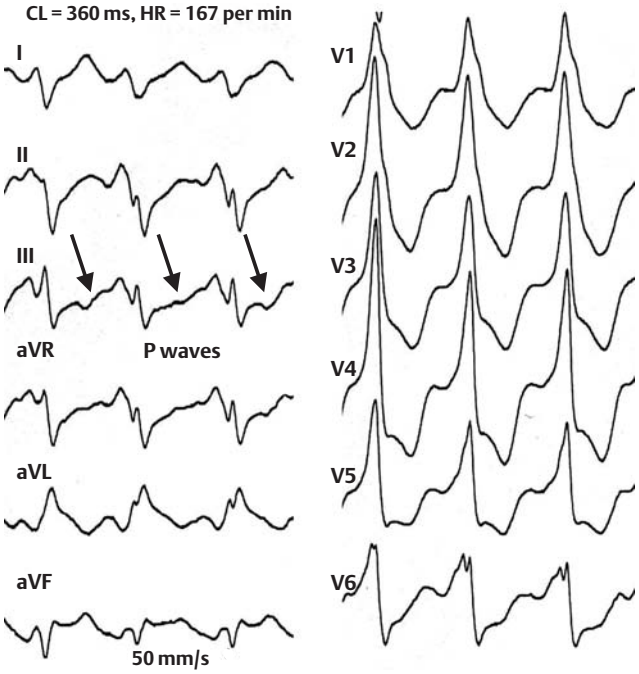
Induction mechanism:
- Vagal blockade of AV conduction with sole antegrade conduction via Kent fiber; retrograde conduction of the impulse via the AV node with initiation of reentry

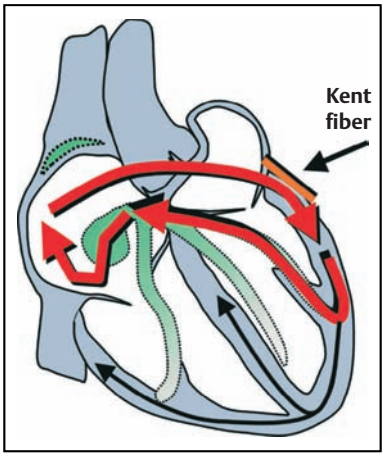
ECG characteristics:
- Tachycardia with wide ventricular complex

Etiology:
- Congenital accessory pathways between the atrium and ventricle, so-called Kent fiber

Treatment:
- Curative: ablation of Kent fiber
- Antiarrhythmics (class Ic, III, ajmaline)
- In acute cases: 12–18 mg adenosine i.v.

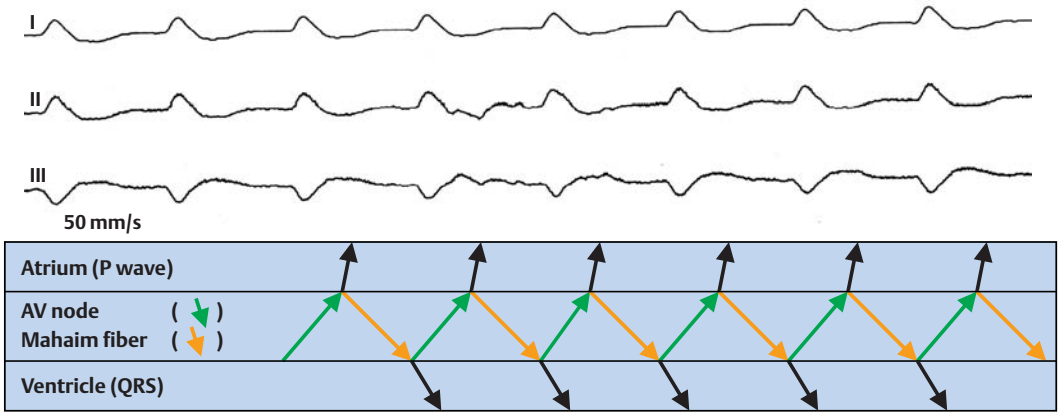
Antidromic WPW Reentry Tachycardia





Differential diagnosis:
- Ventricular tachycardia
- Supraventricular tachycardia with functional bundle branch block
- Mahaim tachycardia

Antidromic Mahaim Reentry Tachycardia



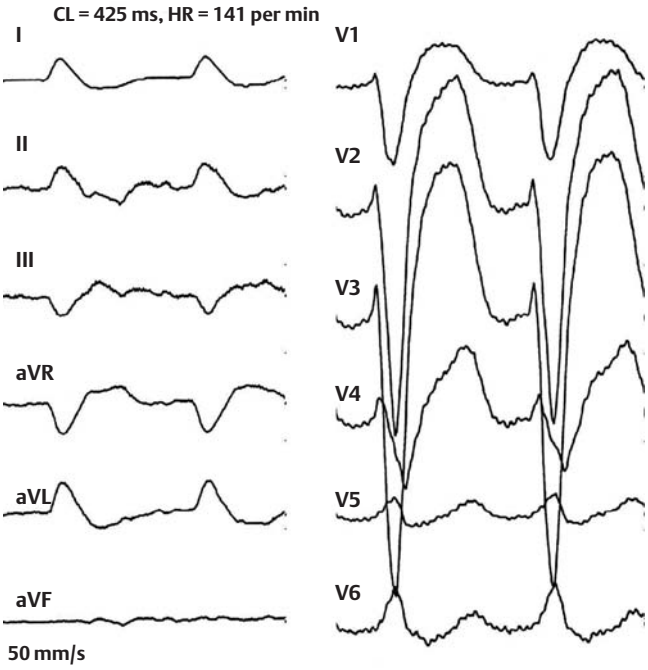
Mechanism:
- Antidromic reentry between the Mahaim fiber and the AV node
- Induction via ventricular extrasystole

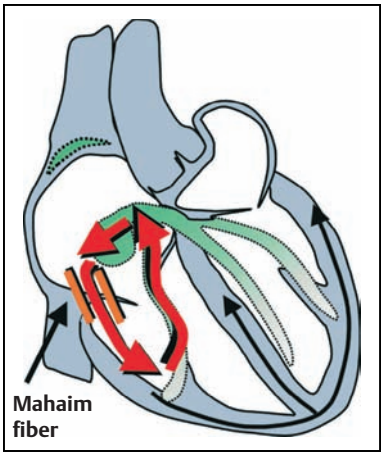
ECG characteristics:
- Constant tachycardia with left bundle branch block pattern
- Retrograde P waves between the QRS complexes

Etiology:
- Right-sided antegrade accessory pathway (Mahaim) with conduction properties similar to the AV node

Treatment:
- Ablation of the Mahaim fiber (often technically difficult)
- Antiarrhythmics (see WPW) as an alternative to ablation

Antidromic Mahaim Reentry Tachycardia

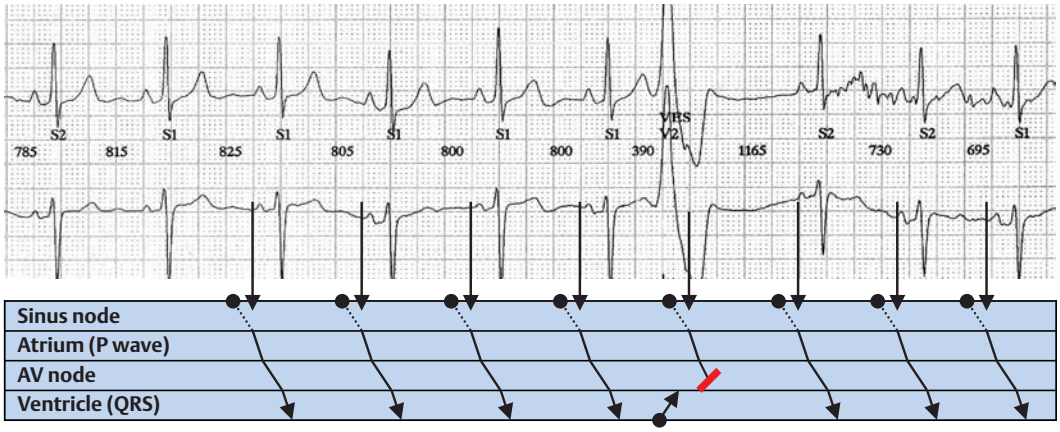




Differential diagnosis:

- Ventricular tachycardia
- Supraventricular tachycardia with functional left bundle branch block pattern
- Antidromic WPW tachycardia

Ventricular Extrasystole—Singular



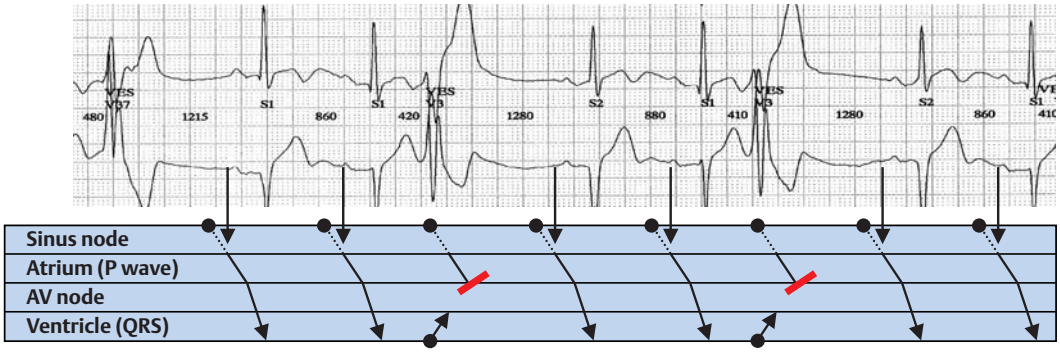
Mechanism:
- Focal activity, interventricular reentry, or reentry around scars (infarct, patch, etc.)

ECG characteristics:
- Premature widened QRS complex with compensatory pause (2 × RR)
- With retrograde atrial excitation, no compensatory pause

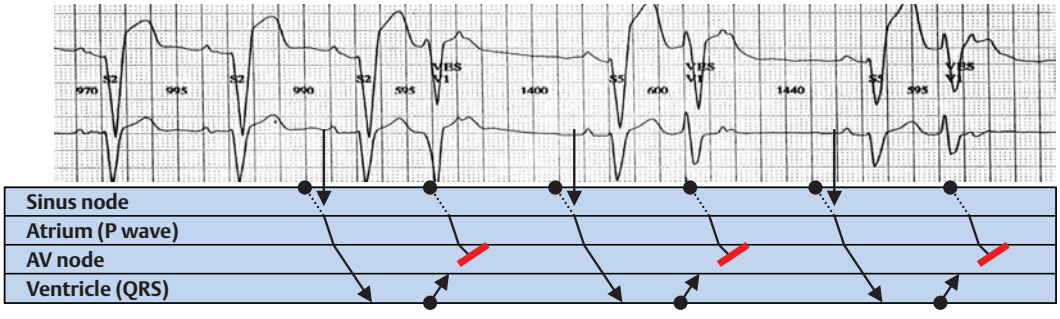
Etiology:
- People with no cardiac disease: idiopathic
- Possible in all types of cardiac disease

Treatment:
- Independent of underlying disease

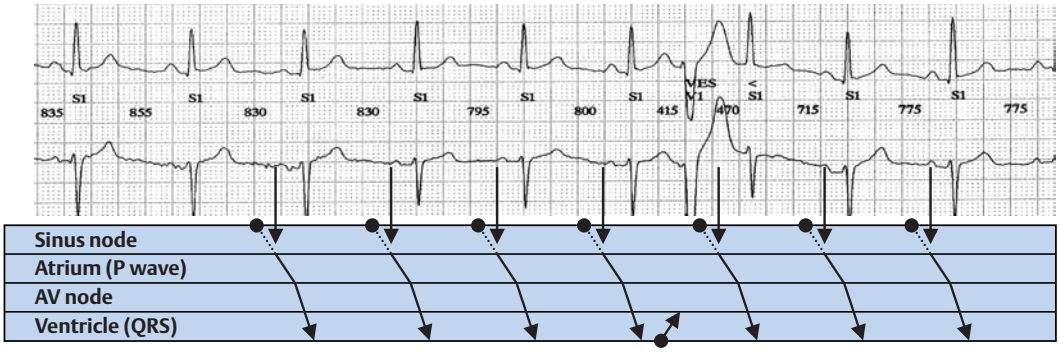
Ventricular Extrasystole—Trigeminy



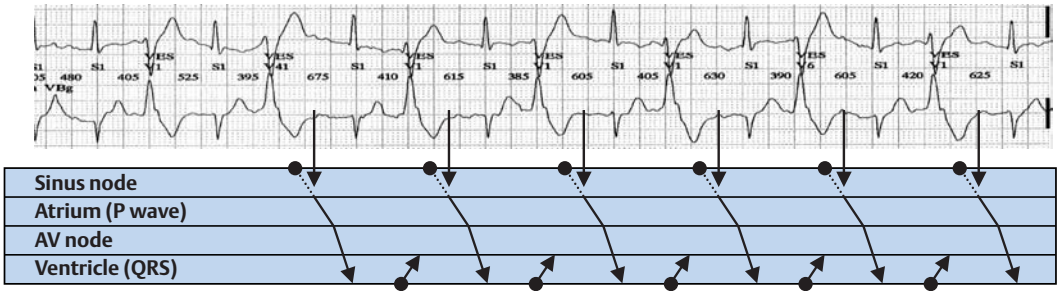
Ventricular Extrasystole—Bigeminy



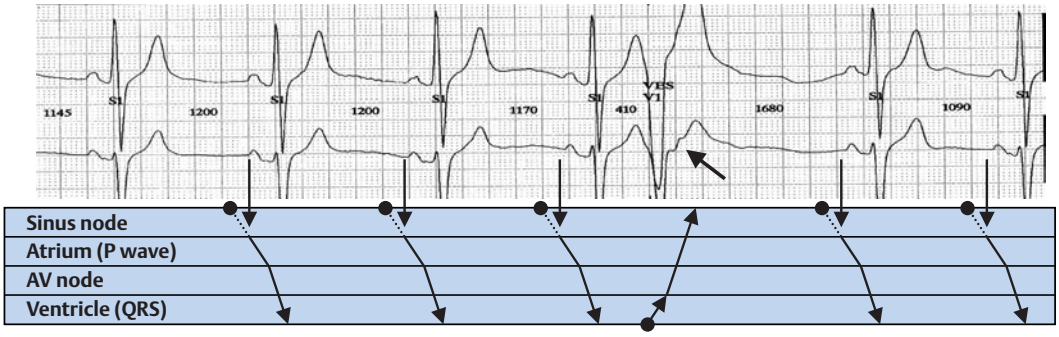
Ventricular Extrasystole—Interposed Ventricular Extrasystole



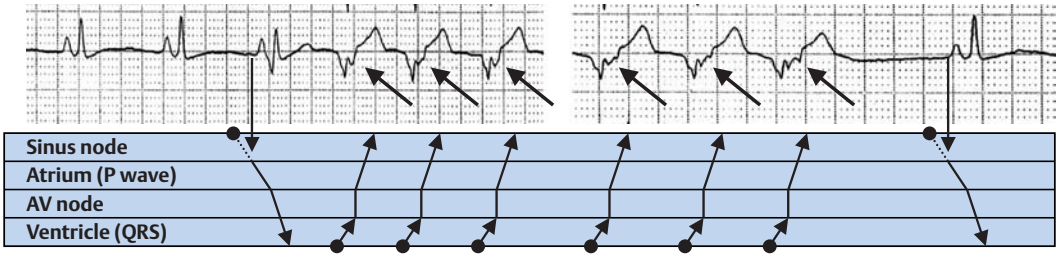
Ventricular Extrasystole—Interposed Bigeminy



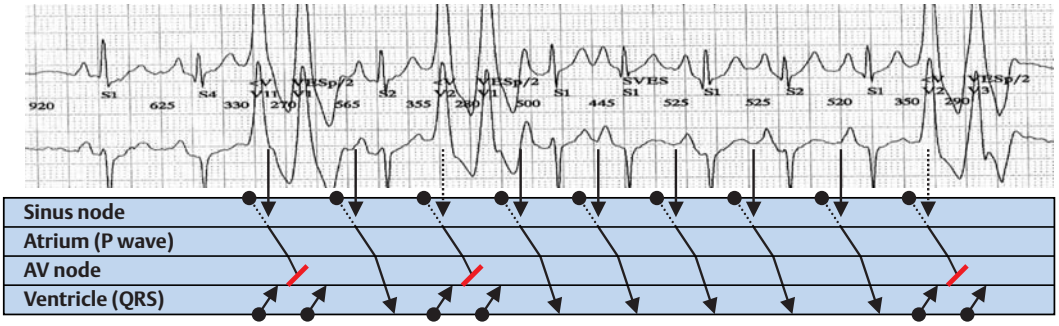
Singular Ventricular Extrasysteole With Retrograde Conduction



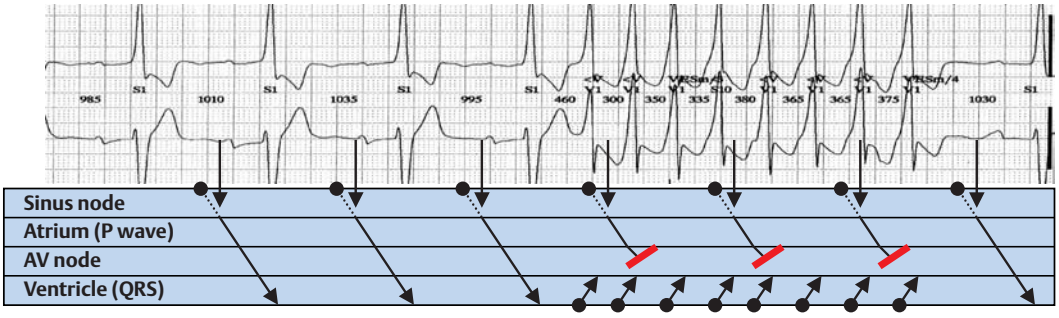
Idioventricular Rhythm With Retrograde Conduction



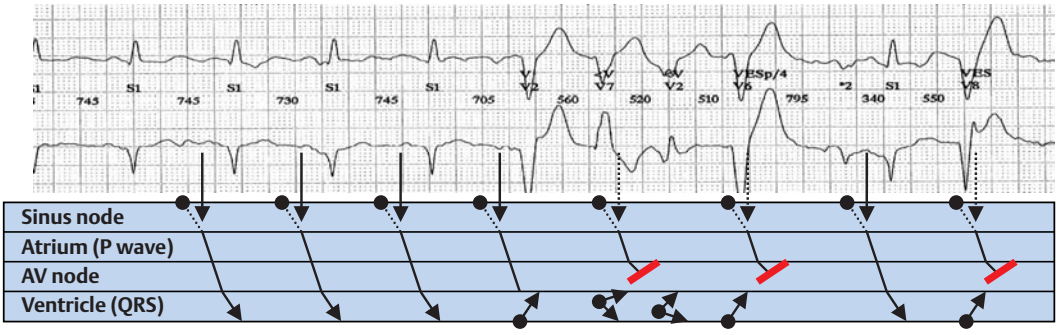
Ventricular Couplets



Ventricular Extrasystole—Monomorphic Salve



Ventricular Extrasystole—Polytopic Salve

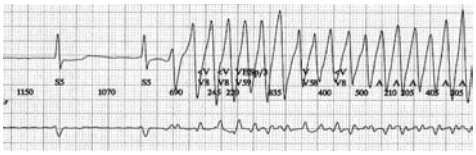


Ventricular Extrasystole—Lown Classification

Significance:

- Judgement on prognosis in patients with acute myocardial infarct

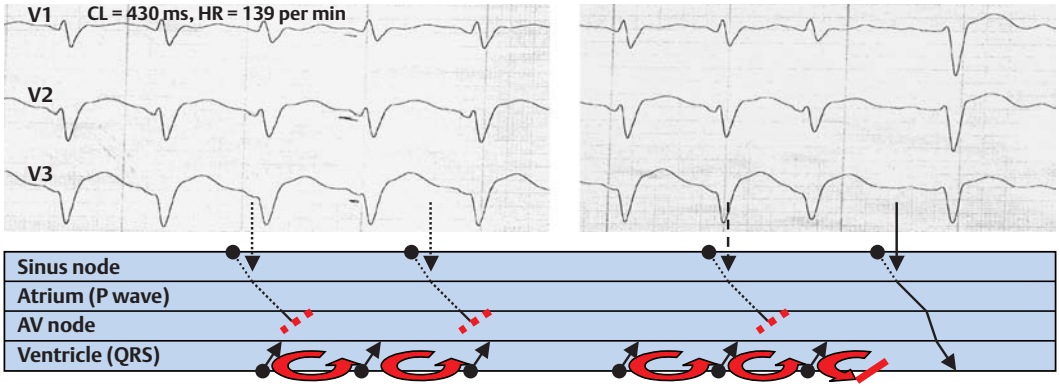
R-on-T-phenomenon



Classes:

