

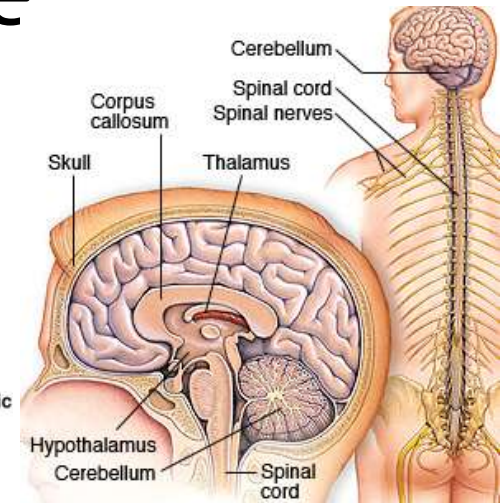
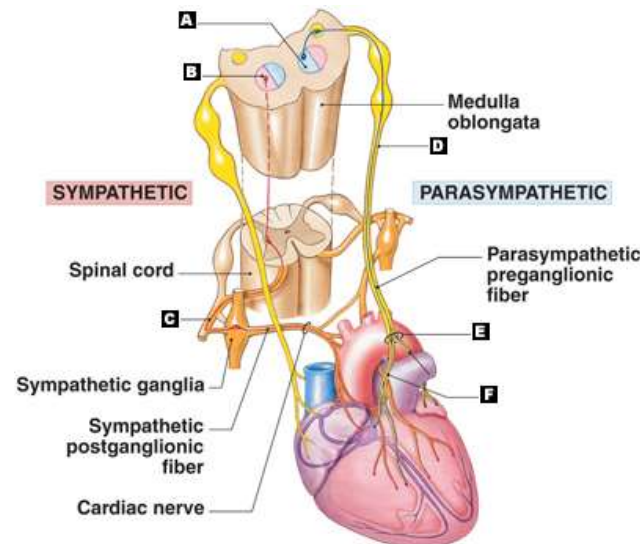
# Heart rhythm disturbances

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

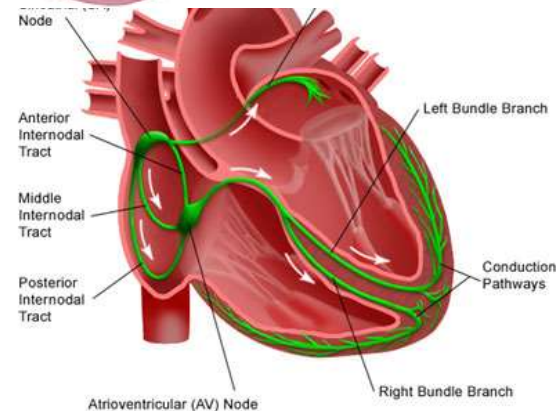
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# Plan of the Lecture

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



the Heart



# Definition

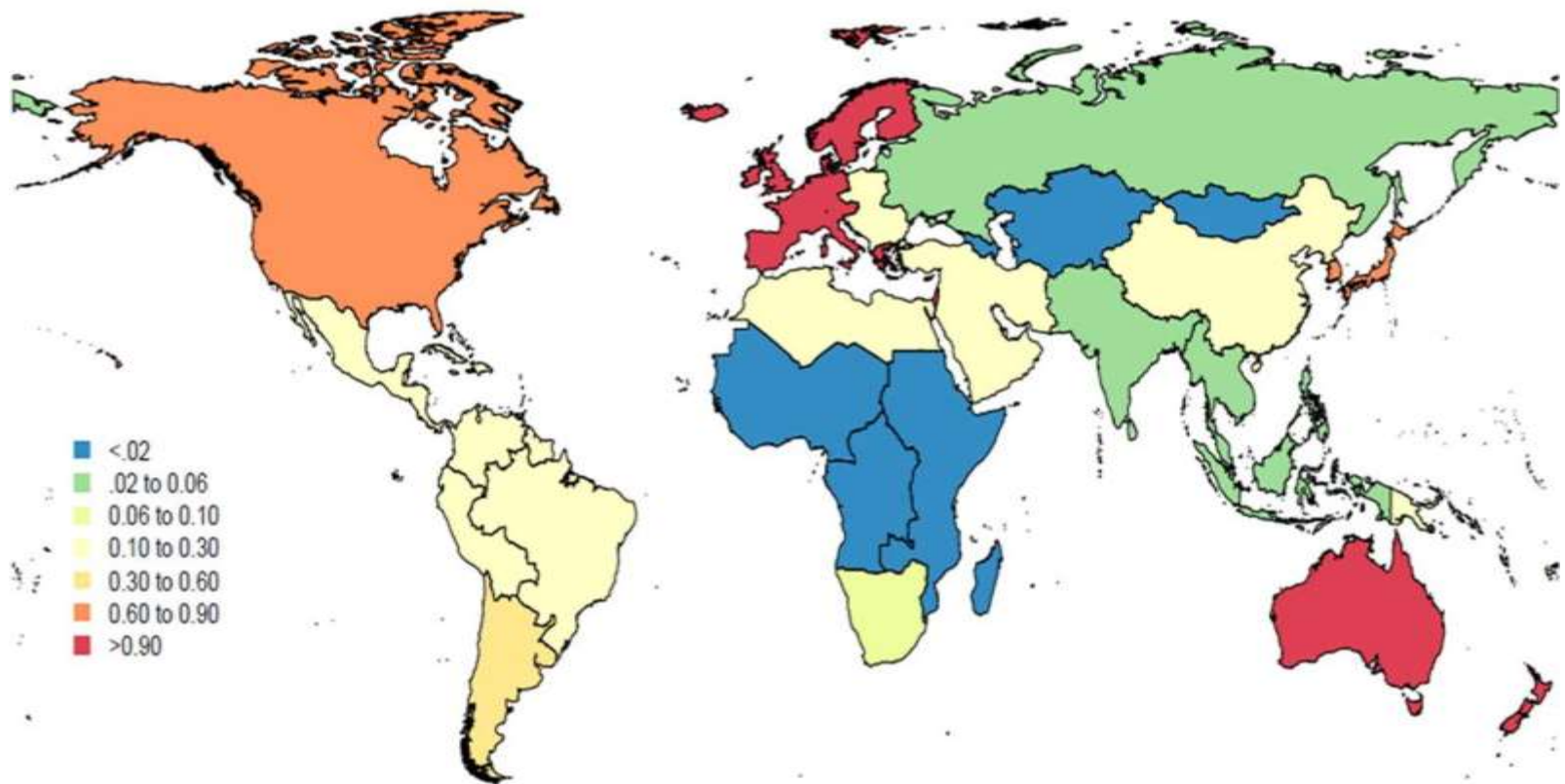
Heart rhythm disturbances (cardiac arrhythmia, cardiac dysrhythmia, irregular heartbeat) is a group of conditions with abnormal variation from the normal heartbeat (irregular, too fast, or too slow) via congenital (e.g., accessory atrioventricular connection, hereditary ion channelopathies) or acquired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

