

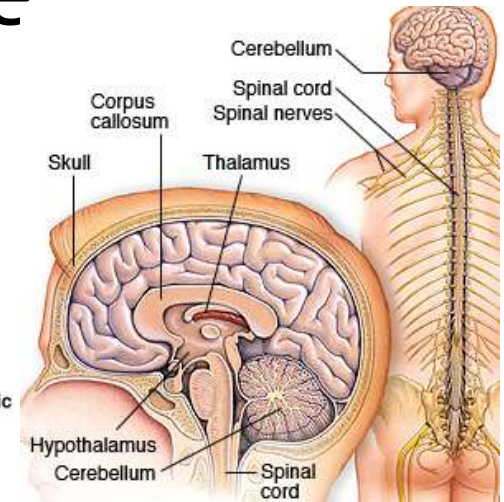
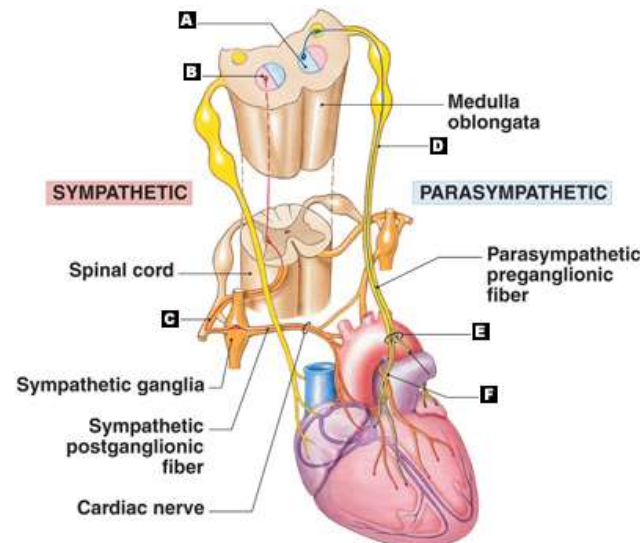
HEART ARRHYTHMIAS

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

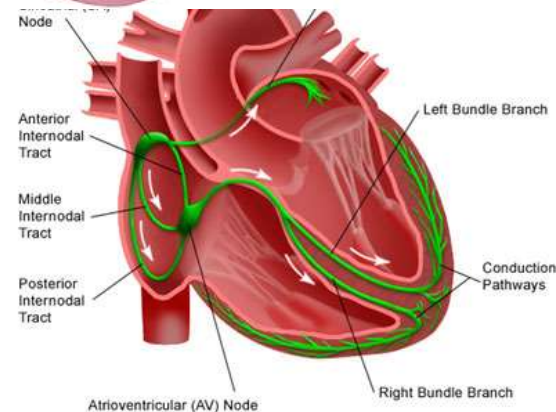
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Plan of the Lecture

- Definition
- Epidemiology
- Risk factors
- Etiology
- Mechanisms
- Classification
- Clinical investigation
- Diagnosis
- Treatment
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic and treatment guidelines



the Heart



Definition

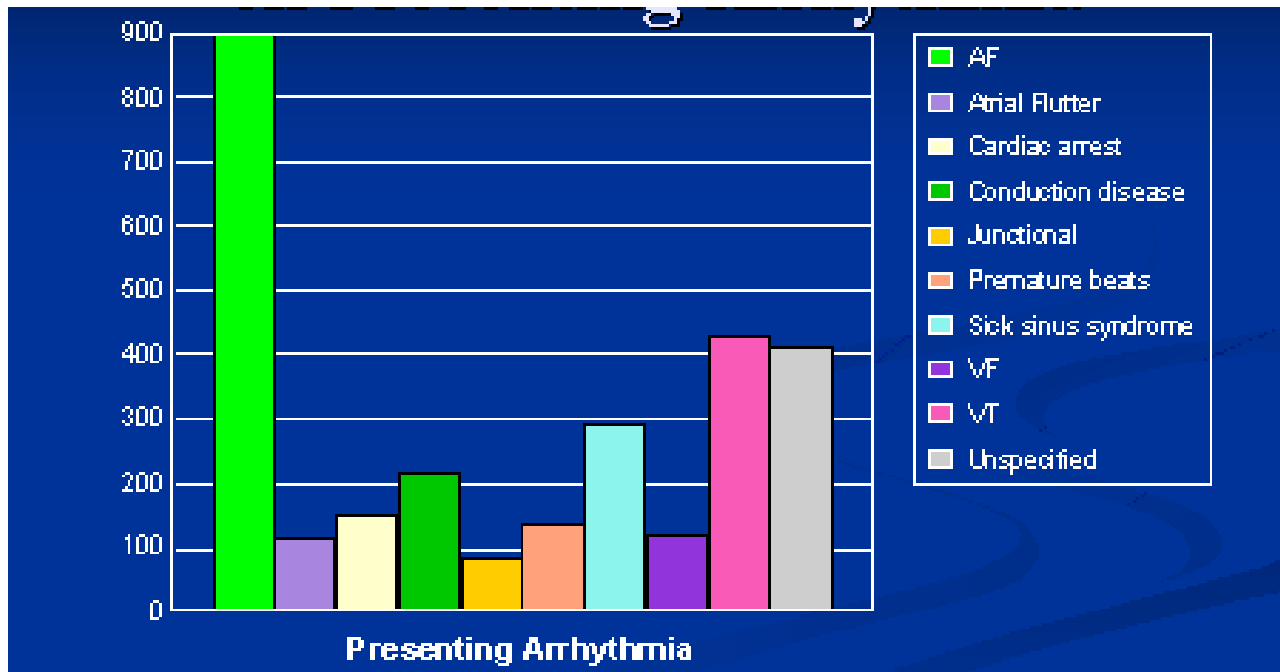
Heart arrhythmias (heart rhythm disturbances, cardiac arrhythmia, cardiac dysrhythmia, irregular heartbeat) is a group of conditions with abnormal variation from the normal heartbeat (irregular, too fast, or too slow) via congenital (e.g., accessory atrioventricular connection, hereditary ion channelopathies) or acquired abnormalities of structure or function of heart and its conduction system or/and systemic abnormalities (electrolyte deviations, hypoxia, neuro and/or humoral (e.g. hormonal) imbalances (hypothyroidism, hyperthyroidism), chronic distress), and drugs and toxins (e.g., alcohol, caffeine).

Epidemiology

- Heart arrhythmias are relatively common, often repetitive, occasionally persistent, and rarely life threatening .
- The precipitants of heart arrhythmias vary with age (most serious arrhythmias affect people older than 60), gender, and associated comorbidity.
- While heart arrhythmias are a frequent cause of emergency room and primary care physician visits, they are infrequently the primary reason for hospital admission.
- A recent study has suggested that 1 in 4 adult Americans over the age of 40 could develop an irregular heartbeat.
- Certain types of heart arrhythmias can cause sudden cardiac death (100,000 people in the UK every year).

Epidemiology

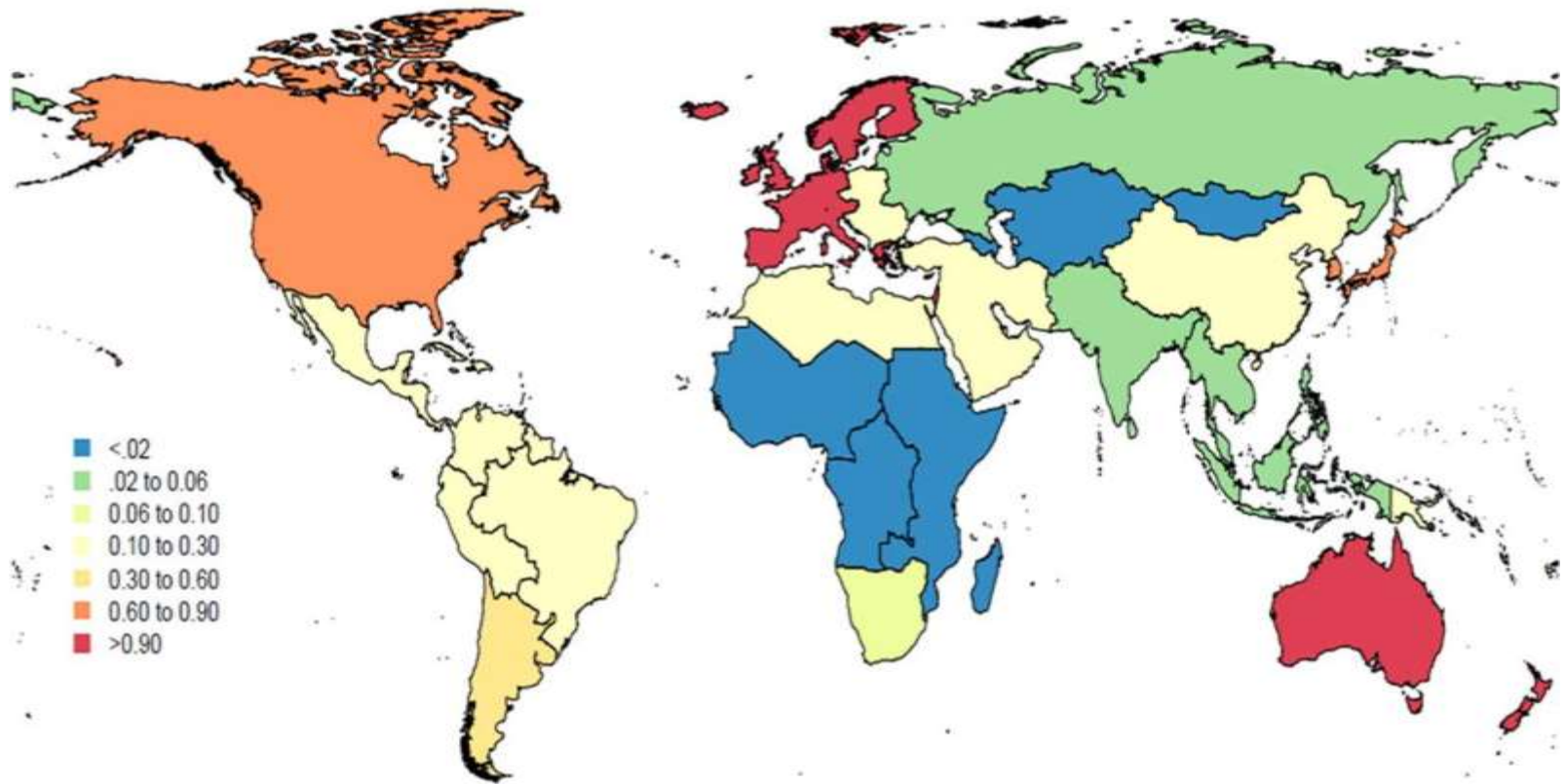
(Total Hospitalization Days)



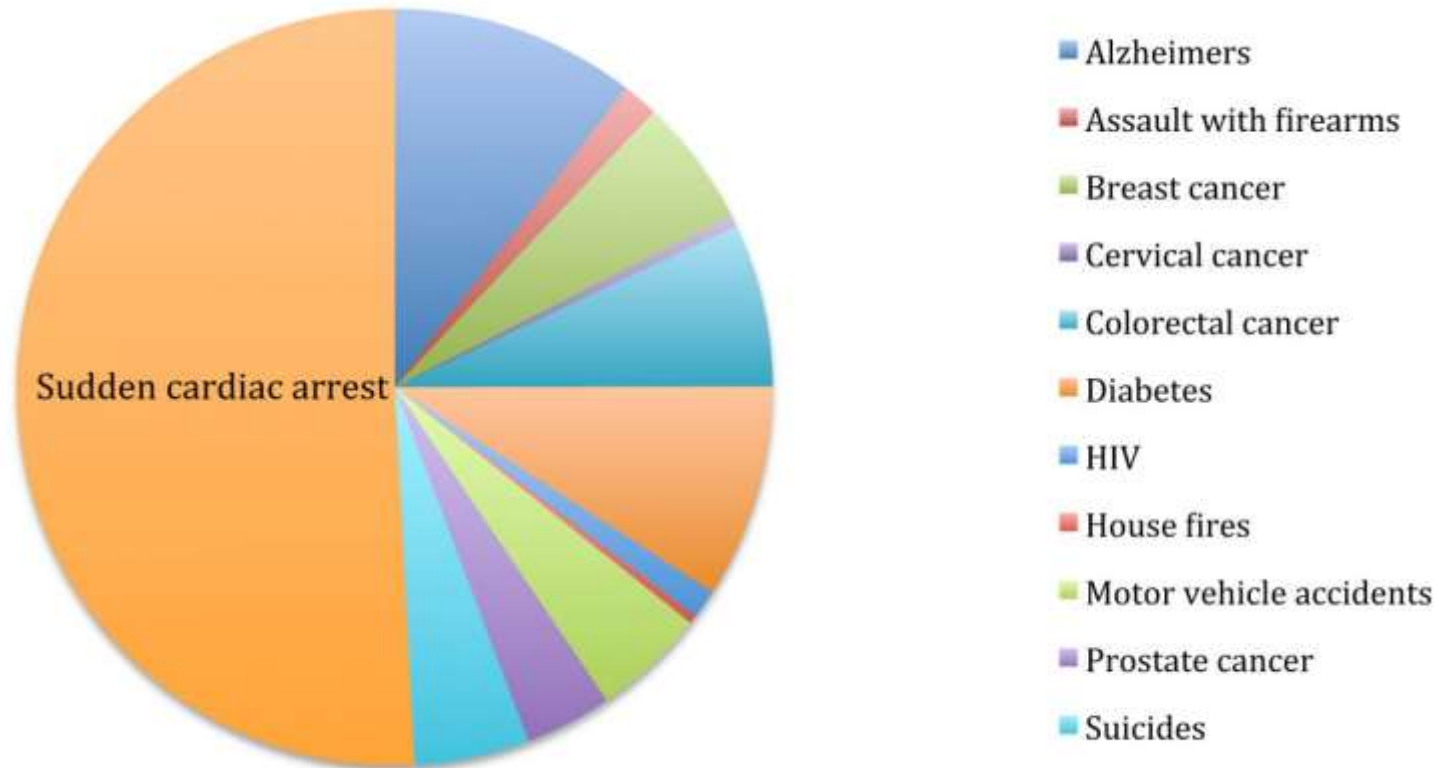
AF – atrial fibrillation, VF – ventricular fibrillation, VT – ventricular tachycardia

Epidemiology

(Worldwide Distribution of Atrial Fibrillation)



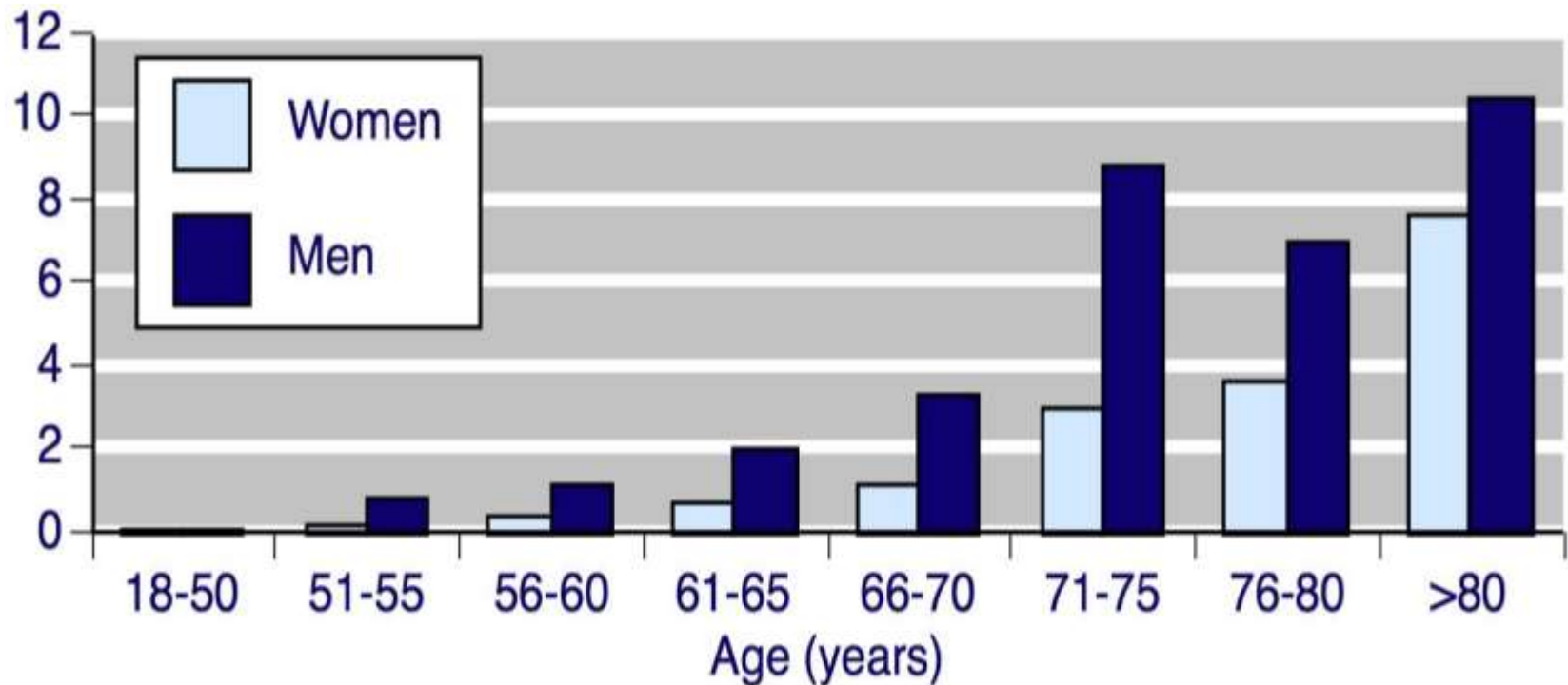
Epidemiology (Sudden Cardiac Arrest)



Sudden cardiac arrest (SCA) is a leading cause of death among adults over the age of 40 in the United States and other countries.

