

Arrhythmias

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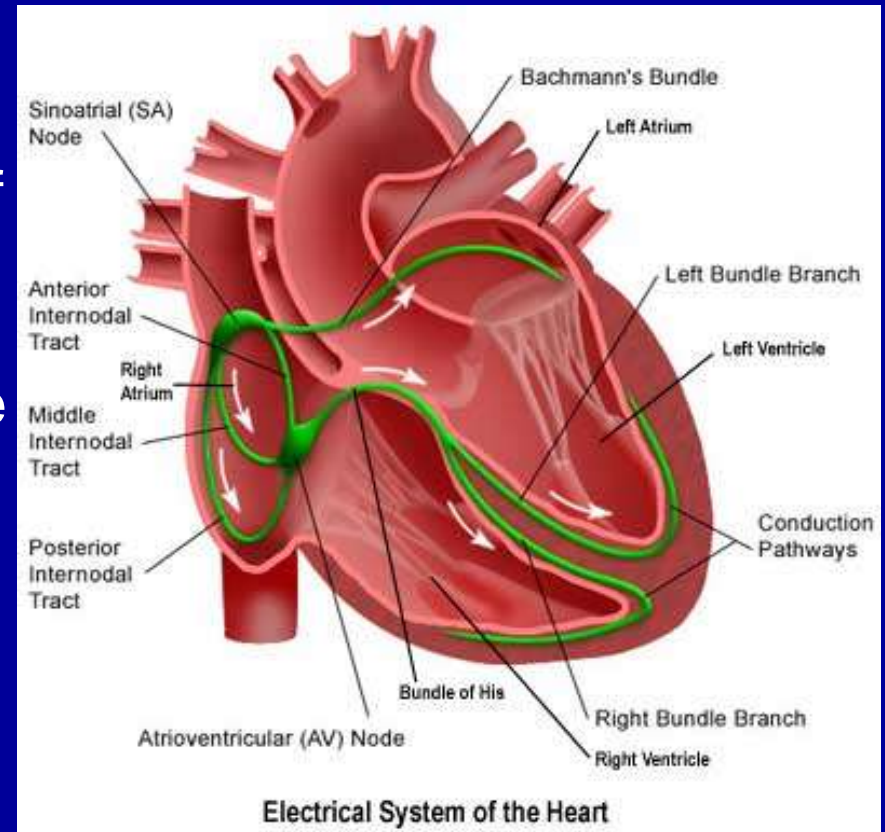
Definition of Arrhythmia

Cardiac arrhythmia (also dysrhythmia) is a term for any of a large and heterogeneous group of conditions in which there is abnormal electrical activity in the heart.

That is the Origin, Rate, Rhythm, Conduct velocity and/or sequence of heart activation are abnormal.

Normal electrical activity in the heart

Each heart beat originates as an electrical impulse from a small area of tissue in the right atrium of the heart called the **sinus node** or Sino-atrial node or SA node. The impulse initially causes both of the atria to contract, then activates the **atrioventricular (or AV) node** which is normally the only electrical connection between the atria and the ventricles, which can be called as main pumping chambers.



The impulse then spreads through both ventricles via the **Bundle of His** and the **Purkinje fibres** causing a synchronised contraction of the heart muscle, and thus, the pulse

Sinus Rhythm Criteria:



1. **Every** QRS complex is preceded by a P-wave
2. **P-waves** appear normal, that is they are of sinus node origin:
 - A. Normal Morphology:
 1. P-wave duration **< 0.12 sec**
 2. P-wave height **< 2.5 mm**
 - B. Normal Axis:
 1. P-waves is upright in **leads II, III & aVF**
 2. P-waves is negative in lead aVR

Etiology of Arrhythmias

- **Congenital - Present at birth due to genetics, or conditions during the perinatal environment**
- **Violations of neurohumoral (including endocrine) regulation of the heart (disregulatory arrhythmias)**
- **Organic (congenital and acquired) heart defects**
- **Other system disease**
- **Electrolyte disturbance and acid-base imbalance**
- **Drugs, toxic and chemical substances**
- **Unknown origin**



Mechanisms of Arrhythmia

- reentry (most common)
- automaticity
- triggered activity

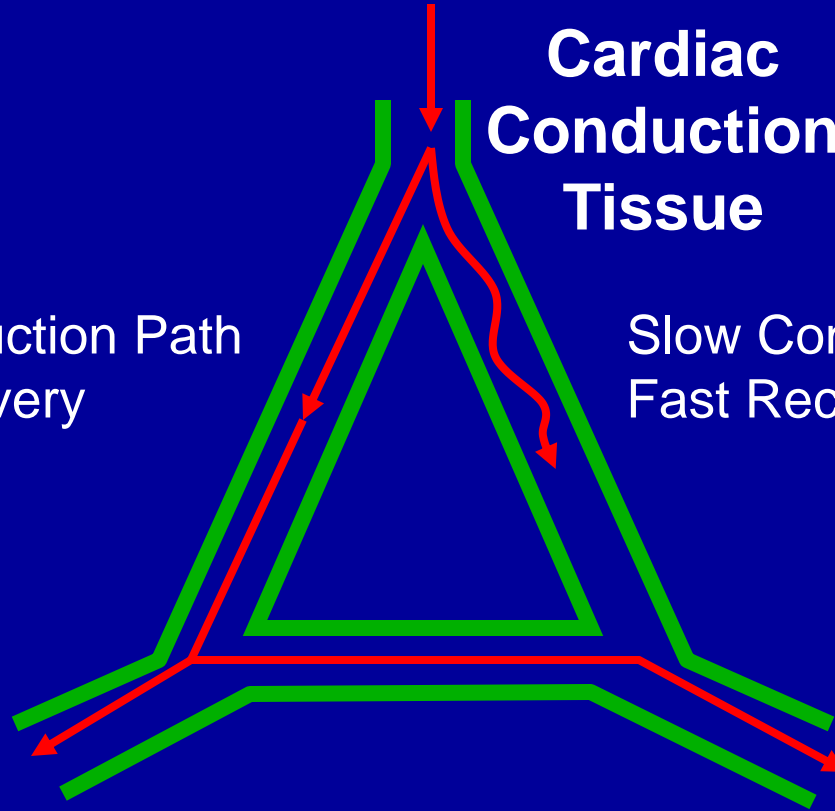
Reentry Requires...