# Epidemiology

- Arrhythmias are relatively common, often repetitive, occasionally persistent, and rarely life threatening .
- The precipitants of arrhythmias vary with age (most serious arrhythmias affect people older than 60), gender, and associated comorbidity.
- While 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 arrhythmia can cause sudden cardiac death (100,000 people in the UK every year).

# Epidemiology

## (Worldwide Distribution of Atrial Fibrillation)



# 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

# Risk Factors and Etiology

- Functional violations and influences (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, myocardiodystrophy, 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, some dietary supplements and herbal treatments).
- 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 three basic mechanisms—enhanced or suppressed automaticity, triggered activity, or re-entry.
- 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.

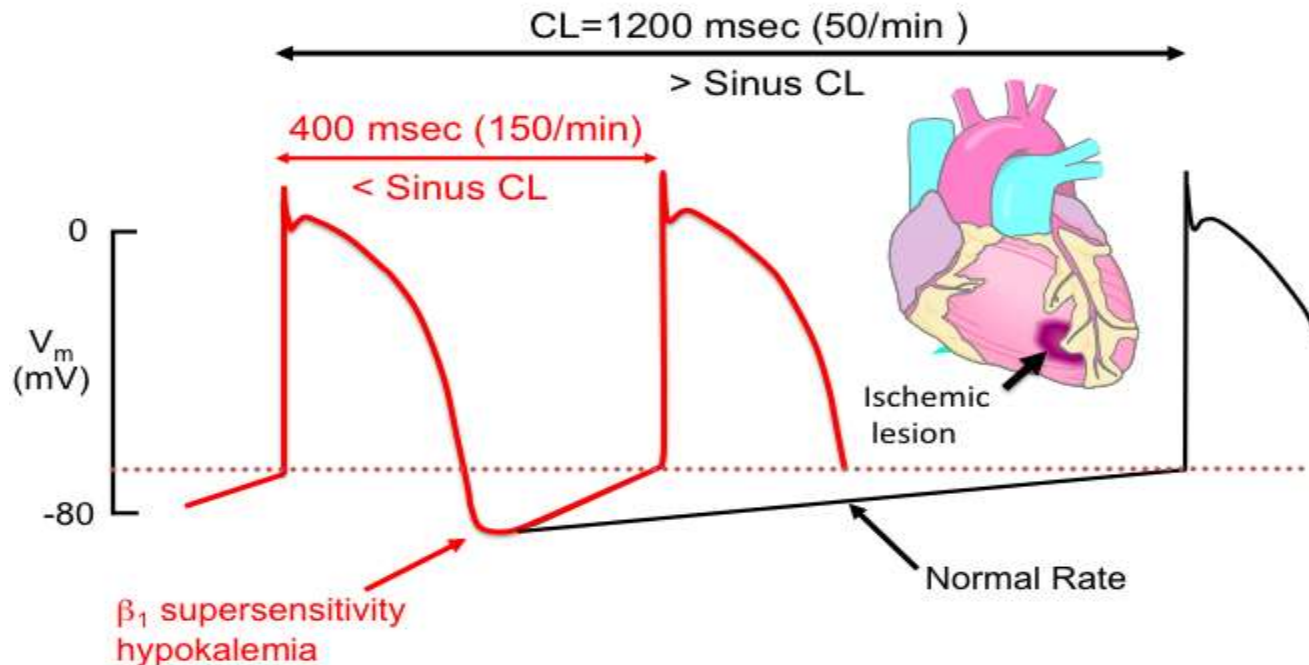


# Mechanisms

## (Bradyarrhythmias and Tachyarrhythmias)

- Bradyarrhythmias typically arise from disturbances in impulse formation at the level of the sinoatrial node or from disturbances in impulse propagation at any level, including exit block from the sinus node, conduction block in the 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 (Enhanced Automaticity)

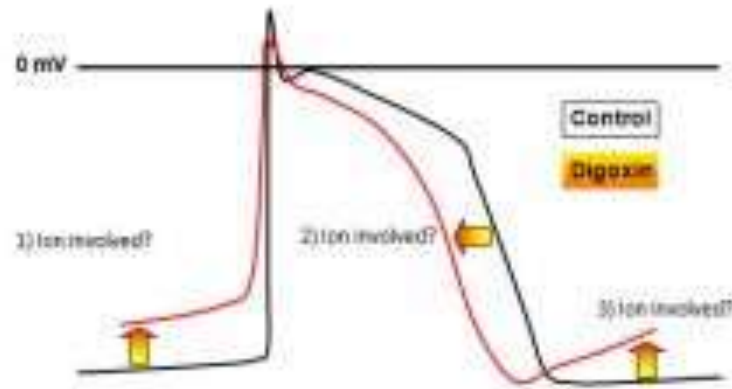


Lowered action potential makes SA node more irritable; makes arrhythmias more likely - Increased intracellular calcium causes earlier phase 4 upstroke.