- 0 No ventricular extrasystole
- I Fewer than 30 monotopic ventricular extrasystoles per hour
- II More than 30 monotopic ventricular extrasystoles per hour
- III A Polytopic ventricular extrasystoles
- III B Ventricular bigeminy
- IV A Ventricular couplets
- VI B Ventricular salves (> three ventricular extrasystoles) and tachycardia
- V R on T phenomenon

Slow Monomorphic Ventricular Tachycardia Following Anterior Infarction, Spontaneous Termination



Etiology:
- Coronary heart disease with status post infarct

Mechanism:
- Area in the region of the infarction scar with delayed impulse conduction facilitates a reentry circuit

ECG characteristics:
- Tachycardia with wide QRS complex

Acute treatment:
- Defibrillation
- If hemodynamically stable, also attempt termination with antiarrhythmics (class IB, amiodarone) in intensive care

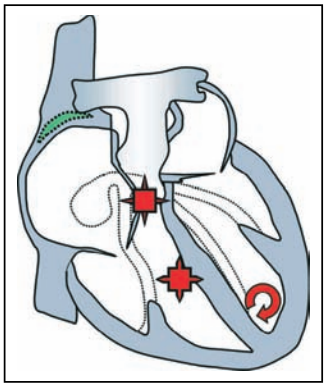
Chronic treatment:
- In the majority of cases implantation of a defibrillator (ICD)
- Antiarrhythmics and ablation treatment rarely curative, more frequently used as adjuvant treatment to reduce the frequency of arrhythmic episodes

Idiopathic Ventricular Tachycardia

Idiopathic ventricular tachycardia arising from the right ventricular outflow tract (RVOT)

Mechanism:
- Triggered activity or abnormal automation

ECG characteristics:
- Tachycardia with wide QRS complex
- Inferior axis (ranging from right to marked right axis deviation); left bundle branch configuration
- V1: "QS" configuration (absent "r")
- Tall narrow "R" in leads II and III
- Small "R" or "rS" in lead I



Repetitive monomorphic right ventricular tachycardia (GALLAVERDIN)

Mechanism:
- Triggered activity

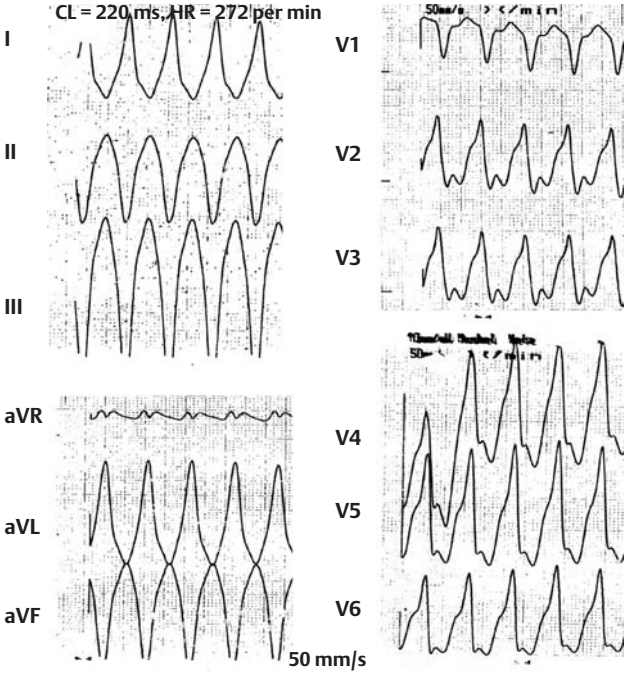
ECG characteristics:
- Tachycardia with wide QRS complex
- Left bundle branch configuration
- Inferior axis (right to marked right deviation)

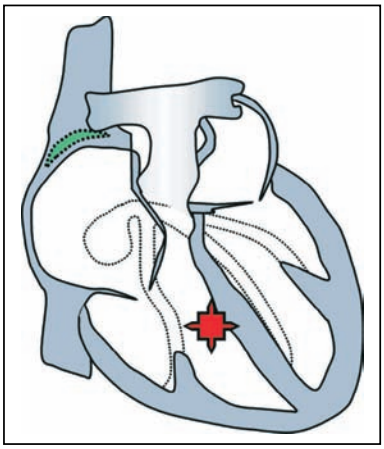
Idiopathic left ventricular tachycardia

Mechanism:
- Mostly reentry

ECG characteristics:
- Tachycardia with wide QRS complex
- However, rarely wider than 120 ms
- Right bundle branch configuration
- Left anterior axis (marked left deviation)

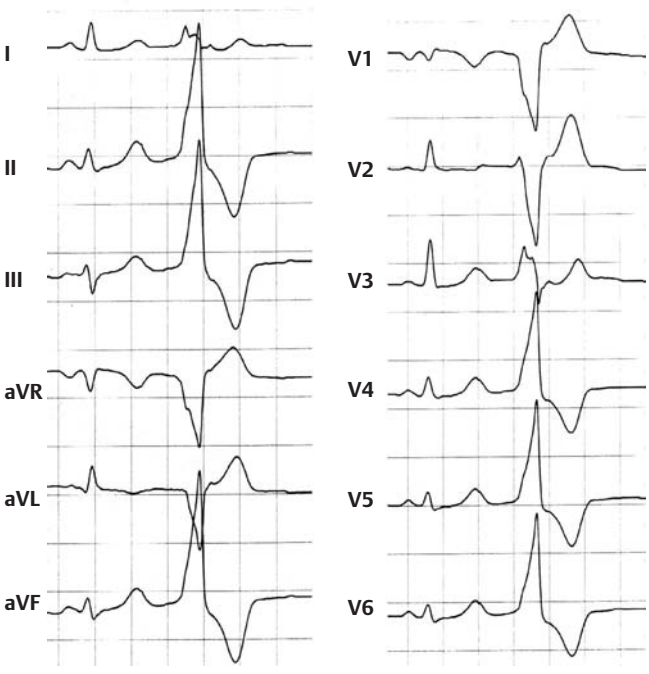
Idiopathic Ventricular Tachycardia—Septal Origin

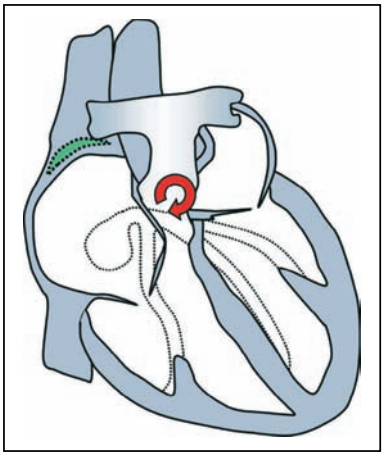




Differential diagnosis:
- Idiopathic ventricular tachycardia (RVOT origin)
- Ventricular tachycardia with structural heart disease
- Supraventricular tachycardia with functional bundle branch block or antidromic excitation

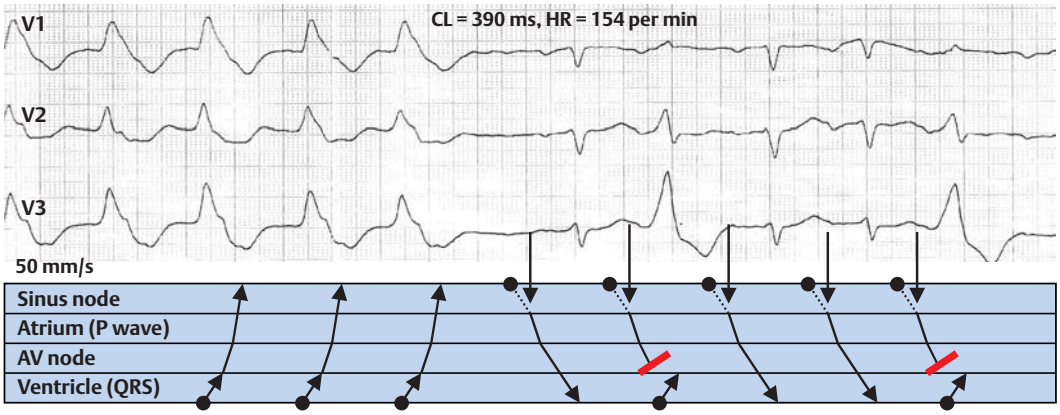
Idiopathic Ventricular Extrasystole—RVOT





Differential diagnosis:
- Other idiopathic extrasystoles
- Ventricular extrasystole in structural heart disease
- SVES with functional left bundle branch block

Catecholamine-Sensitive Ventricular Tachycardia, Spontaneous Termination



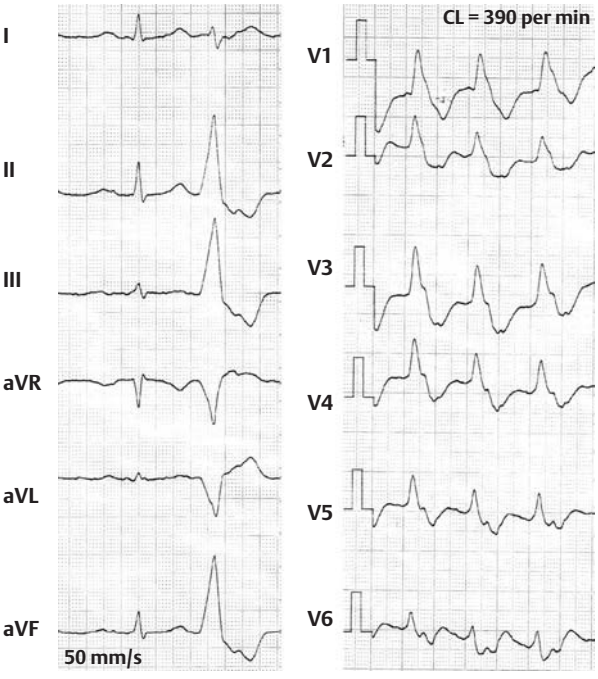
Mechanism:
- Triggered activity as a result of raised catecholamine level (e.g., under stress-including ECG stress test with bicycle, anger/excitement, etc.)

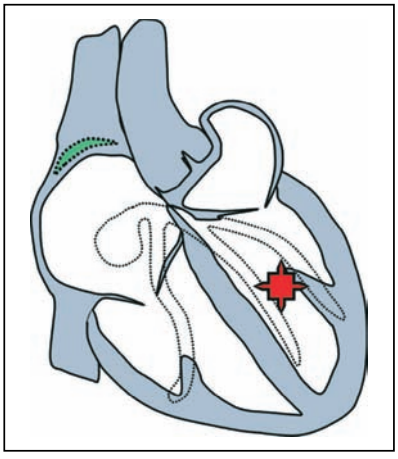
ECG characteristics:
- Wide ventricular complex tachycardia on stress test; right bundle branch block configuration
- Inferior axis; retrograde conduction
- Arrhythmia termination in the recovery phase

Etiology:
- Unclear
- Good prognosis

Treatment:
- Beta-blockade (mostly successful)
- (Ablation/debrillator)

Catecholamine-Sensitive Ventricular Tachycardia

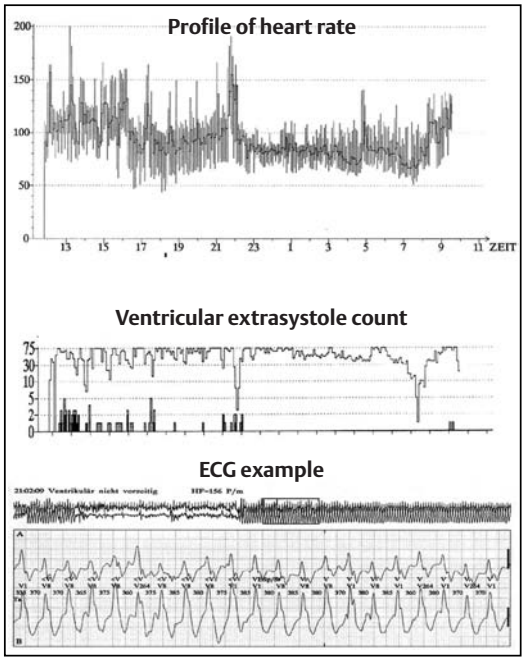




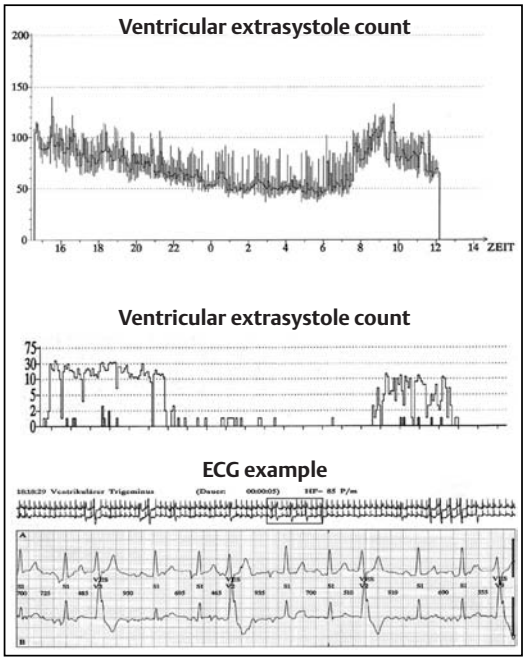
Differential diagnosis:
- Idiopathic ventricular tachycardia
- Ventricular tachycardia in structural heart disease
- Supraventricular tachycardia with functional bundle branch block or antidromic excitation

Catecholamine-Sensitive Ventricular Tachycardia

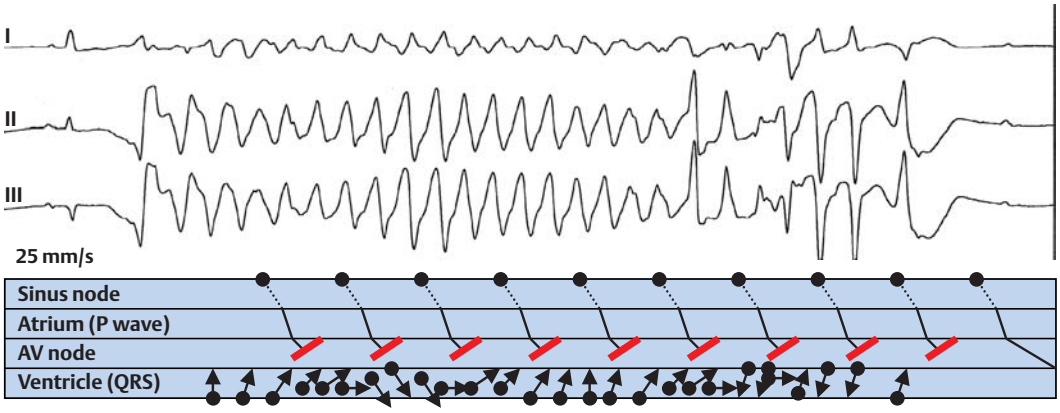
Holter before beta-blockade



Holter after beta-blockade



Torsade de Pointes Tachycardia



Etiology:
- Often with congenital/acquired QT syndrome
- Also possible with other heart disease

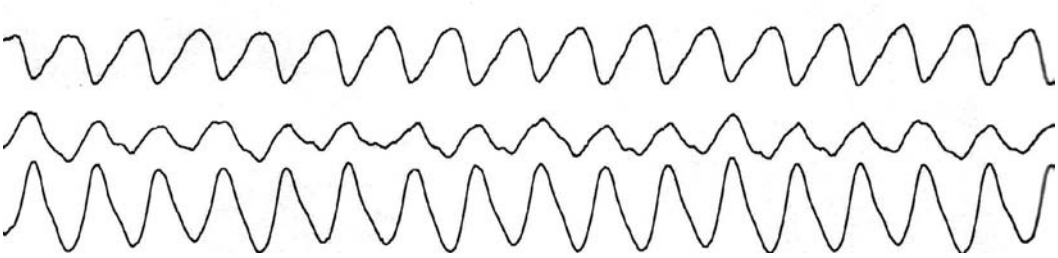
Mechanism:
- Inhomogeneity of repolarization

ECG characteristics:
- Very rapid ventricular contraction with variable amplitude and vector

Acute treatment:
- Defibrillation, magnesium, beta-blocker

Chronic treatment:
- Treatment of the underlying disease (including cessation of antiarrhythmics)
- Beta-blockade
- Defibrillator (ICD)

Ventricular Flutter



Sinus node
Atrium (P wave)
AV node
Ventricle (QRS)

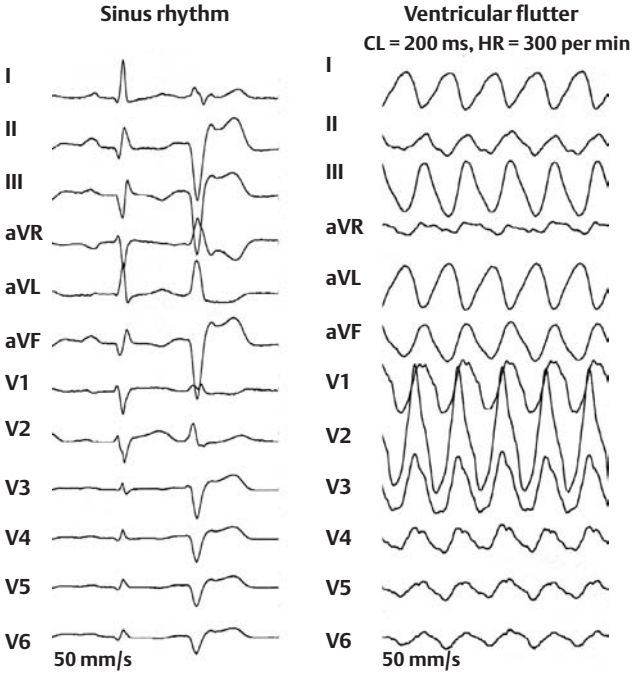
Mechanism:
- Rapid reentry?
- Faster focus?

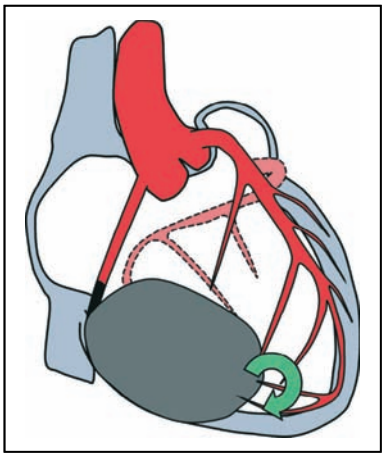
ECG characteristics:
- Homogeneous ventricular complexes with no isoelectric axis
- Hair-pin configuration
- Ventricular rate between 240 and 300 per minute

Etiology:
- Severe cardiac disease
- Very rare as primary arrhythmia

Treatment:
- For hemodynamic reasons—circulatory arrest—immediate defibrillation
- Treatment of underlying disease
- ICD implantation in most cases; adjuvant antiarrhythmics

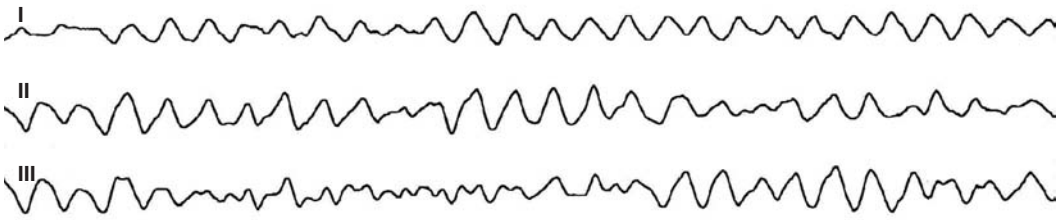
Ventricular Flutter Following Posterior Myocardial Infarction





Differential diagnosis:
- Other ventricular tachycardia
- Supraventricular tachycardia with functional bundle branch block
- Antidromic WPW and Mahaim
- Reentry tachycardia

Ventricular Fibrillation



Sinus node
Atrium (P wave)
AV node
Ventricle (QRS)

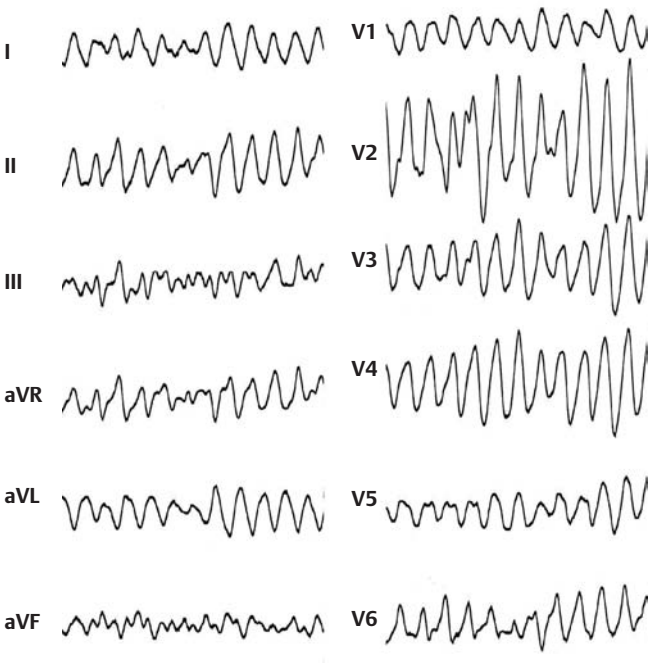
Mechanism:
- Rapid reentry?
- Faster focus?