Epidemiology

(Sudden Cardiac Death, per 1,000 person years)



Risk Factors and Etiology

- Alcohol abuse
- Diabetes
- Drug abuse
- Excessive coffee consumption
- Heart_disease
- Hypertension
- Hyperthyroidism (an overactive thyroid gland)
- Mental stress
- Scarring of the heart, often the result of a heart attack
- Smoking
- Some dietary supplements
- Some herbal treatments
- Some medications
- Etc.

Risk Factors and Etiology

- Functional violations (physical and emotional stress, fever heat, increased intracranial pressure, respiratory disturbances, etc.).
- Changed structure of the heart (coronary artery disease, cardiomyopathy, valvular heart diseases, myocarditis, conductive tissue disease, etc.).
- Systemic diseases (diabetes, lupus erythematosus, etc.).
- Toxic injuries of the myocardium (alcohol, caffeine, tobacco, drugs, some medications (adrenalin, noradrenalin, glucocorticoids, etc.), bacterial toxins, phosphor organic substances, etc.).
- Nervous system disorder.
- Hormone balance disorder (hyperthyroidism, hypothyroidism, etc.).
- Violation of intracellular or extracellular ions balance (sodium, potassium, calcium, magnesium and chlorine).
- Direct mechanical influences on the heart (catheter intervention, surgery, chest trauma).

Mechanisms

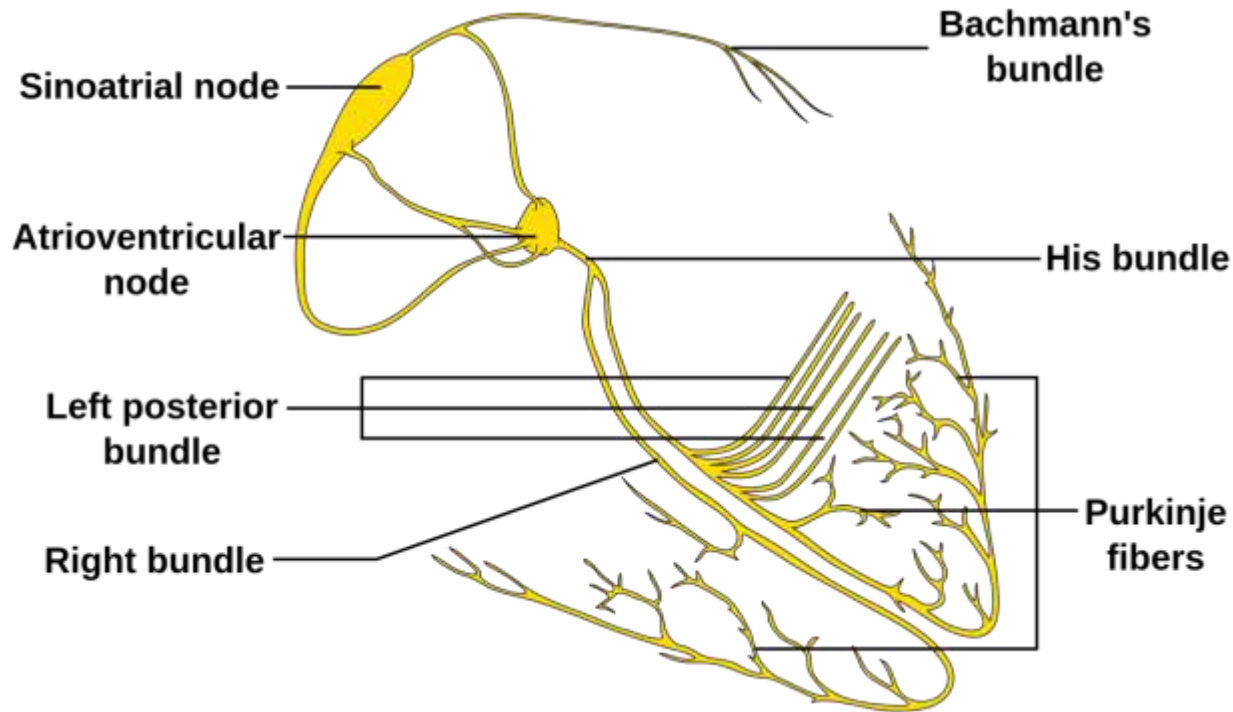
- There are four basic mechanisms— conduction violations, enhanced or suppressed automaticity, triggered activity, or re-entry.
- Conduction violation can result in sinus, atrium, atrium-ventricular and ventricle block; and in pre-excitations (Wolf-Parkinson-White) syndrome.
- Suppression of automaticity of the sinoatrial (SA) node can result in sinus node dysfunction, and sick sinus syndrome (SSS), which is still the most common indication for permanent pacemaker implantation
- Enhanced automaticity can result in multiple arrhythmias, both atrial and ventricular.
- Triggered activity occurs when early afterdepolarizations and delayed afterdepolarizations initiate spontaneous multiple depolarizations, precipitating ventricular arrhythmias.
- The most common mechanism of arrhythmogenesis results from reentry (micro- and macro-); requisites for reentry include bidirectional conduction and unidirectional block.

Mechanisms

(Bradyarrhythmias and Tachyarrhythmias)

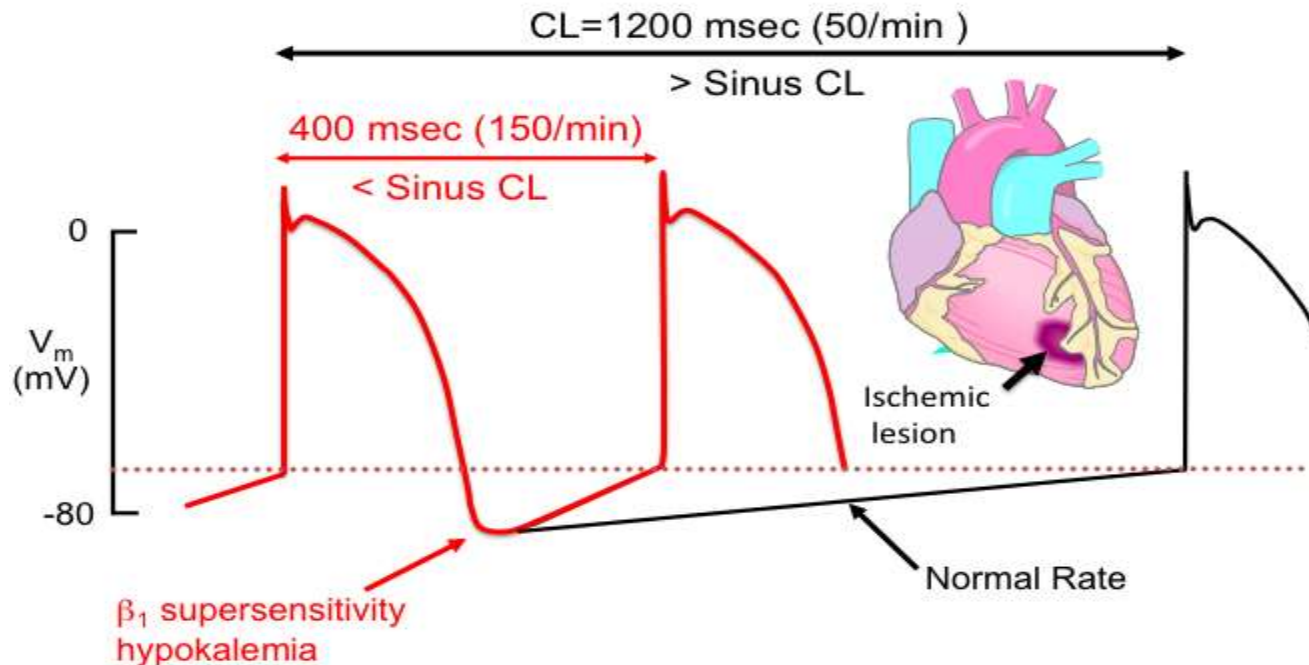
- Bradyarrhythmias typically arise from disturbances in impulse formation at the level of the sinoatrial node (SA) or from disturbances in impulse propagation at any level, including exit block from the SA, conduction block in the atrioventricular node (AVN) and impaired conduction in the His-Purkinje system.
- Tachyarrhythmias can be classified according to mechanism, including enhanced automaticity (spontaneous depolarization of atrial, junctional, or ventricular pacemakers), triggered arrhythmias (initiated by afterdepolarizations occurring during or immediately after cardiac repolarization, during phase 3 or 4 of the action potential), or reentry (circus propagation of a depolarizing wavefront).

Mechanisms (Conduction Block)



A block can be found in every part of the conduction system of the heart.

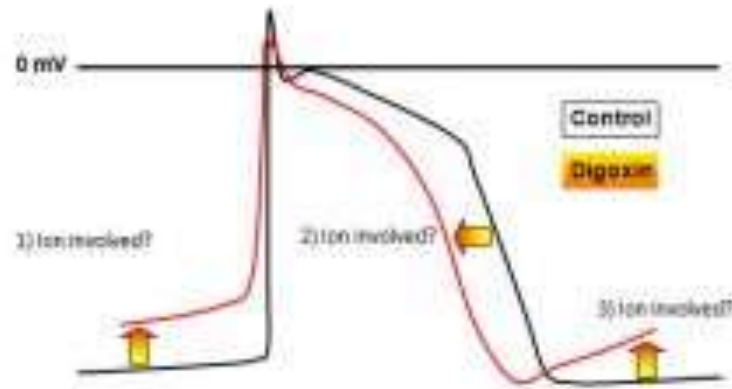
Mechanisms (Enhanced Automaticity)



Lowered action potential makes SA node more irritable; makes arrhythmias more likely - Increased intracellular calcium causes earlier phase 4 upstroke.

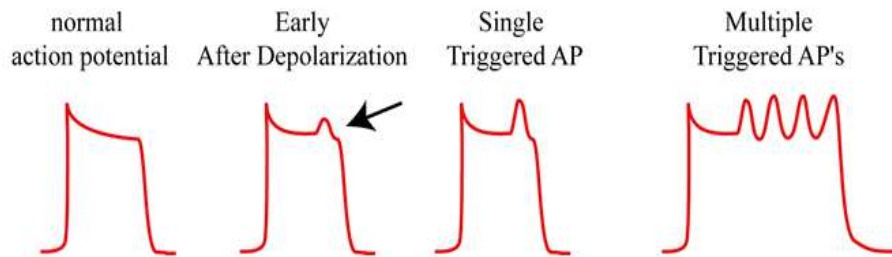
Mechanisms (Suppressed Automaticity)

Effects of Digoxin on Cardiac Action Potential

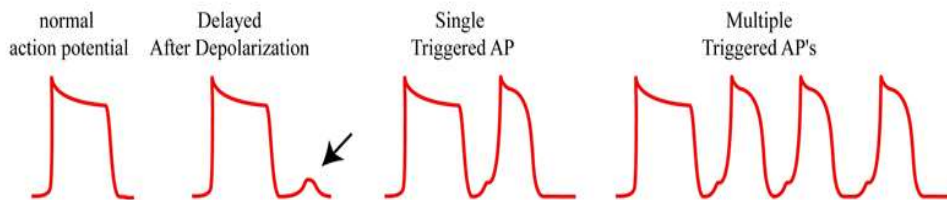


Lowered action potential due to digoxin makes sinoatrial node (SA node) more irritable; makes arrhythmias more likely - Increased intracellular calcium causes earlier phase 4 upstroke.

Mechanisms (Triggered Activity)



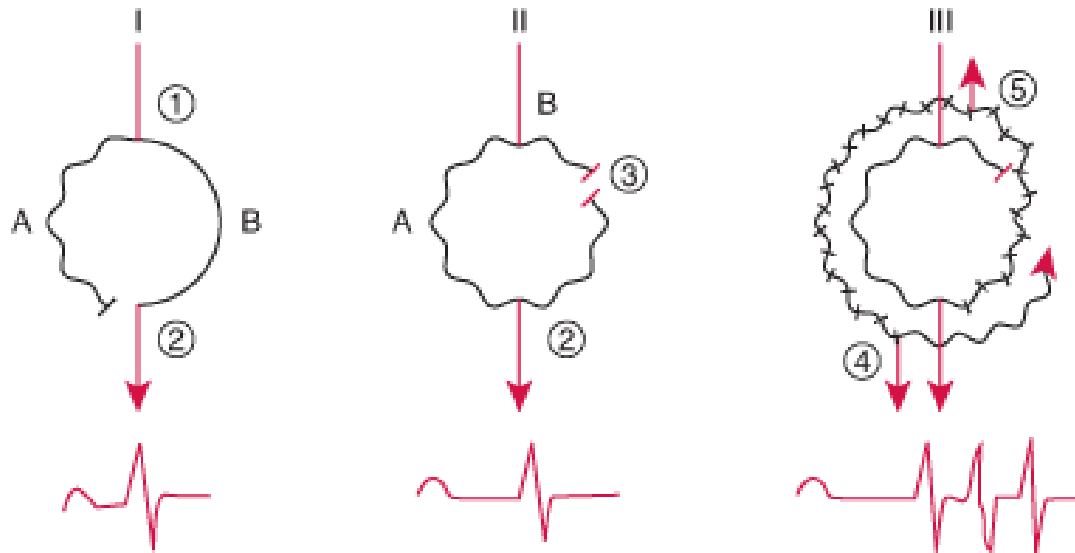
Early After Depolarization: Sometimes, during the plateau phase of the action potential, a spontaneous depolarization may occur. This is often the case when there is too much calcium in the cell. These depolarizations may reach threshold and induce, too soon, a new action potential.



Delayed After Depolarization: This is similar to the early after depolarizations but these occur after full repolarization has taken place, hence their name, delayed (or late) after depolarizations.

Mechanisms

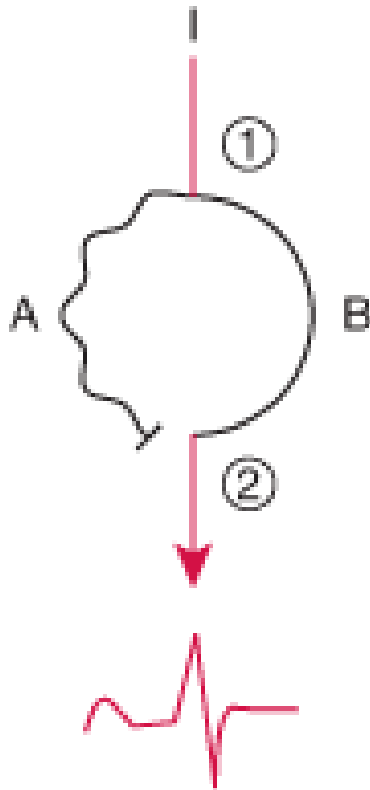
(Reentry: 1)



Atrioventricular nodal reentry is used as an example. Two pathways connect the same points. Pathway A has slower conduction and a shorter refractory period. Pathway B conducts normally and has a longer refractory period.

Mechanisms

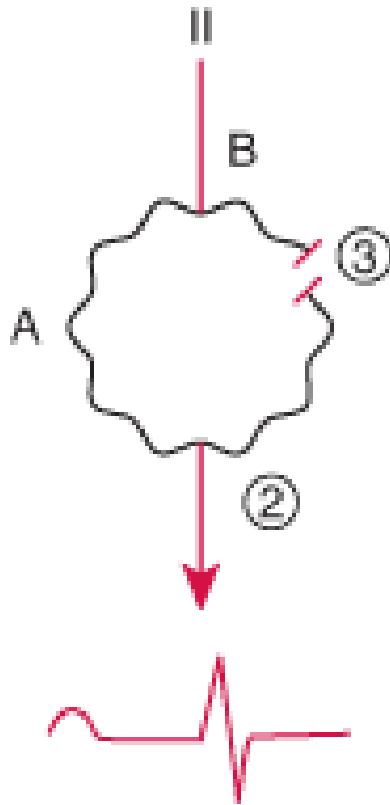
(Reentry: 2)



I. A normal impulse arriving at 1 goes down both A and B pathways. Conduction through pathway A is slower and finds tissue at 2 already depolarized and thus refractory. A normal sinus beat results.

Mechanisms

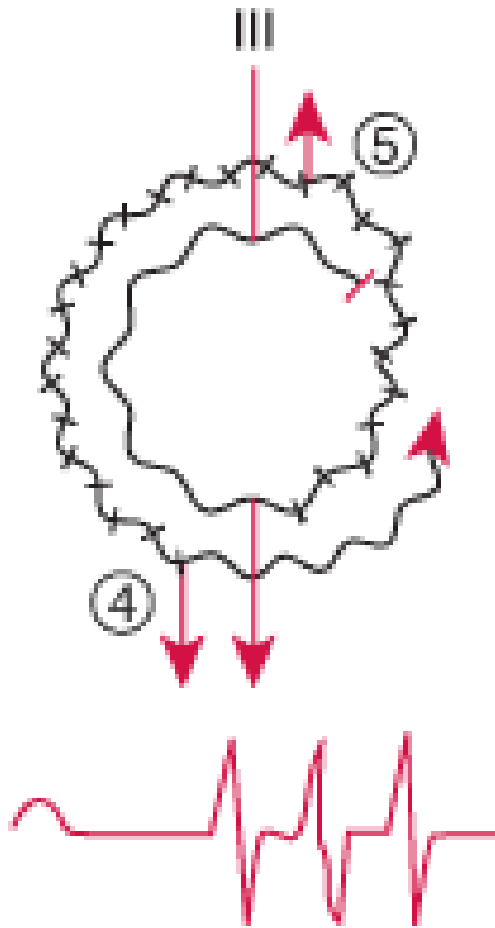
(Reentry: 3)



II. A premature impulse finds pathway B refractory and is blocked, but it can be conducted on pathway A because its refractory period is shorter. On arriving at 2, the impulse continues forward and retrograde up pathway B, where it is blocked by refractory tissue at 3. A premature supraventricular beat with an increased PR interval results.