# Mechanisms

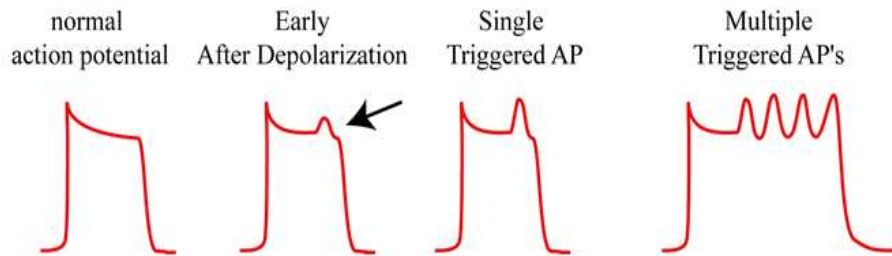
## (Suppressed Automaticity)

Effects of Digoxin on Cardiac Action Potential

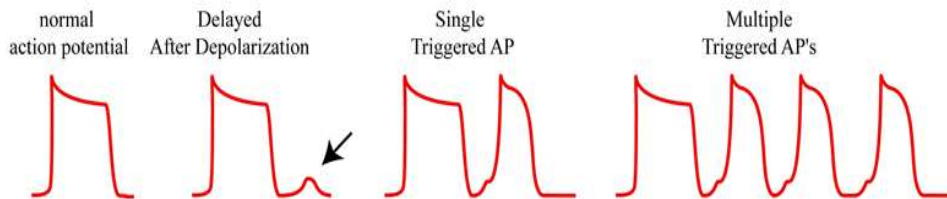


Lowered action potential due to digoxin makes sinoatrial node (SA node) more irritable; makes arrhythmias more likely - Increased intracellular calcium causes earlier phase 4 upstroke.

# Mechanisms (Triggered Activity)



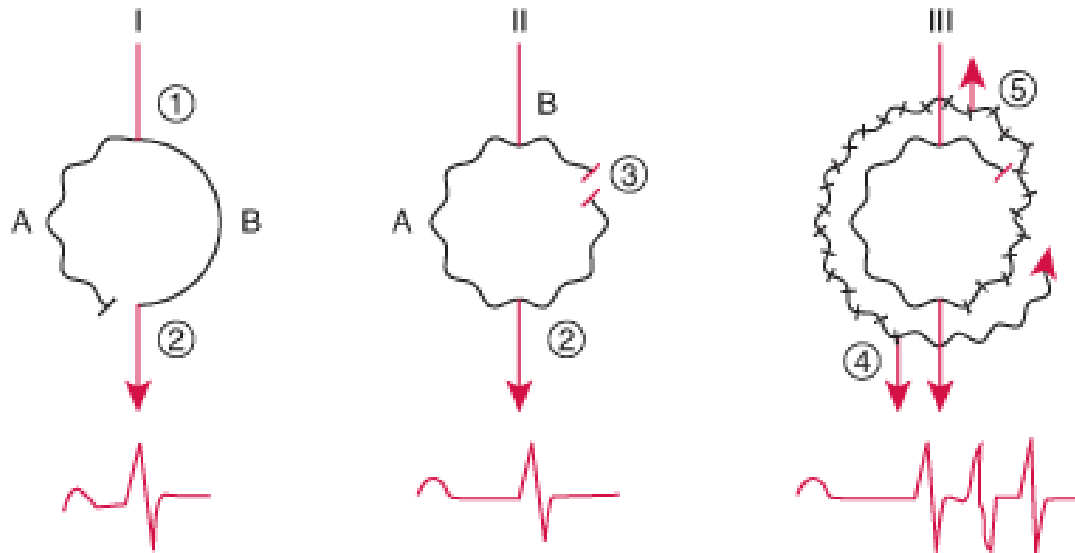
**Early After Depolarization:** Sometimes, during the plateau phase of the action potential, a spontaneous depolarization may occur. This is often the case when there is too much calcium in the cell. These depolarizations may reach threshold and induce, too soon, a new action potential.



**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

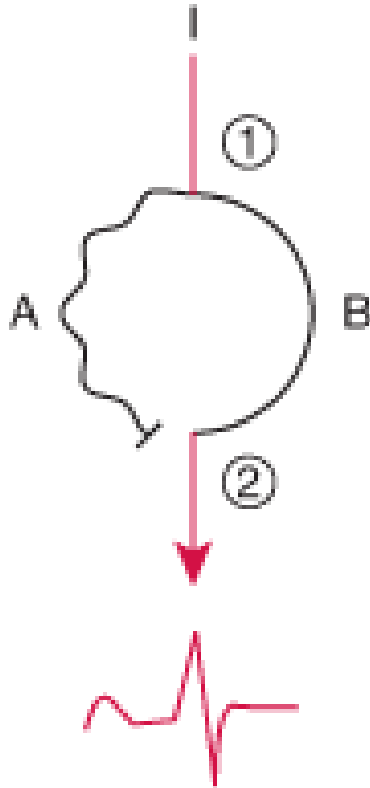
## (Mechanism of Typical Reentry: 1)



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

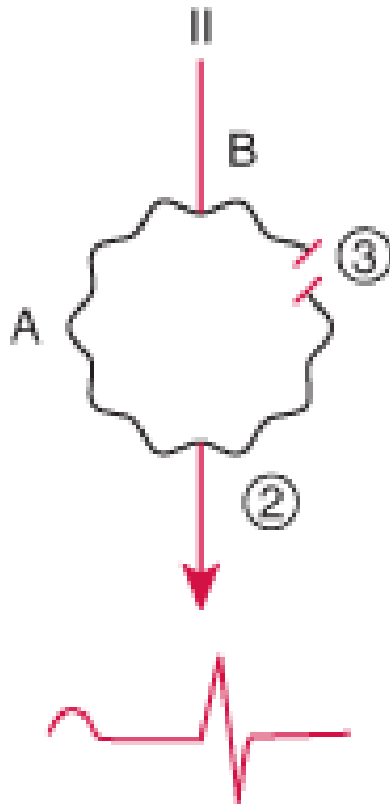
## (Mechanism of Typical Reentry: 2)