Electrical Impulse

Cardiac
Conduction
Tissue

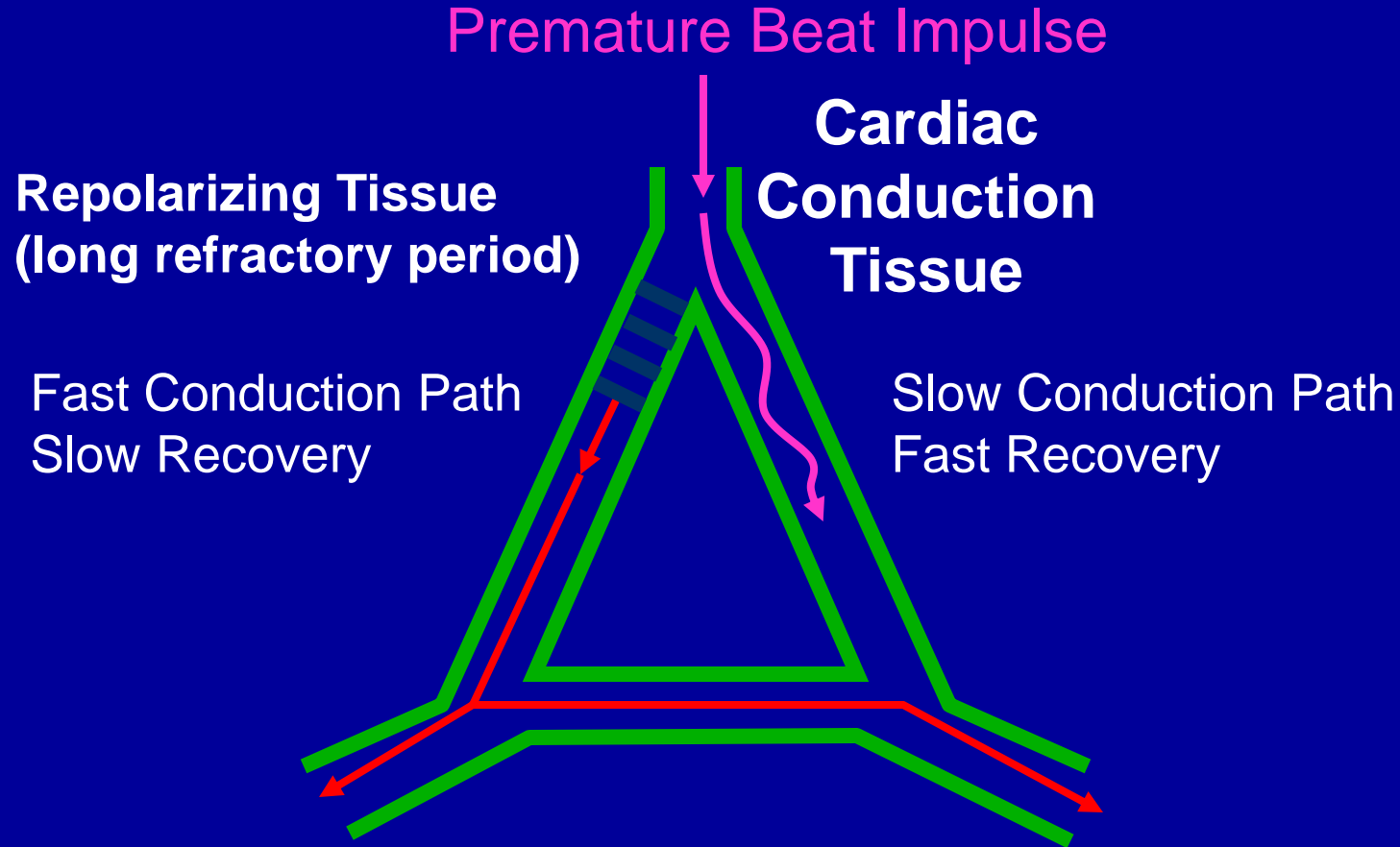
Fast Conduction Path
Slow Recovery

Slow Conduction Path
Fast Recovery



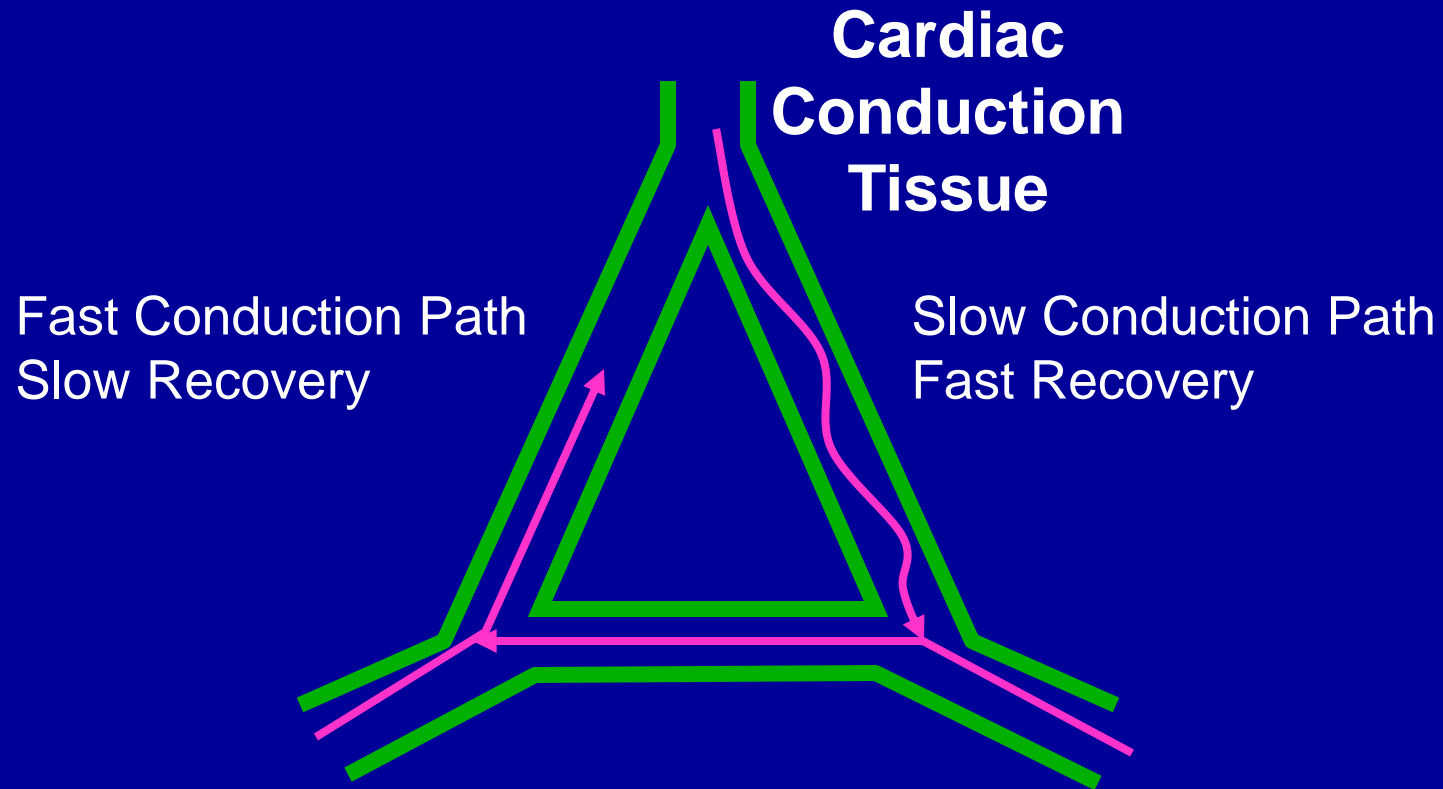
1. 2 distinct pathways that come together at beginning and end to form a loop.
2. A unidirectional block in one of those pathways.
3. Slow conduction in the unblocked pathway.

Reentry Mechanism



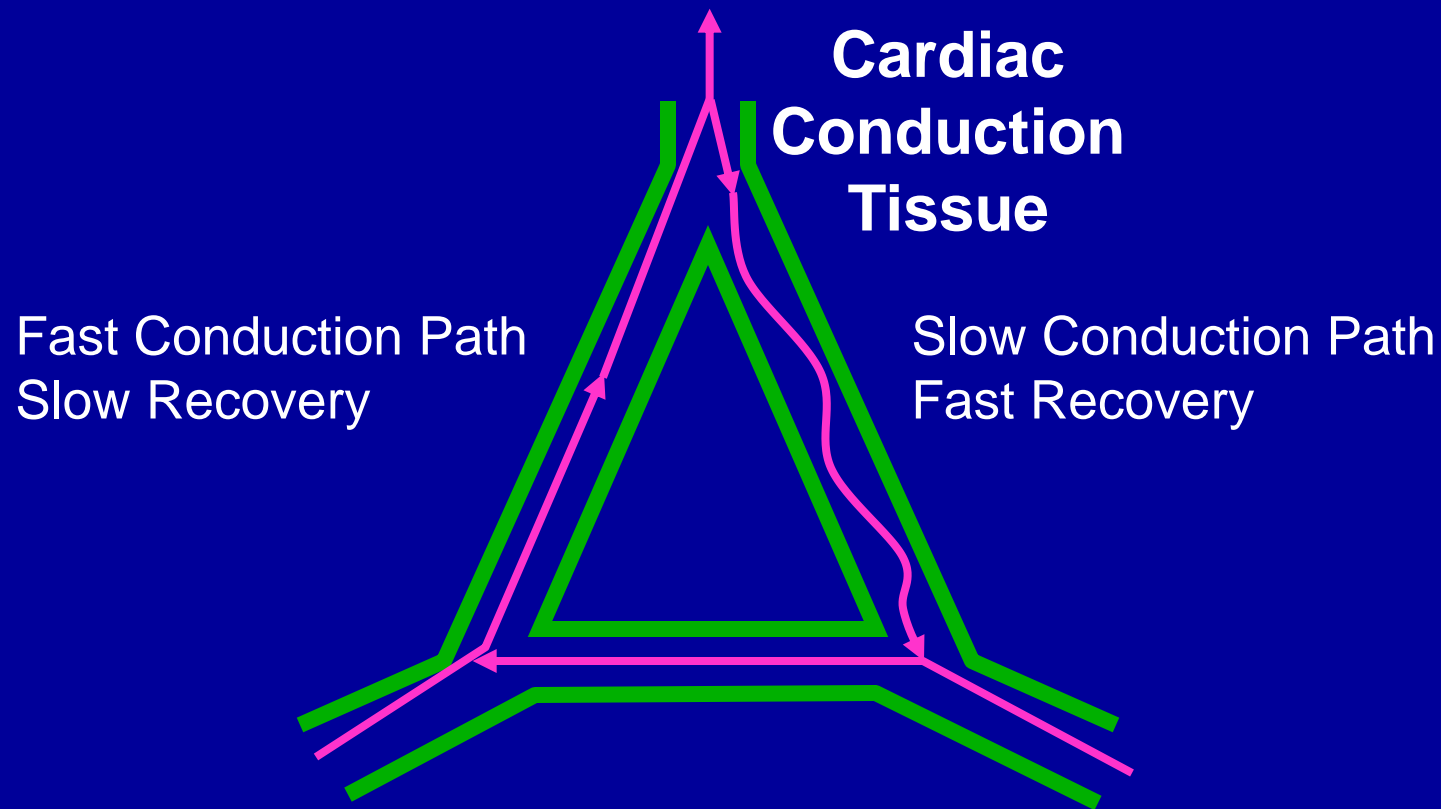
1. An arrhythmia is triggered by a premature beat
2. The fast conducting pathway is blocked because of its long refractory period so the beat can only go down the slow conducting pathway

Reentry Mechanism



3. The wave of excitation from the premature beat arrives at the distal end of the fast conducting pathway, which has now recovered and therefore travels retrogradely (backwards) up the fast pathway