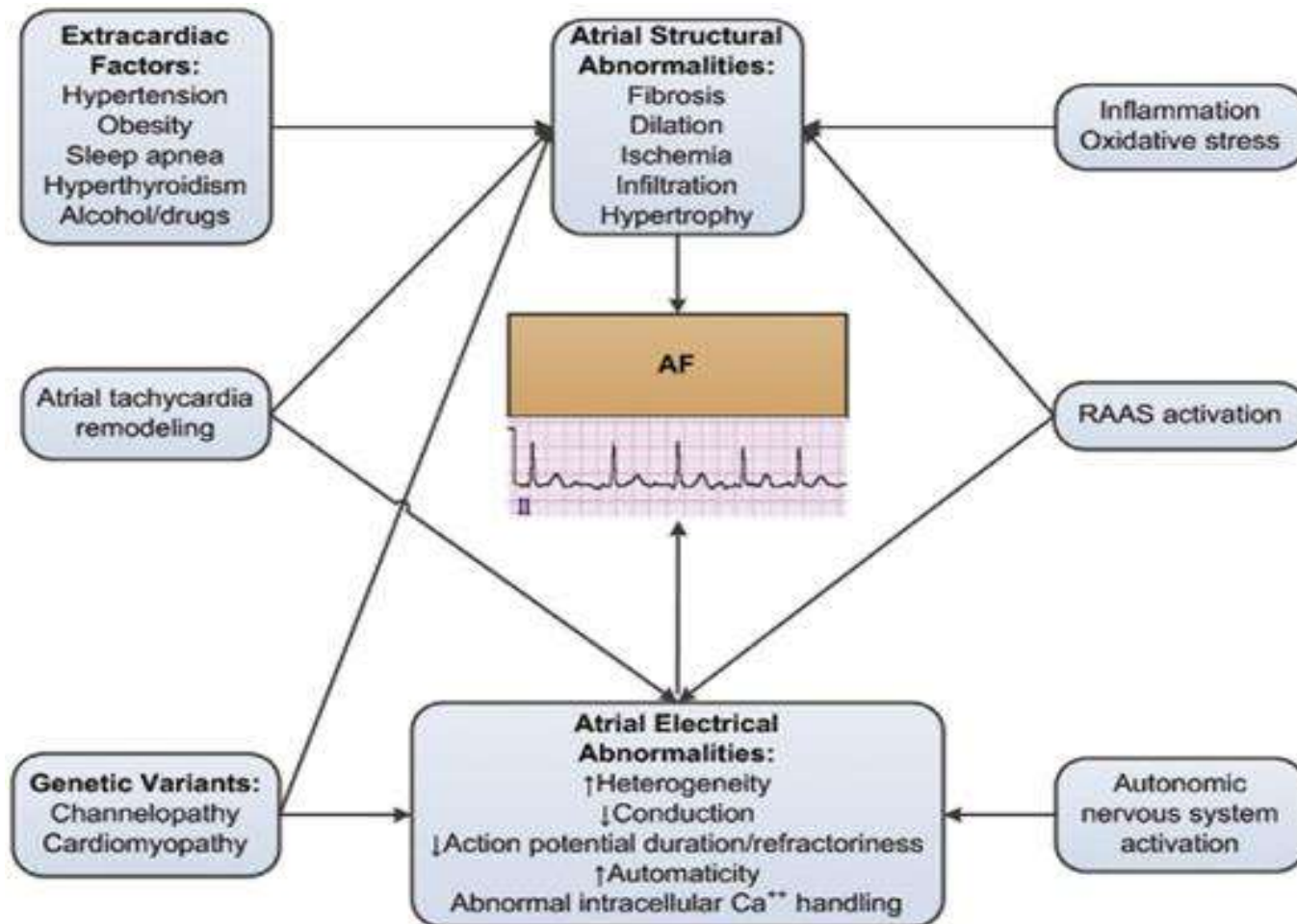
Mechanisms

(Reentry: 4)



III. If conduction over pathway A is sufficiently slow, a premature impulse may continue retrograde all the way up pathway B, which is now past its refractory period. If pathway A is also past its refractory period, the impulse may reenter pathway A and continue to circle, sending an impulse each cycle to the ventricle (4) and retrograde to the atrium (5), producing a sustained reentrant tachycardia.

Mechanisms (Atrial Fibrillation)



Classification

(International Classification of Diseases (ICD): 1)

Chapter IX

I30-I52 Other forms of heart disease

I44 Atrioventricular and left bundle-branch block (I44.0 Atrioventricular block, first degree; I44.1 Atrioventricular block, second degree: type I and II; I44.2 Atrioventricular block, complete; I44.3 Other and unspecified atrioventricular block; I44.4 Left anterior fascicular block; I44.5 Left posterior fascicular block; I44.6 Other and unspecified fascicular block ; I44.7 Left bundle-branch block, unspecified).

I45 Other conduction disorders (I45.0 Right fascicular block; I45.1 Other and unspecified right bundle-branch block; I45.2 Bifascicular block; I45.3 Trifascicular block; I45.4 Nonspecific intraventricular block; I45.5 Other specified heart block; I45.6 Pre-excitation syndrome (Lown-Ganong-Levine syndrome, Wolff-Parkinson-White syndrome); I45.8 Other specified conduction disorders: atrioventricular [AV] dissociation ([R94.3](#)); I45.9 Conduction disorder, unspecified).

I46 Cardiac arrest (I46.0 Cardiac arrest with successful resuscitation; I46.1 Sudden cardiac death; I46.9 Cardiac arrest, unspecified).

Classification

(International Classification of Diseases (ICD): 2)

Chapter IX

I30-I52 Other forms of heart disease

147 Paroxysmal tachycardia (I47.0 Re-entry ventricular arrhythmia; I47.1 Supraventricular tachycardia; I47.2 Ventricular tachycardia, I47.9 Paroxysmal tachycardia, unspecified).

I48 Atrial fibrillation and flutter (I48.0 Paroxysmal atrial fibrillation, I48.1 Persistent atrial fibrillation, I48.2 Chronic atrial fibrillation, I48.3 Typical atrial flutter, Type I atrial flutter, I48.4 Atypical atrial flutter, Type II atrial flutter, I48.9 Atrial fibrillation and atrial flutter, unspecified).

I49 Other cardiac arrhythmias (I49.0 Ventricular fibrillation and flutter, I49.1 Atrial premature beats, I49.2 Junctional premature beats, I49.3 Ventricular premature beats , I49.4 Other and unspecified premature beats, I49.5 Sick sinus syndrome, I49.8 Other specified cardiac arrhythmias (Brugada syndrome, Long QT syndrome, Rhythm disorder: coronary sinus, ectopic, nodal), I49.9 Cardiac arrhythmia, unspecified).

Classification

(International Classification of Diseases (ICD): 3)

Chapter IX

R00 Abnormalities of heart beat.

R00.0 Tachycardia, unspecified.

R00.1 Bradycardia, unspecified:

Bradycardia: sinoatrial

sinus

vagal

Slow heart beat (use additional external cause code (Chapter XX), if desired, to identify drug, if drug-induced).

R00.2 Palpitations

Awareness of heart beat

R00.8 Other and unspecified abnormalities of heart beat

Classification

(Types of Arrhythmia)

- Conduction violations:
 - Block (sinus one, atrium one, atrium-ventricular one, ventricle one).
 - Wolf-Parkinson-White syndrome (pre-excitation syndrome).
- Automatism violations:
 - homotopic automatism violation (sinus tachycardia, sinus bradycardia, sinus arrhythmia)
 - heterotopic automatism violation (atrium-ventricular rhythm, idioventricular rhythm).
- Combined heart rhythm violations (extrasystole, paroxysmal tachycardia, atrial flutter, ventricle flutter, atria fibrillation, ventricle fibrillation).

Classification

(Heart Blocks)

Location	Name
Within the sinoatrial node (SA node or Sinus node)	Sinoatrial nodal blocks (SA nodal block, SA block, Sinoatrial block)
Within the atrioventricular node (AV node)	Atrioventricular block (AV nodal block, AV block or AVB)
At and below the bundle of His	Intra-Hisian blocks and Infra-Hisian blocks respectively
Within the left or right bundle branches	Bundle branch blocks
Within the fascicles of the left bundle branch	"Fascicular block" or Hemiblocks

Classification

(Heart Blocks Detalization)

- Sinoatrial (SA) nodal blocks: SA node Wenckebach (Mobitz I), SA node Mobitz II, SA node exit block (in addition, the SA node can be suppressed by any other arrhythmia that reaches it (retrograde conduction from the ventricles, ectopic atrial beats, atrial fibrillation, and atrial flutter)).
- Atrioventricular (AV) nodal blocks: first-degree AV block, second-degree AV block (type I or Mobitz I, also known as Wenckebach block; type 2 or Mobitz II), third-degree AV block (complete heart block).
- Infra-Hisian block: type 2 second degree heart block or Mobitz II (left anterior fascicular block, left posterior fascicular block).
- Right bundle branch block.

Classification

(Premature Contraction or Extrasystoles)

Level

- Atrial
- Junctional
- Ventricular

Frequency

- Rare
- Frequent

Focus

- Unifocal

Multifocal

Time of occurrence

- Early
- Late

Regularity

- Regular
 - Bigeminy (extrasystole follows every sinus beat)
 - Trigeminy (extrasystole follows every other sinus beat (every third beat is ectopic))
- Irregular

Series

- Single beats
- Couplet beats
- Triplet beats
- Interpolated

Classification

(Lown Criteria of Premature Ventricular Contractions)

Grade 0	No ventricular extrasystoles/ectopics
Grade I	Unifocal and infrequent PVCs; <30 PVCs per hour
Grade II	Unifocal and frequent PVCs, ≥ 30 PVCs per hour
Grade III	Multifocal
Grade IVA	2 consecutive beats (couplets)
Grade IVB	≥ 3 consecutive beats (salvos)
Grade V	"R on T" phenomenon

>5 consecutive PVCs is considered a run of VT. This run of VT may either be sustained or non-sustained; treat as per VT.

Classification

(Atrial Fibrillation)

- Paroxysmal atrial fibrillation (AF): episodes of AF that terminate spontaneously or with intervention within 7 days.
- Persistent AF: episodes of continuous AF that last more than 7 days and do not self-terminate.
- Long-standing persistent AF: episodes of continuous AF that last more than 12 months.
- Permanent AF: Applies when a joint physician/patient decision has been made to accept the presence of AF and stop further attempts to restore and/or maintain sinus rhythm.
- Nonvalvular AF: AF in the absence of rheumatic mitral valve disease, a prosthetic heart valve, or mitral valve repair.

Clinical Investigation

(Signs and Symptoms)

- Completely asymptomatic
- Abnormal heartbeat (palpitations)
- Forceful or painful extra beats
- Shortness of breath
- Temporarily absence of ability to breath
- Chest discomfort or pain
- Fluttering
- Quivering
- Hypotension
- Diaphoresis
- Neck fullness
- Paleness
- Vasovagal type response with light-headedness, dizziness, nausea, or loss of consciousness (vertigo, syncope, fainting)
- Higher risk of blood clotting, embolization and stroke (atrial fibrillation)
- Cardiac arrest, or sudden cardiac death

Diagnosis

(Key Remarks)

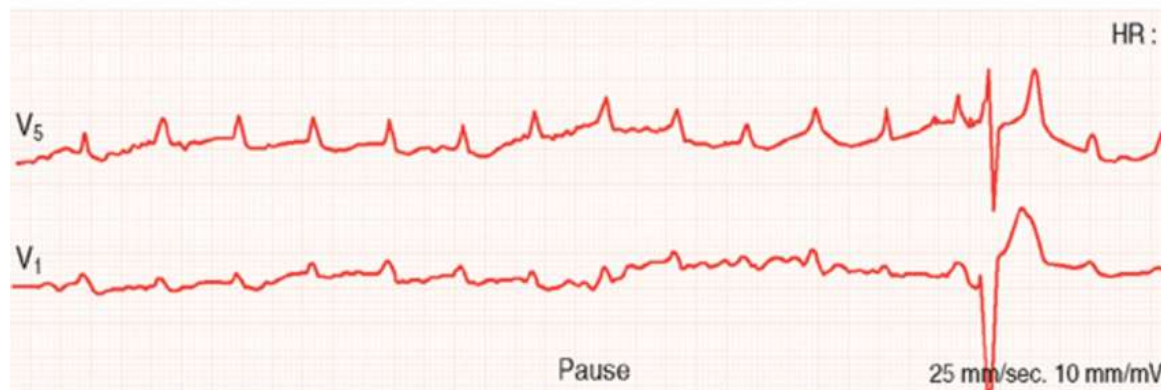
- Because there are a number of tests available for the diagnosis of cardiac arrhythmias, it is important to proceed with a stepwise approach.
- The goal is to obtain a correlation between symptoms and the underlying arrhythmia and initiation of appropriate therapy.
- Additional testing is usually advocated to identify patients with arrhythmias caused by ischemia or who are at risk for sudden cardiac death.

Diagnosis

(Assessment of Structural Heart Disease)

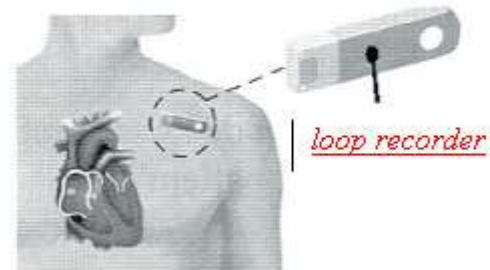
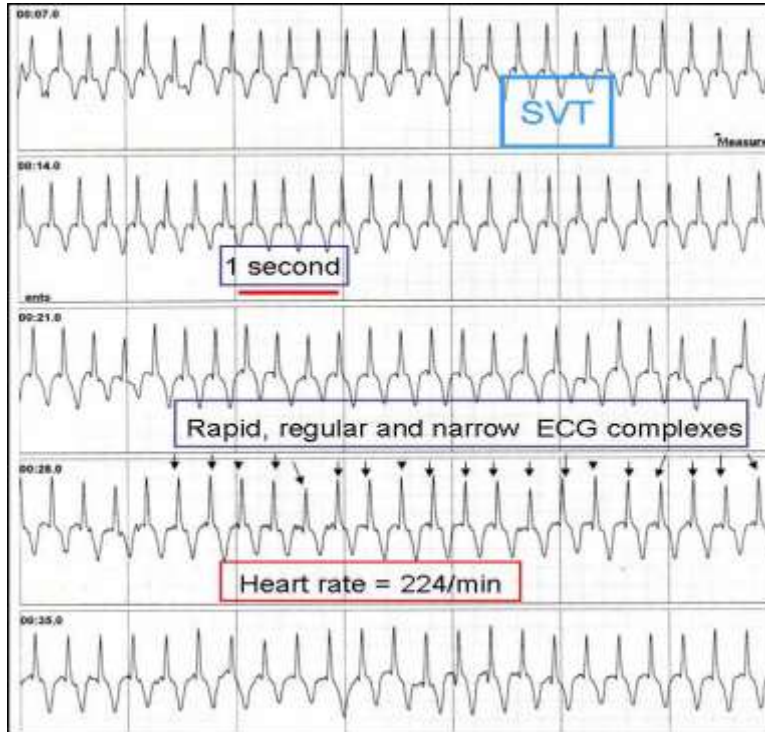
- Interviewing (complaints, history) and physical examination with attention to coronary artery disease or myocardial infarctions, risk factors for coronary artery disease (CAD), and family history of sudden cardiac death are extremely important.
- Auscultation may detect an irregular rhythm or premature beats.
- Careful scrutiny of the electrocardiogram (ECG) is imperative to look for conduction system delays, QRS widening, previous myocardial infarction, or PVCs.
- Stress testing, usually with imaging (e.g., stress echocardiography or stress thallium and echocardiography) can demonstrate the presence of CAD, LV dysfunction, or valvular heart disease.
- Patients may present with a wide complex tachycardia, possibly VT versus SVT with aberrancy. The rule is that sustained or nonsustained wide complex tachycardia in patients with known CAD or previous myocardial infarction (MI) is VT until proven otherwise.

Diagnosis (Holter Monitoring)



Ambulatory 24- to 48-hour baseline Holter monitoring is useful in quantitating and qualifying arrhythmias in patients with frequent symptoms. Here intermittent complete heart block in patient with syncope. HR – heart rate.