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

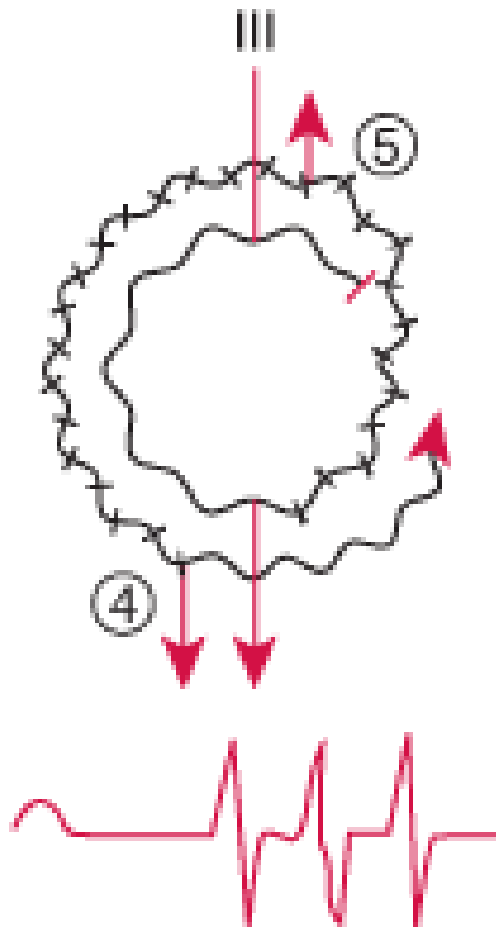
## (Mechanism of Typical Reentry: 3)



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

## (Mechanism of typical 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.



# Classification

## (International Classification of Diseases (ICD): 1)

### 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): 2)

### 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)

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

**The discussion subject of the next lecture**

# Classification

## (Lown-Wolf Grading of PVCs)

Grade 0	No ventricular extrasystoles/ectopics
Grade I	Unifocal and infrequent PVCs; <30 PVCs per hour
Grade II	Unifocal and frequent PVCs, $\geq 30$ PVCs per hour
Grade III	Multifocal
Grade IVA	2 consecutive beats (couplets)
Grade IVB	$\geq 3$ 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.

# 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)

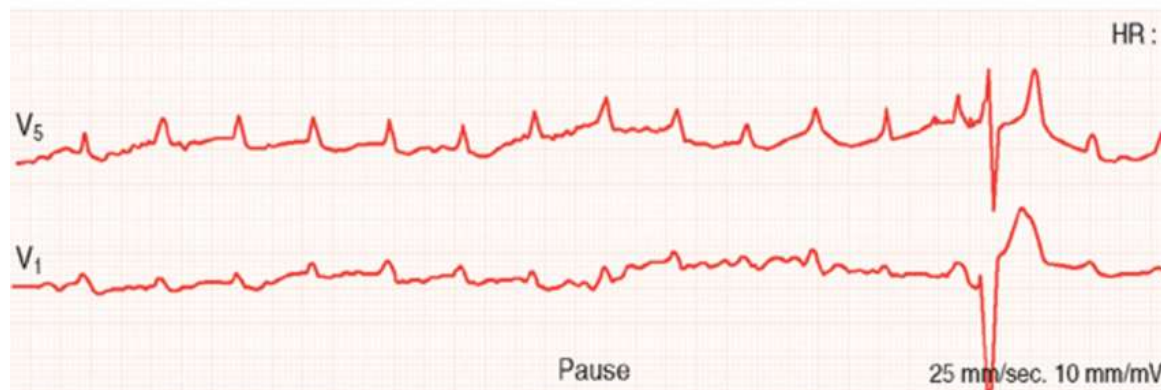
- Because there are a number of tests available for the diagnosis of cardiac arrhythmias, it is important to proceed with a stepwise approach.
- The goal is to obtain a correlation between symptoms and the underlying arrhythmia and initiation of appropriate therapy.
- Additional testing is usually advocated 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.

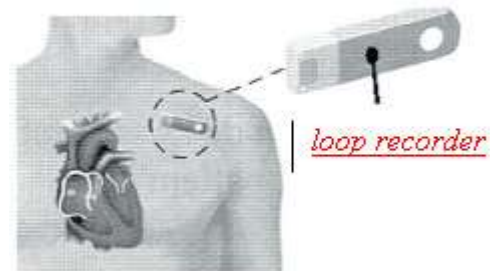
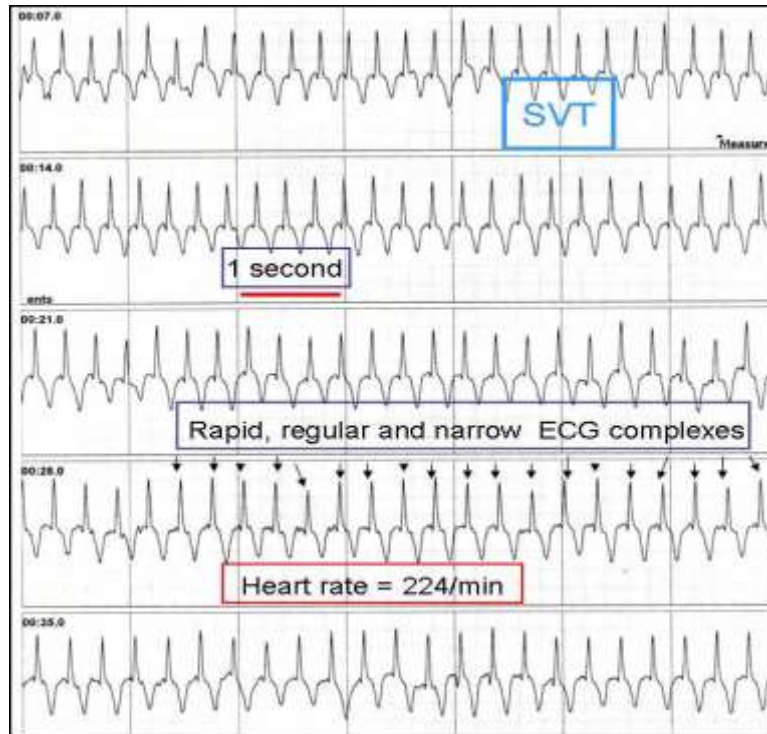
# Diagnosis (Holter Monitoring)