Reentry Mechanism



4. On arriving at the top of the fast pathway it finds the slow pathway has recovered and therefore the wave of excitation 're-enters' the pathway and continues in a 'circular' movement. This creates the re-entry circuit

Reentry Circuits

AV Nodal Reentry

- SVT

Atrial Reentry

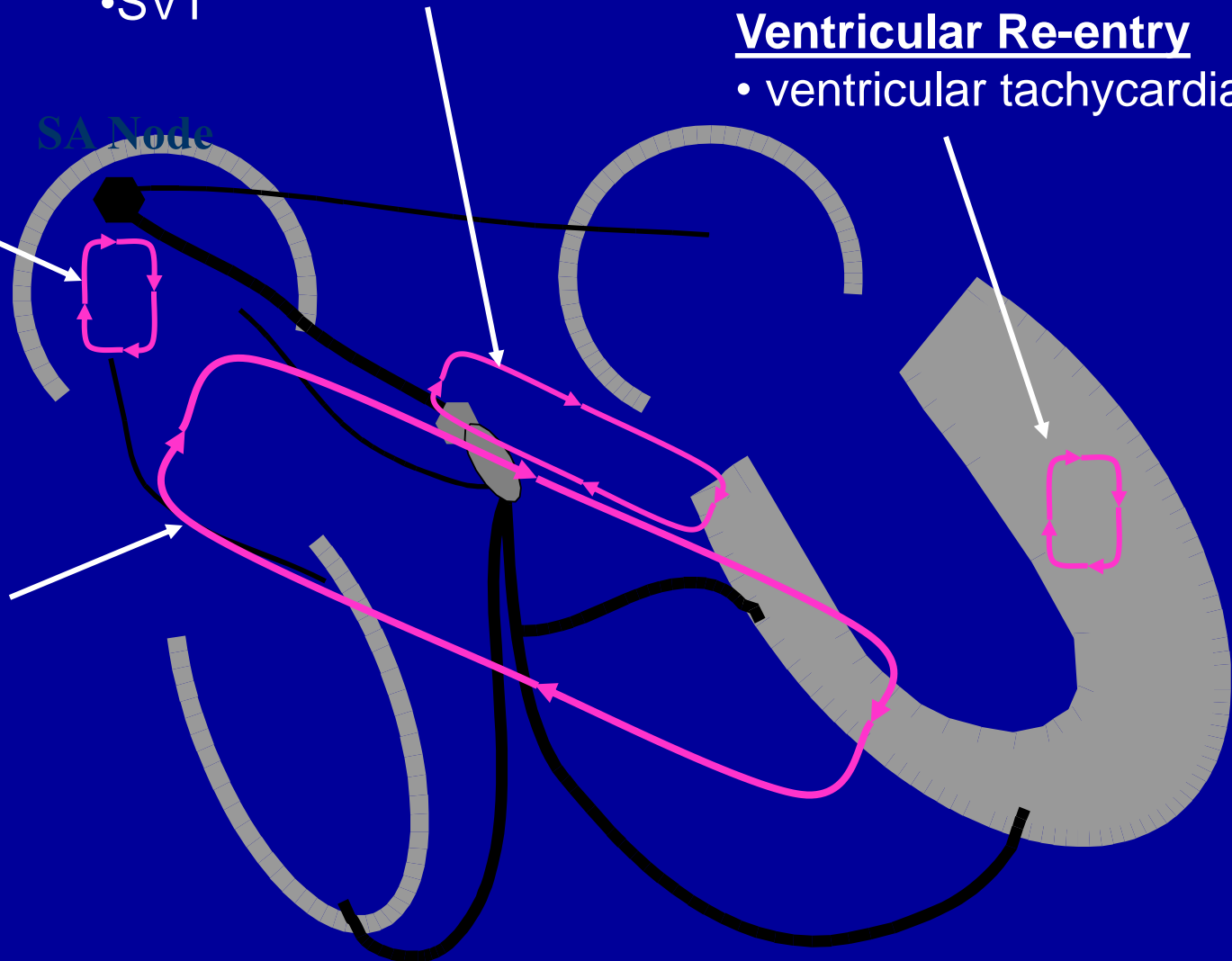
- atrial tachycardia
- atrial fibrillation
- atrial flutter

Ventricular Re-entry

- ventricular tachycardia

Atrio-Ventricular Reentry

- WPW
- SVT



Reentry Requires...

1. 2 distinct pathways that come together at beginning and end to form a loop.
2. A unidirectional block in one of those pathways.
3. Slow conduction in the unblocked pathway.

Large reentry circuits, like a-flutter, involve the atrium.

Reentry in *WPW* involves atrium, AV node, ventricle and accessory pathways.

Terminating Reentry

- Spontaneous termination
 - Another premature beat that disturbs the underlying conduction/refractoriness relationships
- Pace the heart at a rate above the tachycardia rate
 - Abruptly stop pacing
 - This is how implantable cardioverter-defibrillators can stop VT without a shock (ATP)

Automaticity

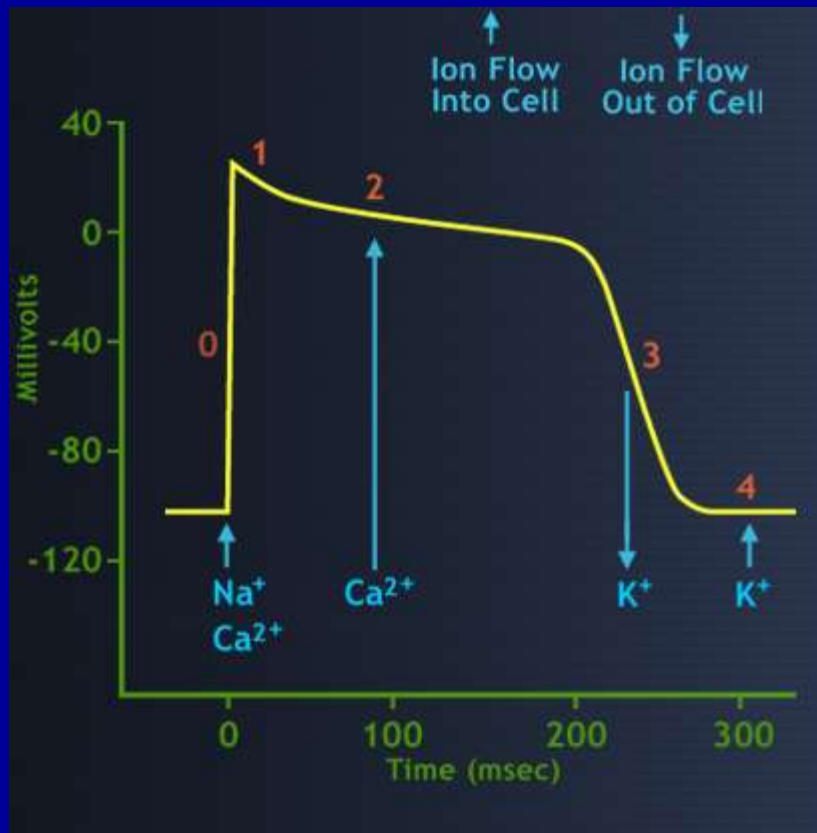
- Heart cells other than those of the SA node depolarize faster than SA node cells, and take control as the cardiac pacemaker.
- Factors that enhance automaticity include:
 - ↑ SANS, ↓ PANS, ↑ CO₂, ↓ O₂, ↑ H⁺, ↑ stretch, hypokalemia and hypocalcaemia.

Examples: *ventricular ectopy after MI*

Mechanisms of Rhythm Disorders

Triggered Activity

Depolarization occurring in Phase 3 (2) or 4 of the action potential can trigger arrhythmias



Early Afterdepolarization arise during the plateau phase (2) or the repolarization phase (3) of the last beat:

- Low potassium blood levels
- Slow heart rate
- Drug toxicity (ex. Quinidine causing Torsades de Pointes type of VT)

Late Afterdepolarization arise during the resting phase (4) of the last beat

- Potential Causes:
 - Premature beats
 - Increased calcium blood levels
 - Increased adrenaline levels
 - Digitalis toxicity

Diagnosis of Arrhythmia

Interviewing (irregular, rapid heart beats, shortness of breath, palpitation, fainting)

Physical examination (rhythm abnormalities, signs of underlying cardiac or non-cardiac disease)

Lab tests- usual + electrolytes (K, Na), thyroid function

Echocardiography (structural heart diseases, LV EF)

ECG; Holter ECG monitoring

Stress tests ("sit-stand", a test with 20 squats, bicycle ergometry, treadmill test, isometric test (hand, foot))

Transesophageal electrical cardio stimulation

Psychoemotional tests

Event Monitors

Holter monitoring: Document symptomatic and asymptomatic arrhythmias over 24-48 hours. Can also evaluate treatment effectiveness in a-fib, pacemaker effectiveness and identify silent MIs.

Trans-telephonic event recording: patient either wears monitor for several days or attaches it during symptomatic events and an ECG is recorded and transmitted for evaluation via telephone. Only 20% are positive, but still helpful.