Diagnosis (Event Recording)



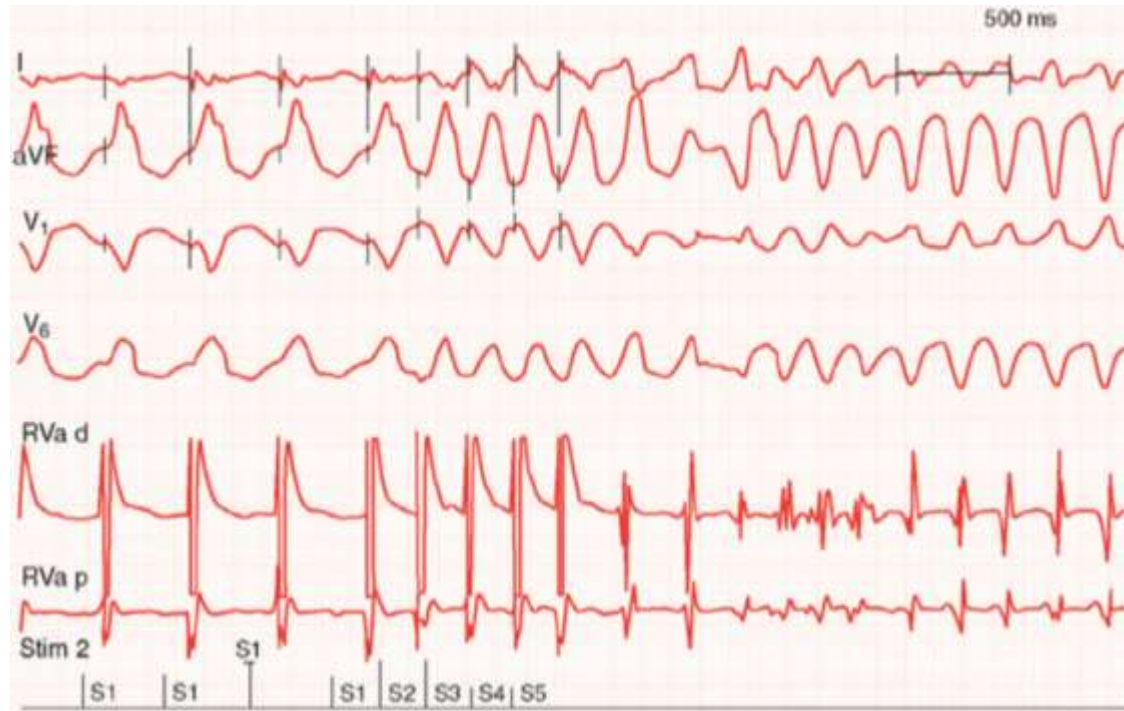
Event recording monitoring systems, or loop recorders (e.g., King of Hearts, Instromedix, Rosemont, III) can be worn for longer intervals (usually a month) and can document infrequent arrhythmia episodes and provide symptom-arrhythmia correlation.

Diagnosis (Remote Magnetic Navigation)



Diagnosis

(Electrophysiologic Testing)



Electrophysiologic testing is the gold standard for evaluating patients with recurrent syncope and can help identify underlying His-Purkinje disease, inducible VT, SVT, and sinus node dysfunction. Induced sustained monomorphic tachycardia.

Diagnosis

(Bradyarrhythmias)

- A regular QRS bradyarrhythmia with a 1:1 relationship between P waves and QRS complexes indicates absence of AV block; P waves preceding QRS complexes indicate sinus bradycardia (if P waves are normal) or sinus arrest with an escape atrial bradycardia (if P waves are abnormal).
- P waves after QRS complexes indicate sinus arrest with a junctional or ventricular escape rhythm and retrograde atrial activation.
- An irregular QRS rhythm with a 1:1 relationship between P waves and the following QRS complexes indicates sinus arrhythmia with gradual acceleration and deceleration of the sinus rate (if P waves are normal).
- No relationship between P waves and QRS complexes indicates AV block; the escape rhythm can be junctional (narrow QRS complex) or ventricular (wide QRS complex).
- Pauses in an otherwise regular QRS rhythm may be caused by blocked P waves (an abnormal P wave can usually be discerned just after the preceding T wave or distorting the morphology of the preceding T wave), sinus arrest, or sinus exit block, as well as by AV block.

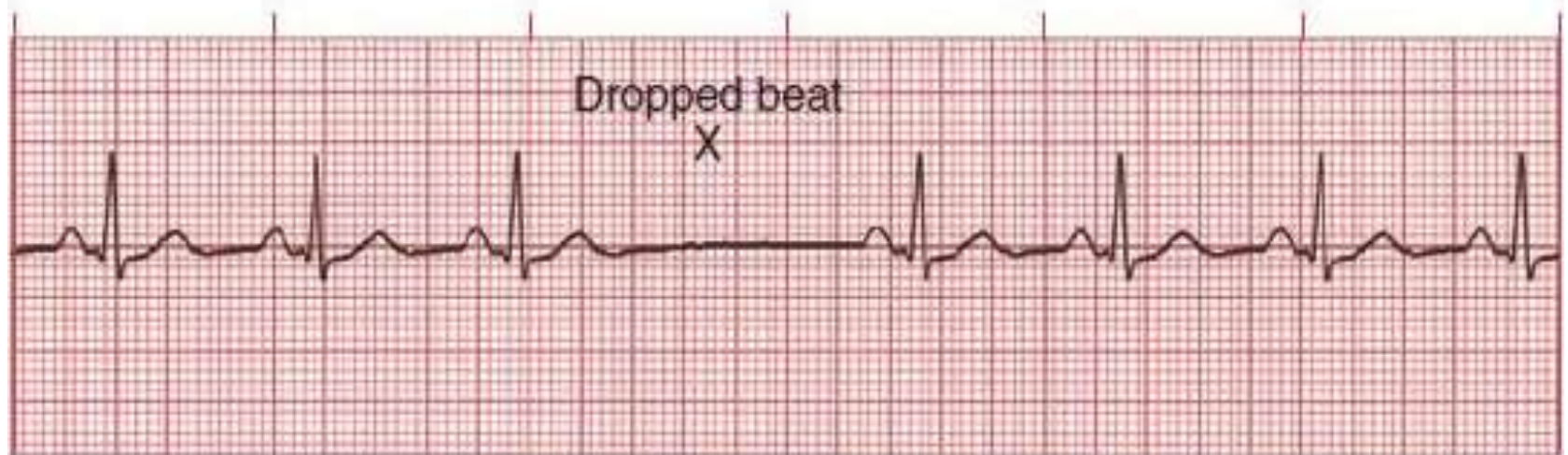
Diagnosis

(SA blocks)

- SA blocks rarely give severe symptoms, because the secondary pacemaker of the heart (AVN) is enough to retain consciousness in the resting state.
- The SA can be suppressed by any other arrhythmia that reaches it (retrograde conduction from the ventricles, ectopic atrial beats, atrial fibrillation, and atrial flutter).
- In SA block contrary to the SA suppression an electrical impulse is generated by the SA node that doesn't make the atria contract.

Diagnosis (SA block)

- The block occurs in some multiple of the P-P interval.
- After the dropped beat, cycles continue on time.



Rate: Normal to slow; determined by duration and frequency of SA block

Rhythm: Irregular whenever an SA block occurs

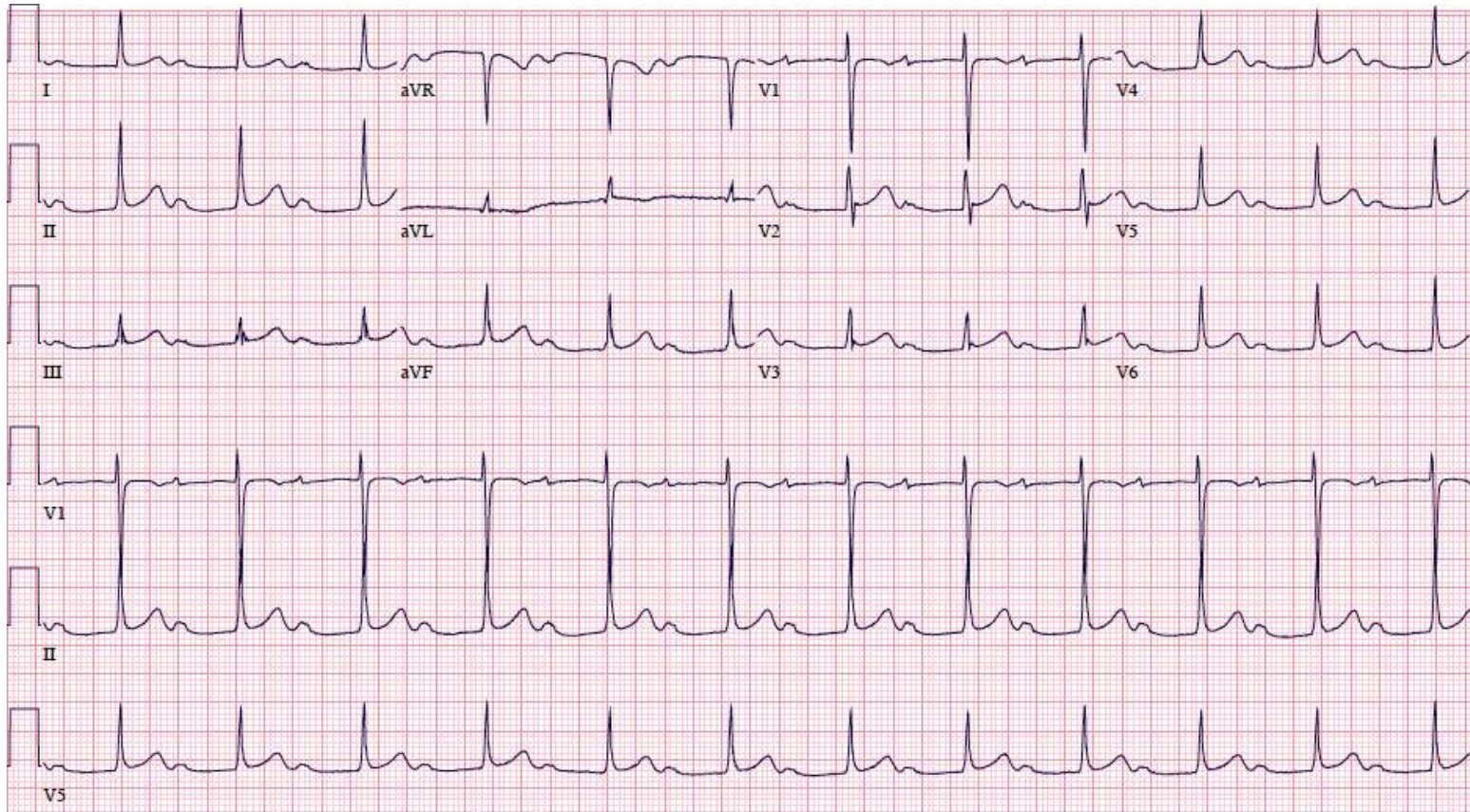
P Waves: Normal (upright and uniform) except in areas of dropped beats

PR Interval: Normal (0.12–0.20 sec)

QRS: Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Cardiac output may decrease, causing syncope or dizziness.

Diagnosis (First-Degree AV Block)



First-Degree degree AV block is defined as a prolonged interval between atrial and ventricular activation $>200\text{ms}$. This delay results from disease in the AV-node or His-Purkinje system. An AV block is not the cause of bradycardia, because every atrial impulse results in conduction to the ventricles.

Diagnosis

(Second-Degree AV Block)

Mobitz I or Wenckebach



Mobitz II



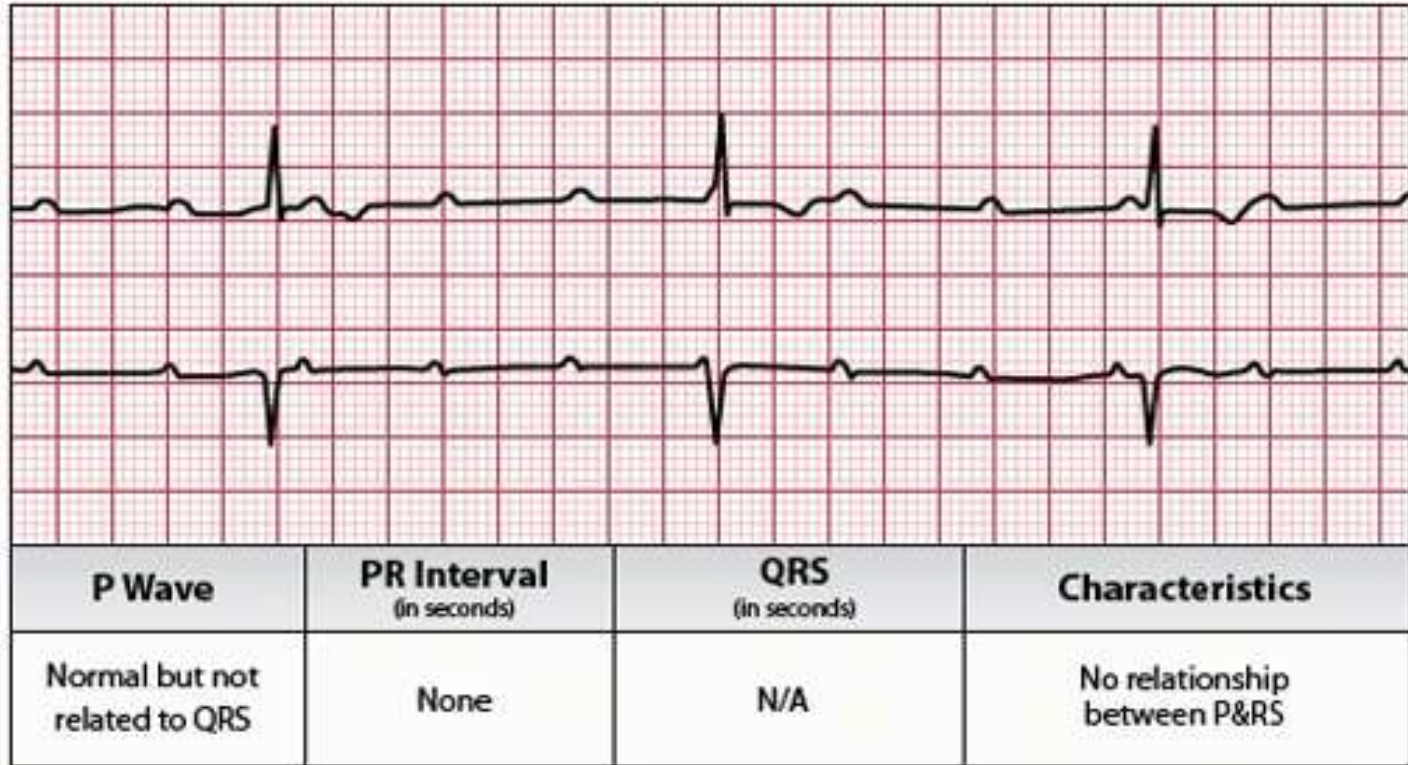
2:1 block



The presence of second-degree AV block is diagnosed when one or more (but not all) of the atrial impulses fail to conduct to the ventricles due to impaired conduction.

Diagnosis

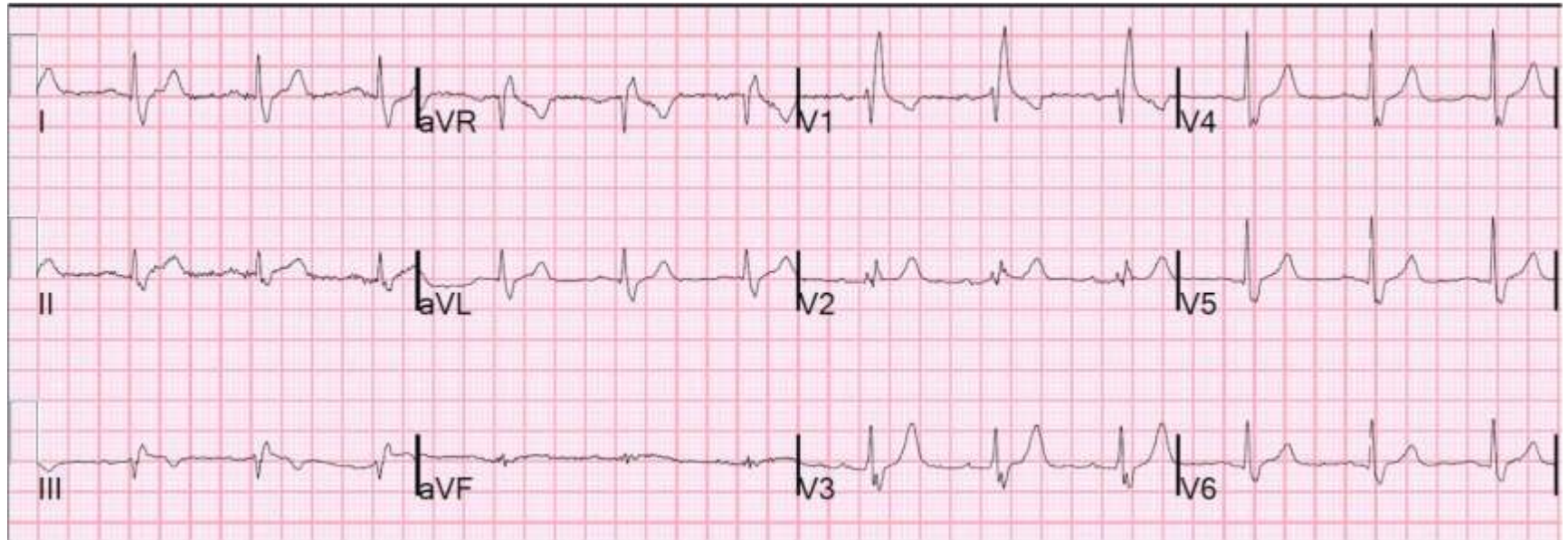
(Third-Degree AV Block)



Most patients are more symptomatic: syncope, near syncope, confusion (Adams Stokes attacks), dyspnea, severe angina, myocardial infarction, sudden death.