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

## (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

## (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).

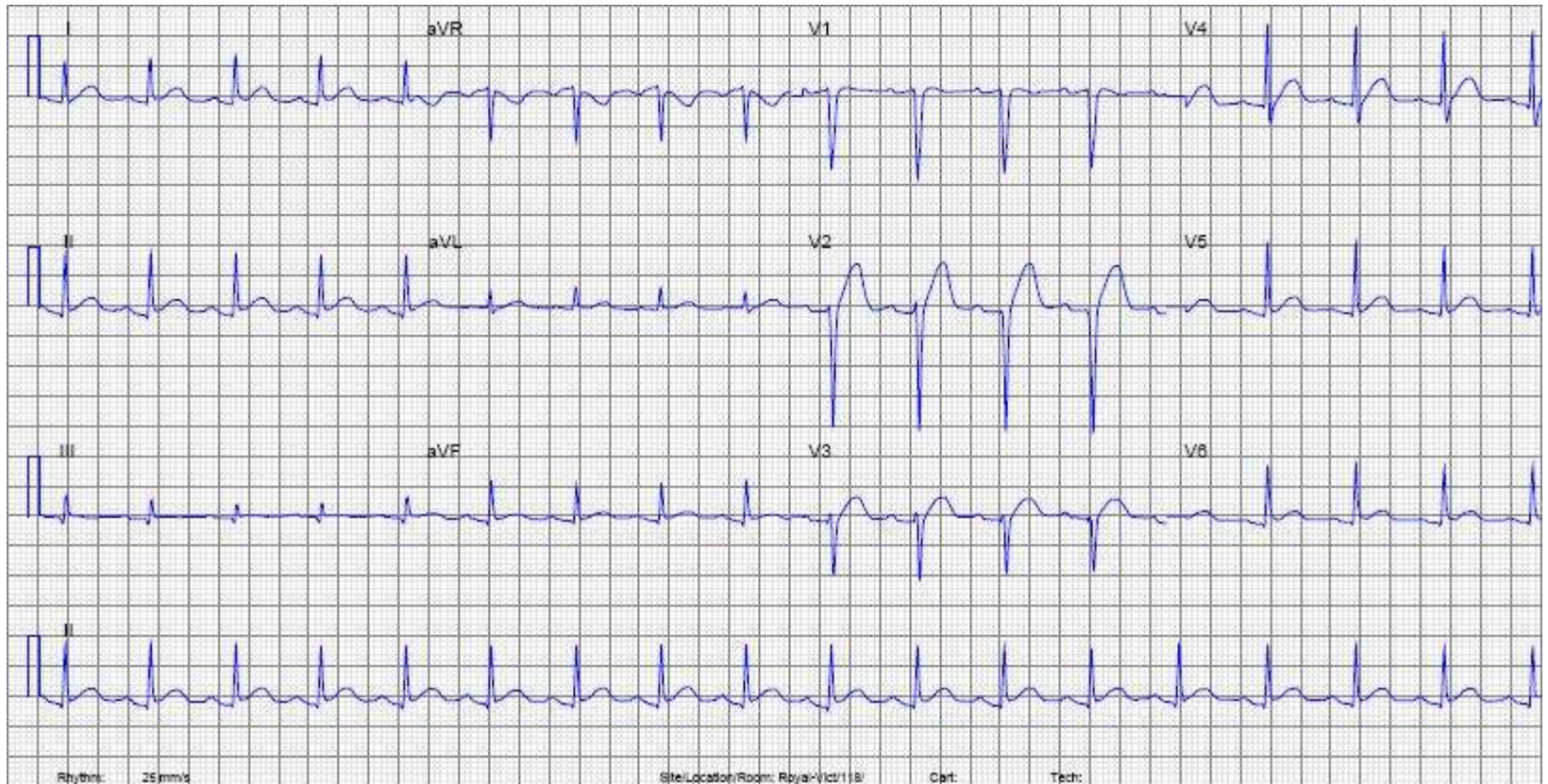
# Diagnosis

## (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.