Exercise testing

- Symptoms only appear or worsen with exercise.
- Premature ventricular contractions (PVCs) occur in 10% without and 60% of patients with CAD. PVCs DO NOT predict severity of CAD (neither for nor against)!
- Also used to evaluate medication effectiveness (esp. flecanide & propafenone)
 - ♥ You can assess SA node function with exercise testing.

*

Electrophysiologic Testing...

- Catheters are placed in RA, AV node, Bundle of HIS, right ventricle, and coronary sinus (to monitor LA and LV).
- Used to evaluate cardiogenic syncope of unknown origin, symptomatic SVT, symptomatic WPW, and sustained v-tach.
- *Ablative therapy is beneficial in AV node reentry, WPW, atrial tachycardia, a-flutter, and some v-tach. Complication is 1%

Therapy Principal

- Pathogenesis therapy – treatment underlying condition
- Stop the arrhythmia immediately if the hemodynamic was unstable
- Individual therapy

Anti-arrhythmia Agents

- **Anti-tachycardia agents**
- **Anti-bradycardia agents**

Anti-bradycardia agents

- **β -adrenic receptor activator**
(epinephrine, adrenaline, isoprenaline)
- **M-cholinergic receptor blocker**
(Atropine)
- **Non-specific activator (Aminophylline)**

Anti-tachycardia agents

Modified Vaughan Williams classification

1. I class: Sodium channel blocker
2. II class: β -receptor blocker
3. III class: Potassium channel blocker
4. IV class: Calcium channel blocker
5. Others: Adenosine, Digital

Class 1A agents: Procainamide, Quinidine, Disopyramide

Uses

Wide spectrum, but **side effects limit usage**

Quinidine : maintain sinus rhythms in atrial fibrillation and flutter and to prevent recurrent tachycardia and fibrillation

Procainamide: acute treatment of supraventricular and ventricular arrhythmias (**no longer in production**)

Disopyramide: treat certain types of serious (possibly fatal) ventricular arrhythmias

Side effects

Hypotension, reduced cardiac output

Proarrhythmia (generation of a new arrhythmia) eg.

Torsades de Points (\uparrow QT interval)

Dizziness, confusion, insomnia, seizure (high dose)

Gastrointestinal effects (common)

Lupus-like syndrome (esp. procainamide)

Class 1B agents: **Lidocaine,** **Phenytoin, Mexiletine**

Uses

during acute : Ventricular tachycardia and fibrillation (esp. ischemia)

Not used in atrial arrhythmias or AV junctional arrhythmias

Side effects

Less proarrhythmic than Class 1A (less QT effect)
CNS effects: dizziness, drowsiness

Class 1C agents: **Flecainide, Propafenone**

Uses

- Wide spectrum

- Used for **supraventricular arrhythmias** (fibrillation and flutter)

- Premature ventricular contractions (caused problems)

- Wolff-Parkenson-White syndrome

Side effects

- Proarrhythmia and sudden death** especially with chronic use (CAST study)

- Increase ventricular response to supraventricular arrhythmias

- CNS and gastrointestinal effects like other local anesthetics

Class II agents: Beta-blockers

Uses

**treating sinus and catecholamine dependent tachy
arrhythmias**

converting reentrant arrhythmias in AV

**protecting the ventricles from high atrial rates (slow AV
conduction)**

Side effects

bronchospasm

hypotension

beware in partial AV block or ventricular failure

Class III agents: **Amiodarone, Sotalol, Ibutilide**

Amiodarone

Uses

Very wide spectrum: **effective for most arrhythmias**

Side effects: many serious that increase with time

Pulmonary fibrosis

Hepatic injury

QT prolongation

Increase LDL cholesterol

Thyroid disease

Photosensitivity

May need to reduce the dose of digoxin and class 1 antiarrhythmics

Class III agents: **Amiodarone, Sotalol, Ibutilide**

Sotalol

Uses

Wide spectrum: supraventricular and ventricular tachycardia

Side effects

**Proarrhythmia,
fatigue,
insomnia**

Contraindicated in systolic ventricular dysfunction

Class III agents: **Amiodarone, Sotalol, Ibutilide**

Ibutilide

Uses

conversion of atrial fibrillation and flutter with rapid IV infusion

Side effects

Torsades de pointes

Class IV agents: **Verapamil and Diltiazem**

Uses

- control ventricular rate during **supraventricular tachycardia**

- convert supraventricular tachycardia (re-entry around AV)

Side effects

- Caution when partial AV block is present.

- Can get asystole if β blocker is on board

- Caution when hypotension, decreased CO or sick sinus syndrome

- Some gastrointestinal problems

Additional agents

Adenosine

Administration

rapid i.v. bolus, very short T_{1/2} (seconds)

Cardiac effects

Slows AV conduction

Uses

convert re-entrant supraventricular arrhythmias
hypotension during surgery, diagnosis of CAD

Magnesium

Torsades de point from any reason

Arrhythmias in a patient with known hypomagnesaemia.

Consider its use in acute ischaemia to prevent early ventricular arrhythmias.

Digoxin induced arrhythmias

Proarrhythmia effect of antiarrhythmia agents

- **Ia, Ic class: Prolong QT interval, may cause VT or VF in coronary artery disease and heart failure patients**
- **III class: Like Ia, Ic class agents**
- **II, IV class: Bradycardia**

Non-drug therapy

- **Cardioversion:** For tachycardia especially hemodynamic unstable patient
- **Radiofrequency catheter ablation (RFCA):** For those tachycardia patients (SVT, VT, AF, AFL)
- **Artificial cardiac pacing:** For bradycardia, heart failure and malignant ventricular arrhythmia patients.

Classification of arrhythmias

Violations of automaticity

- Nomotopic (pacemaker - in the sinus node)
 - sinus tachycardia (ST)
 - sinus bradycardia (SB)
 - sinus arrhythmia (SA)
 - sick sinus syndrome (SSS)
- Heterotopic (pacemaker - outside the sinus node)
 - atrial rhythm
 - atrioventricular rhythm
 - idioventricular rhythm

Classification of arrhythmias

Violations of excitability

- Premature complex
 - by site: atrial, atrioventricular, ventricular
 - according to the number of sources: monotopic, politopic
 - according to time of occurrence: early interpolated, late
 - according to frequency: single (up to 5 per minute), multiple (more than 5 per minute), pair (couplet)
 - According to ordering: unordered, allorhythmias (bigeminy, trigeminy, quadrigeminy)
- Paroxysmal tachycardia (atrial, AV, ventricular)

Classification of arrhythmias

Conduction abnormalities

- The increase in conductivity (Wolff-Parkinson White (WPW) syndrome)
- The decrease in conductivity (blockade: sinoauricular, intraatrial, AV, bundle-branch block)
- **Mixed** (atrial / ventricular flutter / fibrillation)

Sinus tachycardia



Sinus rate > 100 beats/min (100-180)

Causes:

1. Some physical condition: exercise, anxiety, exciting, alcohol, coffee
2. Some disease: fever, hyperthyroidism, anemia, myocarditis
3. Some drugs: Atropine, Isoprenaline

Clinical significance:

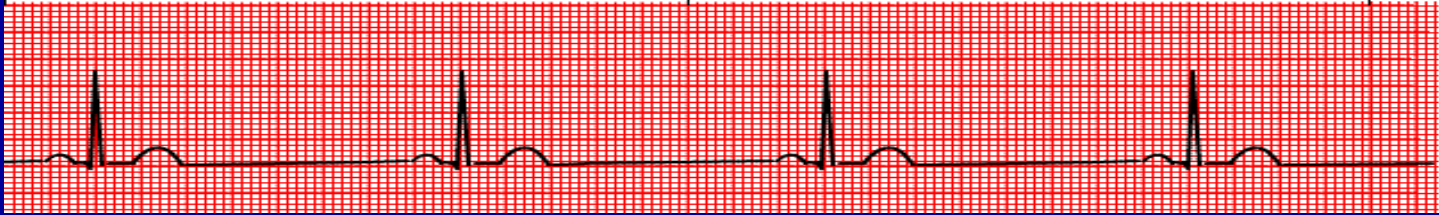
Benign and needn't therapy in most cases

But sometimes:

- Dizziness and hypotension due to decreased CO
- Increased myocardial oxygen consumption may lead to angina

Treatment : address underlying cause and/or determining if it is even a problem (adenosine, beta blockers).