Diagnosis

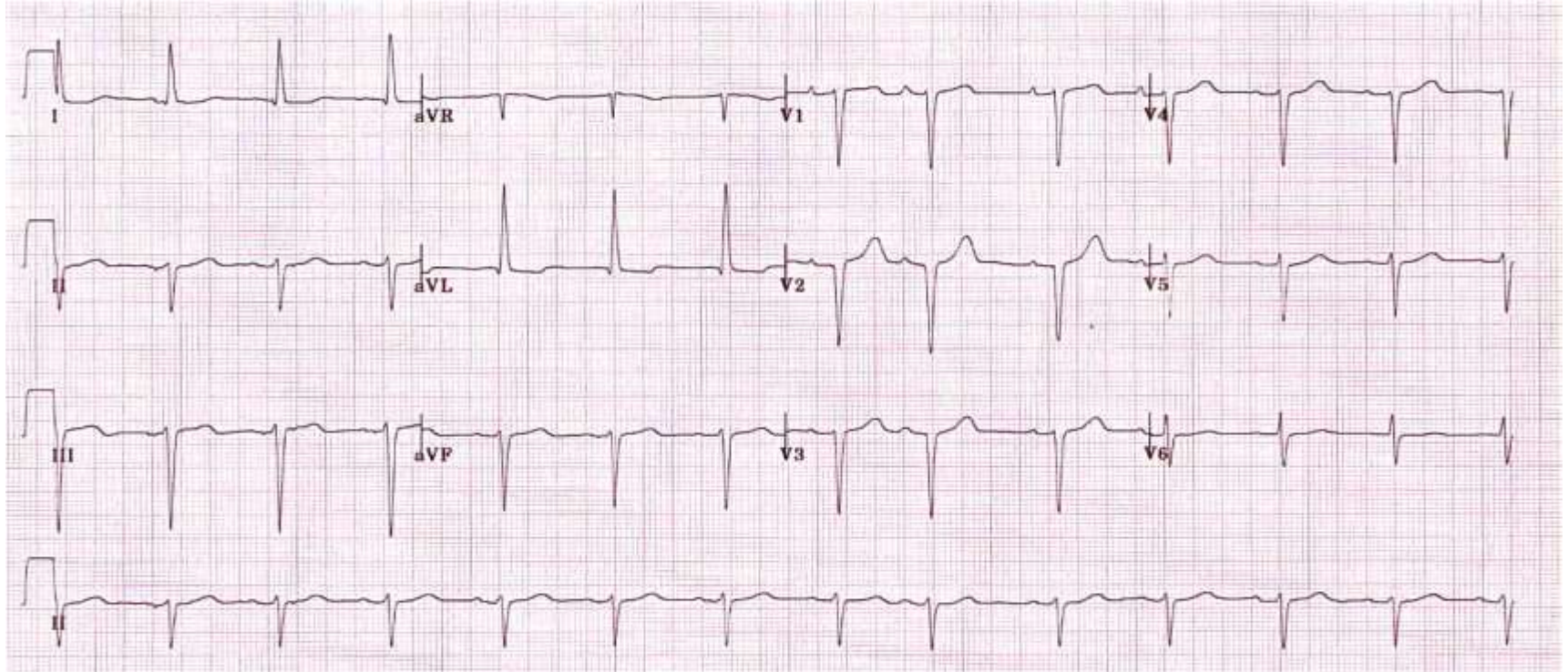
(Infra-Hisian Block: Right Bundle Branch Block)



- Right bundle branch block (RBBB) is a unifascicular block of the right bundle and can be found in healthy people and is represented by a broad QRS complex ($>120\text{ms}$).
- A new RBBB in a patient with a history of normal ventricular conduction warrens further investigation. The last activity is to the right and results in a RSR' pattern in V1 where $R' > R$, , T-waves are usually inverted in V2 and V3, with some ST depression.. In V6 a slurred S wave can be seen at the end of the QRS complex.

Diagnosis

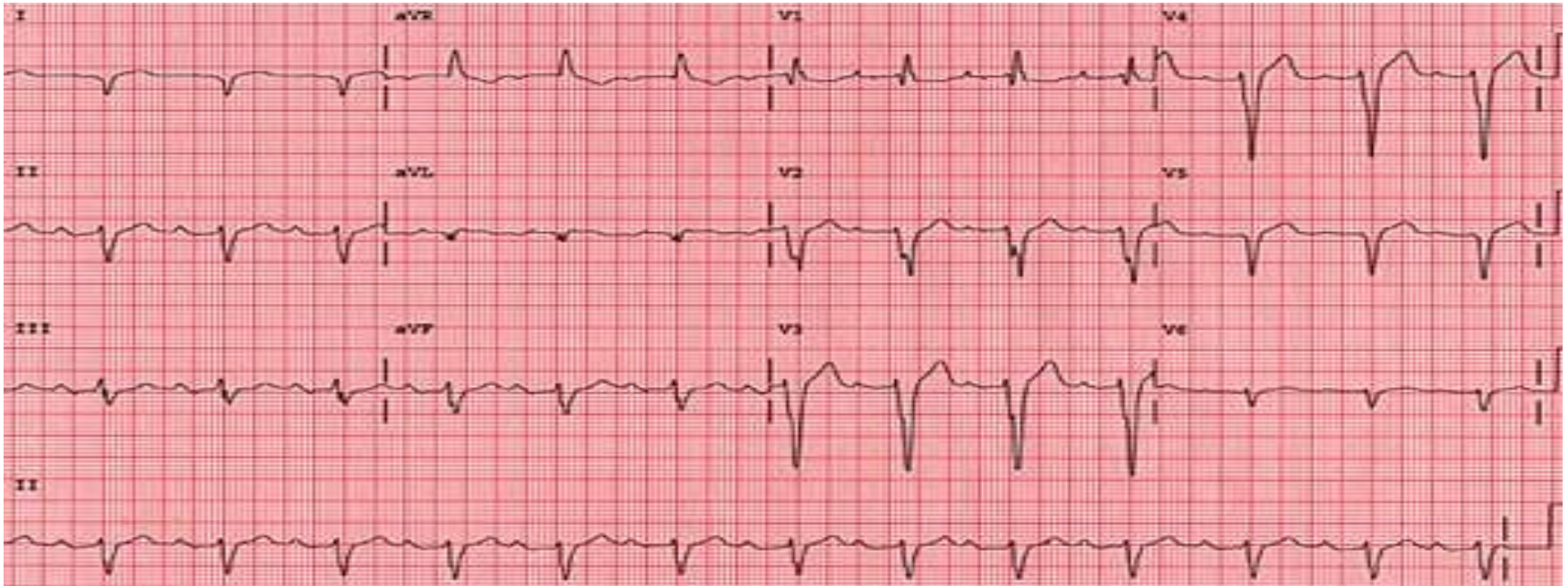
(Infra-Hisian Block: Left Anterior Fascicular Block)



- Left anterior fascicular block (LAFB) is composed of two fascicles.
- One of the fascicle has an anterior location and activates the interventricular septum and the anterior of the ventricle.
- Clinically a LAFB is represented by a left axis deviation and an absent or very small S and normal q in lead I and a S>R in lead II and III. QRS duration should be <120ms.

Diagnosis

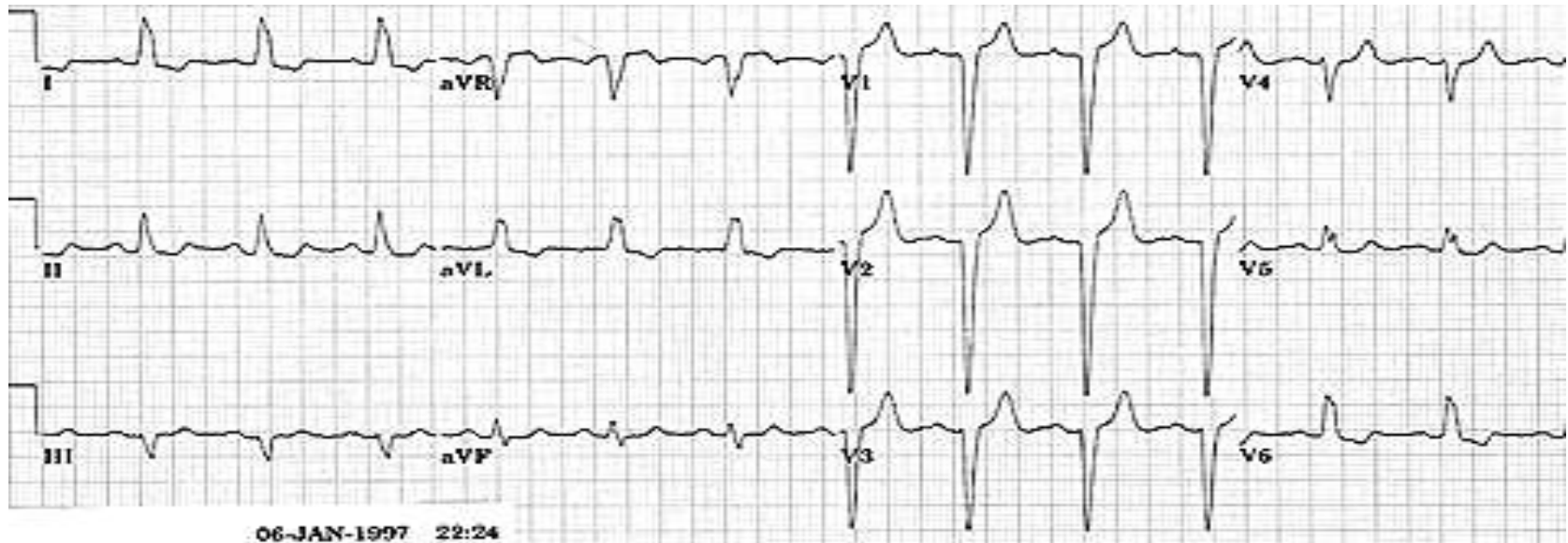
(Infra-Hisian Block: Left Posterior Fascicular Block)



- Left posterior fascicular block (LPFB) is composed of two fascicles, one of the fascicle has an anterior location and activates the interventricular septum and the anterior of the ventricle.
- Clinically a LAFB is represented by a left axis deviation and an absent or very small S and normal q in lead I and a S>R in lead II and III. QRS duration should be <120ms.

Diagnosis

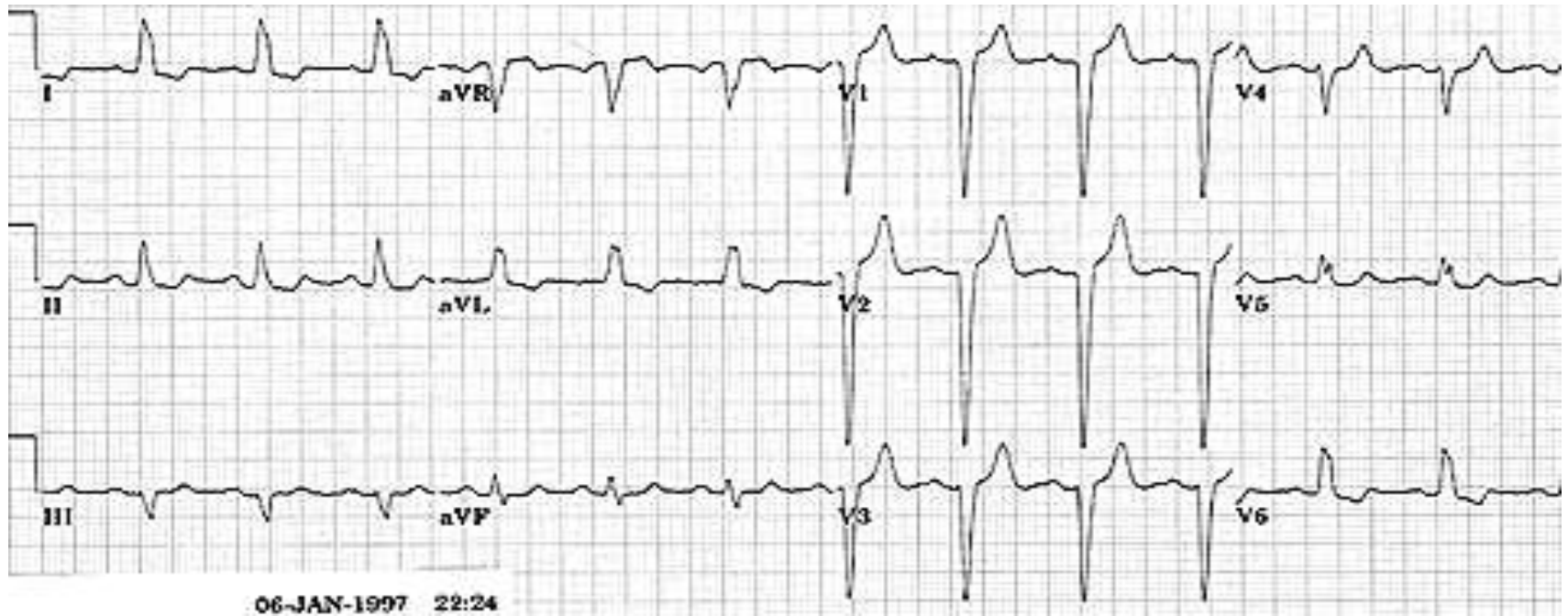
(Infra-Hisian Block: Left Bundle Branch Block)



- Left bundle branch block (LBBB) is uncommon in healthy and need to be performed to screen for underlying disease.
- LBBB results, most importantly a broad QRS of >120ms. In V1 a broad monomorphic S wave can be seen (sometimes with a small r wave) representing slow left ventricular activation. In the lead V6 a broad monomorphic R wave is seen with no Q waves

Diagnosis

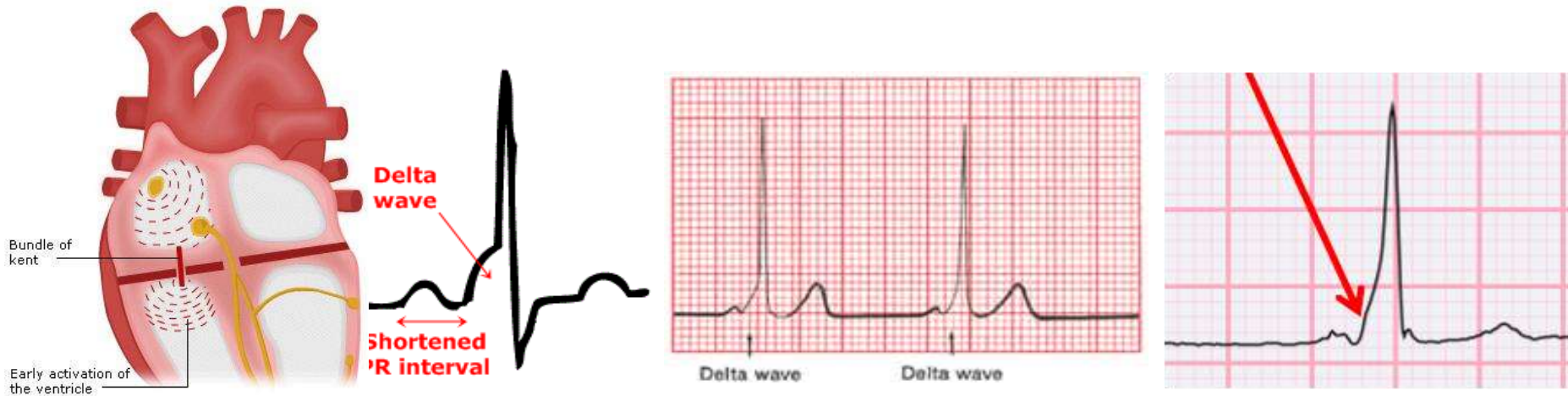
(Infra-Hisian Block: Bifascicular Blocks)



- RBBB plus either LAFB (common) or LPFB (uncommon)
- Features of RBBB plus frontal plane features of the fascicular block (axis deviation, etc.)

Diagnosis

(Wolff-Parkinson-White Syndrome)



Wolff–Parkinson–White syndrome (WPW) referred to as pre-excitation syndromes, and is caused by the presence of an abnormal accessory electrical conduction pathway between the atria and the ventricles. Electrical signals traveling down this abnormal pathway (known as the bundle of Kent) may stimulate the ventricles to contract prematurely, resulting in a unique type of supraventricular tachycardia referred to as an atrioventricular re-entrant tachycardia.

Diagnosis

(Tachyarrhythmias: Irregular, Narrow QRS Complex)

- Irregular, narrow QRS complex tachyarrhythmias include atrial fibrillation (AF), atrial flutter or true atrial tachycardia with variable AV conduction, and multifocal atrial tachycardia.
- Differentiation is based on atrial ECG signals, which are best seen in the longer pauses between QRS complexes.
- Atrial ECG signals that are continuous, irregular in timing and morphology, and very rapid (> 300 beats/min) without discrete P waves indicate AF.
- Discrete P waves that vary from beat to beat with at least 3 different morphologies suggest multifocal atrial tachycardia.
- Regular, discrete, uniform atrial signals without intervening isoelectric periods (usually at rates > 250 beats/min) suggest atrial flutter.
- Regular, discrete, uniform, abnormal atrial signals with intervening isoelectric periods (usually at rates < 250 beats/min) suggest true atrial tachycardia.

Diagnosis

(Tachyarrhythmias: Irregular, Wide QRS Complex)

- Irregular, wide QRS complex tachyarrhythmias include the above 4 atrial tachyarrhythmias, conducted with either bundle branch block or ventricular preexcitation, and polymorphic ventricular tachycardia (VT).
- Differentiation is based on atrial ECG signals and the presence in polymorphic VT of a very rapid rate (> 250 beats/min).