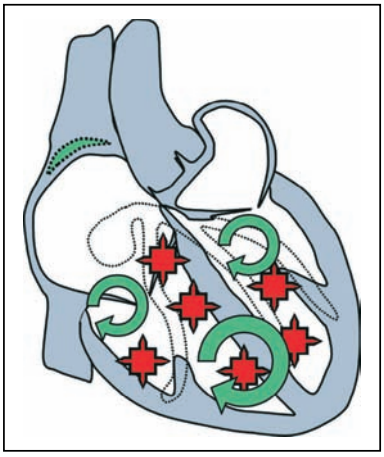
ECG characteristics:
- Nonhomogeneous oscillating ventricular complexes with no isoelectric axis
- Wave and tooth configuration
- Ventricular rate > 300 per minute

Etiology:
- Severe cardiac disease
- Very rare as primary arrhythmia

Treatment:
- For hemodynamic reasons—circulatory arrest—immediate defibrillation
- Treatment of underlying disease
- ICD implantation in most cases; adjuvant antiarrhythmics

Ventricular Fibrillation

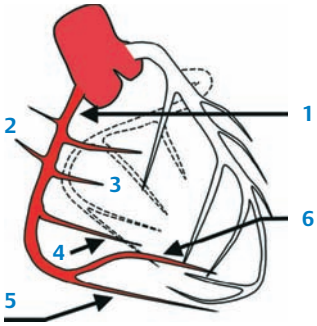




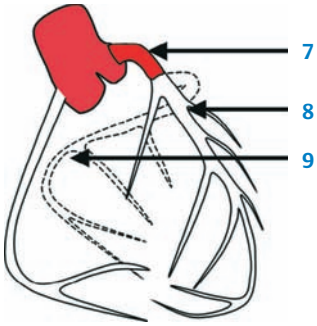
Differential diagnosis:
- Ventricular flutter
- Atrial fibrillation with 1 : 1 conduction with WPW syndrome

4 Coronary Heart Disease and Myocardial Infarction

Right and Left Coronary Arteries

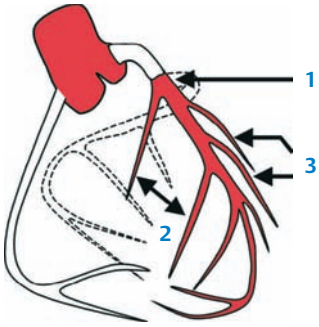


Right coronary artery (RCA) [1]:
Supply:
Parts of the lateral and posterior wall, atria, sinus and AV node
Branches:
– Before crux cordis:
  • Atrial branches: right atrial branches [2]
  • Right ventricular branches [3]
  • Marginal branches [4]
  Division at the crux cordis:
  • Posterior interventricular branch [5]
  • Posterior lateral branches (one to three branches) [6]

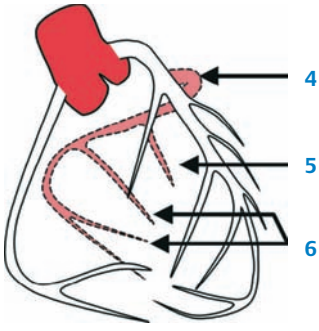


Left coronary artery (LCA, main branch) [7]:
Supply:
Of the anterolateral anterior wall, of the ventral and apical septum, parts of the lateral and posterior wall, atria, sinus and AV node
Branches:
– To the anterior wall: left anterior descending artery (LAD) [8]
– To the lateral wall: circumflex branch (RCX) [9]

Left Anterior Descending Artery and Circumflex Artery

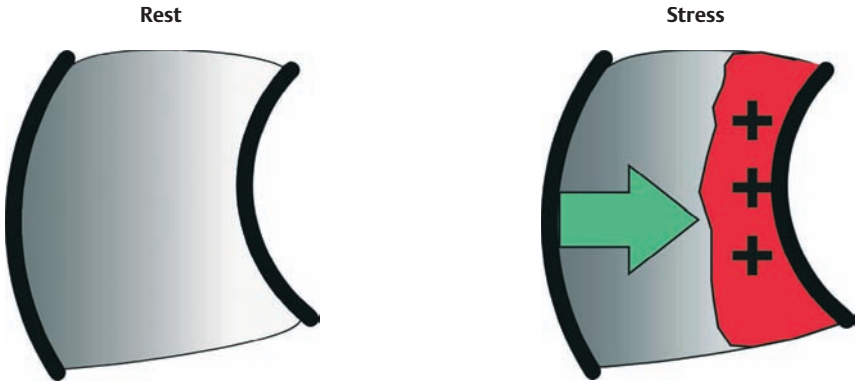


Left anterior descending artery (LAD) [1]:
Supply:
Of the anterolateral wall, of the ventral and apical septum
Branches:
– To the septum: septal branches [2]
– To the anterolateral wall: diagonal branches [3]



Circumflex branch (RCX) [4]:
Supply:
Of parts of the lateral and posterior wall, of parts of the atria, sinus and AV node
Branches:
– Side branches of proximal origin: marginal branches [5]
– Side branches of distal origin: posterolateral branches [6]
– In very large RCX: also posterior interventricular branch (otherwise belongs to the RCA)

Stress-Induced Ischemia in Coronary Heart Disease

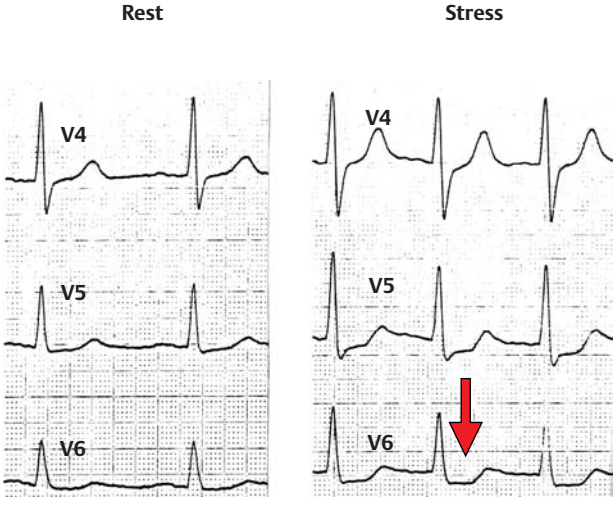


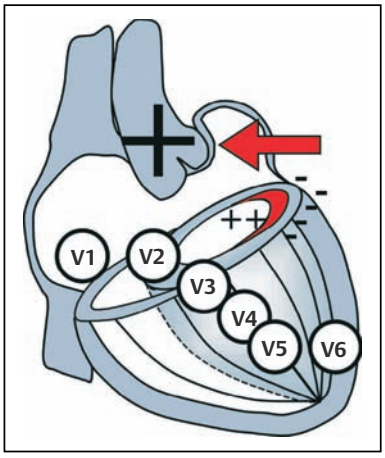
Mechanism:
- In the affected area the inner myocardial layer (endocardium) is particularly susceptible to ischemia (so-called last meadows), under stress → reduced perfusion with decreased electrical excitability, ischemic area becomes electropositive compared to the rest of the myocardium; thereby flow of charge from the healthy electronegative myocardium to the ischemic zone

Etiology:
- Coronary heart disease with at least one significant stenosis

Treatment:
- Interventional: PCTA/bypass
- Medications: ASA, beta-blocker, nitrate, CSE-inhibitor, ACE-inhibitor if necessary

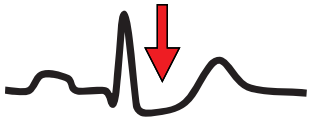
Stress-Induced Ischemia in Coronary Heart Disease



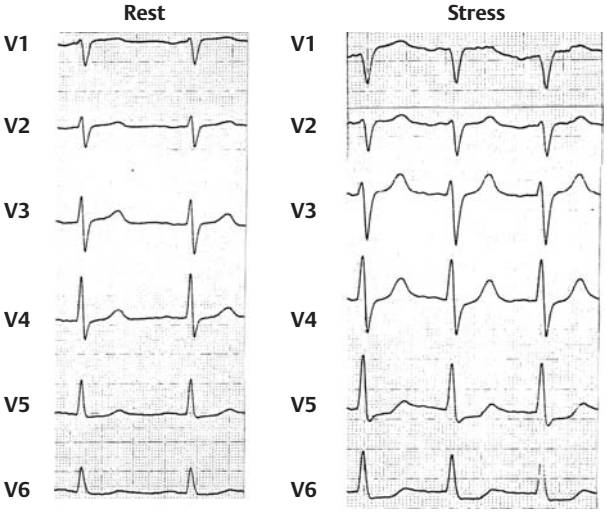


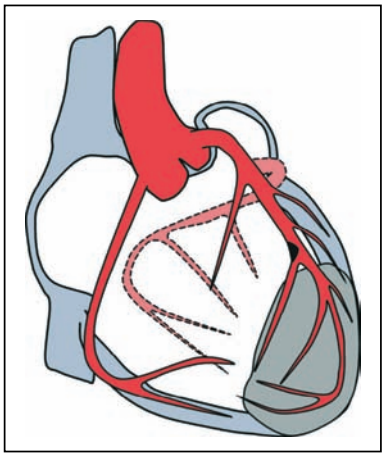
ECG characteristics:
- Horizontal to descendent ST segment depression (significant if more than 0.1 mV in the limb leads, if more than 0.2 mV in the precordial leads)
- Also, possibly additional changes of the T wave configuration (inversion)

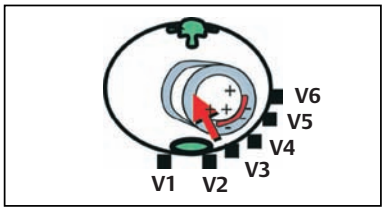
ECG characteristics:



Stress-Induced Ischemia in Coronary Heart Disease (Anterior Wall) Without Infarction

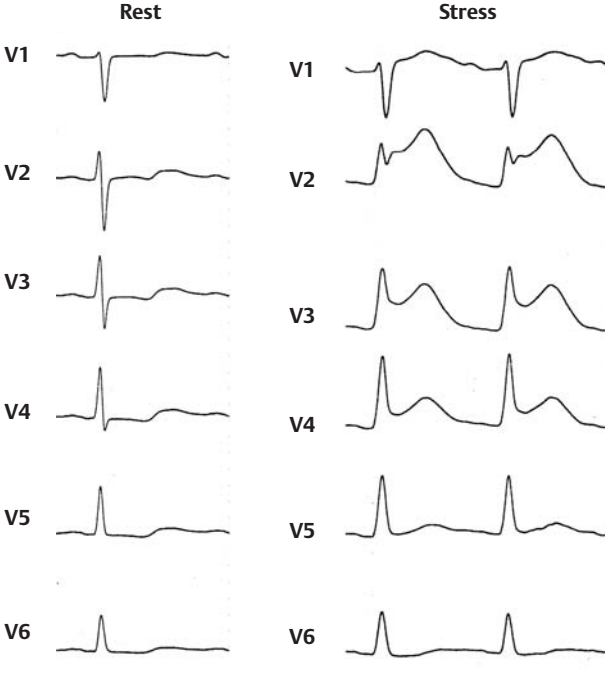


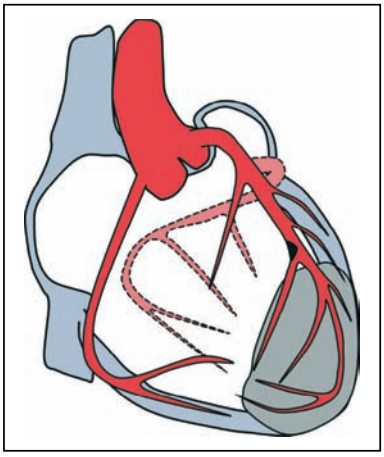


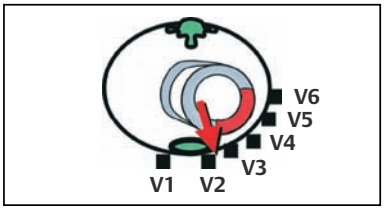


Differential diagnosis:
- Left ventricular hypertrophy, cardiomyopathy
- Digitalis, antiarrhythmics
- Bundle branch block, WPW syndrome
- Sympathetic tone, hypokalemia

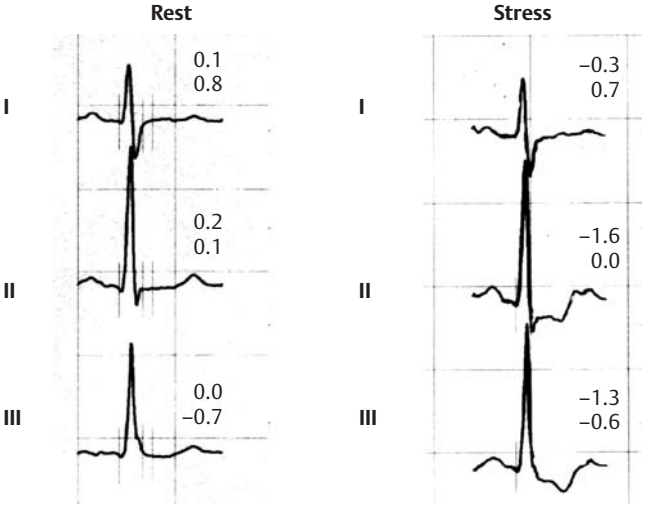
Vasospasm in the Region of the Anterior Wall With Acute Transmural Ischemia Under Stress

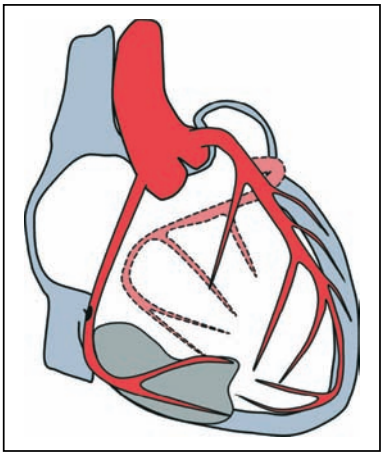


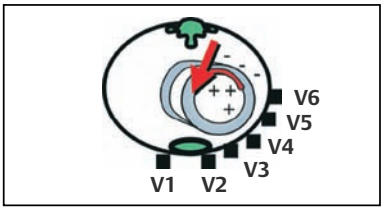




Stress-Induced Ischemia in Coronary Heart Disease (Posterior Wall) Without Infarction



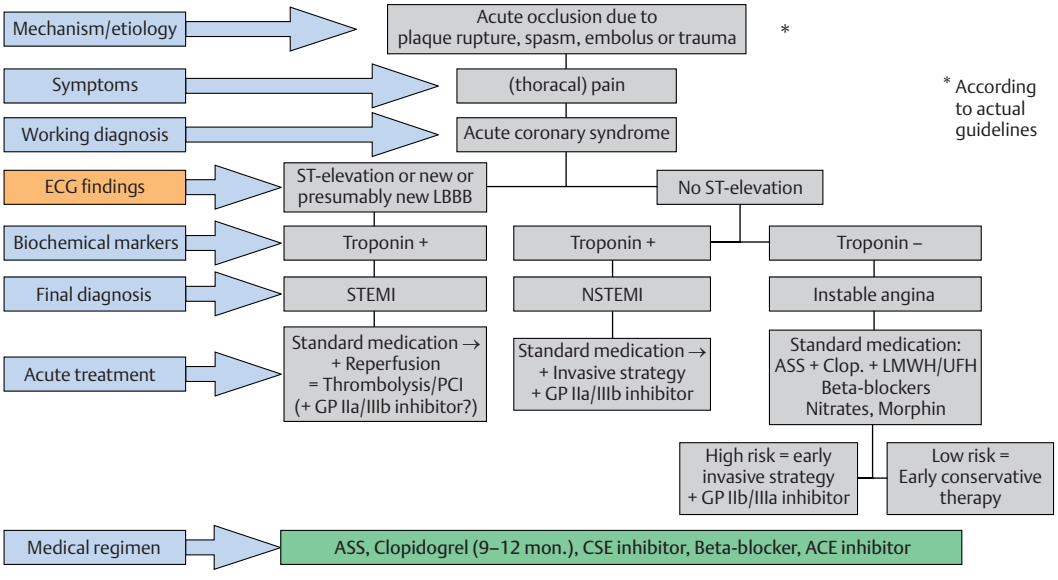




ECG characteristics:
- Horizontal to descendent ST segment depression (significant if more than 0.1 mV in the limb leads, if more than 0.2 mV in the pre-cordial leads)
- Also, possibly additional changes of the T wave configuration (inversion)

Acute Coronary Syndrome (ACS)

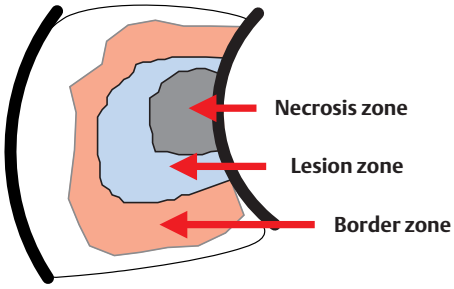
Definition:
- Clinical signs during the period between the occurrence of acute arterial occlusion and the course to clinical stabilization or to development of myocardial infarction



Acute Myocardial Infarction

Anatomical pathology:
- **Necrosis zone:** Electrically inactive zone (infarction Q)
- **Lesion zone:** Cells markedly damaged by ischemia form abnormal potentials without participating in excitation, damaged site from which current arises — represented by ST elevation
- **Border zone:** Cells participate in excitation with delayed repolarization (negative T wave)

Diagnosis:
- Clinical, ECG changes, enzyme profile (creatine phosphokinase, troponin)
- If two out of three criteria are positive, then infarct is confirmed



Definition:
- Acute myocardial necrosis as a result of interruption of coronary perfusion
- STEMI (ST-Elevation Myocardial Infarction): ST-elevation at least in two limb leads ≥ 0.1 mV or in two precordial leads ≥ 0.2 mV or LBBB with typical symptoms

Etiology:
- Coronary heart disease
- Inflammatory, trauma, spasm, embolism

Complications:
- Bradycardia, ventricular arrhythmias
- Aneurysm, shock, papillary muscle rupture
- Rupture of the wall, ventricular septal defect
- Pericarditis, Dressler syndrome

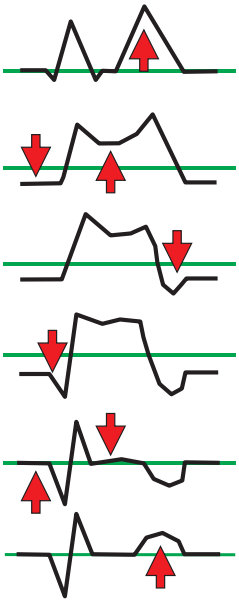
Acute treatment:
- Reperfusion: lysis/PCTA/heparin/bypass
- Adjuvant: nitrate, beta-blocker, sedation, oxygen
- Treatment of complications

Chronic treatment:
- Beta-blocker, thrombocyte aggregation inhibitor
- CSE- and ACE-inhibitors
- Treatment of cardiac insufficiency and arrhythmia