Sinus Bradycardia



Sinus rate < 60 beats/min

Normal variant in many normal and older people

Causes: Cause-vagal stimulation, athlete, during sleep, drugs (Beta blockers; digoxin), head injuries, MI, hypothyroidism,

Clinical significance- Dependent on symptoms

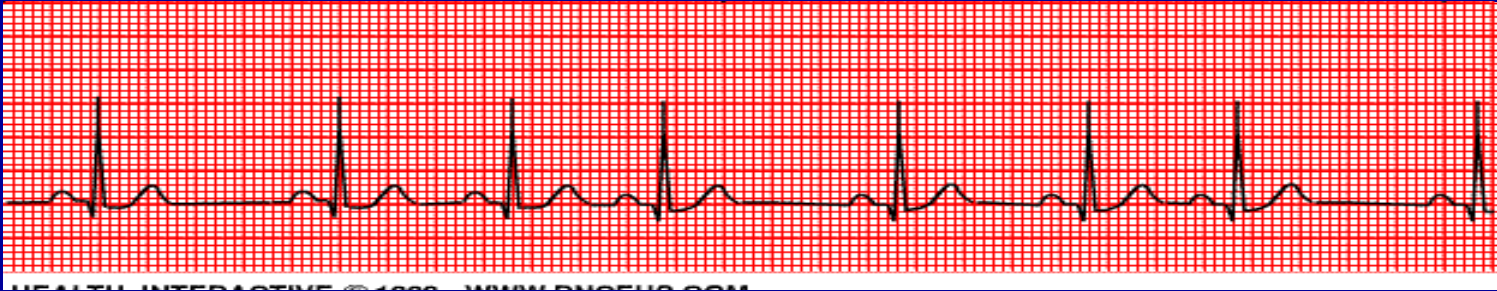
Most patients have no symptoms.

Severe bradycardia may cause dizziness, confusion or disorientation, shortness of breath, fatigue, palpitation, even syncope.

Needn't specific therapy

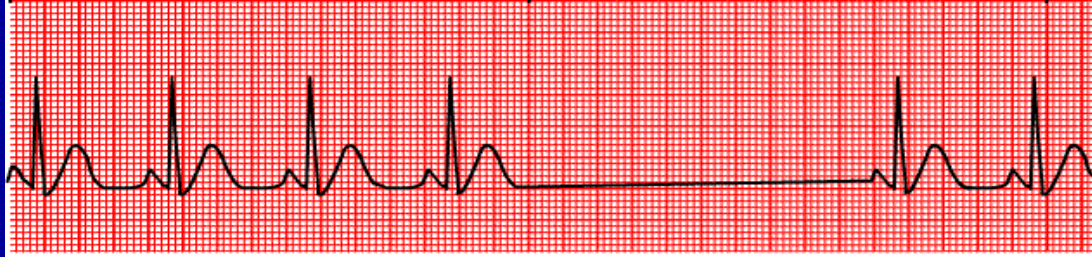
If the patient has severe symptoms, *atropine* or planted an pacemaker may be needed.

Sinus Arrhythmia



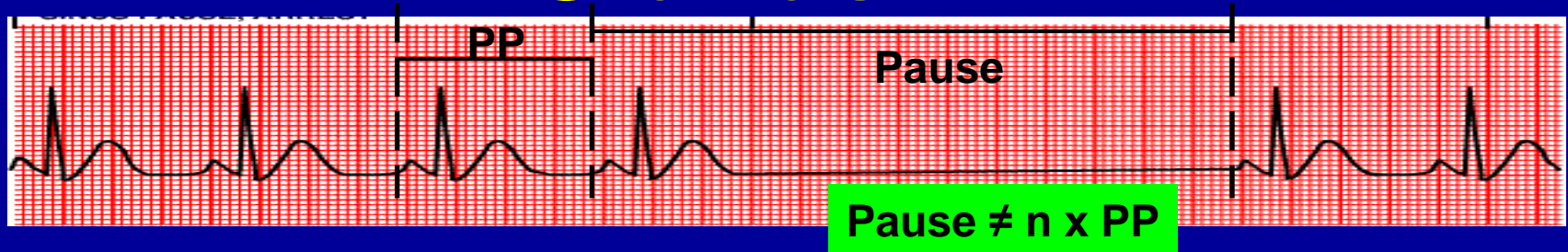
- Rate 60-100
- Irregular rhythm- increases with inspiration, decreases with expiration
- P, QRS, T wave normal
- **Cause-** children, myocardial ischemia
- Treatment- none (underlying condition)

Sinus Arrest



- See pauses
- May see ectopic beats (PAC's PVC's) do not treat
- Cause myocardial ischemia
- Treatment
 - Atropine
 - Isoprenaline
 - Pacemaker

Sinus Arrest or Sinus Standstill



- Sinus arrest or standstill is recognized by a pause in the sinus rhythm.
- **Causes:** myocardial ischemia, hypoxia, hyperkalemia, higher intracranial pressure, sinus node degeneration and some drugs (digitalis, β -blocks).
- Symptoms: dizziness, amaurosis, syncope
- Therapy – atropine, pacemaker

Sick Sinus Syndrome (SSS)

- SSS: The function of sinus node was degenerated. SSS encompasses both disordered SA node automaticity and SA conduction.
- **Causes:** CAD, SN degeneration, myopathy, connective tissue disease, metabolic disease, tumor, trauma and congenital disease.
- With marked sinus bradycardia, sinus arrest, sinus exit block or junctional escape rhythms
- Bradycardia-tachycardia syndrome

Sick Sinus Syndrome (SSS)

ECG Recognition:

1. Sinus bradycardia ≤ 40 bpm;
2. Sinus arrest (pauses $> 3s$)
3. Type II SAB
4. Nonsinus tachyarrhythmia (SVT, AF or Af).

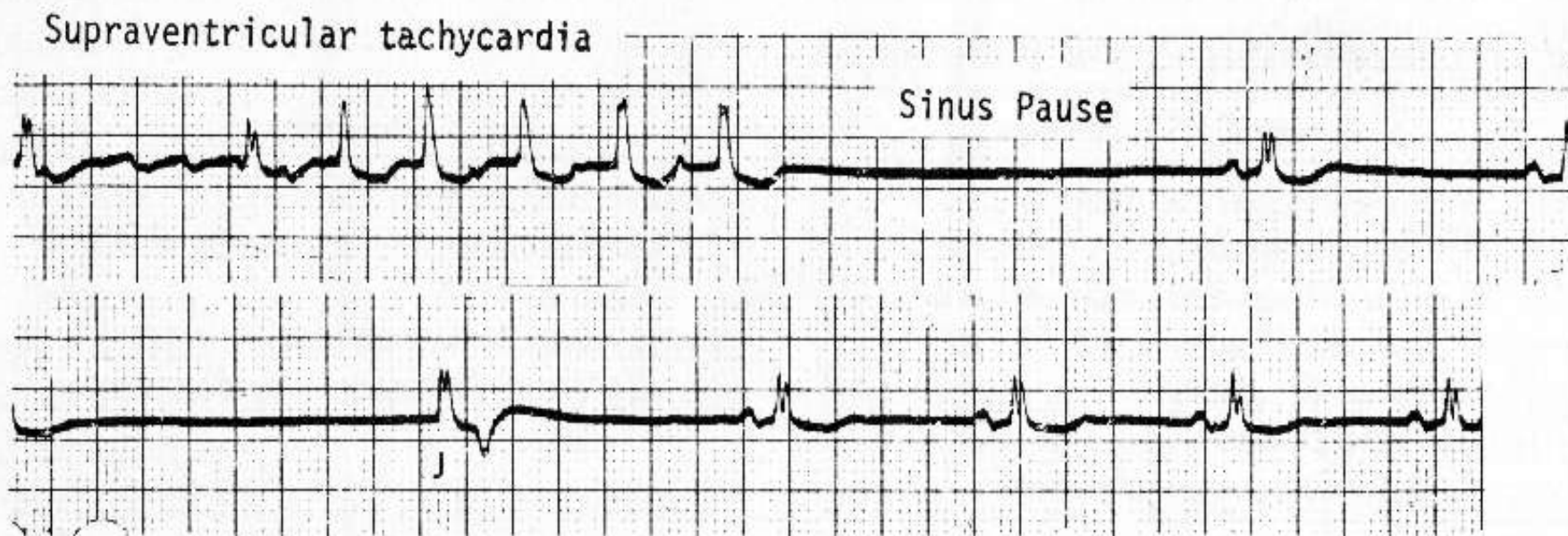


Fig. 18-13. Brady-tachy (sick sinus) syndrome. This rhythm strip shows supraventricular tachycardia (probably atrial flutter) followed by a sinus pause, an AV junctional escape beat (*J*), and then sinus rhythm.