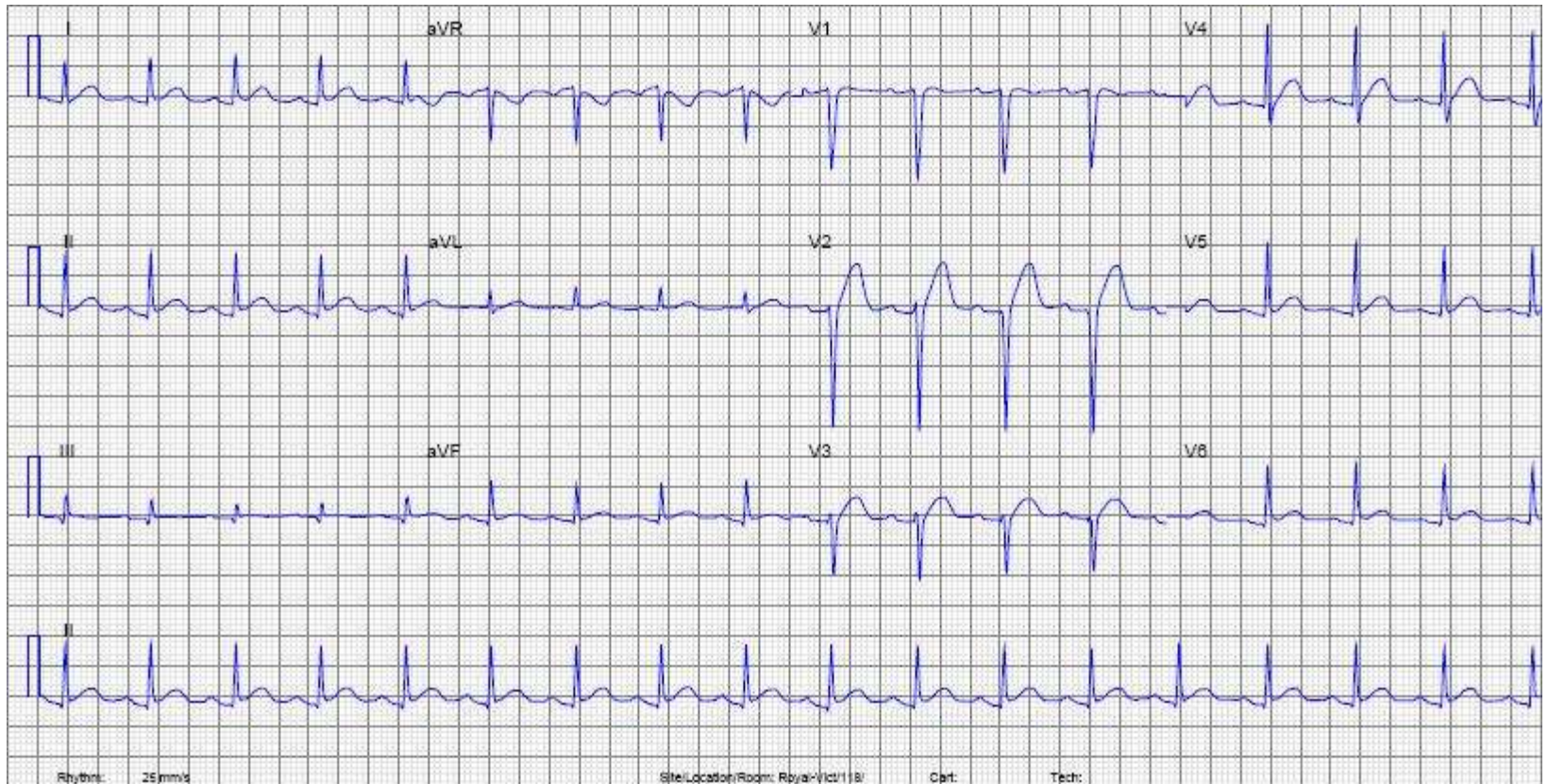
Diagnosis

(Tachyarrhythmias: Regular, Narrow QRS Complex)

- Regular, narrow QRS complex tachyarrhythmias include sinus tachycardia, atrial flutter or true atrial tachycardia with a consistent AV conduction ratio, and paroxysmal SVTs (AV nodal reentrant SVT, orthodromic reciprocating AV tachycardia in the presence of an accessory AV connection, and SA nodal reentrant SVT).
- Vagal maneuvers or pharmacologic AV nodal blockade can help distinguish among these tachycardias.
- With these maneuvers, sinus tachycardia is not terminated, but it slows or AV block develops, disclosing normal P waves.
- Similarly, atrial flutter and true atrial tachycardia are usually not terminated, but AV block discloses flutter waves or abnormal P waves.
- The most common forms of paroxysmal SVT (AV nodal reentry and orthodromic reciprocating tachycardia) must terminate if AV block occurs.

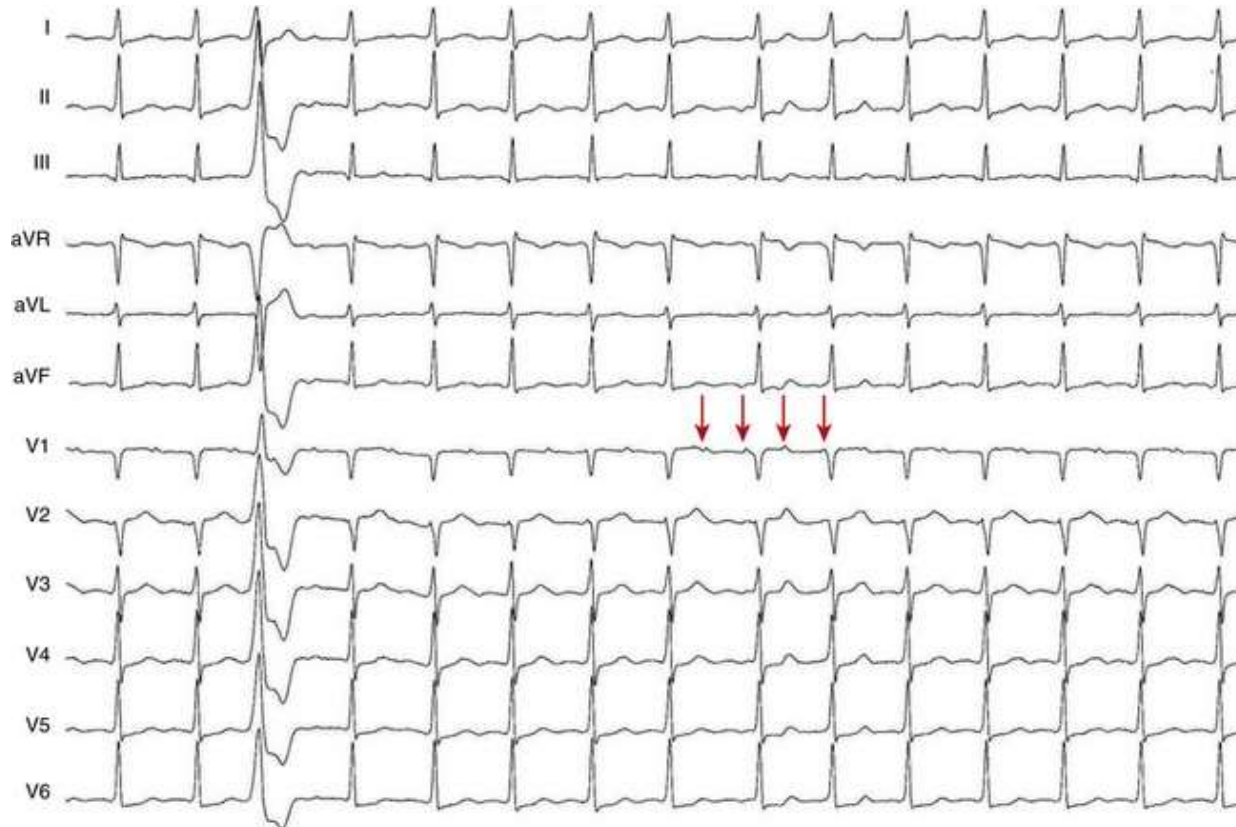
Diagnosis

(Sinus Tachycardia)



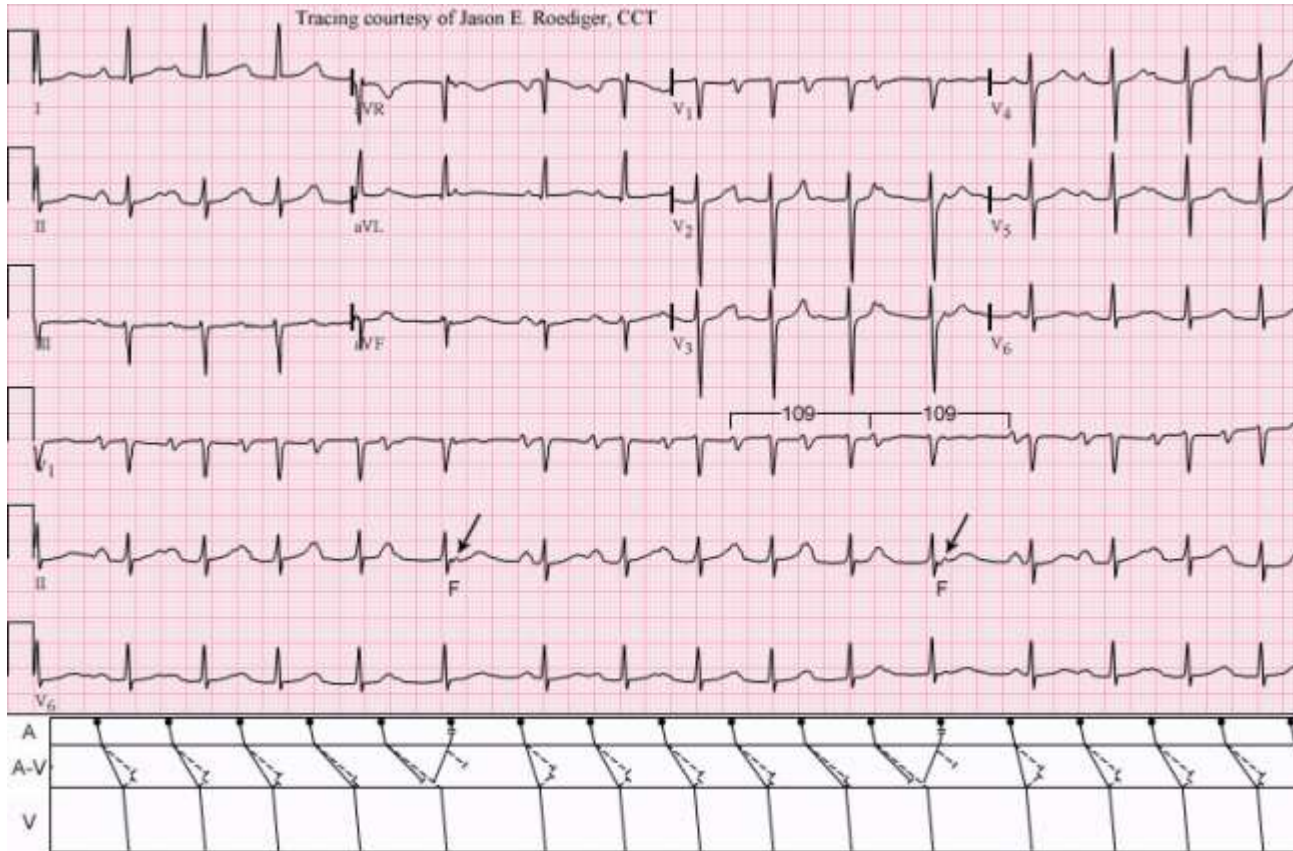
Rate around 108. Regular sinus rhythm - there is a P wave before each QRS and the P waves are up in II and down in aVR.

Diagnosis (Atrial Tachycardia)



Surface ECG of multifocal atrial tachycardia. Note the varying morphology of the P waves and the PR intervals.

Diagnosis (AV nodal reentrant SVT)



Atrioventricular nodal reentrant tachycardia (AVNRT).

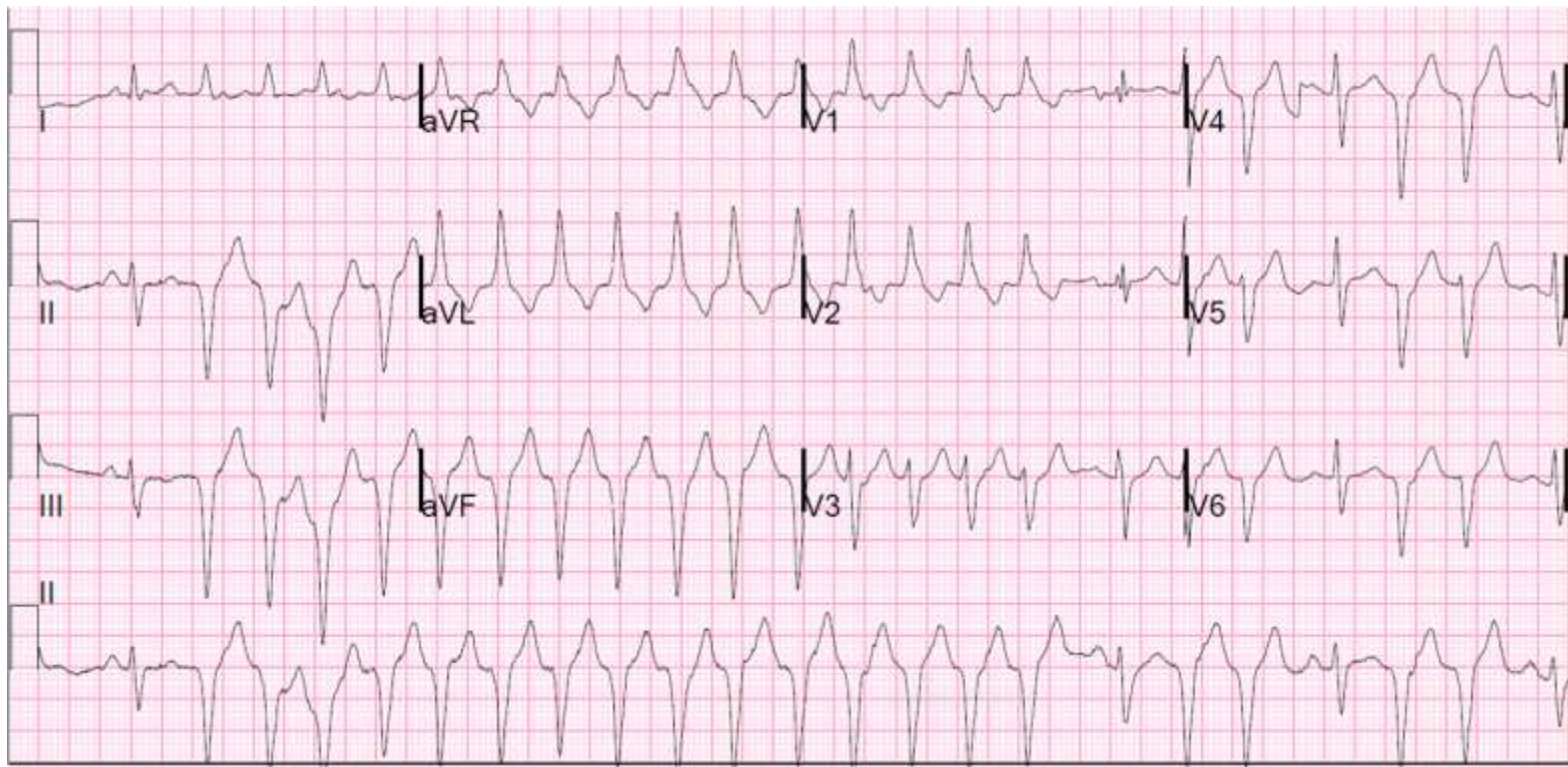
Diagnosis

(Tachyarrhythmias: Regular, Wide QRS Complex)

- Regular, wide QRS complex tachyarrhythmias include those listed for a regular, narrow QRS complex tachyarrhythmia, each with bundle branch block or ventricular preexcitation, and monomorphic VT.
- Vagal maneuvers can help distinguish among them.
- ECG criteria to distinguish between VT and SVT with an intraventricular conduction defect are often used.
- When in doubt, the rhythm is assumed to be VT because some drugs for SVTs can worsen the clinical state if the rhythm is VT; however, the reverse is not true.

Diagnosis

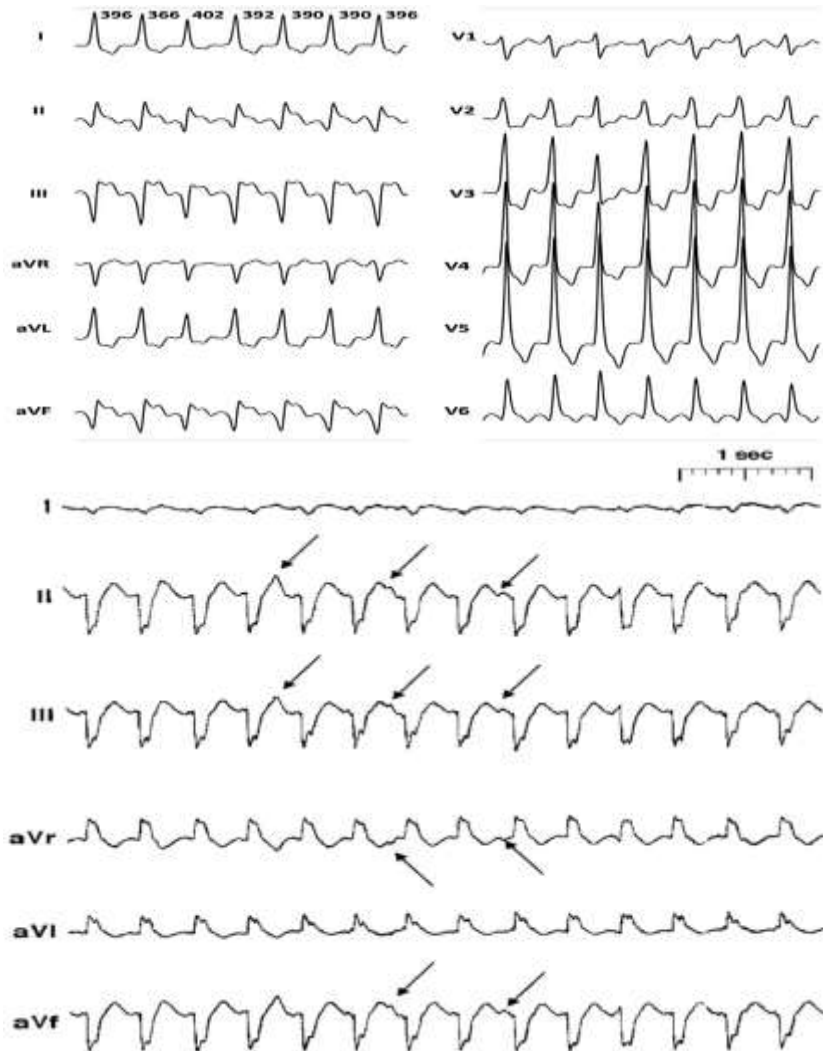
(Regular Wide QRS Complex Tachyarrhythmia)



Regular monomorphic wide complex tachycardia with no P-waves.