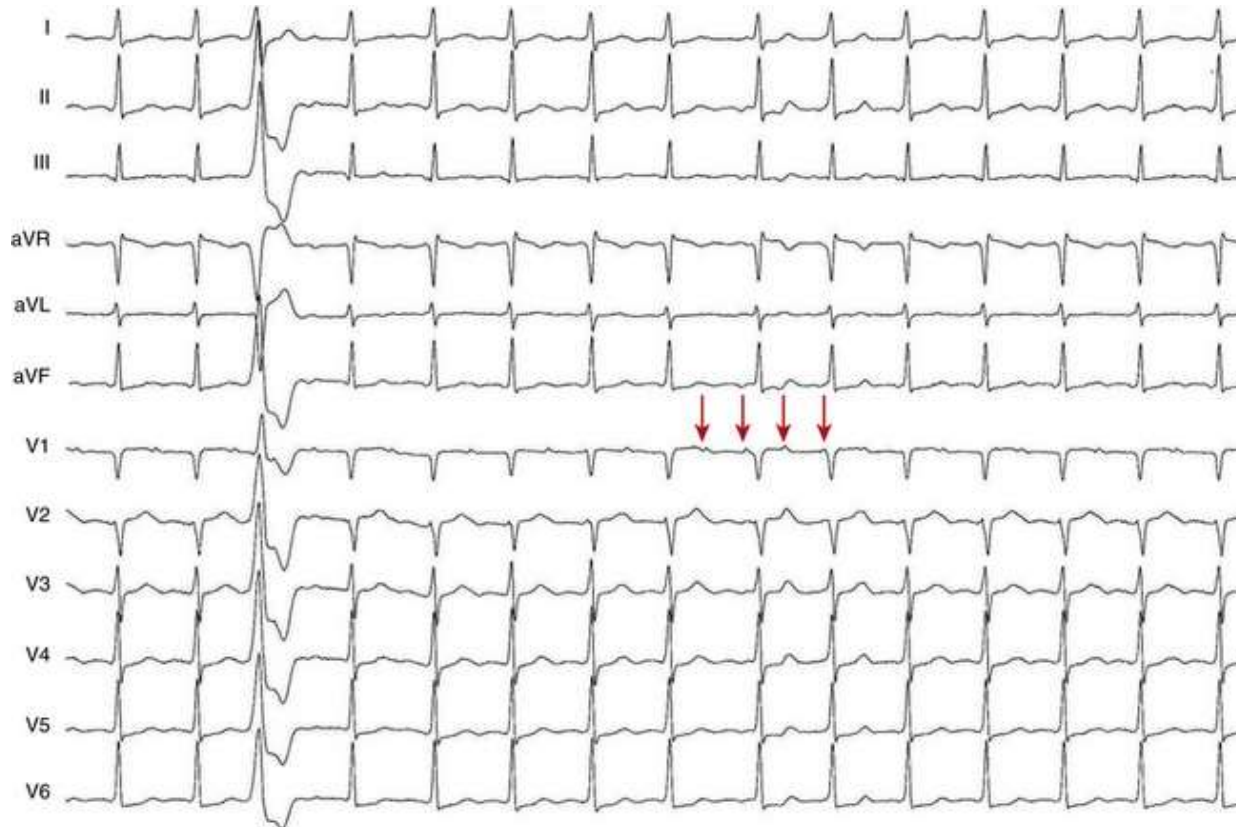
# Diagnosis

## (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.

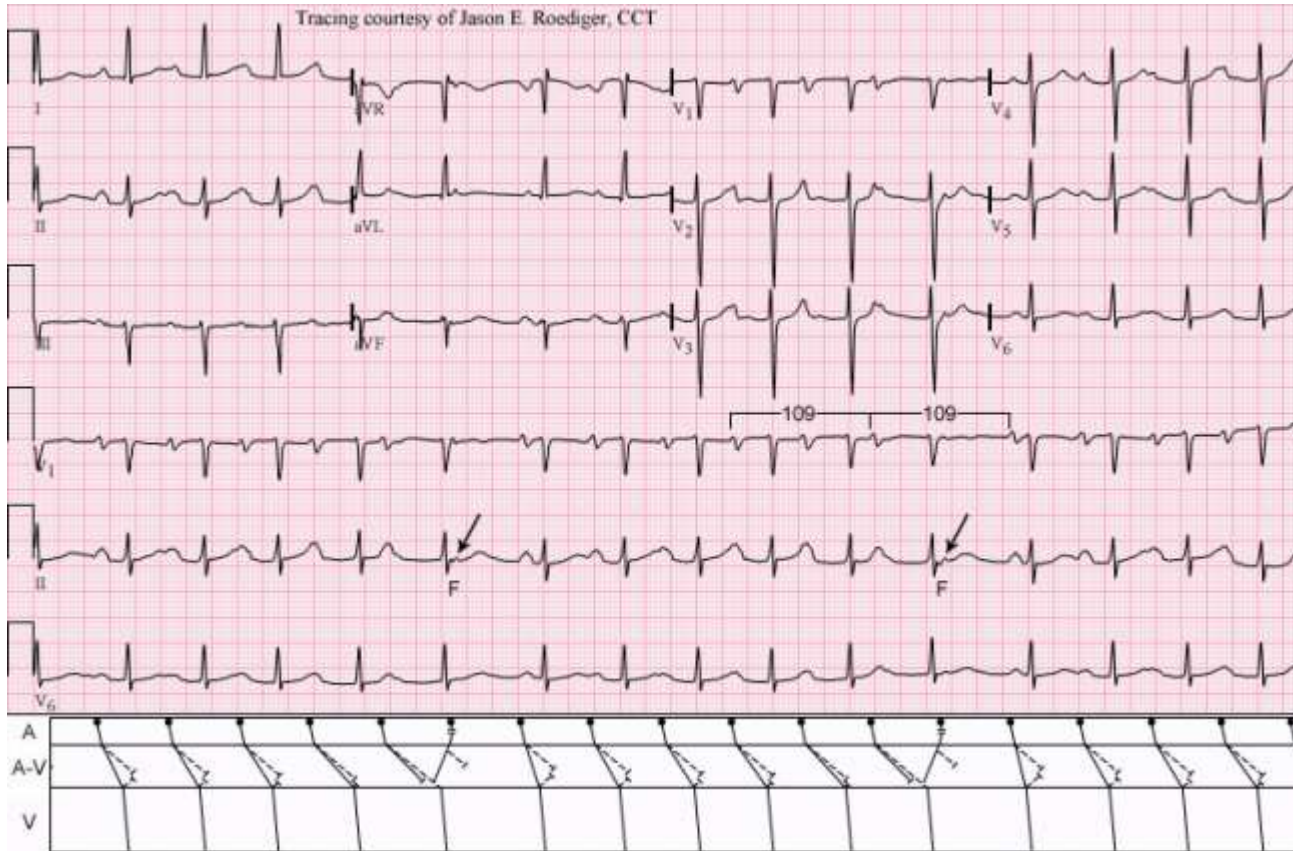
# Diagnosis (Atrial Tachycardia)



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).