Sick Sinus Syndrome (SSS)

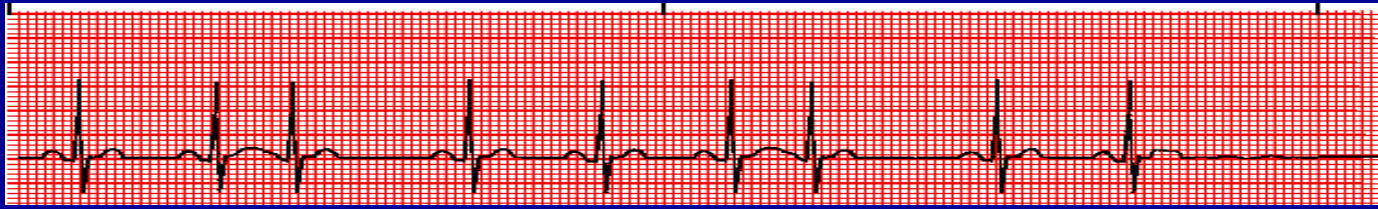
Therapy:

- 1. Treat the etiology**
- 2. Treat with drugs: anti-bradycardia agents, the effect of drug therapy is not good.**
- 3. Artificial cardiac pacing.**

Premature contractions

- The term “premature contractions” are used to describe non sinus beats.
- Common arrhythmia
- The morbidity rate is 3-5%

Atrial premature contractions (APCs)



- APCs arising from somewhere in either the left or the right atrium.
- **ECG:** P wave abnormally shaped, PR interval shorter, QRS normal, incomplete compensatory pause

A-V premature contractions

With simultaneous excitation of the atria and ventricles

ECG signs:

- **P wave is not determined**
- premature QRS complex is not expanded
- Incomplete compensatory pause

With preceding by excitation of the ventricles

ECG signs:

- P wave after QRS
- premature QRS complex is not expanded
- complete compensatory pause

Atrial and A-V premature contractions

- **Causes:** may occur in normal person, smoking, caffeine, rheumatic heart disease, CAD, hypertension, hyperthyroidism, hypokalemia
- **Symptoms:** many patients have no symptom, some have palpitation, chest uncomfortable.
- **Therapy:** Needn't therapy in the patients without heart disease. Can be treated with β -blocker, *propafenone*? or verapamil (watch for SVT).

Ventricular Premature Contractions (VPCs)

Etiology:

- 1. Occur in normal person**
- 2. Myocarditis, CAD, valve heart disease, hyperthyroidism, Drug toxicity (digoxin, quinidine and anti-anxiety drug)**
- 3. Electrolyte disturbance, anxiety, drinking, coffee**

Premature Ventricular Contractions

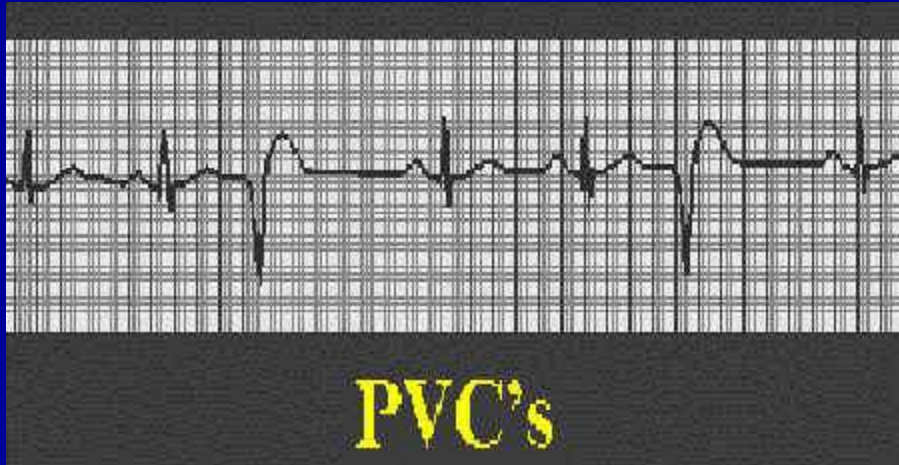
- Clinical significance
 - In normal heart, usually benign
 - In heart disease, PVCs may decrease CO and precipitate angina and HF
 - **Patient's response to PVCs must be monitored
 - PVCs often do not generate a sufficient ventricular contraction to result in a peripheral pulse, so apical-radial pulse rate should be assessed to determine if pulse deficit exists

Premature Ventricular Contractions (PVC's)-ectopic

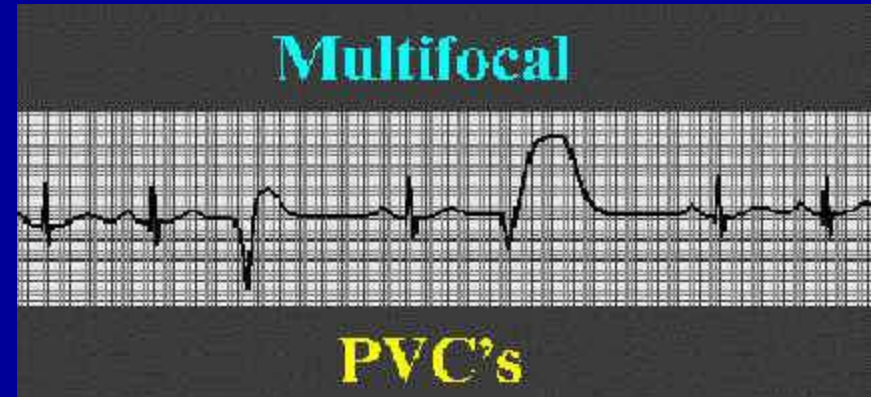
- QRS wide and bizarre
- no P waves
- T opposite deflection of PVC
- complete compensatory pause

PVC's

PVC's uni-focal



PVC's multi-focal



Multifocal- from more than one foci

Bigeminy- every other beat is a PVC

trigeminy- every third beat is a PVC

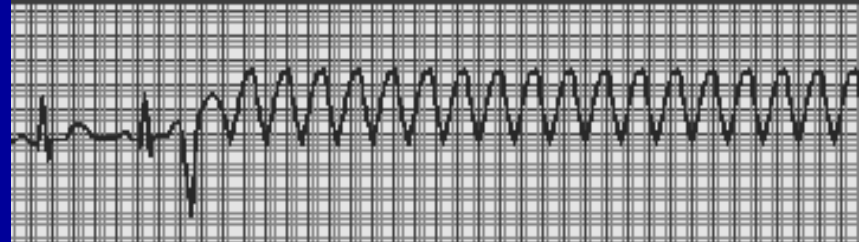
Couplet- 2 PVC's in a row

Treat if:

- >5 PVC's a minute
- Runs of PVC's (≥ 3 PVC's)
- Multi focal PVC's
- "R on T"



PVC on T-Wave



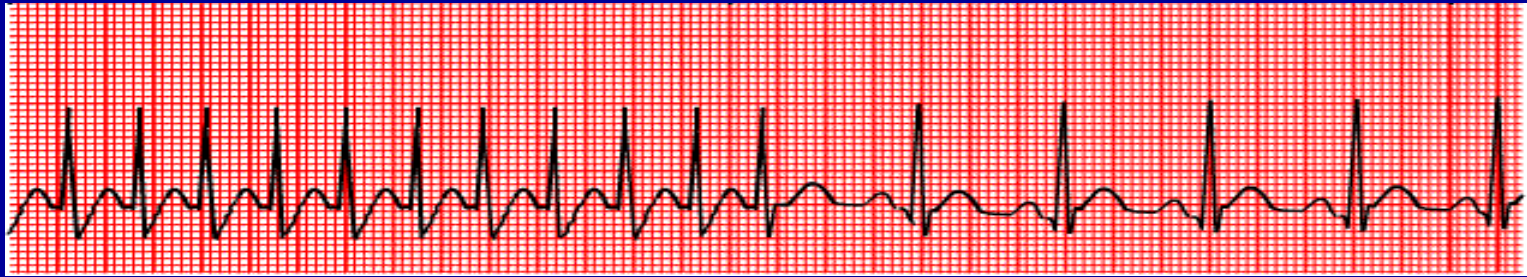
**Initiates Ventricular
Tachycardia**

PVCs: Treatment

Therapy: treat underlying disease, antiarrhythmia

- **No structure heart disease:**
anxiety agents, β -blocker and mexiletine to relieve the symptom.
- **With structure heart disease (CAD, LVH):**
 1. Treat the underlying disease
 2. β -blocker, amiodarone
 3. **Class I especially class Ic agents should be avoided**
because of proarrhythmia and lack of benefit of prophylaxis

Supraventricular Tachycardia (SVT)/PSVT (paroxysmal SVT)



- Rate- 150-250 (Very fast!)
- Atria is pacemaker (may not see p waves)
- Cause-SNS stimulation, MI, CHF, sepsis
- Treatment- *adenosine*, digoxin, calcium channel blockers, beta-blockers, vagal stimulation

Atrial flutter

Etiology:

1. It can occur in patients with normal atrial or with abnormal atrial.
2. It is seen in rheumatic heart disease (mitral or tricuspid valve disease), CAD, hypertension, hyperthyroidism, congenital heart disease, COPD.
3. Related to enlargement of the atria
4. Most AF have a reentry loop in right atrial

ATRIAL FLUTTER



Fig. 13-3. Note the variable ventricular rate in this patient with atrial flutter.