Myocardial Infarction: Stages and ECG Changes

Acute stages:
1. Steeplelike/tented T waves (delayed repolarization of the inner layer as a result of acute ischemia) —early stage
2. Depression of the isoelectric line and elevation of the ST segment (diastolic and systolic current arising from damage)—transmural ischemia
3. Inversion of T wave (delayed repolarization) —intermediate stage
4. Formation of an "infarction Q" (myocardial necrosis)—intermediate stage

Chronic stages:
5. Normalization of the ST segment (following stage)
6. Normalization of the T wave (chronic)



1 h

2–3 h

4–6 h

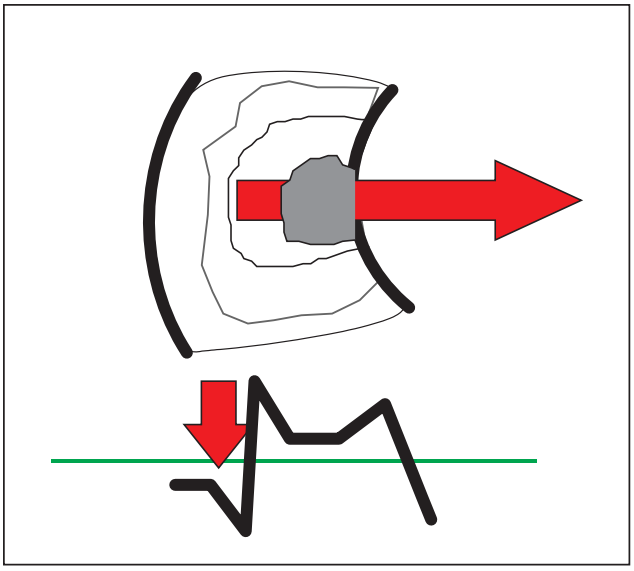
6 h

2–3 weeks

4 weeks

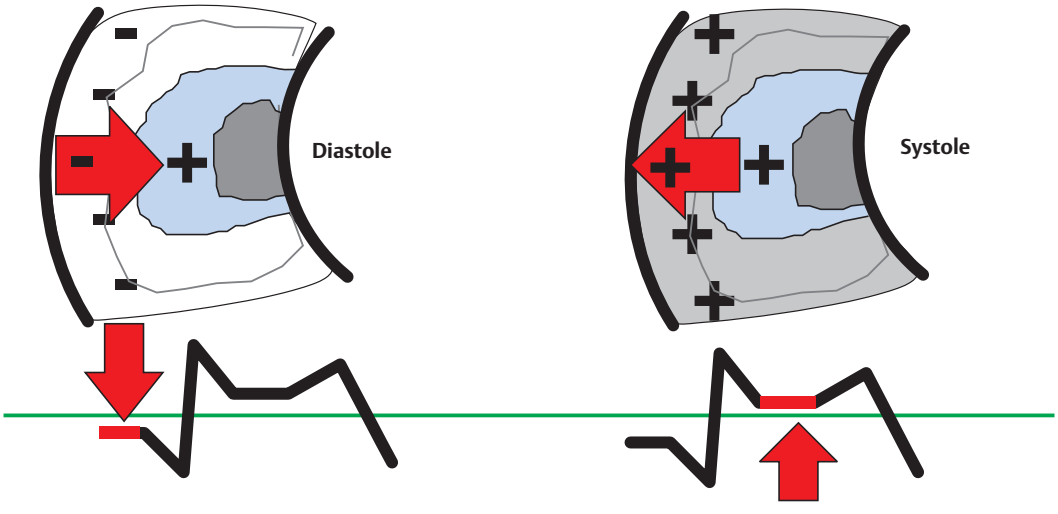
"Infarction Q Wave"

The infarcted tissue is electrically passive and forms a so-called electric hole.
The electrical vector moves forward from the infarct; a negative deflection arises in the form of a Q wave.



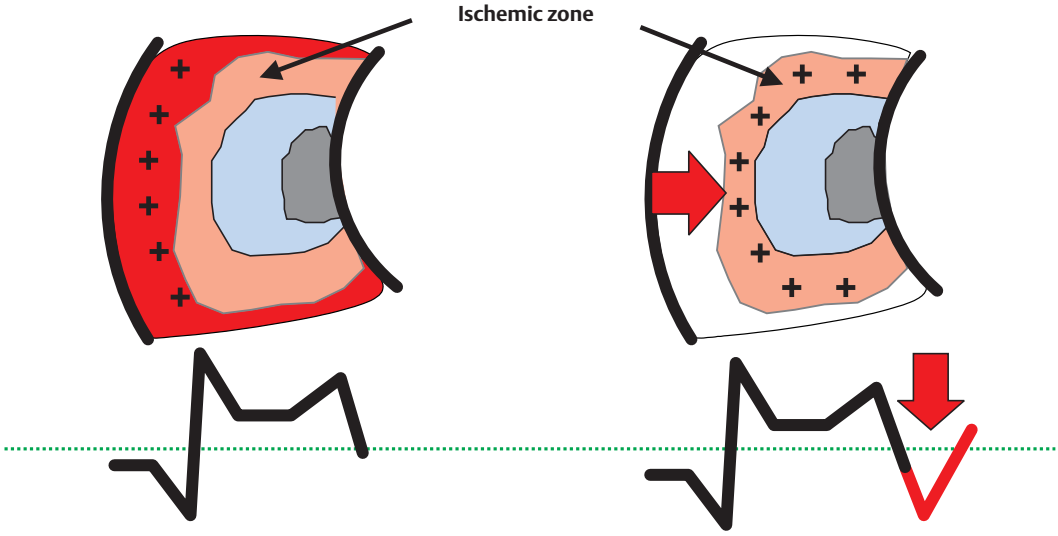
Changes in the ST Segment in Infarction

Zone of cell damage (injury) with abnormal resting potential. In diastole the cells are more electropositive than the healthy myocardium, causing the flow of current to the damaged zone with depression of the isoelectric line. In systole normal depolarization of the healthy myocardium, reversal of the flow of current to the healthy myocardium with ST elevation.

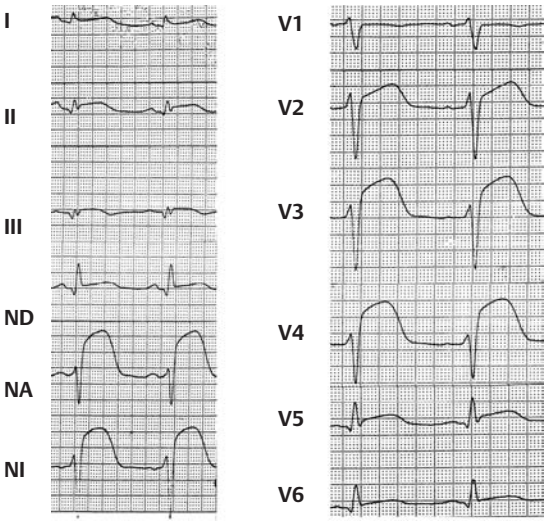


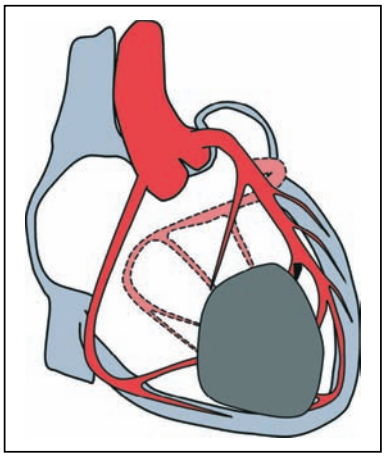
T Inversion in Infarction

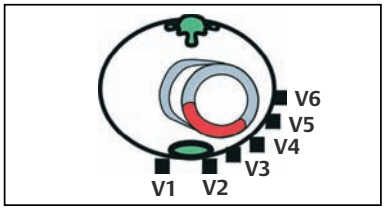
Whilst the healthy myocardium repolarizes within a normal time frame, the ischemic zone at the border of the infarct region remains electrically active due to delayed repolarization, causing a flow of current from the healthy myocardium to the ischemic zone with occurrence of a negative T wave.



Myocardial Infarction—Anterior Wall Septal Infarction—Acute Stage



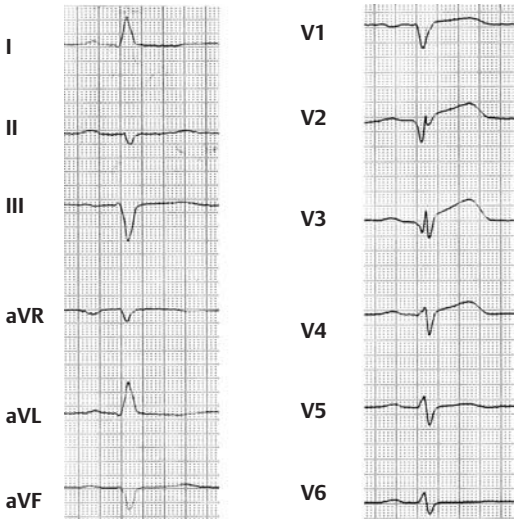


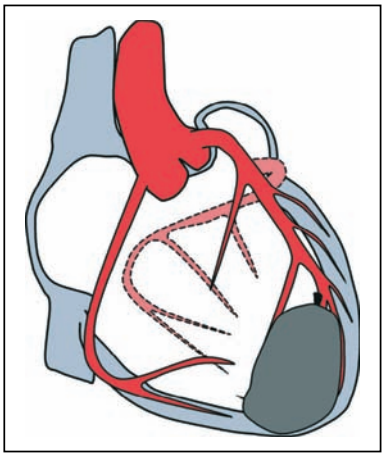


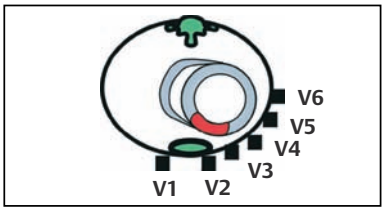
ECG characteristics:
- Direct signs of infarction: I, II, V2–5

Coronary findings:
- Variable (often septal branch of LAD or LAD itself)

Myocardial Infarction—Septum Apex Infarction



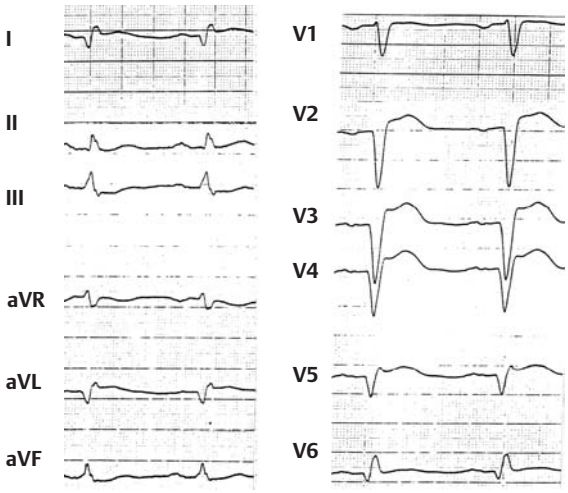


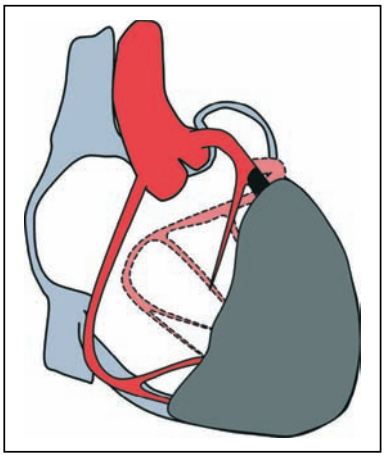


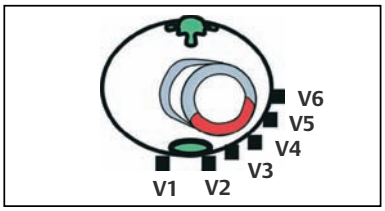
ECG characteristics:
- Direct signs of infarction: (I, II),(V1), V2–4

Coronary findings:
- Occlusion: distal LAD

Extensive Anterior Myocardial Infarction—Chronic Stage With Aneurysm Formation



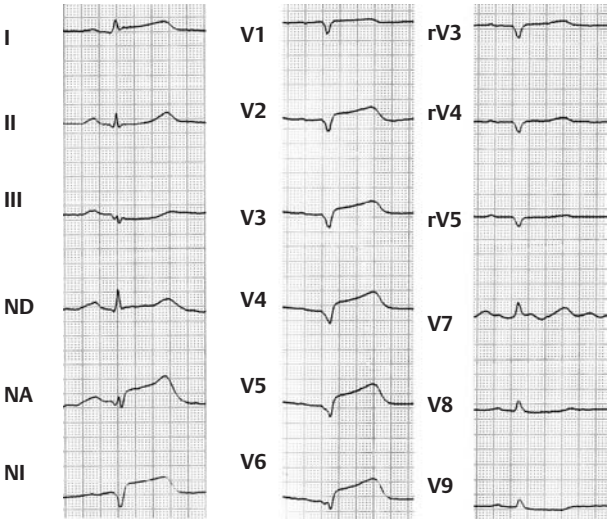


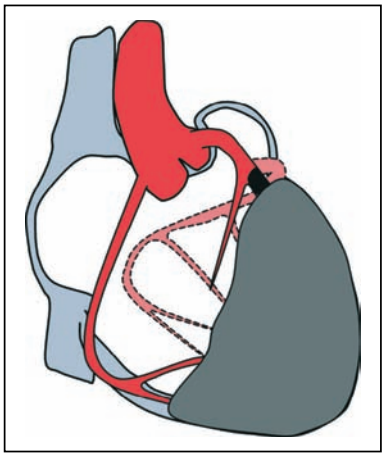


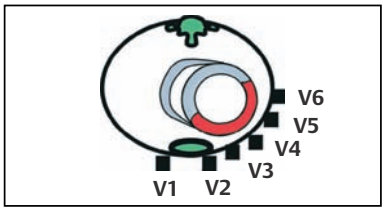
ECG characteristics:
- Direct signs of infarction: I, (II), aVL, (V1), V2-5, (V6)
- Persistent ST elevation as a result of formation of aneurysm

Coronary findings:
- Occlusion: proximal LAD

Extensive Acute Anterior Wall Infarction



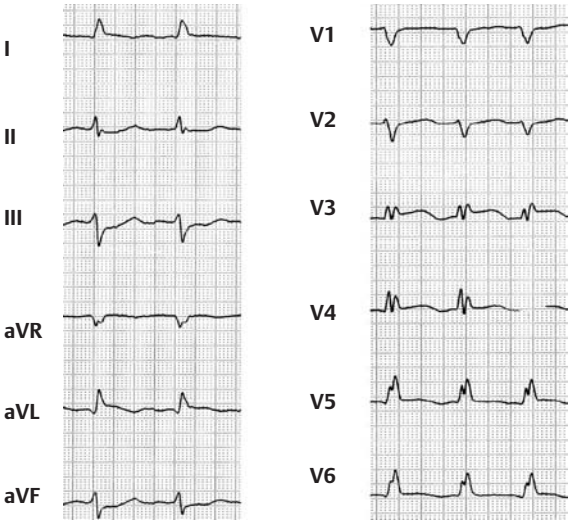


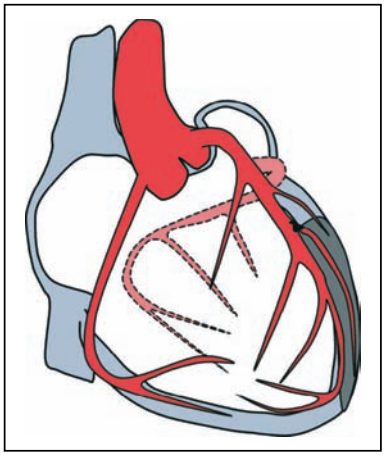


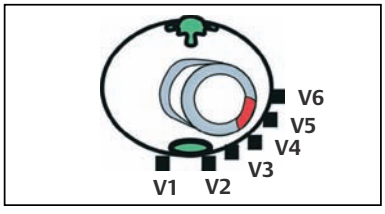
ECG characteristics:
- Direct signs of infarction: I, (II), aVL, (V1), V2–5, (V6), NA, NI

Coronary findings:
- Occlusion: proximal LAD

Anterolateral Infarction With Atrial Fibrillation



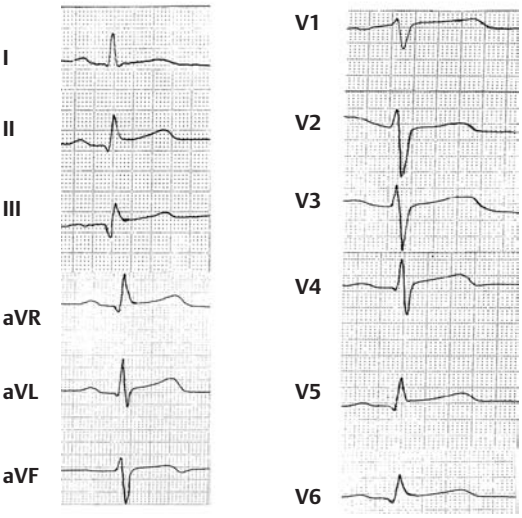


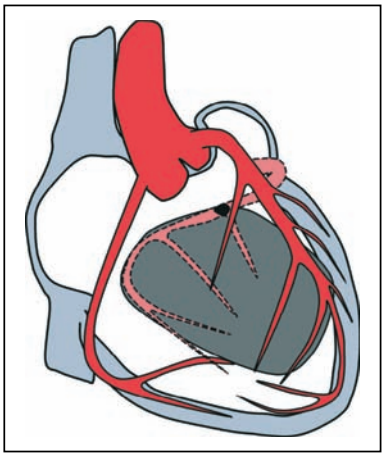


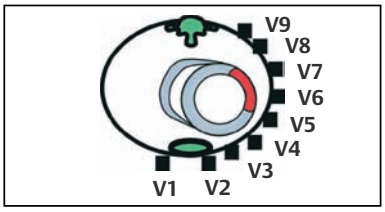
ECG characteristics:
- Direct signs of infarction: I, aVL, (V3)4–V6

Coronary findings:
- Occlusion: often diagonal branch of LAD

Lateral Posterior Wall Infarction (Posterolateral Infarction)



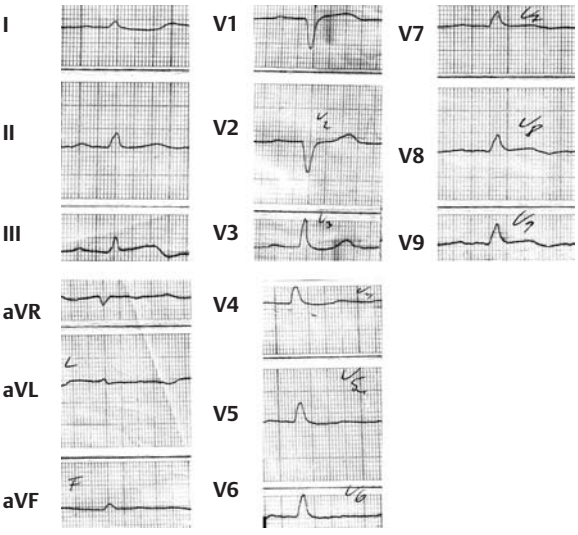


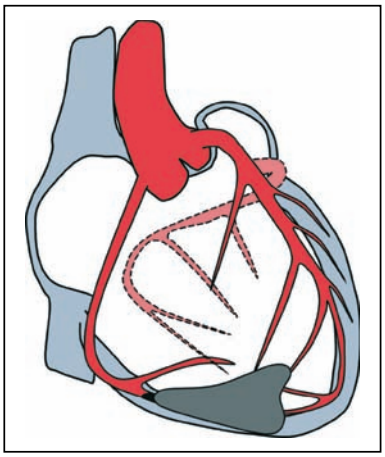


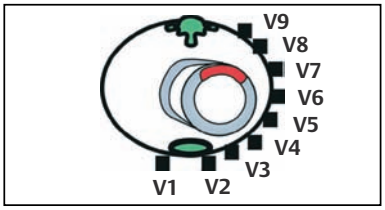
ECG characteristics:
- Direct signs of infarction: II, III, aVF, V5–7

Coronary findings:
- Occlusion: RCX branch or posterolateral branch of the RCX or RCA