Diagnosis

(Narrow and Wide QRS Complexes)

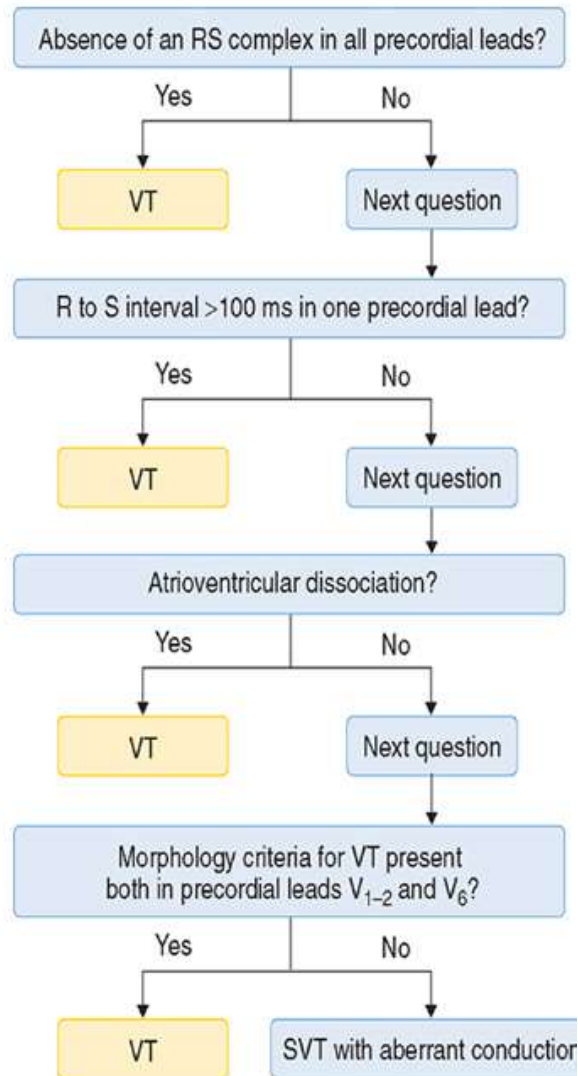


A narrow QRS complex (< 0.12 sec) indicates a supraventricular origin (above the His bundle bifurcation).

A wide QRS complex (≥ 0.12 sec) indicates a ventricular origin (below the His bundle bifurcation) or a supraventricular rhythm conducted with an intraventricular conduction defect or with ventricular preexcitation in the Wolff-Parkinson-White syndrome.

Diagnosis

(Brugada Criteria for Ventricular Tachycardia)



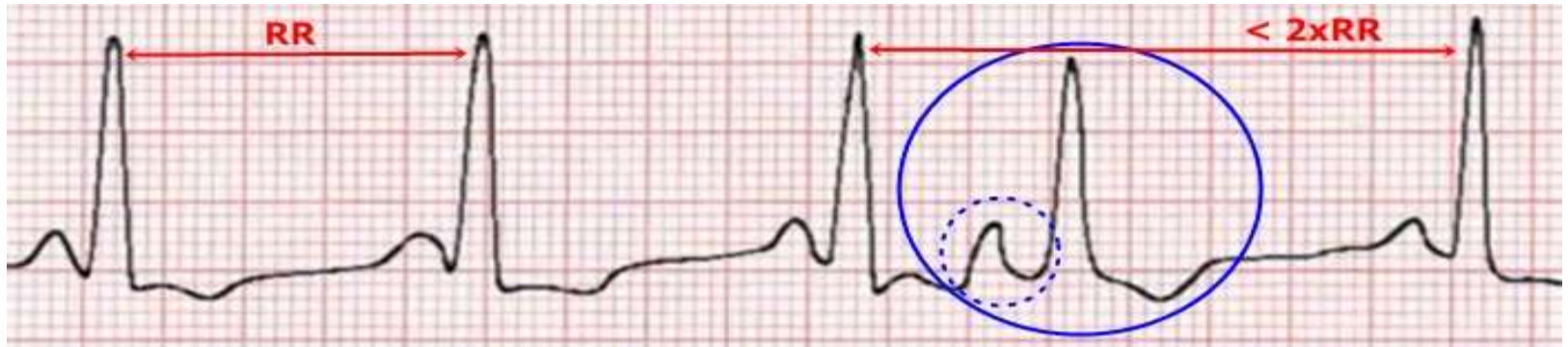
Diagnosis

(Tachyarrhythmias: Regular, Wide QRS Complex)

- Regular, wide QRS complex tachyarrhythmias include those listed for a regular, narrow QRS complex tachyarrhythmia, each with bundle branch block or ventricular preexcitation, and monomorphic VT.
- Vagal maneuvers can help distinguish among them.
- ECG criteria to distinguish between VT and SVT with an intraventricular conduction defect are often used.
- When in doubt, the rhythm is assumed to be VT because some drugs for SVTs can worsen the clinical state if the rhythm is VT; however, the reverse is not true.

Diagnosis

(Supraventricular Extrasystoles)



- Supraventricular extrasystoles originate in the atrial myocardium, or in the AV node and manifest as an early P wave, which has a morphology corresponding to the origin of the focus (negative if lower atrial focus, biphasic if left atrial, etc.).
- The QRS complex can be normal or widened because of aberrancy.
- Frequently, the conduction of the supraventricular extrasystoles is blocked, so that a pause occurs, which is further extended by the subsequent sinus pause.
- They have no prognostic significance but can induce sustained arrhythmias.

Diagnosis

(Ventricular Extrasystoles)



Ventricular extrasystoles can be differentiated from supraventricular extrasystoles by the following:

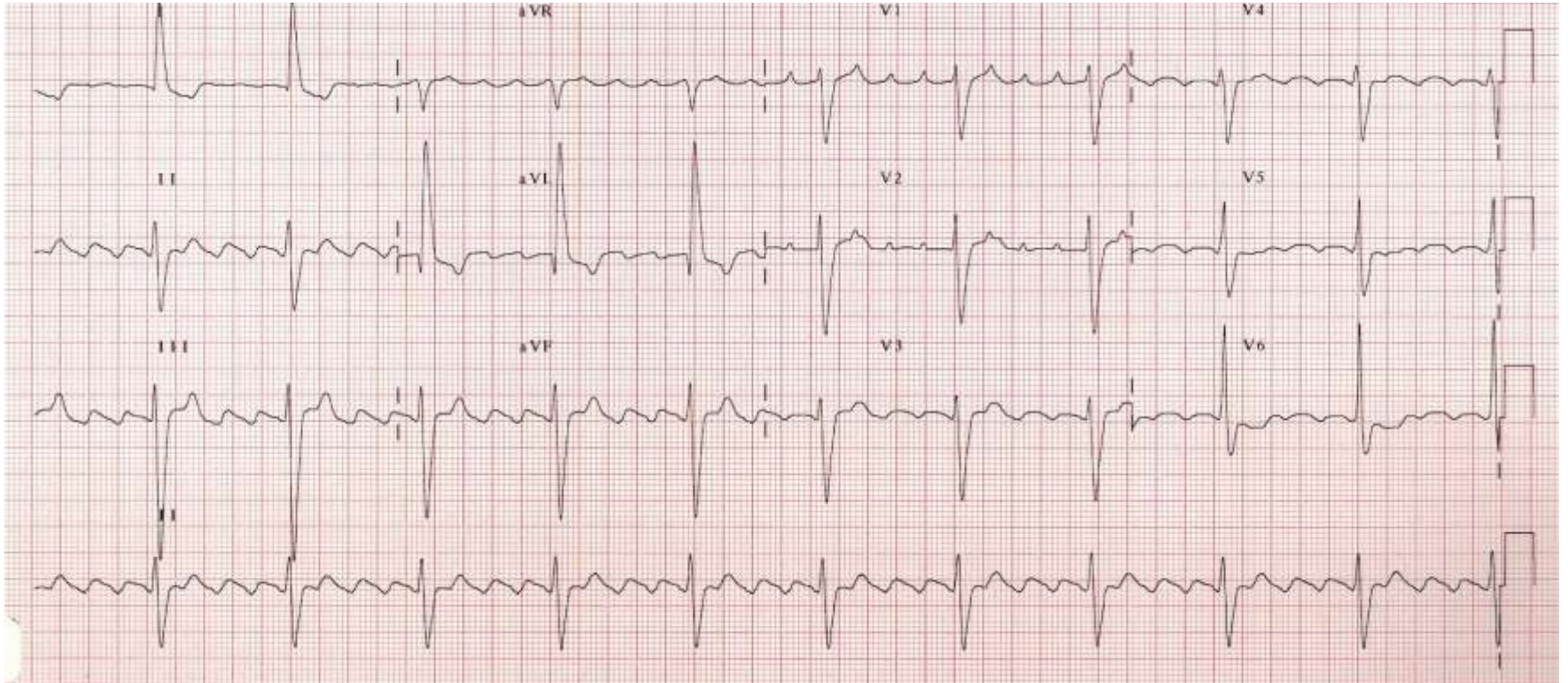
- no preceding P wave (no constant PQ interval)
- different QRS morphology, width, and axis
- altered repolarization (T wave opposite to QRS vector)
- compensatory pause.

Single ventricular extrasystoles have no proven prognostic value, especially in the absence of structural heart disease.

More than 3 consecutive ventricular extrasystoles are considered to be ventricular tachycardia, which represents a risk factor in patients with coronary heart disease or hypertrophic cardiomyopathy.

Diagnosis

(Combined Heart Rhythm Violations: Atrial Flutter)

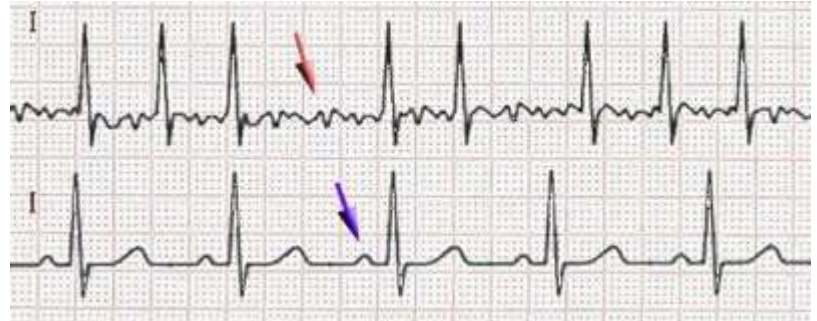


Atrial flutter is a cardiac arrhythmia characterized by atrial rates of 240-400 beats/min and some degree of AVN block.

Diagnosis

(Combined Heart Rhythm Violations: Atrial Fibrillation)

In atrial fibrillation (AF), the normal regular electrical impulses generated by the sinoatrial node in the right atrium of the heart are overwhelmed by disorganized electrical impulses usually originating in the roots of the pulmonary veins with irregular conduction of ventricular impulses that generate the heartbeat.



ECG of atrial fibrillation (top) and normal sinus rhythm (bottom). The purple arrow indicates a P wave, which is lost in atrial fibrillation.

Treatment

(General Principles)

- Treatment of cause.
- Asymptomatic arrhythmias without serious risks do not require treatment even if they worsen.
- Reducing coffee, alcohol, or tobacco use or increasing the amount of rest may help to alleviate symptoms.
- Symptomatic arrhythmias may require treatment to improve quality of life.
- Potentially life-threatening arrhythmias require treatment.
- The method of treatment depends firstly on whether or not the affected person is stable or unstable.
- Treatments may include physical maneuvers, antiarrhythmic drugs, electricity conversion, catheter ablation, or electrosurgery.
- Patients with arrhythmias that have caused or are likely to cause symptoms of hemodynamic compromise may have to be restricted from driving until response to treatment has been assessed.

Treatment

(Physical Maneuvers)

- A number of physical acts can increase parasympathetic nervous supply to the heart, resulting in blocking of electrical conduction through the atrio-ventricular (AV) node.
- Physical (vagal) maneuvers can slow down or stop a number of arrhythmias that originate above or at the AV.
- The Valsalva maneuver should be the first vagal maneuver tried and works by increasing intra-thoracic pressure and affecting baroreceptors within the arch of the aorta. It is carried out by asking the patient to hold his/her breath while trying to exhale forcibly as if straining during a bowel movement.
- Other vagal maneuvers include holding one's breath for a few seconds, coughing, plunging the face into cold water, drinking a glass of ice cold water, etc.
- Carotid sinus massage is effective but is often not recommended in the elderly due to the potential risk of stroke.

Treatment

(Vaughn-Williams Classification of Antiarrhythmic Medications)

Class	Actions (Examples)
I	Sodium channel blockers
IA	Depress phase 0 of action potential; delay conduction, prolong repolarization—phase III or IV (quinidine, procainamide, disopyramide)
IB	Little effect on phase 0 of action potential in normal tissues; depress phase 0 in abnormal tissues; shorten repolarization or little effect (lidocaine, tocainide, mexiletine, diphenylhydantoin)
IC	Depress phase 0 of action potential; markedly slow conduction in normal tissues (flecainide, propafenone, moricizine)
II	β -Adrenergic blocking agents (acebutolol, atenolol, bisoprolol, carvedilol, metoprolol, nadolol, pindolol, propranolol)
III	Prolong action potential duration by increasing repolarization and refractoriness (amiodarone, sotalol, bretylium, dofetilide, azimilide, ibutilide)
IV	Calcium channel blockers (diltiazem, verapamil)
Others	Digoxin, adenosine

Treatment

(Sinoatrial Block and Atrioventricular Block)

- Sinoatrial blocks are typically well-tolerated and most often do not require treatment; emergency treatment, if deemed necessary, consists of administration of atropine sulfate or transcutaneous pacing.
- First-degree AVN block and Mobitz I second-degree AVN block do not generally require treatment unless they cause symptoms and are not due to a reversible cause.
- Mobitz II second-degree AVN block and third-degree AVN block usually require temporary and/or permanent cardiac pacing.
- Pacemaker implantation is a routine surgical procedure, generally performed with conscious sedation and local anesthesia in the electrophysiology laboratory.

Treatment

(Revolution in Implanted Pacemakers)



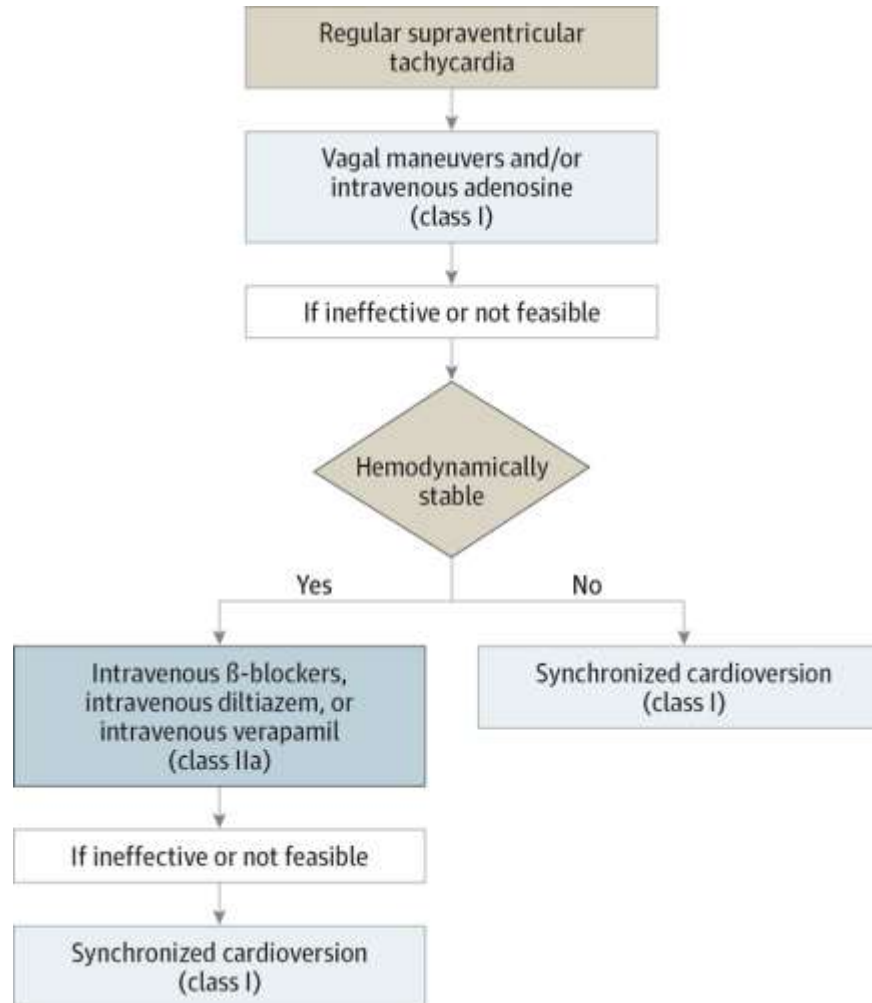
Treatment

(Supraventricular Tachycardia (SVT))

- Most SVTs are unpleasant rather than life-threatening, although very fast heart rates can be problematic for those with underlying ischemic heart disease or the elderly.
- Episodes require treatment when they occur, but interval therapy may also be used to prevent or reduce recurrence.
- SVTs can be classified by whether the AV node is involved in maintaining the rhythm: if so, slowing conduction through the AV node will terminate it; if not, AV nodal blocking maneuvers will not work, although transient AV block is still useful as it may unmask an underlying abnormal rhythm.
- Adenosine, an ultra-short-acting AV nodal blocking agent, is indicated if vagal maneuvers are not effective; if unsuccessful or the permanent SVT (PSVT) recurs diltiazem or verapamil are recommended.¹
- SVT that does not involve the AV node may respond to sotalol or amiodarone.
- If the patient is hemodynamically unstable or other treatments have not been effective, synchronized electrical cardioversion may be used.