Rate of atria is 250-300, vent rate varies
P waves saw tooth, ratio 2:1, 3:1, 4:1
Flutter waves- No PR interval

Atrial flutter

- Symptoms: depend on underlying disease, ventricular rate, the patient is at rest or is exerting**
- With rapid ventricular rate: palpitation, dizziness, shortness of breath, weakness, faintness, syncope, may develop angina and CHF.**

Atrial flutter

Therapy:

1. Treat the underlying disease
2. To restore sinus rhythm: Cardioversion, Esophageal Pulsation Modulation, radiofrequency catheter ablation (RFCA) of the AV junction , Drug (III, Ia, Ic class).
3. Control the ventricular rate: digitalis, CCB, β -block
4. Anticoagulation (as in atrial fibrillation)

Atrial fibrillation

Chaotic atrial rhythm due to multiple reentrant wavelets, 350-500 bpm

Ventricular rate irregular and rapid due to variable AV block

Etiology:

- 1. Morbidity rate increase in older patients**
- 2. Etiology just like atrial flutter**
- 3. Idiopathic**

Mechanism:

- 1. Multiple wavelet re-entry;**
- 2. Rapid firing focus in pulmonary vein, vena cava or coronary sinus.**

ATRIAL FIBRILLATION

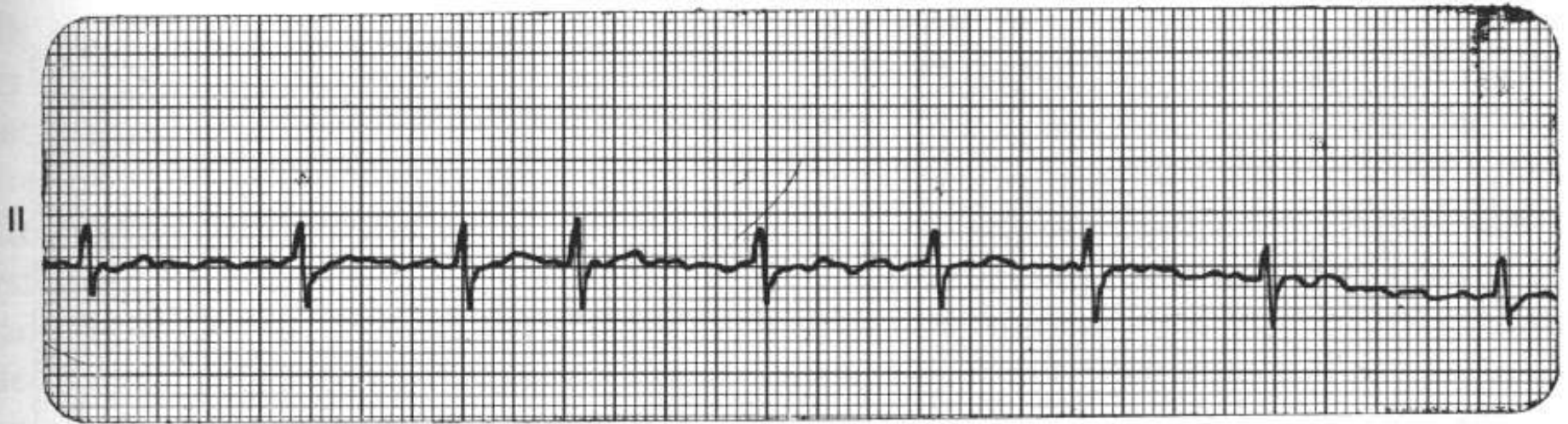


Fig. 13-4. Irregular undulation of the baseline because of fibrillatory (f) waves. There are no true P waves, and the ventricular (QRS) rate is irregular.

Classification of atrial fibrillation (AF)

1. First diagnosed AF - every patient who presents with AF for the first time, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms.
2. Paroxysmal AF is self-terminating, usually within 48 h. Although AF paroxysms may continue for up to 7 days, the 48 h time point is clinically important—after this the likelihood of spontaneous conversion is low and anticoagulation must be considered.

Classification of atrial fibrillation (AF)

3. Persistent AF - is present when an AF episode either lasts longer than 7 days or requires termination by cardioversion, either with drugs or by direct current cardioversion (DCC).
4. Long-standing persistent AF has lasted for ≥ 1 year when it is decided to adopt a rhythm control strategy.
5. Permanent AF - when the presence of the arrhythmia is accepted by the patient (and physician).

Atrial fibrillation

Manifestation:

- Affected by underlying diseases, ventricular rate and heart function.
- May develop embolism in left atrial. Have high incidence of stroke.
- The heart rate, S1 and rhythm is irregularly irregular

Atrial fibrillation

Therapy:

1. Treat the underlying disease
2. Restore sinus rhythm: Drug, Cardioversion, RFCA
3. Rate control: digitalis. CCB, β -block
4. Antithrombotic therapy: Aspirine, Warfarin (INR 2.0–3.0)

Table 15 Drugs for rate control

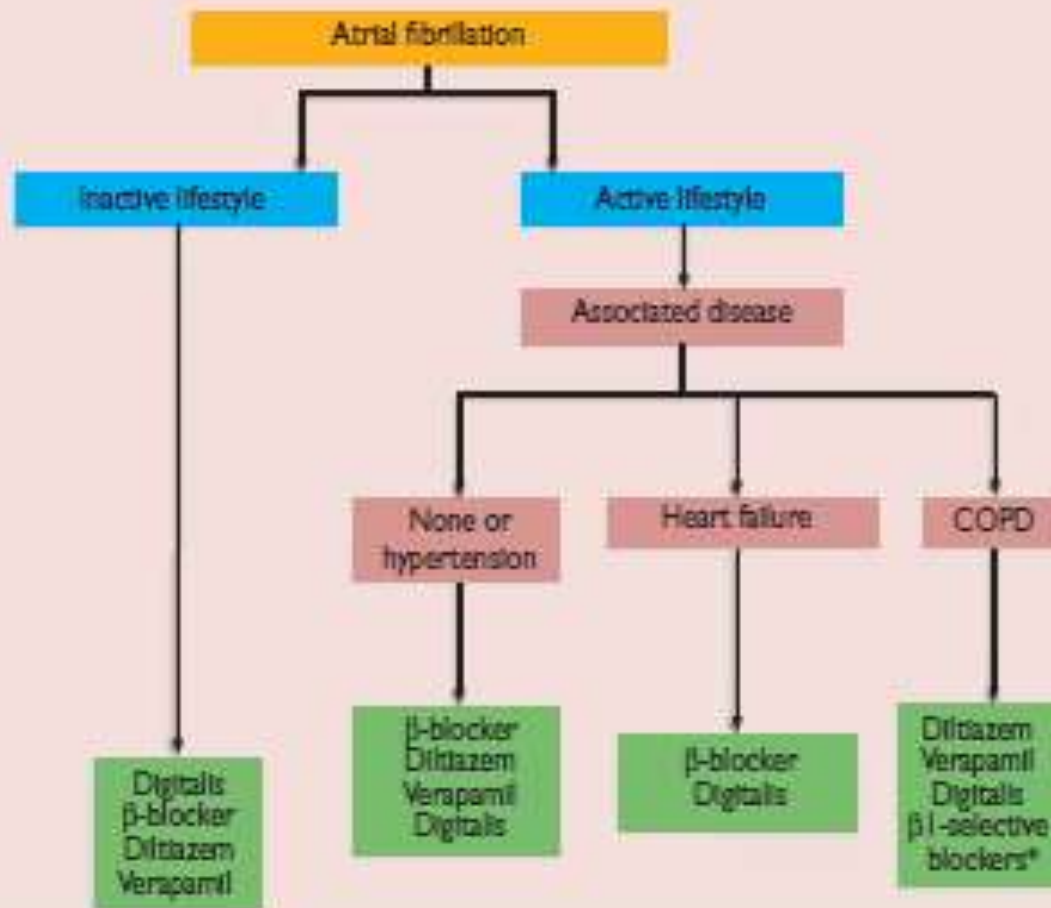
	Intravenous administration	Usual oral maintenance dose
β-Blockers		
Metoprolol CR/XL	2.5–5 mg iv bolus over 2 min; up to 3 doses	100–200 mg o.d. (ER)
Bisoprolol	N/A	2.5–10 mg o.d.
Atenolol	N/A	25–100 mg o.d.
Esmolol	50–200 µg/kg/min iv	N/A
Propranolol	0.15 mg/kg iv over 1 min	10–40 mg t.i.d.
Carvedilol	N/A	3.125–25 mg b.i.d.
Non-dihydropyridine calcium channel antagonists		
Verapamil	0.0375–0.15 mg/kg iv over 2 min	40 mg b.i.d. to 360 mg (ER) o.d.
Diltiazem	N/A	60 mg t.i.d. to 360 mg (ER) o.d.
Digitalis glycosides		
Digoxin	0.5–1 mg	0.125 mg–0.5 mg o.d.
Digitoxin	0.4–0.6 mg	0.05 mg–0.1 mg o.d.
Others		
Amiodarone	5 mg/kg in 1 h, and 50 mg/h maintenance	100 mg–200 mg o.d.
Dronedarone ²	N/A	400 mg b.i.d.

ER = extended release formulations; N/A = not applicable.

²Only in patients with non-permanent atrial fibrillation.