Strict Posterior Infarction



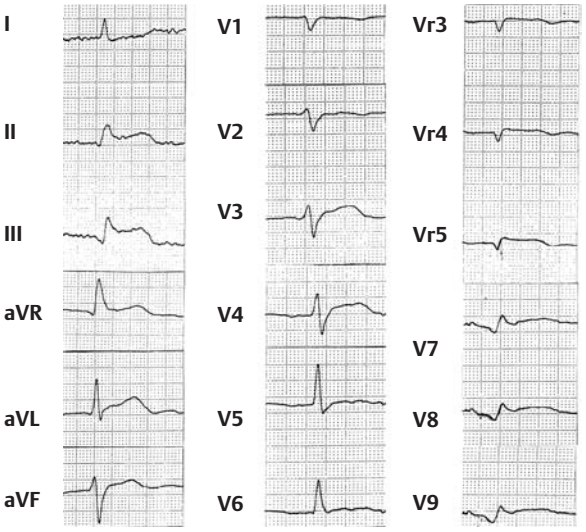


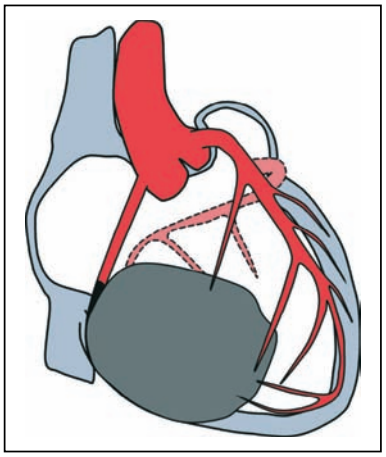


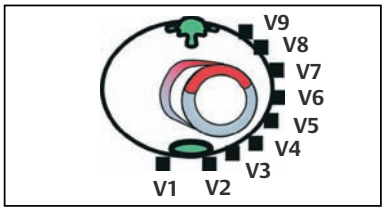
ECG characteristics:
- Direct signs of infarction: aVF, V8–V9

Coronary findings:
- Occlusion: often interventricular posterior branch of the RCA

Extensive Posterior Wall Infarction With Involvement of the Right Ventricle



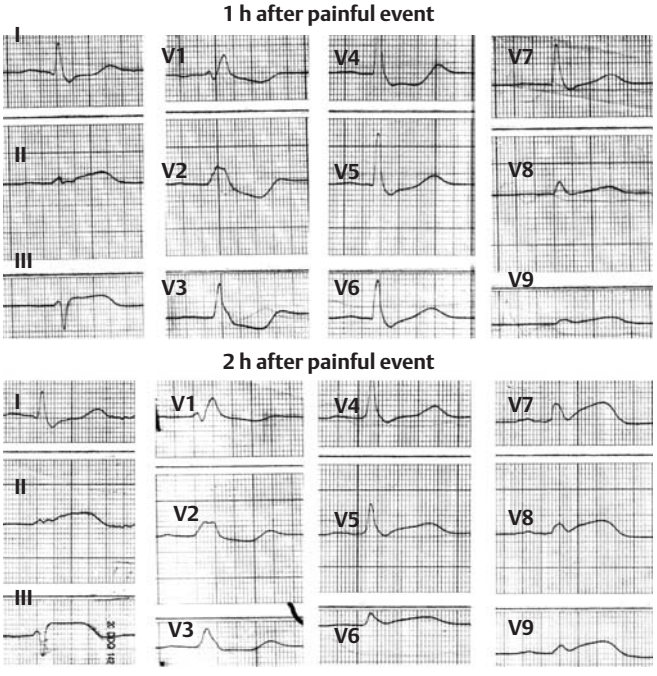


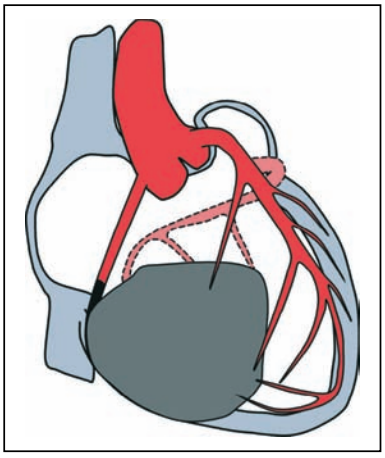


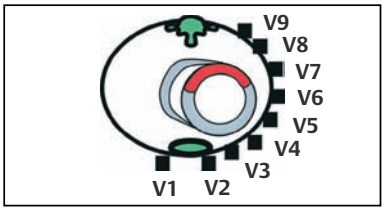
ECG characteristics:
- Direct signs of infarction in leads: II, III, V(6)–7–9
- Sign of right ventricular infarct rV3–rV5

Coronary findings: – Occlusion: often RCA

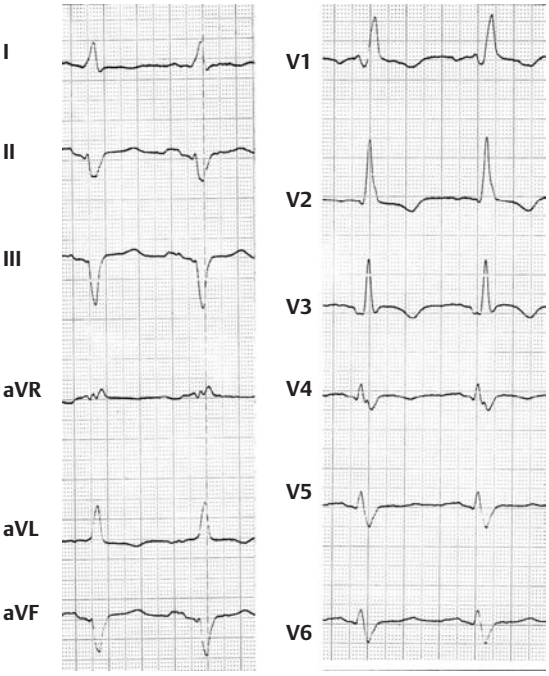
Extensive Posterior Wall Infarction With Complete Right Bundle Branch Block

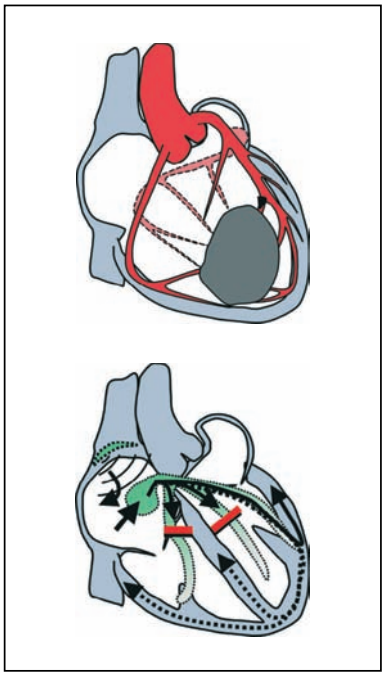




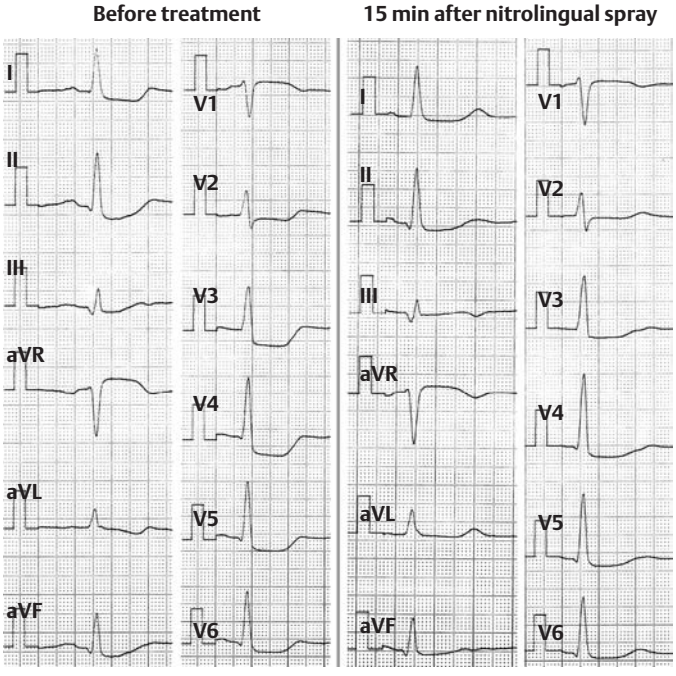


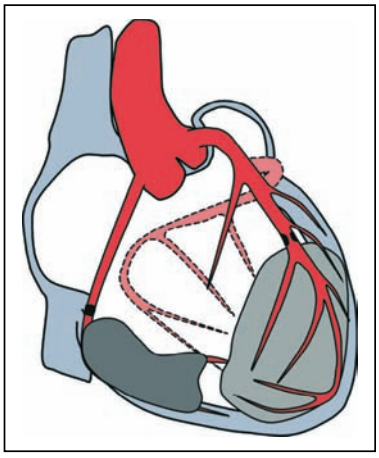
Status Post Septal Infarction With Bifascicular Block

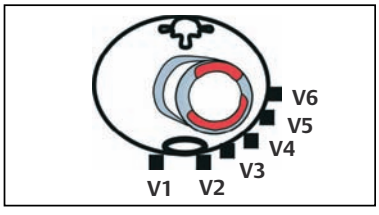




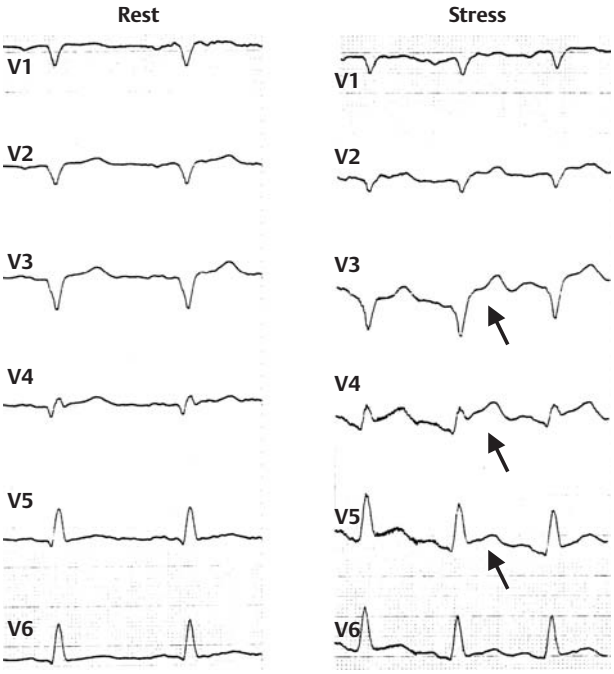
Resting Ischemia in the Anterior Wall Region Following Posterior Wall Infarction

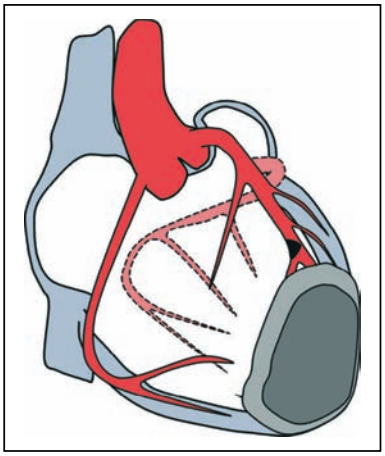






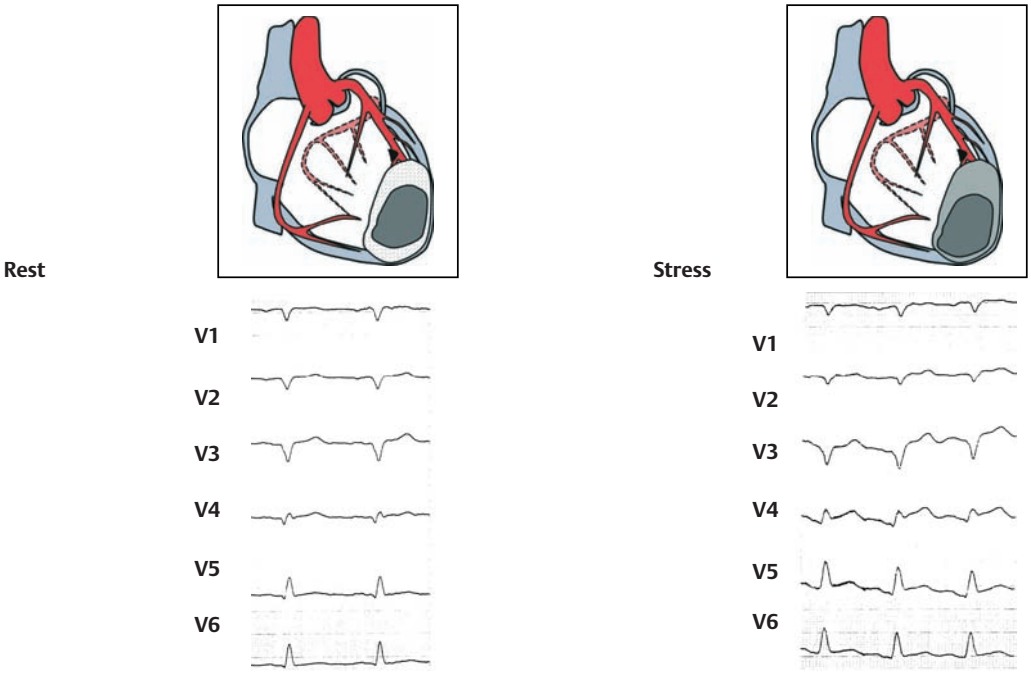
Stress-Induced Ischemia in the Infarct Region Following Anterior Myocardial Infarction



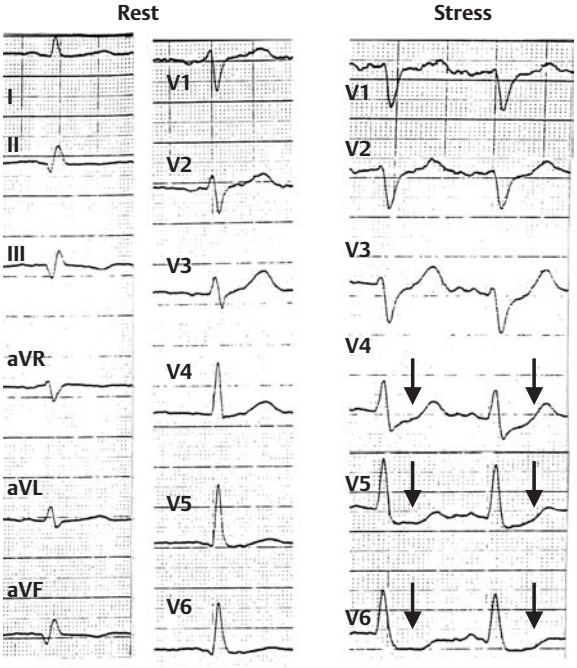


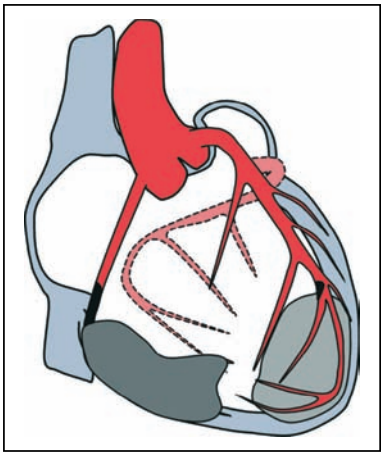
Differential diagnosis:
- Condition post anterior wall infarction
- Occurrence of ST elevation in the area of infarction—V3–5 (reconstruction of the infarction pattern) under stress

Stress-Induced Ischemia in the Infarct Region Following Anterior Myocardial Infarction



Stress-Induced Ischemia in the Anterior Wall Region Following Posterior Myocardial Infarction



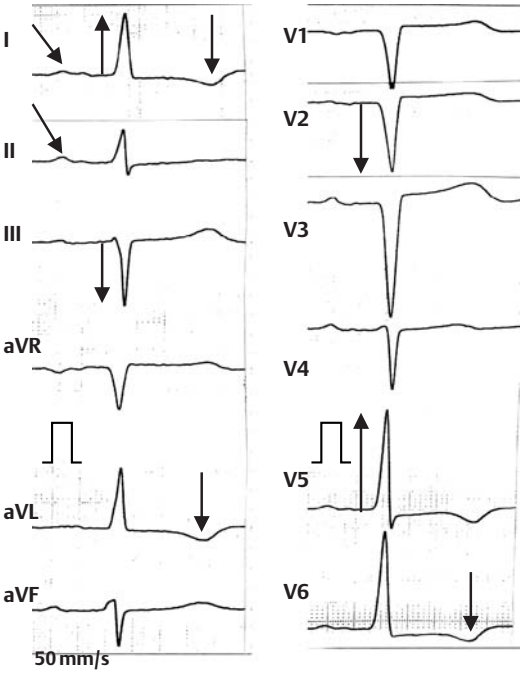


ECG characteristics:

- Condition post posterior myocardial infarction (III)
- Occurrence of ST elevation in the anterior wall region (I, V3–5) under stress

5 Other ECG Changes

Left Ventricular Hypertrophy



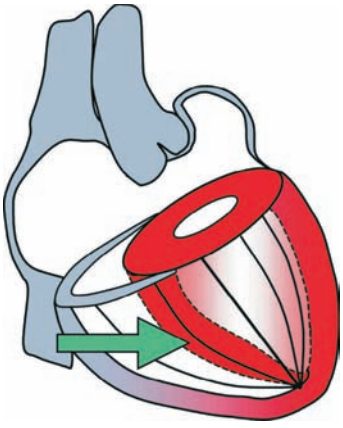
Mechanism:
- Hypertrophy of the left ventricular musculature as a consequence of systolic or diastolic overload
- This determines:
  - Rotation of the cardiac axis in the superior and posterior direction
  - Increase in voltage
  - Lengthening of impulse conduction
  - Relative ischemia of the inner layer with disturbed repolarization resulting in current flow from the outer to the inner layer

ECG characteristics:
- Left axis to marked left axis deviation
- Widened QRS complexes (QRS up to 0.11 s; turning point in V5/6 > 0.05 s)
- High voltage of the R/S amplitudes (see indices)
- Left ventricular repolarization changes (T wave inversion, ST depression)
- Other criteria with left bundle branch block !