Treatment

(Regular Supraventricular Tachycardia of Uncertain Type)



Treatment

(Ventricular tachycardia (VT): 1)

- Patients suffering from pulseless VT or unstable VT are hemodynamically compromised and require immediate cardioversion.
- If a person still has a pulse, it is usually possible to terminate the episode using cardioversion that should be synchronized to the heartbeat if the waveform is monomorphic if possible; an initial energy of 100J is recommended; if the waveform is polymorphic, then higher energies and an unsynchronized shock should be provided (also known as defibrillation).
- For those who are stable with a monomorphic waveform the medications procainamide or sotalol may be used and are better than lidocaine; evidence does not show that amiodarone is better than procainamide.

Treatment

(Cardioversion and Defibrillation)



Treatment

(Ventricular tachycardia (VT): 2)

- As hypomagnesemia is a common cause of VT, magnesium sulphate can be given for torsades de pointes or if hypomagnesemia is found/suspected.
- Long-term anti-arrhythmic therapy may be indicated to prevent recurrence of VT; beta-blockers and a number of class III antiarrhythmics are commonly used.
- Catheter ablation is a possible treatment for those with recurrent VT; remote magnetic navigation is one effective method to do the procedure.
- An implantable cardioverter defibrillator (ICVD) is more effective than drug therapy for prevention of sudden cardiac death due to VT and VF, but may be constrained by cost issues, as well as patient co-morbidities and patient preference.

Treatment

(Extrasystoles)

- No heart disease (normal left ventricular function)
 - Avoid provocative factors
 - Mild asymptomatic ventricular extrasystoles (PVCs) no management required
 - Symptomatic PVCs accounting for <20% of total beats consider beta blocker for disabling symptoms
 - PVCs account for >20% of total beats consider Cardiac Catheter Ablation
- Heart disease (with left ventricular dysfunction)
 - PVCs increase risk of morbidity and mortality
 - Consider management for Low Class 3-5
 - Consider Cardiac Catheter Ablation

Treatment

(Cardiac Catheter Ablation)



Treatment

(Implantable Cardioverter Defibrillator (ICVD))



Treatment

(Atrial Fibrillation: Main Goals)

- The main goals of treatment are to prevent circulatory instability and stroke.
- Rate or rhythm control are used to achieve the former, whereas anticoagulation is used to decrease the risk of the latter.
- If cardiovascular system unstable due to uncontrolled tachycardia, immediate cardioversion is indicated.

Treatment

(Atrial Fibrillation: Anticoagulants)

- Anticoagulation reduce the risk of stroke and is recommended in most people other than those at low risk of stroke or those at high risk of bleeding.
- The risk of stroke from non-valvular AF can be estimated using the CHA₂DS₂-VASc score, and anticoagulation is recommended if there is a score of 2 or more.
- Anticoagulation can be achieved through a number of means including warfarin, heparin, dabigatran, rivaroxaban edoxaban, and apixaban.

Treatment

(Atrial Fibrillation: Anticoagulants)

- Anticoagulation reduce the risk of stroke and is recommended in most people other than those at low risk of stroke or those at high risk of bleeding.
- The risk of stroke from non-valvular AF can be estimated using the CHA2DS2-VASc score, and anticoagulation is recommended if there is a score of 2 or more.
- Anticoagulation can be achieved through a number of means including warfarin, heparin, dabigatran, rivaroxaban edoxaban, and apixaban.

Treatment

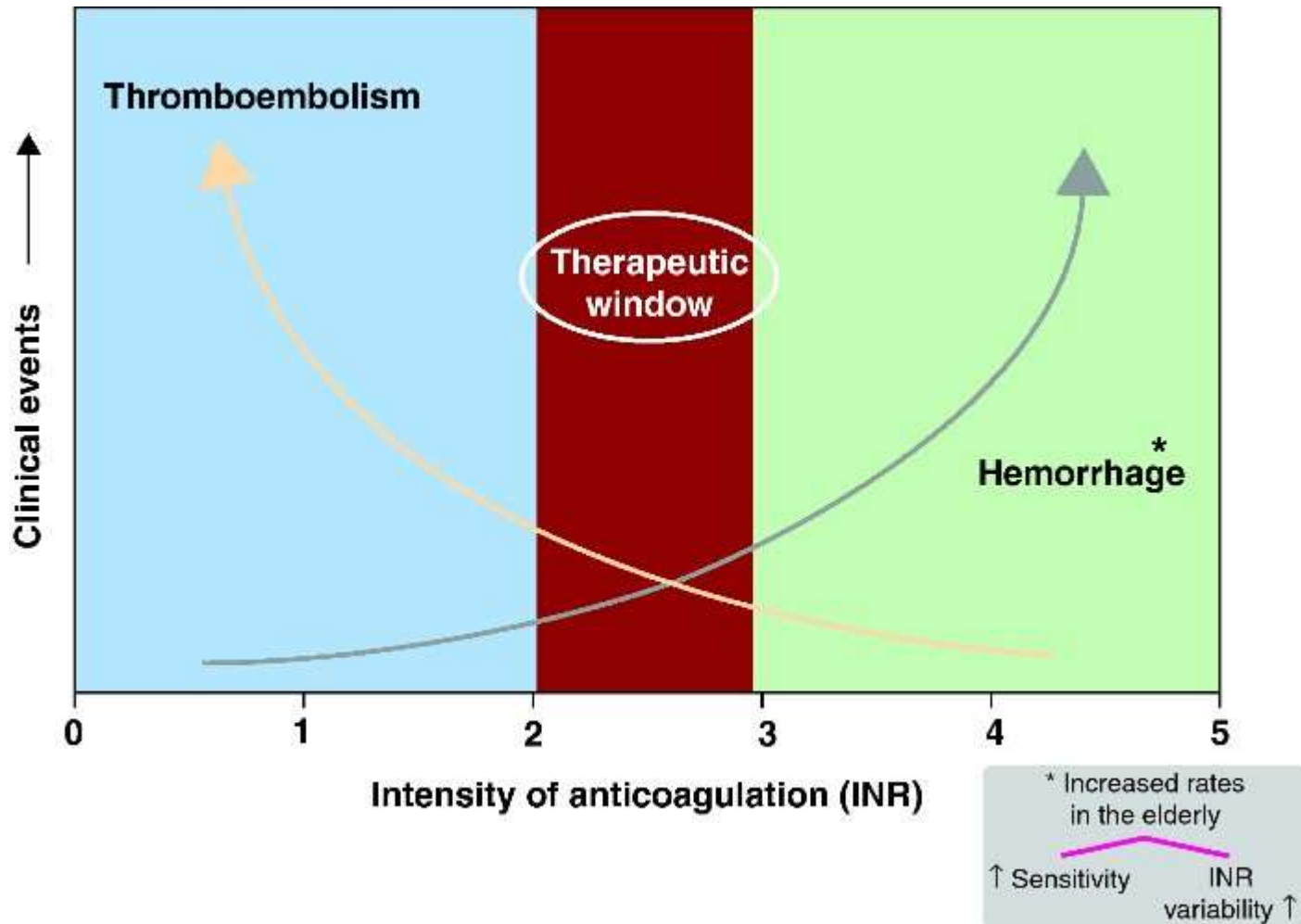
(Atrial Fibrillation: CHA2DS2-VASc Score)

Risk Factor	Score
<u>C</u> ongestive heart failure/LV dysfunction	1
<u>H</u> ypertension	1
<u>A</u> ge ≥ 75 y	2
<u>D</u> iabetes mellitus	1
<u>S</u> troke/TIA/TE	2
<u>V</u> ascular disease (prior myocardial infarction, peripheral artery disease, or aortic plaque)	1
<u>A</u> ge 65-74 y	1
<u>S</u> ex category (ie female gender)	1

LV = left ventricular; TE = thromboembolism. See Table 1 for expansion of other abbreviations.

Treatment

(Atrial Fibrillation: Therapeutic Window of Warfarin)



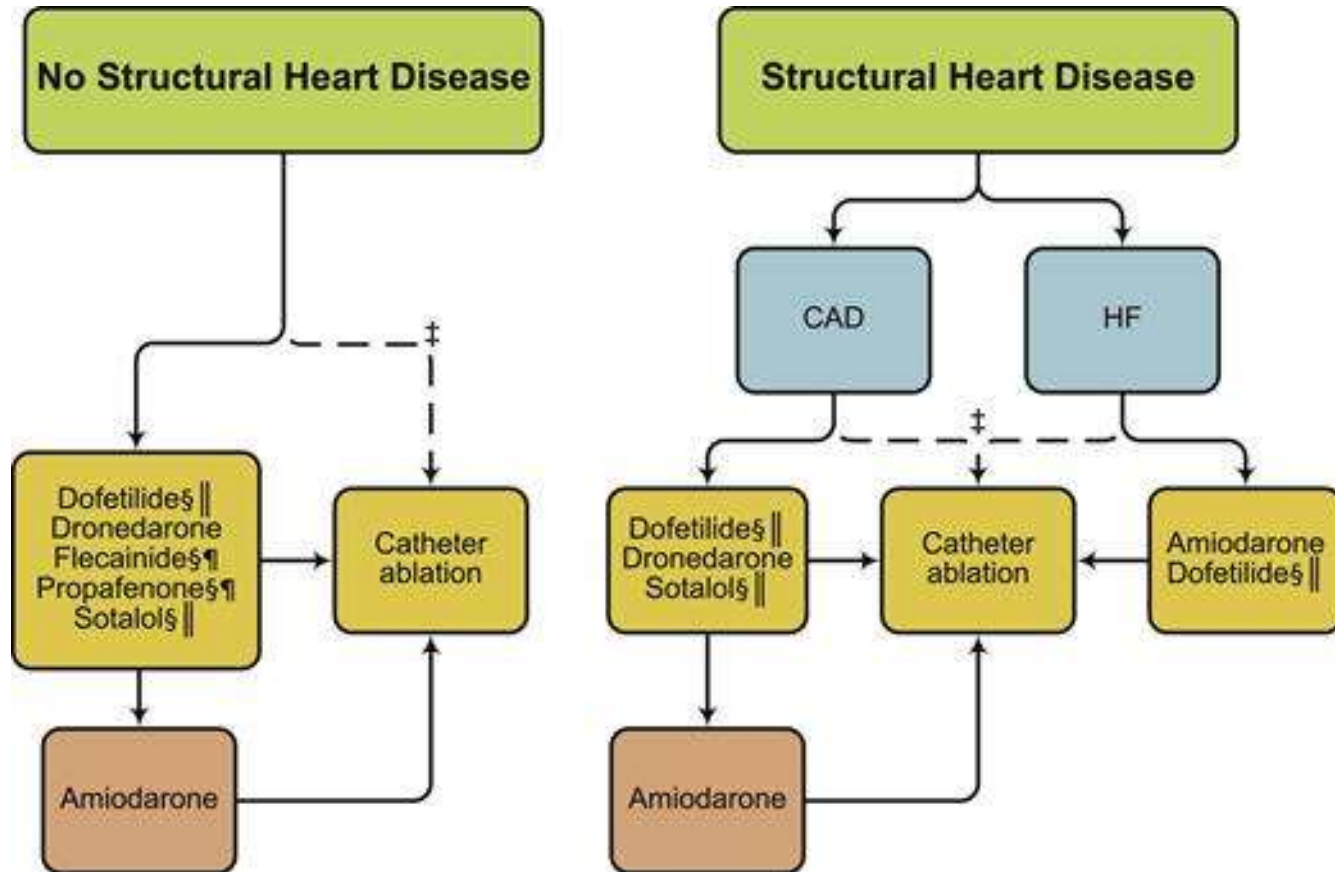
Treatment

(Atrial Fibrillation: Rate Versus Rhythm Control)

- Both methods have similar outcomes.
- *Rate control* lowers the heart rate closer to normal, usually 60 to 100 bpm, without trying to convert to a regular rhythm.
- *Rhythm control* tries to restore a normal heart rhythm in a process called cardioversion and maintains the normal rhythm with medications.
- Rhythm control is more important in the acute setting AF, whereas rate control is more important in the chronic phase.

Treatment

(Atrial Fibrillation: Strategies for Rhythm Control)



Treatment

(Atrial Fibrillation: Rate Control)

- Rate control to a target heart rate of 110 bpm is recommended in most people.
- Lower heart rates may be recommended in those with left ventricular hypertrophy or reduced left ventricular function.
- Rate control can be done with:
- Beta blockers (preferably the cardioselective beta blockers such as metoprolol, atenolol, bisoprolol, nebivolol)
- Non-dihydropyridine calcium channel blockers (e.g., diltiazem or verapamil)
- In those with chronic disease either beta blockers or calcium channel blockers are recommended.
- Amiodarone can be used when other agents are contraindicated or ineffective (particularly due to hypotension).

Treatment

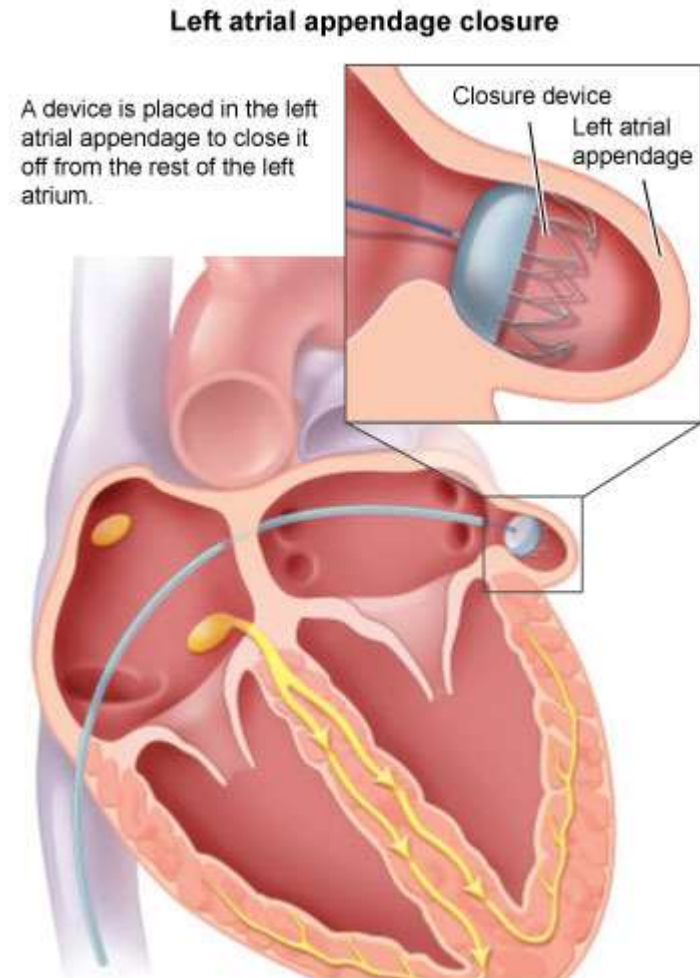
(Atrial Fibrillation: Cardioversion)

- *Electrical cardioversion* involves the restoration of normal heart rhythm through the application of a DC electrical shock
- *Chemical cardioversion* is performed with drugs, such as amiodarone, procainamide, dofetilide, ibutilide, propafenone, or flecainide.
- After successful cardioversion the heart may be in a stunned state, which means that there is a normal rhythm but restoration of normal atrial contraction has not yet occurred.

Treatment

(Atrial Fibrillation: Interventional Procedures)

- Surgery
- Ablation
- Left atrial appendage occlusion



Treatment

(Atrial Flutter)

- General treatment goals for symptomatic atrial flutter are similar to those for atrial fibrillation and include the following:
- Control of the ventricular rate
- Restoration of sinus rhythm
- Prevention of recurrent episodes or reduction of their frequency or duration
- Prevention of thromboembolic complications
- Minimization of adverse effects from therapy.

Prognosis

- The outlook for cardiac arrhythmias depends on the type of rhythm disturbance and whether the person has coronary artery disease, congestive heart failure, or some other heart muscle or other disorder.
- The prognosis for ventricular fibrillation is grave, and death follows quickly without emergency treatment.
- Most atrial arrhythmias have an excellent prognosis.
- The availability of permanent pacemakers, implanted cardioversion/defibrillation devices and effective medications has improved the prognosis for many people with serious cardiac arrhythmias.

Prophylaxis

- Once an acute arrhythmia has been terminated, ongoing treatment may be indicated to prevent recurrence.
- In general, patients with more frequent or disabling symptoms warrant some form of prevention.
- A variety of drugs including simple AVN nodal blocking agents such as beta-blockers and verapamil, as well as antiarrhythmic may be used, usually with good effect, although the risks of these therapies need to be weighed against potential benefits.
- Radiofrequency ablation has revolutionized the treatment of tachycardia caused by a reentry pathway.

Abbreviations

AF - atrial fibrillation

AV - atrio-ventricular

AVB - atrioventricular block

AVNRT - atrioventricular nodal
reentrant tachycardia

AF - atrial fibrillation

CAD - coronary artery disease

ECG - electrocardiogram

IHR – heart rate

CD - International Classification of
Diseases

ICVD - Implantable Cardioverter
Defibrillator

LAFB - Left anterior fascicular block

LBBB - left bundle branch block

LPFB - left posterior fascicular block LV -
left ventricle

MDP - membrane diastolic potential

MI - myocardial infarction

PSVT -permanent SVT

PVCs – premature ventricular contractions

RAAS - renin–angiotensin–aldosterone
system

RBBB - Right bundle branch block SVT-
supraventricular Tachycardia

SA node –sinoatrial node

Th - threshold

VF – ventricular fibrillation

VT – ventricular tachycardia

WPW - Wolff–Parkinson–White syndrome

Diagnostic and treatment guidelines

- [ACC/AHA/ESC Guidelines for the Management of Patients With Supraventricular Arrhythmias](#)
- [ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities](#)
- [2012 focused update of the ESC Guidelines for the management of atrial fibrillation](#)
- [2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: Executive Summary](#)
- [2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia](#)
- [2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death](#)