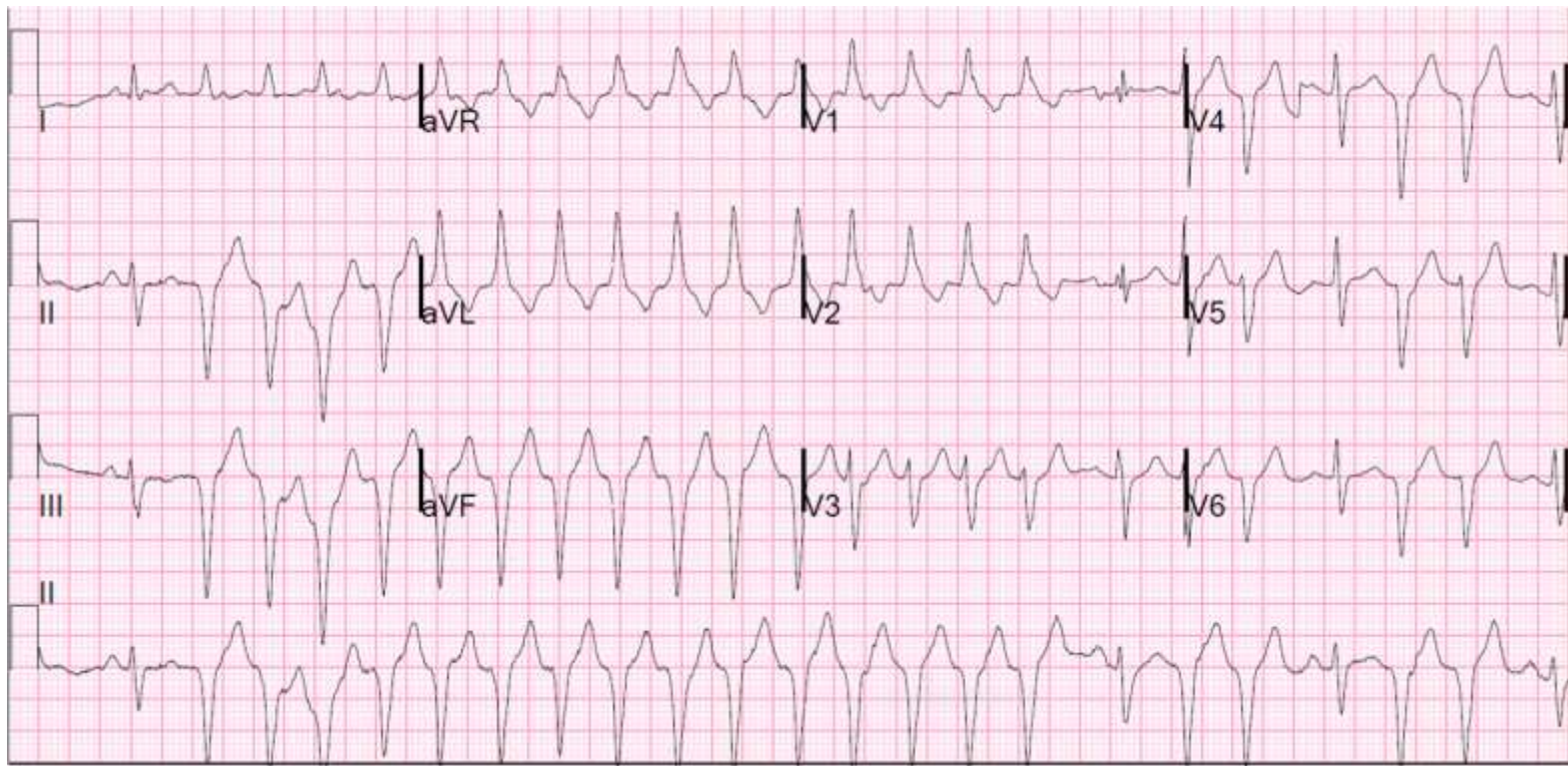
# Diagnosis

## (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

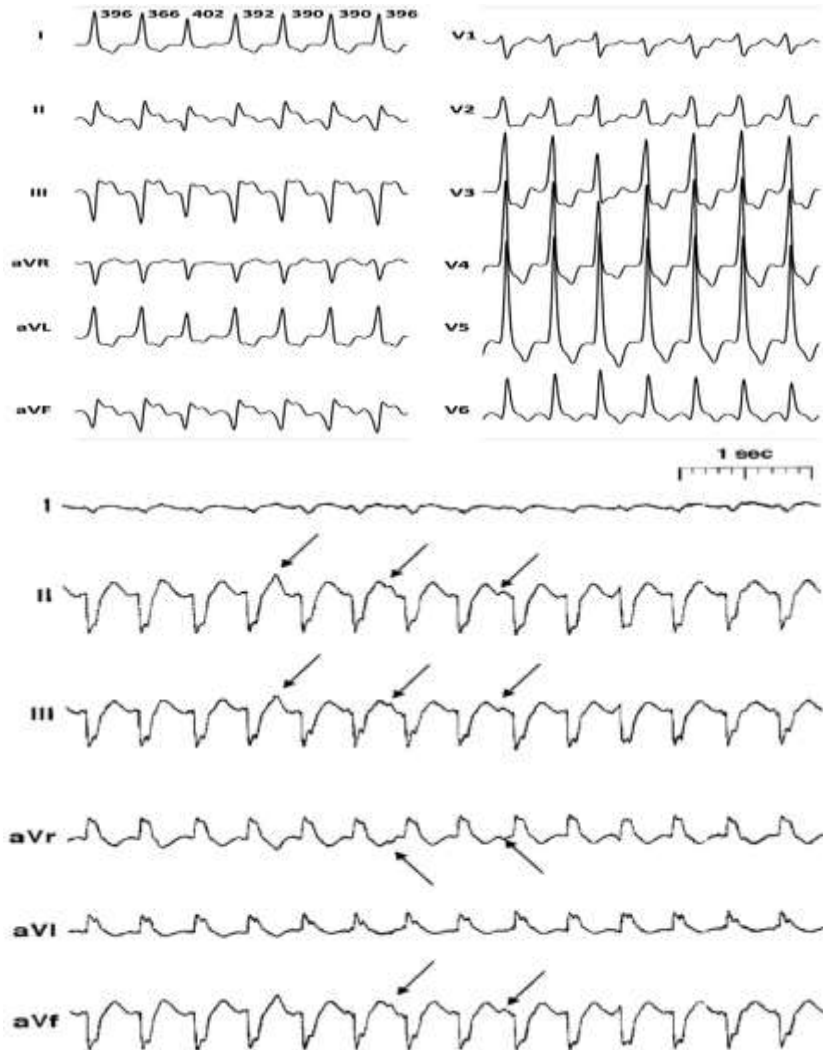
(Regular Wide QRS Complex Tachyarrhythmia)



Regular monomorphic wide complex tachycardia with no P-waves.