Left Ventricular Hypertrophy

Indices with left ventricular hypertrophy:
- Sokolow index (S V1 + R V5 > 3.5 mV)
  - Sensitivity 25–43%, specifity 95%
- Lewis-Index (R I + S III – R III – S I > 1,7 mV)
- Cornell-Index (R aVL + S V3 > 35 mm)
- Romhilt-Estes point system with assessment of:
  - Amplitude (R or S in EA ≥ 2.0 mV S V1–3 ≥ 2.5 mV, R V4–6 ≥ 2.5 mV) — 3 points
  - Cardiac axis (left axis greater than –30°) — 2 points
  - ST–T changes — 1–3 points
  - QRS width (> 0.09 s, turning point V5/6 > 0.05 s) — 1 point each
  - Left atrial dilatation (P mitrale) — 3 points

  → 5 points or more = criteria for left-ventricular hypertrophy
  - Sensitivity: 50–55%; specificity 95–98%



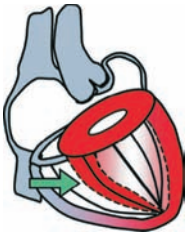
Etiology:
- Arterial hypertension, aortic defects, aortic isthmus stenosis, mitral insufficiency, HOCM, congenital defects (e.g., ductus arteriosis, ventricular septal defect)

Treatment:
- Treatment of the underlying disease

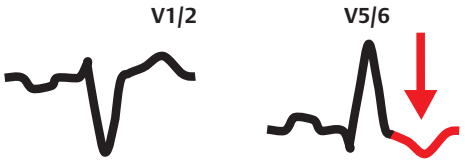
Differential diagnosis:
- Myocardial infarction
- Cardiomyopathy
- Peri(myo)carditis
- Medications

Left Ventricular Hypertrophy

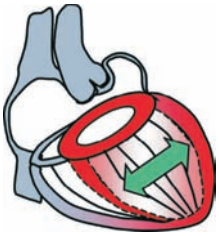
Pressure overload of the left ventricle (so-called resistance hypertrophy or systolic overload):
- High amplitude R waves
- Discordance of the ventricular repolarization (ST depression, T wave inversion in V5–6)



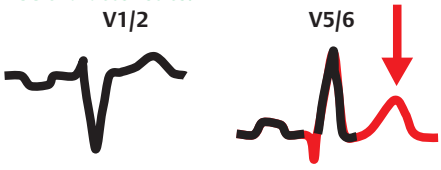
ECG characteristics:



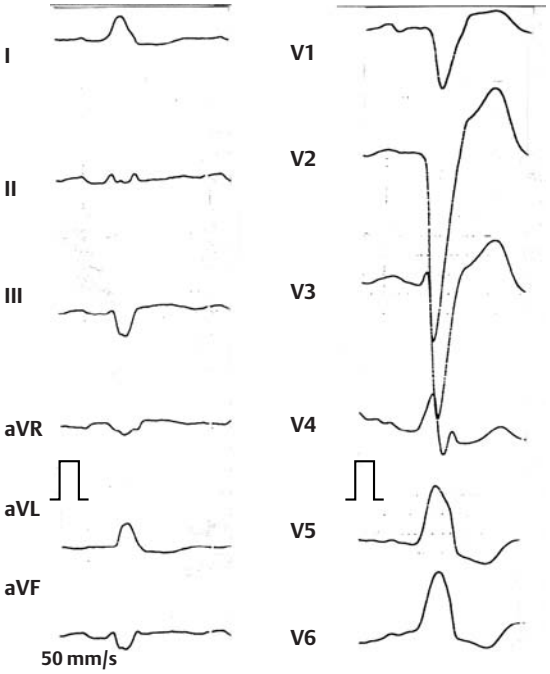
Volume loading of the left ventricle (so-called volume hypertrophy or diastolic overload):
- Prominent Q saw-tooth in I, aVL, V5–6
- Prominent R saw-tooth in V1–2
- Tall T waves in V5–6 ("voluminous T")



ECG characteristics:

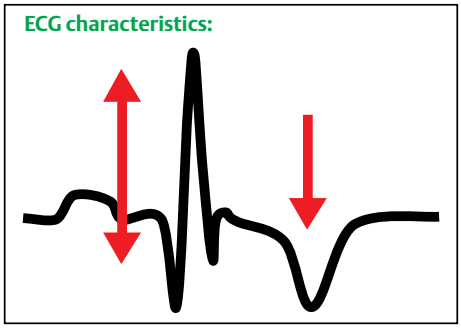


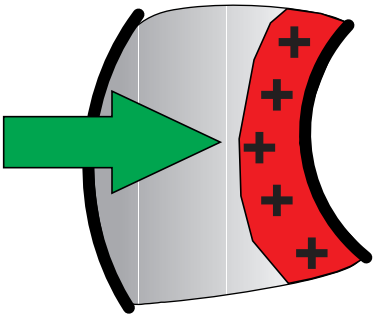
Left Ventricular Hypertrophy in Left Bundle Branch Block



ECG characteristics:
- Left bundle branch block makes ECG diagnosis of left ventricular hypertrophy significantly more difficult
- Indicators are:
  - QRS complex width > 160 ms
  - S in V1/2 + R in V6 > 4.5 mV
  - P wave changes (left atrial dilatation)

Hypertrophic Obstructive Cardiomyopathy





Etiology:
- Genetic defect in chromosome 1, 11, 14, or 15; encoding of pathological myofibrils

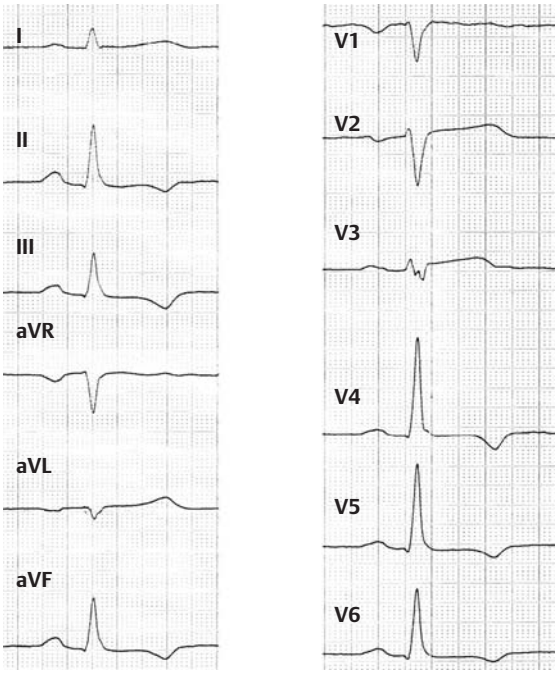
ECG characteristics:
- Often highly positive Sokolow index (see 5.1 Left Ventricular Hypertrophy)
- Marked T inversion in the precordial leads (pseudo-infarction ECG)

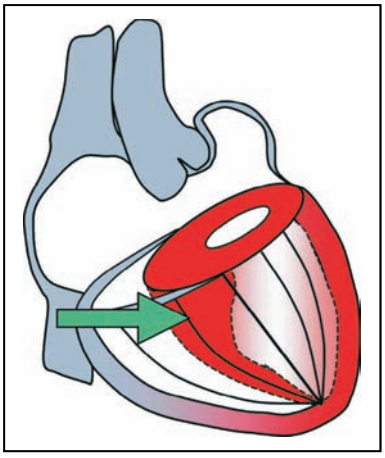
Mechanism:
- Hypertrophy-related relative ischemia of the inner layer causing disturbance of repolarization in this region, flow of current from outer to inner layer

Obstruction:
- LVOT, apical, midseptal
- No obstruction (HNCM)

Treatment:
- Beta-blocker, calcium antagonists (verapamil)
- Pacemaker
- Interventional sclerosis of septal branch (TASH), operative intervention (myectomy)
- Arrhythmia prophylaxis (amiodarone)
- Insertion of debrillator with occurrence of malignant ventricular arrhythmias

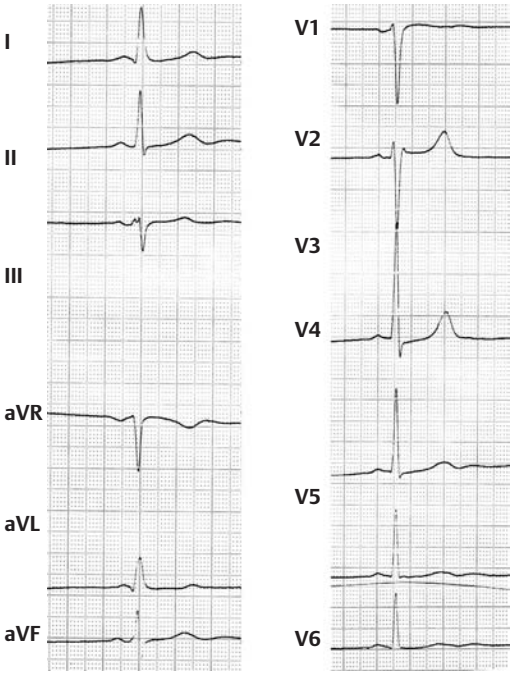
Hypertrophic Obstructive Cardiomyopathy





Differential diagnosis:
- Myocardial infarction
- Left-sided cardiac overload
- Peri(myo)carditis
- Medications (antiarrhythmics)

Mitral Valve Prolapse Syndrome



Definition:
- Symptoms that are attributed to mitral valve anomaly or to neuroendocrine dysfunction, which cannot be solely explained by mitral valve anomaly

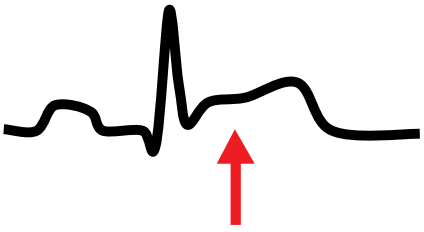
ECG characteristics:
- ST segment depression under stress (up to 40%)
- T wave inversion in II, III, aVF (10–40%)
- Prolongation of QT interval
- Ventricular arrhythmias

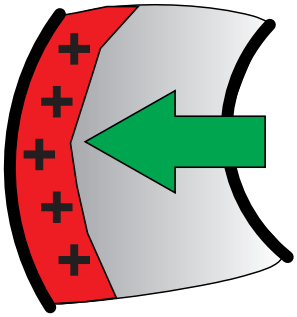
Etiology:
- Several genetic defects (autosomal dominant inheritance postulated)

Treatment:
- Mild MPS: none
- Moderate MPS: reduction of afterload (ACE-inhibitors)
- Severe MPS: valve replacement
- Treatment of the arrhythmias with beta-blocker
- Endocarditis prophylaxis (if prolapse audible on auscultation)

Pericarditis

ECG characteristics:





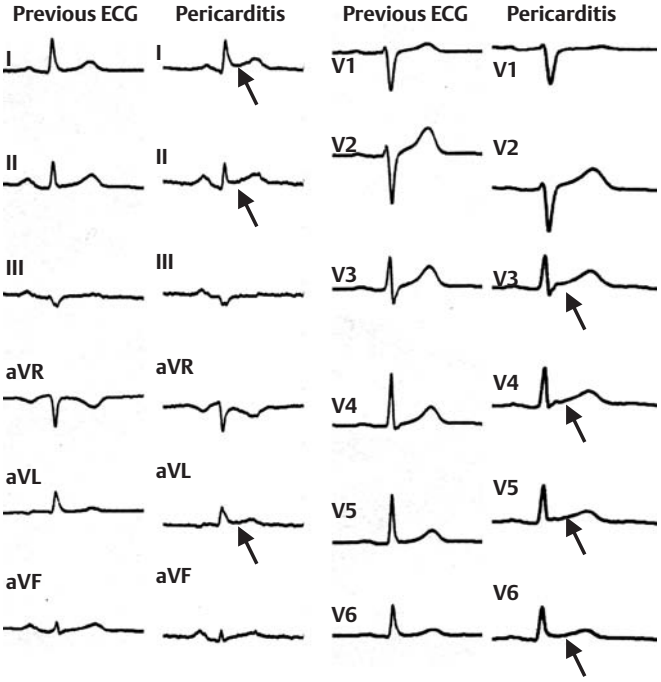
Etiology:
- Inflammatory (viral, bacterial, fungi, TB)
- Post infarct (Dressler syndrome)
- Metabolic
- Systemic disease

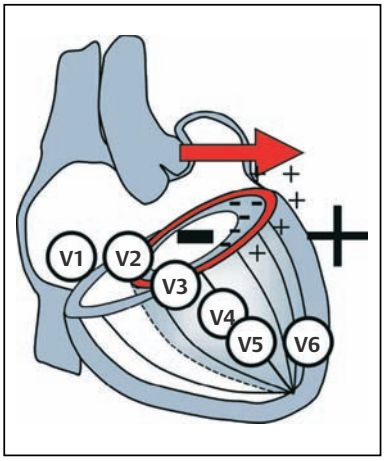
ECG characteristics:
- ST segment elevation with S saw-toothing
- Initially positive T wave, later negative
- Low voltage with pericardial effusion

Mechanism:
- Damaged outer myocardial layer is electropositive compared to the inner layer
- Flow of current from inner to outer layer

Treatment:
- Treatment of the underlying disease
- Anti-inflammatories
- Pericardial punction if effusion compromising hemodynamic stability

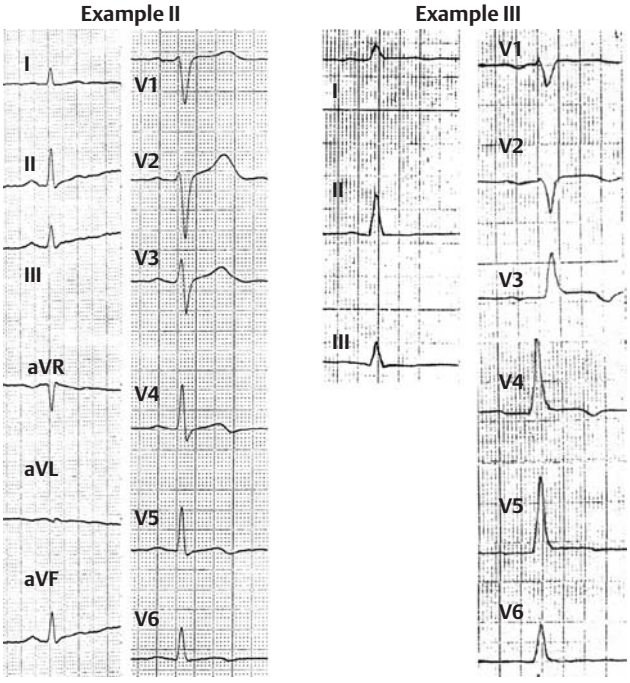
Pericarditis

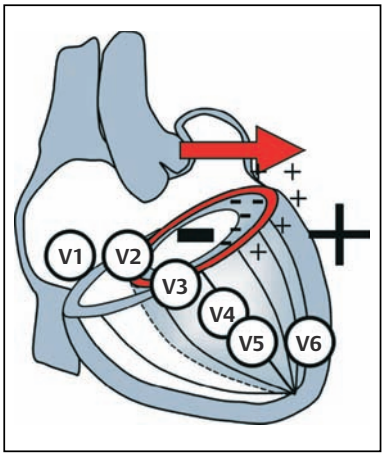




Differential diagnosis:
- Myocardial infarction
- Left-sided cardiac overload
- Embolus
- Vagotonia
- Electrolyte shifts
- Cardiomyopathy

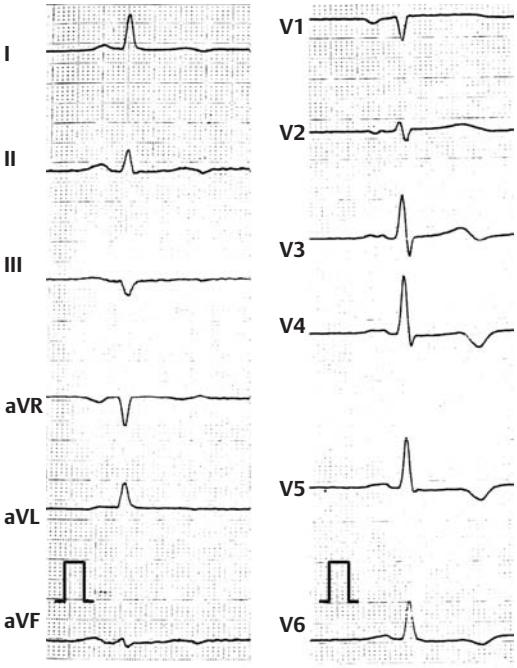
Pericarditis





Differential diagnosis:
- Myocardial infarction
- Left-sided cardiac overload
- Embolus
- Vagotonia
- Electrolyte shifts
- Cardiomyopathy

Myocarditis



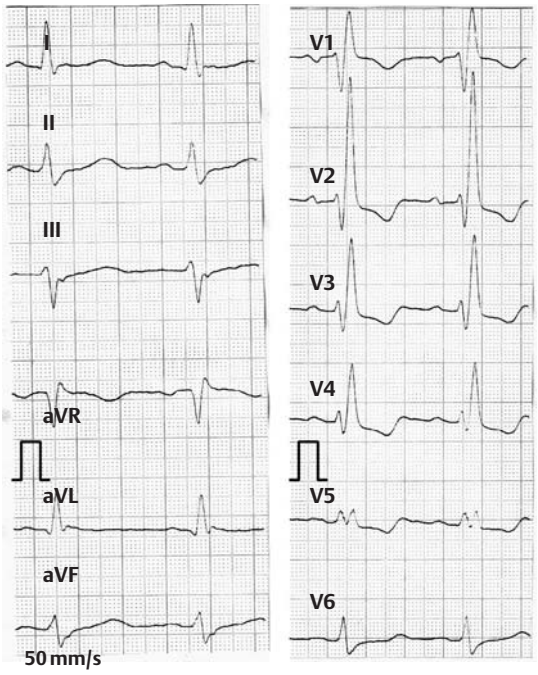
Mechanism:
- Inflammation-related disturbance of repolarization and disturbance of impulse formation and conduction

ECG characteristics:
- Nonspecific repolarization ventricular changes
- Cave: occurrence of supraventricular and ventricular arrhythmias (including ventricular fibrillation) and SA, AV, and bundle branch blocks

Etiology:
- Viral, bacterial, spirochetes (including *Borrelia*), fungi, rickettsia, protozoa
- Rheumatic disease
- Systemic disease

Treatment:
- Treatment of the underlying disease
- Anti-inflammatories
- Rest, monitoring

Right Ventricular Hypertrophy



Mechanism:
- Hypertrophy of the right ventricular musculature as a consequence of systolic or diastolic overload

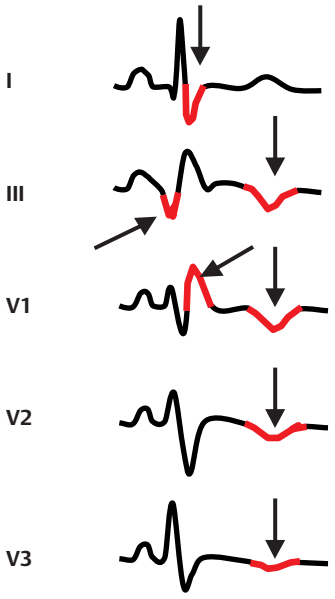
ECG characteristics:
- Often right axis
- P pulmonale (P wave in II, III > 0.3 mV)
- Positive Sokolow–Lyon index (RV1 + SV5 > 1.05 mV)
- Right ventricular repolarization changes
- Right bundle branch block (possible)

Etiology:
- Congenital defects (including ASD, VSD, Fallot, pulmonary stenosis, etc.)
- Cor pulmonale
- Mitral valve defects and left heart failure with pulmonary hypertension

Treatment:
- Treatment of the underlying disease

Acute Pulmonary Embolism

ECG characteristics:



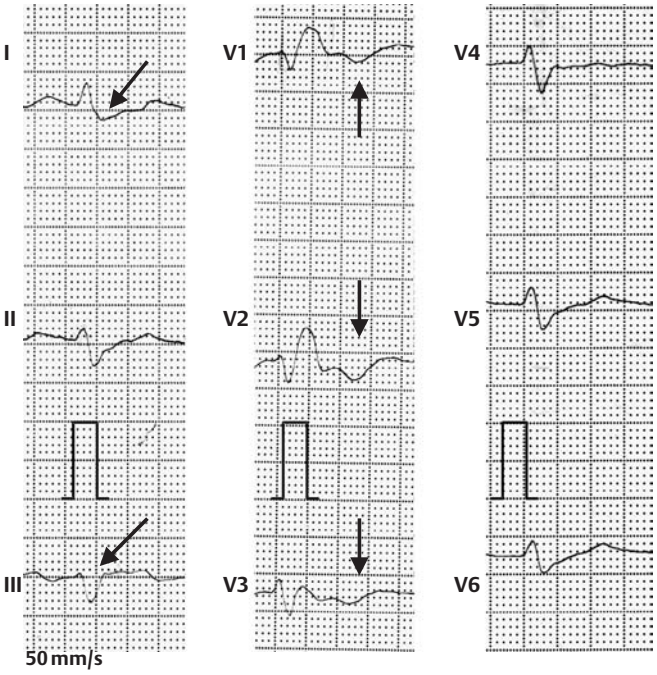
Mechanism:
- Acute systolic overload of the right ventricle as a result of occlusion of one or more arteries in the pulmonary end circulation

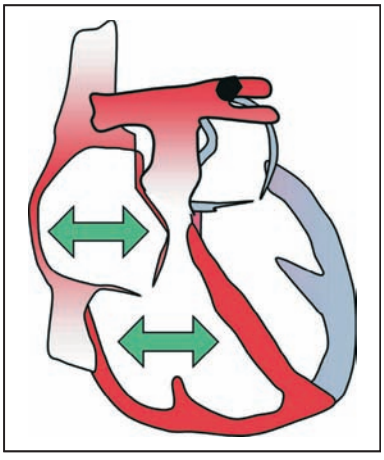
ECG characteristics:
- Often renewed occurrence of right bundle branch block
- SI–qIII type
- S in V5 and V6
- ST elevation and T wave inversion in III, V1–3
- P pulmonale
- Atrial and ventricular arrhythmias

Etiology:
- Embolization of thrombosis in the leg, pelvis, or other venous systems
- Tumor embolization

Treatment:
- Acute treatment of severe embolus (mostly in intensive care)
- Treatment of the underlying disease