# Diagnosis

## (Narrow and Wide QRS Complexes)

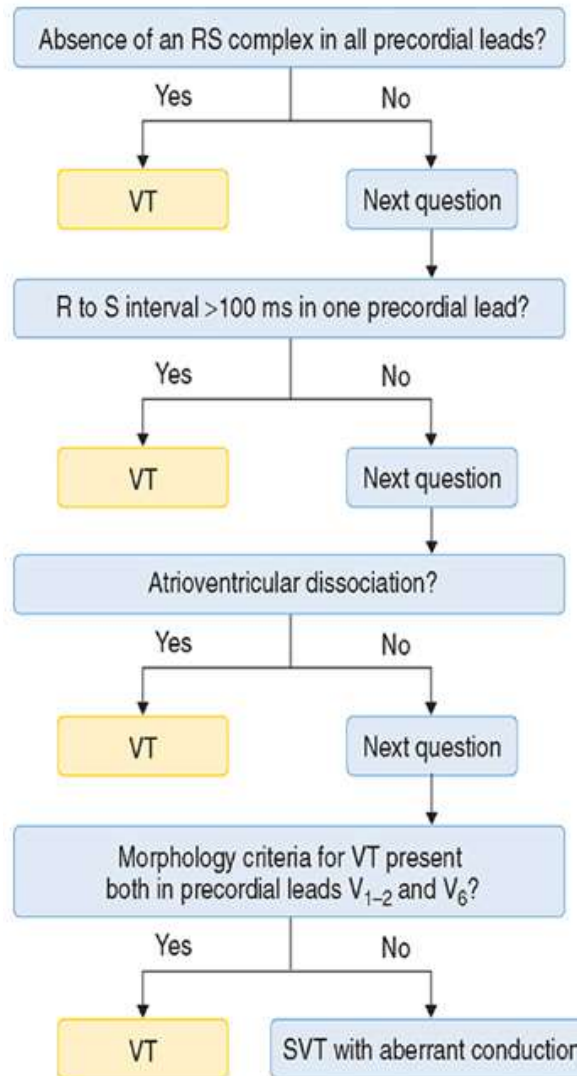


A narrow QRS complex ( $< 0.12$  sec) indicates a supraventricular origin (above the His bundle bifurcation).

A wide QRS complex ( $\geq 0.12$  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)



# 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	$\beta$ -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

## (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.<sup>1</sup>
- 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

## (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

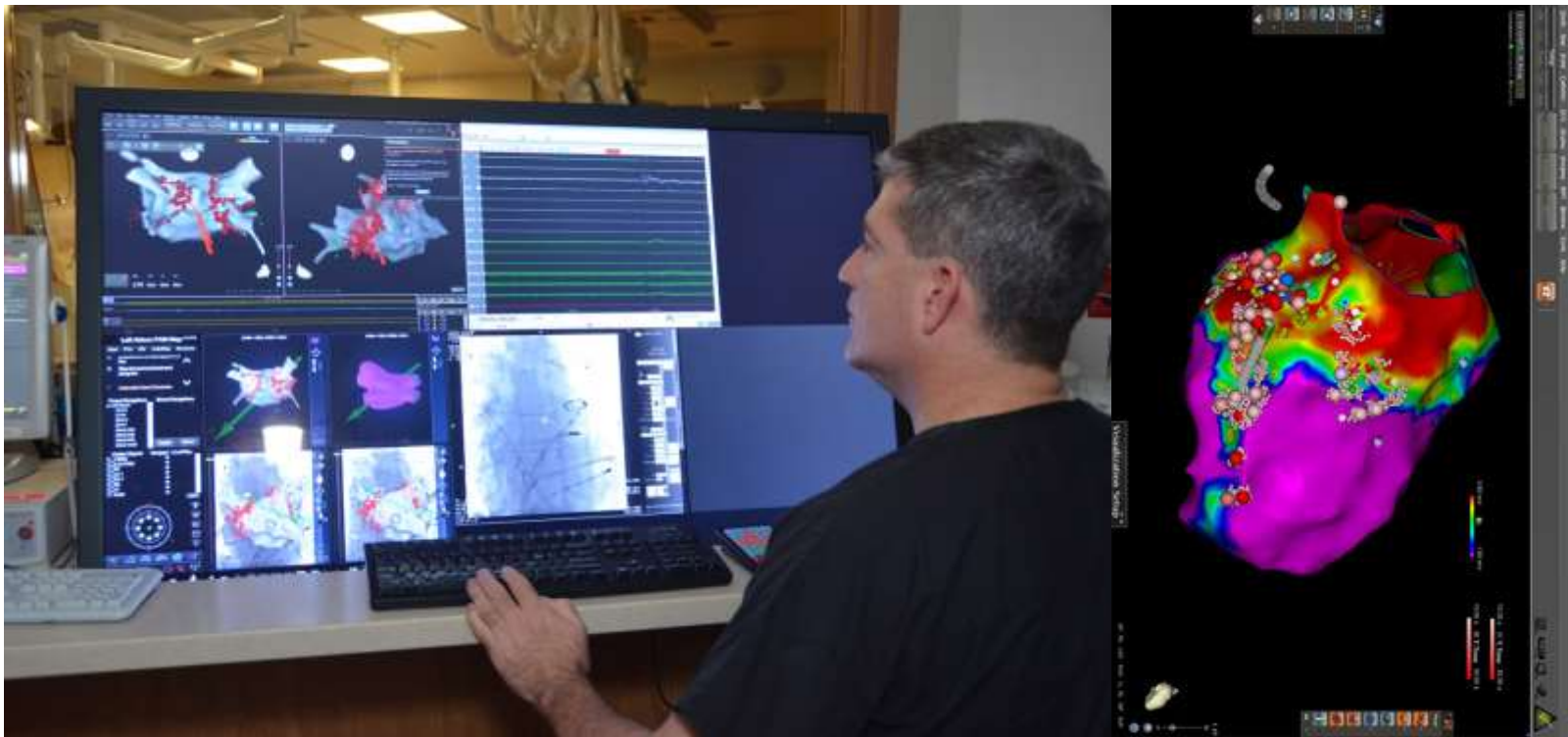
## (Ventricular tachycardia (VT): 2)

- 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 (Catheter Ablation)



# Treatment (Remote Magnetic Navigation)



# Treatment

(Implantable Cardioverter Defibrillator (ICVD))



# 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 AV 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

AV - atrio-ventricular

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

LV - left ventricle

MDP - membrane diastolic potential

MI - myocardial infarction

PSVT -permanent SVT

SVT- supraventricular Tachycardia

Th - threshold

SA node –sinoatrial node

PVCs – premature ventricular contractions

# Diagnostic and treatment guidelines

- [ACC/AHA/ESC Guidelines for the Management of Patients With Supraventricular Arrhythmias](#)
- [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](#)