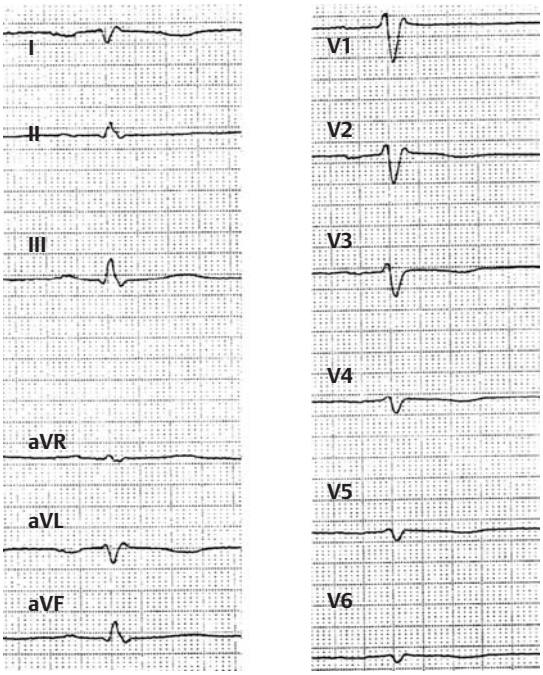
Acute Pulmonary Embolism





Differential diagnosis:
- Posterior myocardial infarction with possible involvement of the right ventricle
- Pericarditis

Dextrocardia



Mechanism:
- Right-sided location of the heart

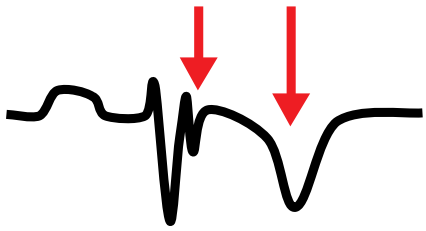
ECG characteristics:
- Low voltage
- Absent R formation in the precordial leads with rS configuration
- Nonspecific apical ventricular changes

Etiology:
- Congenital

Treatment:
- None

Arrhythmogenic Right Ventricular Dysplasia

ECG characteristics:



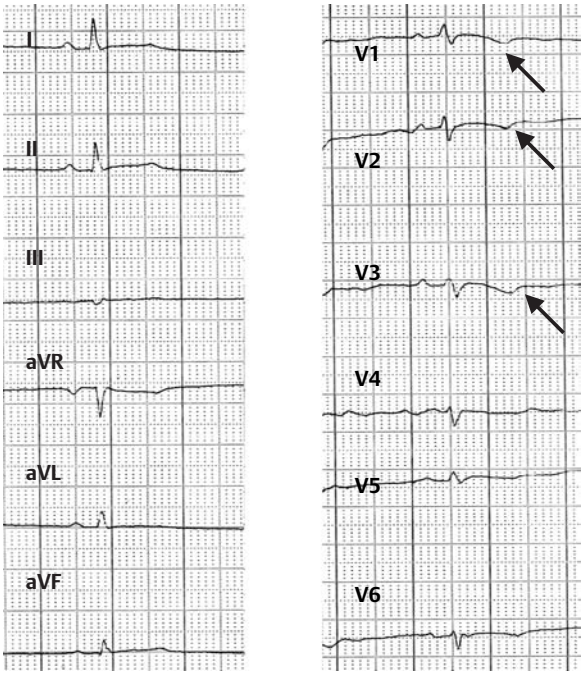
Etiology:
- Unknown (most likely heterogeneous group of genetic defects); additionally viral infection (coxsackie?)

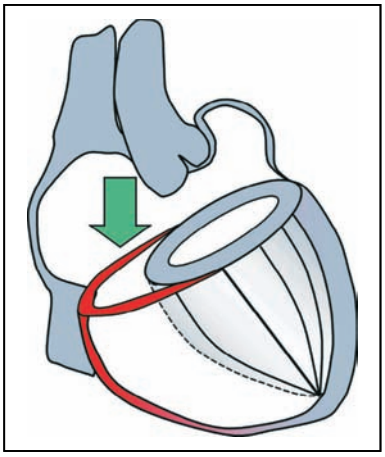
Pathology:
- Substitution of parts of the right ventricular musculature with fibrotic adipose tissue (infundibular, RV apex, or inferior tricuspid area) leads to right ventricular dilatation
- Left ventricle also often affected—transition to DCM

ECG characteristics:
- T wave inversion in the right precordial leads (V1–3)
- Right bundle branch block, epsilon waves (post depolarization at the end of the QRS complex)
- Ventricular tachycardia (left bundle branch block type)

Treatment:
- Debrillation with ventricular arrhythmias, ablation, beta-blocker
- Treatment of cardiac insufficiency
  Monitoring of family members

Arrhythmogenic Right Ventricular Dysplasia

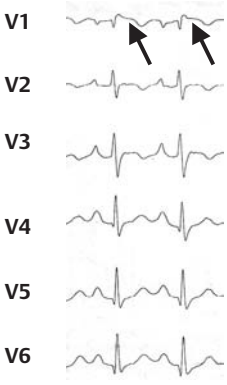




Differential diagnosis:
- Brugada syndrome
- Uhl disease
- Pericarditis
- Right ventricular infarction
- Pulmonary emboli

Brugada Syndrome

ECG characteristics:



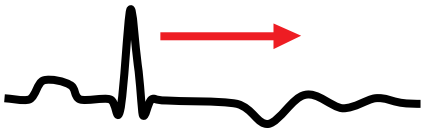
Etiology:
- Genetic defects with up to three mutations in SCN5A (sodium channel) with frequent familial occurrence of ventricular fibrillation

ECG characteristics:
- Three types of repolarisation patterns:
  - Type 1: "tentlike" coved ST-segment elevation displaying a "J-wave" amplitude or elevated ST-segment ≥ 0.2 mV in V1–2 (V3), negative T wave
  - Type 2: "saddle back"-configuration of ST-segment elevation ≥ 0.1 mV, positive T wave
  - Type 3: "saddle back"-configuration of ST-segment elevation ≤ 0.1 mV, positive T wave
- exaggeration or unmasking after drug challenging (ajmaline, flecainide, procainamid)
- Occurrence of malignant ventricular arrhythmias (in particular ventricular fibrillation)

Treatment:
- No specific treatment of the underlying disease
- Debrillator insertion with malignant ventricular arrhythmias (syncope, post cardiac resuscitation)
- Monitoring of family members

QT Syndrome

ECG characteristics:

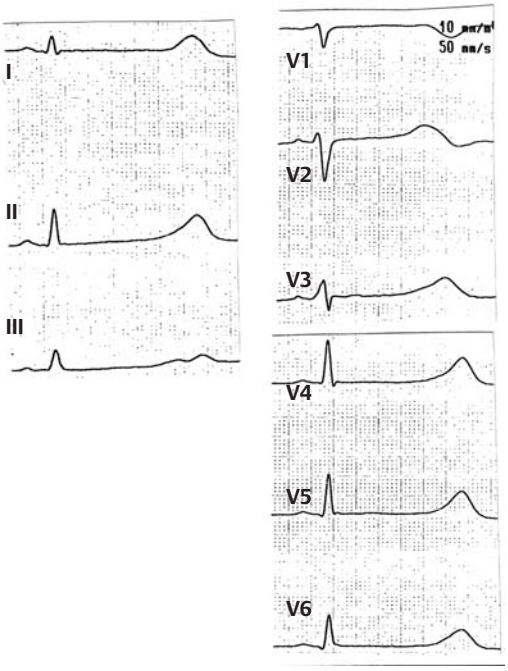


Etiology:
- *Congenital:* Genetic defect of the voltage-dependent potassium or sodium channels (total of five different types)
- *Acquired:* Medication or ischemia

ECG characteristics:
- Prolongation of the QT interval
- Ventricular apical changes
- Occurrence of U waves
- Malignant ventricular arrhythmias (Torsade de pointes)

Treatment:
- Because the trigger of the ventricular arrhythmias is sympathetic activation → beta-blocker sympathetic blockade (stellate ganglion)
- ICD implantation

QT Syndrome

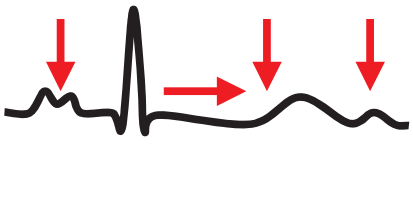


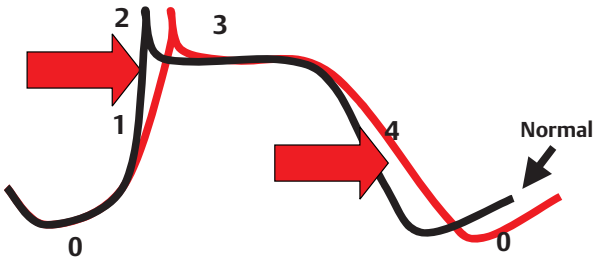
Congenital QT syndromes:
- Romano–Ward syndrome (autosomal dominant, no deafness)
- Jerwell–Lange–Nielsen syndrome (autosomal recessive, deafness)

Acquired QT syndromes:
- Antiarrhythmics
  - Class IA (quinine)
  - Class III (sotalol, amiodarone)
- Phenothiazines
- Tricyclic antidepressants
- Ketaconazole, fluconazole
- Erythromycin
- Nonsedating antihistamines
  - Terfenadine
  - Astimazole
- Combination of the medications listed above!!!
- Ischemia

Class IA Antiarrhythmics (E.g., Quinidine)

ECG characteristics:



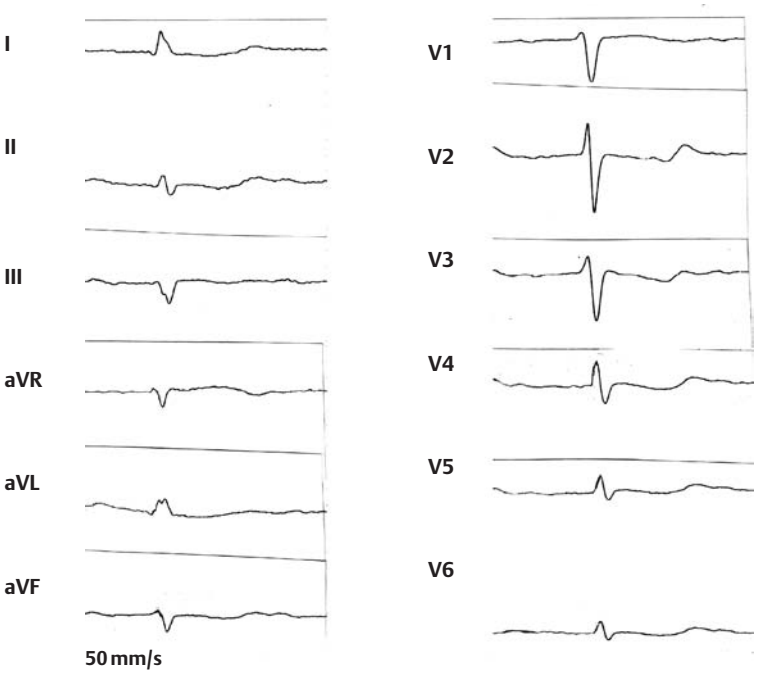


Class IA:
Quinidine, disopyramide, procainamide

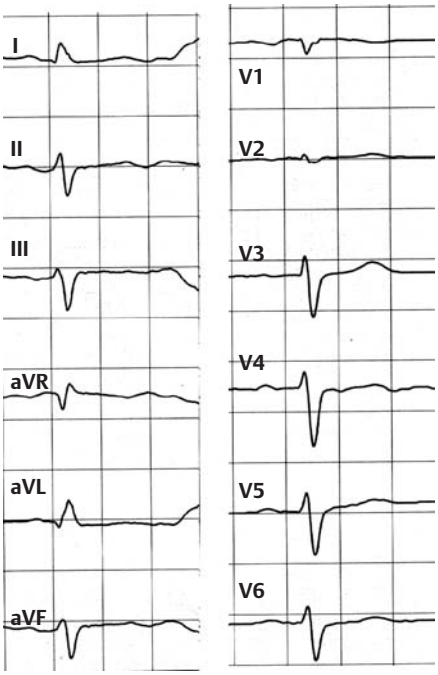
Mechanism of action:
- Blockade of the voltage-dependent sodium channels raising the threshold potential (phase 0), delay of the rise of the slope (phase I, slope flattening), delay of phase IV of the action potential, and anticholinergic effect on the AV node

ECG characteristics:
- Knotting of the P wave
- Shortening of the PT interval
- Widening of the QRS complex
- Depression of the ST segment
- Prolongation of the QT interval

Class IA Antiarrhythmics (E.g., Quinidine)

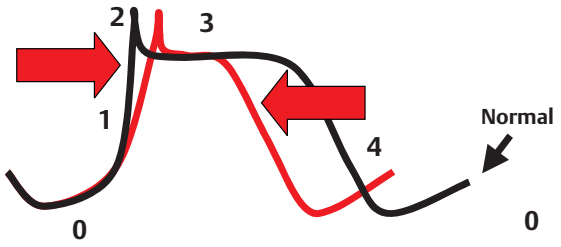


Class IB Antiarrhythmics (E.g., Mexiletine)

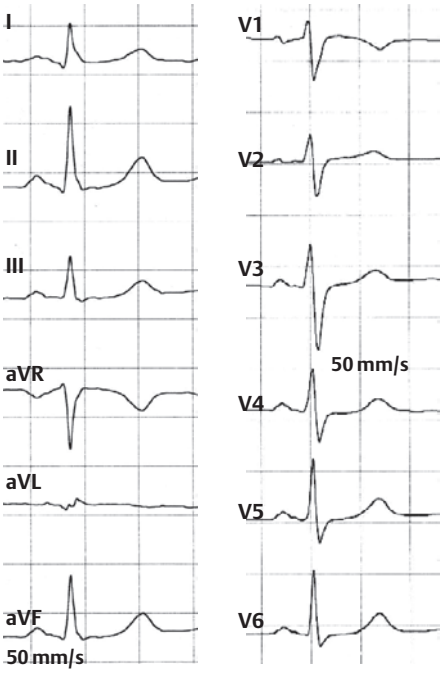


Class IB:
Lidocaine, mexiletine, tocainide
Mechanism of action:
- Blockade of the fast sodium channels
- Shortening of the duration of the action potential
- Reduction in the speed of the rise of the action potential (class I property)

ECG characteristics:
- No typical ECG changes

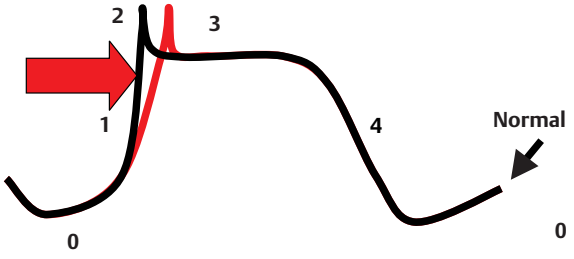


Class IC Antiarrhythmics (E.g., Flecanide)



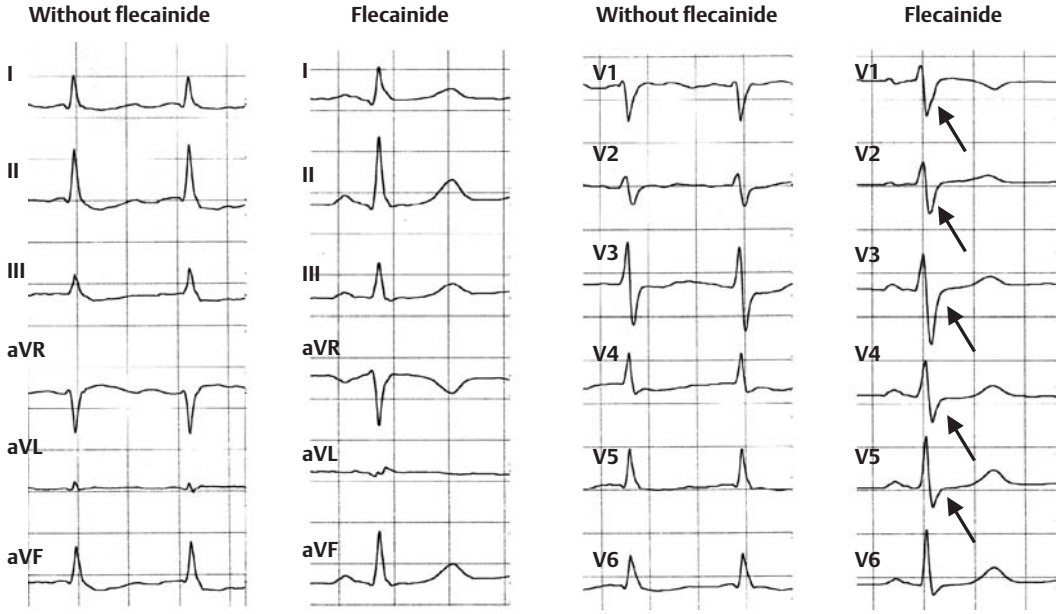
Class I C:
Flecanide, propafenone, ajmaline
Mechanismus of action:
- Blockade of the fast sodium channels causing reduction in the speed of the rise of the action potential (class I property); no changes in the duration of the action potential
- Prolongation of impulse conduction

ECG characteristics:
- Prolongation of the PQ interval
- Widening of the QRS complex
- Only minimal prolongation of the QT interval

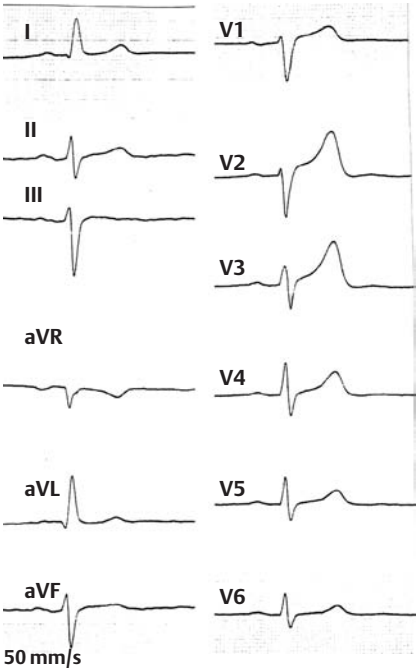


IC Antiarrhythmics (E.g., Flecanide)

Widening of the QRS complex (particularly widening of the S deflection) following oral flecainide (FL), in comparison to the ECG without flecainide; with absolute arrhythmia with atrial fibrillation (oFL), however.



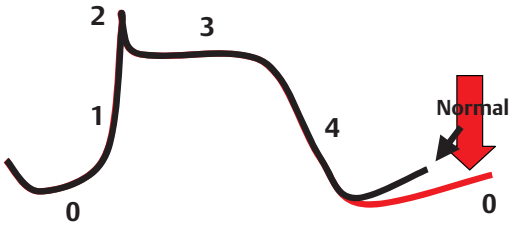
Vagotonia and Class II Antiarrhythmics (E.g., Beta-blockers)



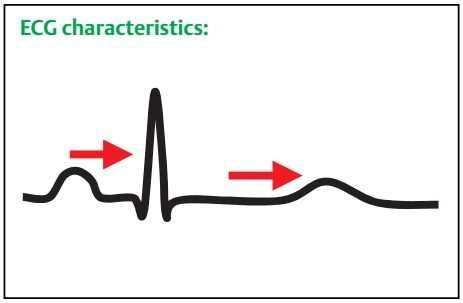
Characteristics:
- Direct cholinergic effect or indirect via blockade of the beta-1 receptor

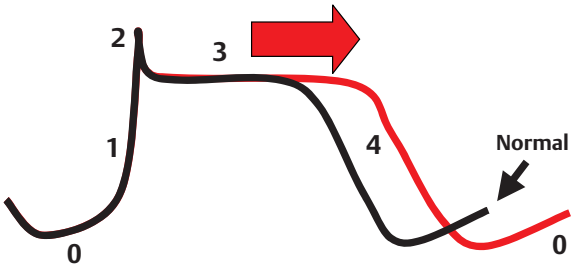
Mechanism of action:
- Acetylcholine activates muscarinic receptors, thereby increasing membrane permeability for potassium and decreasing spontaneous diastolic depolarization (decrease of cardiac frequency and impulse conduction)

ECG characteristics:
- Sinus bradycardia with flat P waves, also 1st degree AV block possible
- Ascending ST elevation and tall, tented T waves



Class III Antiarrhythmics (E.g., Sotalol)





Class III:
Sotalol, amiodarone (azimilide, dofetilide)

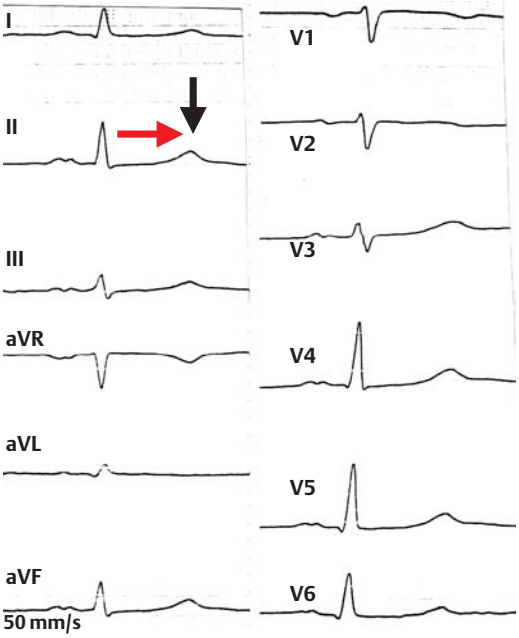
Sotalol characteristics:
- Racemic mixture of D-sotalol (class III antiarrhythmic) and L-sotalol (class II, beta-blocker)

Mechanism of action:
- Prolongation of phase III of the action potential (plateau phase) via action on the potassium channels

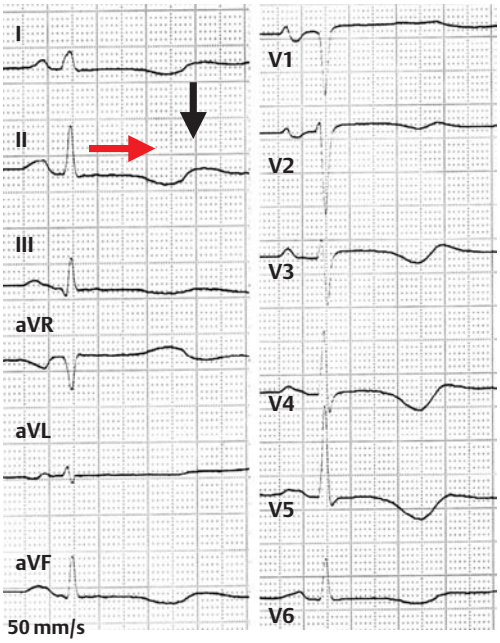
ECG characteristics:
- Sinus bradycardia
- Marked prolongation of the QT interval!!!
- No changes in the QRS complex or the ST segment

Class III Antiarrhythmics (E.g., Sotalol)

Example I

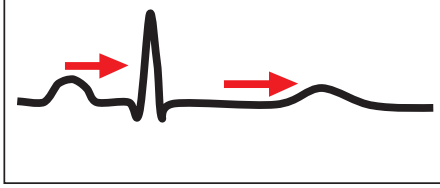


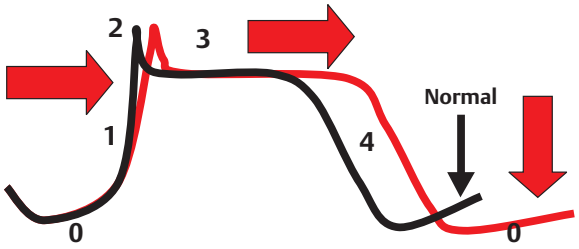
Example II



Class III Antiarrhythmics (E.g., Amiodarone)

ECG characteristics:





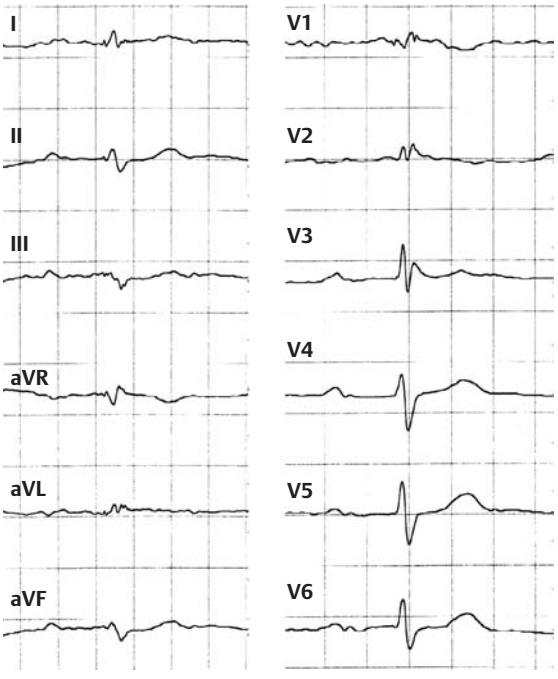
Class III:
Sotalol, amiodarone (azimilide, dofetilide)

Amiodarone characteristics:
- Iodine-containing antiarrhythmic with predominantly class III characteristics

Mechanism of action:
- Influence on the potassium channels—prolongation of phase III (plateau phase)
- Influence on the sodium channels—prolongation of phase I
- Influence on the calcium channels in the sinus and AV nodes—delay of phase 0
- Beta-blocking properties

Electrophysiological characteristics:
- Prolongation of spontaneous diastolic depolarization of the sinus node
- Delay of AV conduction
- Prolongation of the refractory period in the atrial myocardium; not significant in the ventricle
- Raises threshold to defibrillation

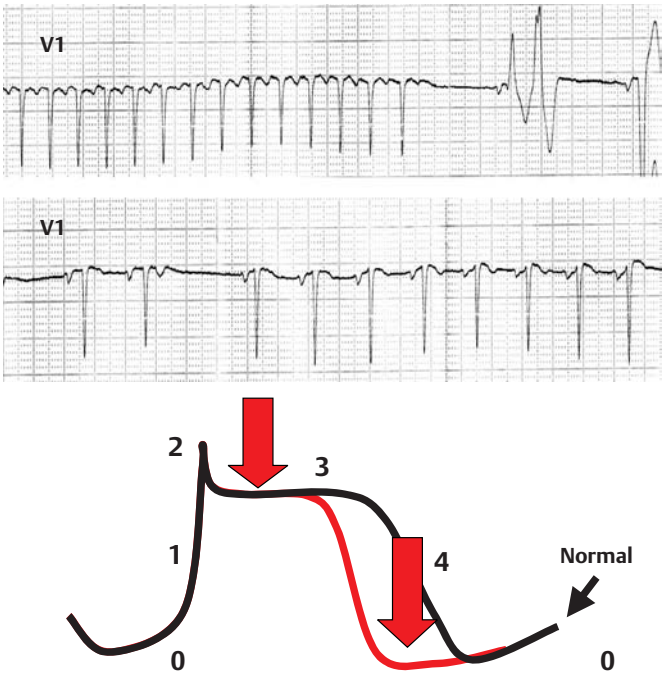
Class III Antiarrhythmics (E.g., Amiodarone)



ECG characteristics:
- Sinus bradycardia
- Prolongation of the PQ interval
- Prolongation of the QT interval
- No relevant changes of the QRS complex or the ST segment

Noncardiac side effects:
- Corneal deposits (reversible)
- Hyperthyroidosis (raised T3 and clinical signs!; serious)
- Hypothyroidosis (hormonal replacement)
- Interstitial pneumonitis (reversible) ranging to pulmonary fibrosis (irreversible)
- Increased photosensitivity, exfoliative dermatitis (frequent, reversible)
- Liver function disorder—rise in transaminases (rare, mostly reversible)
- Insomnia (rare, reversible)
- Neuritis, neuropathies (rare, reversible)

Adenosine



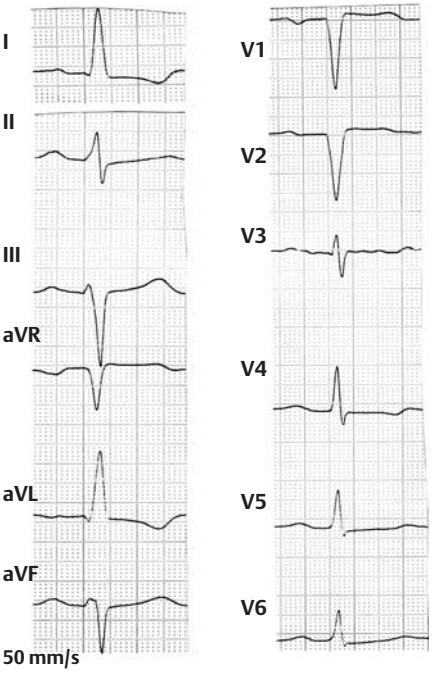
Characteristics:
- Direct effect on the adenosine receptors (A1)
- Hyperpolarization of the cell membrane via effect on the influx of potassium

Mechanism of action:
- Shortening of the action potential (plateau phase)
- Reduction of diastolic depolarization

ECG characteristics:
- Sinus bradycardia following i.v. administration, then blockade of AV conduction

Use:
- Termination of supraventricular reentry tachycardia via intermittent complete blockade of AV conduction

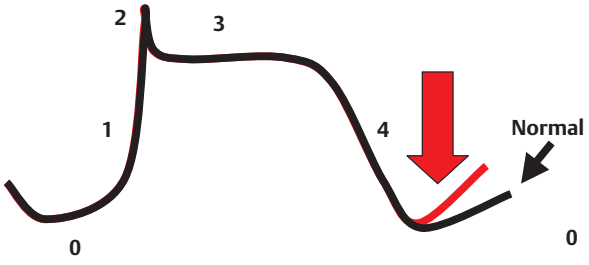
Influence of Sympathetic Tone and of Betamimetics



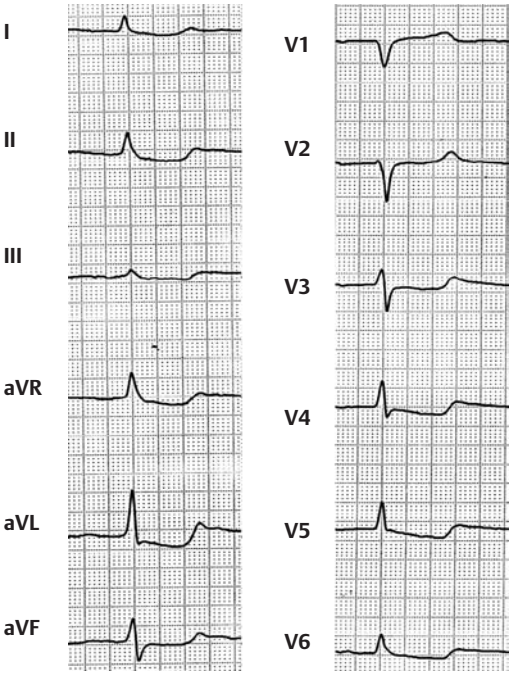
Characteristics:
- Adrenergic effect

Mechanism of action:
- Norepinephrine activates beta-1 receptors, causing reduction of potassium permeability and increases spontaneous diastolic depolarization (increase in cardiac rate and impulse conduction)

ECG characteristics:
- Sinus tachycardia with tall P waves
- Shortened PQ interval
- Increasing ST depression, flattened T waves



Digitalis

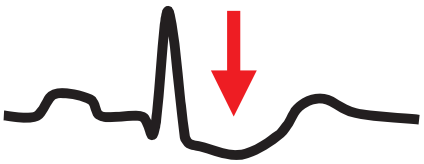


Characteristics:
- Positive inotropic substance

Mechanism of action:
- Blockade of sodium–potassium–ATPase, causing an increase in intracellular calcium
- Among other features, slowing of conduction at the AV node

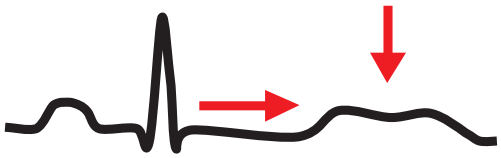
ECG characteristics:
- Trough-shaped ST segment depression

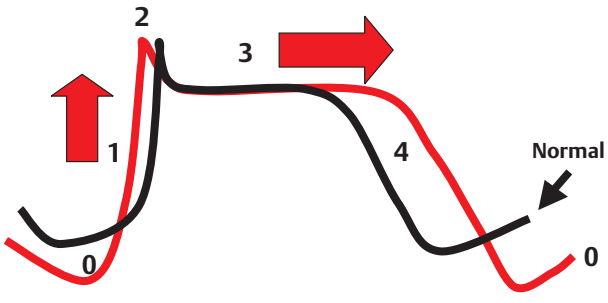
ECG characteristics:



Hypokalemia

ECG characteristics:



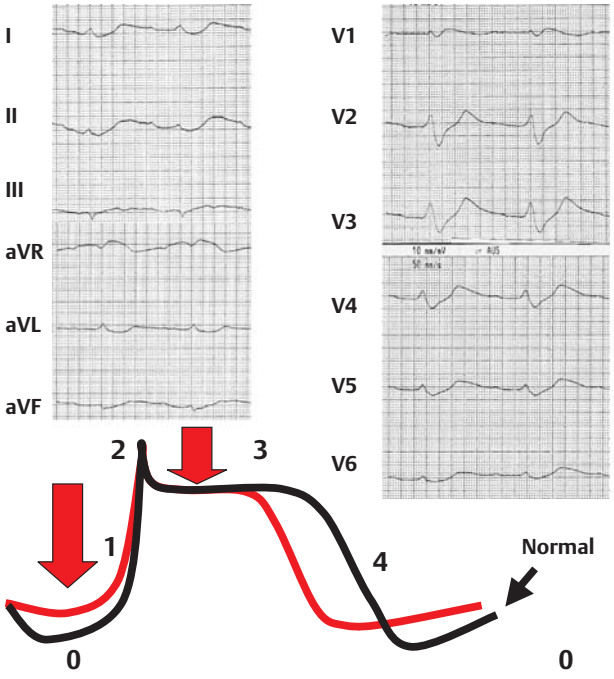


Etiology:
- Chronic laxative abuse, vomiting, diarrhea
- Diuretic treatment, hyperaldosteronism (Conn syndrome, liver cirrhosis, hepatic coma), renal potassium loss
- Diabetic coma
- Idiopathic hypokalemia

Mechanism of action:
- Hyperpolarization of the cell membrane via increase of the intracellular/extracellular gradient
- Acceleration of phase 0
- Prolongation of phase 3
- Increase in the speed of electrical conduction

ECG characteristics:
- No changes in the QRS complex
- Depression of the ST segment
- Prolongation of the QT interval
- TU amalgamation

Hyperkalemia



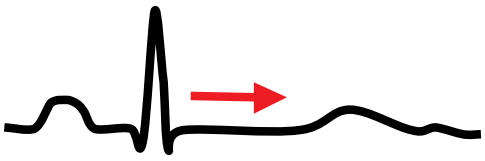
Etiology:
- Renal insufficiency, Addison disease
- Acidosis, hemolysis
- Potassium infusion
- Potassium-sparing diuretics

Mechanism of action:
- Hypopolarization of the cell membrane via decrease of the intracellular/extracellular gradient
- Resting depolarization of the cells with slowing of phase 0 to a degree
- Shortening of phase 3
- Decrease in the speed of electrical conduction

ECG characteristics:
- Flattening of the P wave up to its disappearance (isoelectr. p)
- Widening of the QRS complex
- Elevation of the ST segment
- Shortening of the QT interval (ranging to amalgamation of QRS and T)

Hypocalcelmia/Hypercalcemia

ECG characteristics:

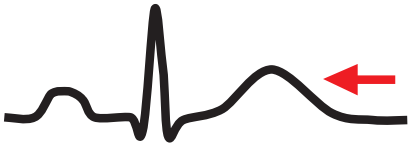


Hypocalcemia – Etiology:
- Gastrointestinal losses (including sprue, vomiting, diarrhea), hypoparathyroidism, pharyngitis, uremia, spasmophilia, hepatic coma

Mechanism of action:
- Prolongation of phase 2

ECG characteristics:
- Prolongation of the QT interval (ranging to T wave flattening)

ECG characteristics:



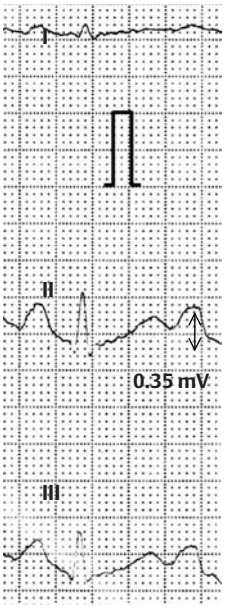
Hypercalcemia – Etiology:
- Osteolysis in tumor disease, vitamin D poisoning

Mechanism of action:
- Shortening of phase 2

ECG characteristics:
- Shortening of the QT interval (ranging to T wave flattening)

Morphological Changes of the P Wave (I)

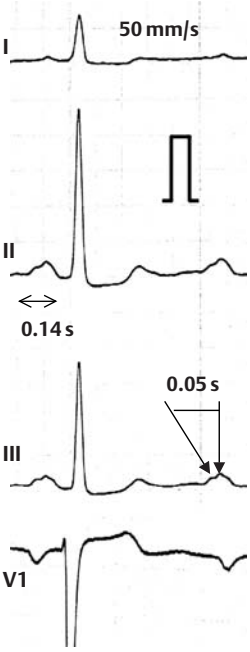
P pulmonale



Mechanism:
Systolic or diastolic overload of the right atrium resulting in hypertrophy

ECG characteristics:
- P in II, III ≥ 0.3 mV

P mitrale

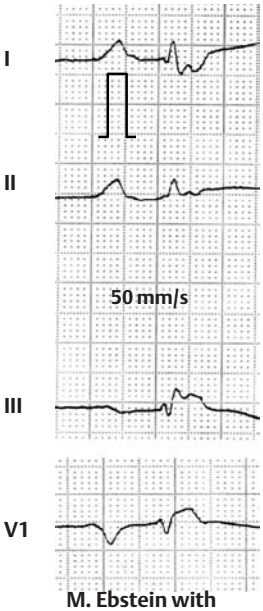


Mechanism:
Systolic or diastolic overload of the left atrium resulting in hypertrophy

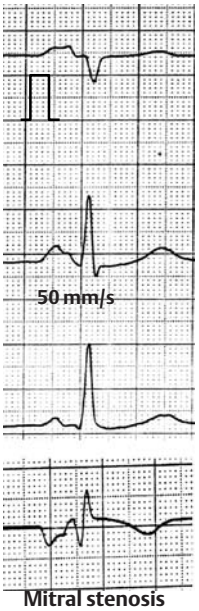
ECG characteristics:
- P in I, II > 0.11 s
- Bifid I,II
- Distance between peaks > 0.03 s
- Deep negative deflection in V1 > –0.15 mV

Morphological Changes of the P Wave (II)

P biatriale



M. Ebstein with atrial septal defect



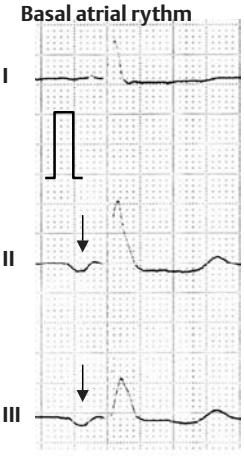
Mitral stenosis

Mechanism:
Systolic or diastolic overload of the right atrium resulting in hypertrophy

ECG characteristics:
- P in II, III > 0.3 mV
- P > 0.11 s
- Bifid
- Negative deflection in V1

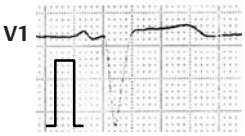
Morphological Changes of the P Wave (III)

Basal atrial rythm

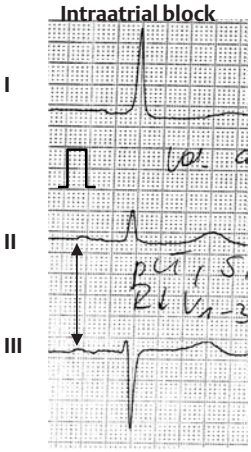


Mechanism:
Excitation of the atria by an ectopic focus (mostly near to the coronary sinus)

ECG characteristics:
- Negative P in II, III
- And aVR
- Often shortened PQ interval

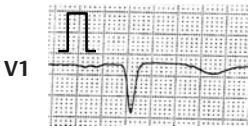


Intraatrial block



Mechanism:
Disturbed and delayed impulse conduction from the right to the left atrium

ECG characteristics:
- Bifid P
- P > 0.11 s
- However, not deep in V1



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