Rate control

The choice of drugs depends on life-style and underlying disease



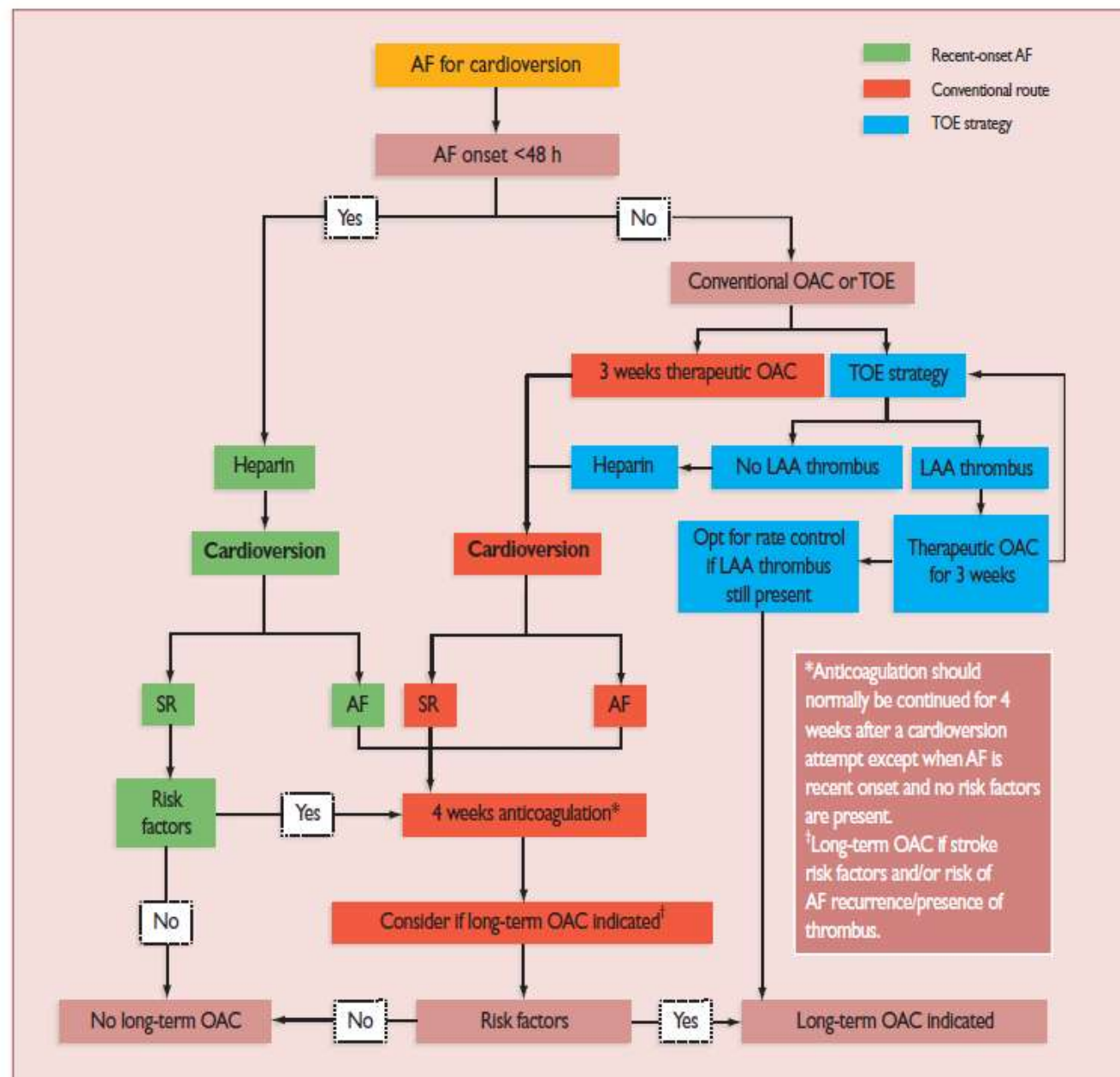
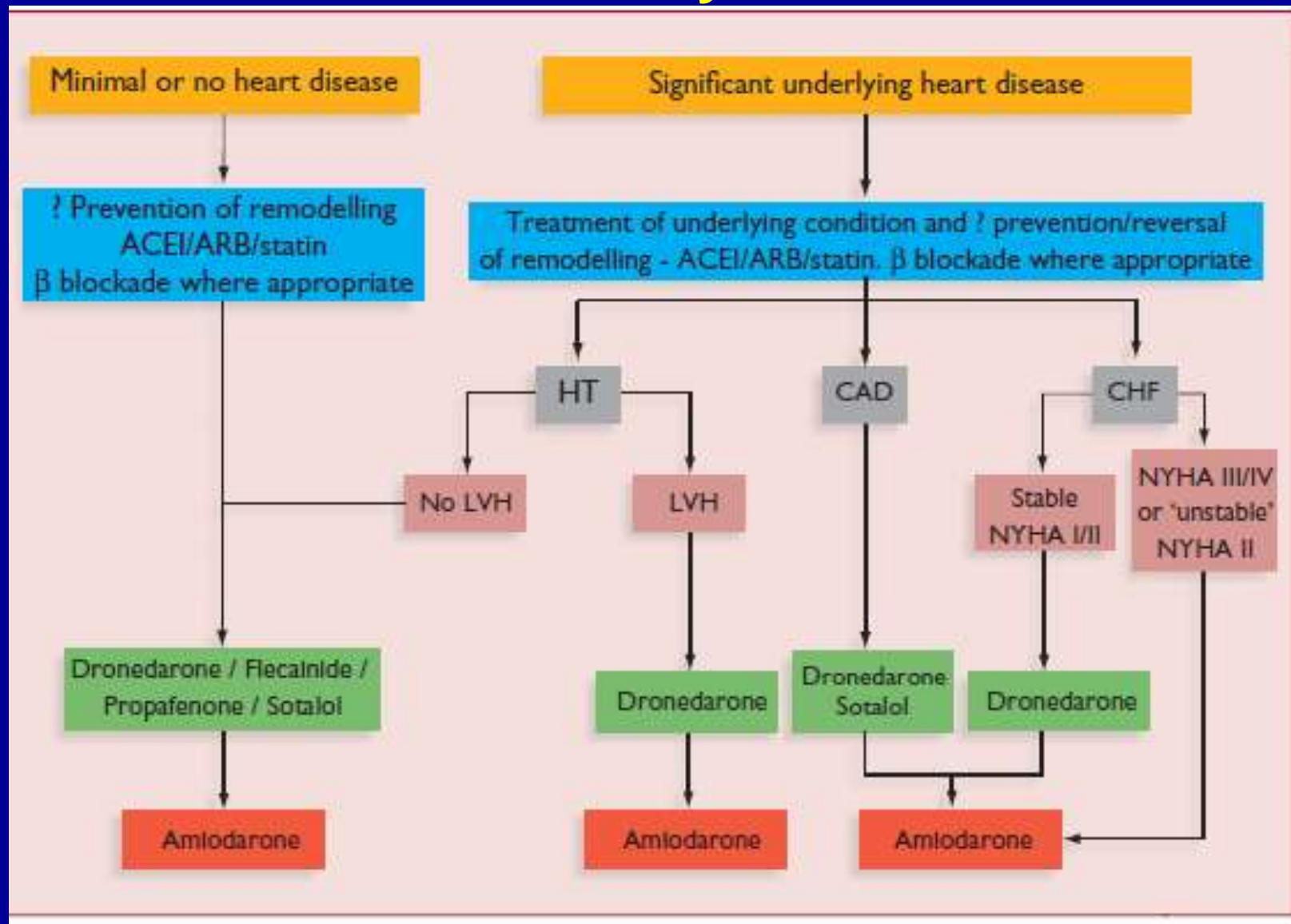


Figure 5 Cardioversion of haemodynamically stable AF, the role of TOE-guided cardioversion, and subsequent anticoagulation strategy. AF = atrial fibrillation; DCC = direct current cardioversion; LA = left atrium; LAA = left atrial appendage; OAC = oral anticoagulant; SR = sinus rhythm; TOE = transoesophageal echocardiography.

Table 12 Drugs and doses for pharmacological conversion of (recent-onset) AF

Drug	Dose	Follow-up dose	Risks
Amiodarone	5 mg/kg i.v. over 1 h	50 mg/h	Phlebitis, hypotension. Will slow the ventricular rate. Delayed AF conversion to sinus rhythm.
Flecainide	2 mg/kg i.v. over 10 min, or 200–300 mg p.o.	N/A	Not suitable for patients with marked structural heart disease; may prolong QRS duration, and hence the QT interval; and may inadvertently increase the ventricular rate due to conversion to atrial flutter and 1:1 conduction to the ventricles.
ibutilide	1 mg i.v. over 10 min	1 mg i.v. over 10 min after waiting for 10 min	Can cause prolongation of the QT interval and torsades de pointes; watch for abnormal T-U waves or QT prolongation. Will slow the ventricular rate.
Propafenone	2 mg/kg i.v. over 10 min, or 450–600 mg p.o.		Not suitable for patients with marked structural heart disease; may prolong QRS duration; will slightly slow the ventricular rate, but may inadvertently increase the ventricular rate due to conversion to atrial flutter and 1:1 conduction to the ventricles.

Treatment of atrial fibrillation: maintaining sinus rhythm



The Task Force for the Management of Atrial Fibrillation of the ESC, 2010

Identifying the risk of thromboembolic complications

(b) Risk factor-based approach expressed as a point based scoring system, with the acronym CHA₂DS₂-VASc
(Note: maximum score is 9 since age may contribute 0, 1, or 2 points)

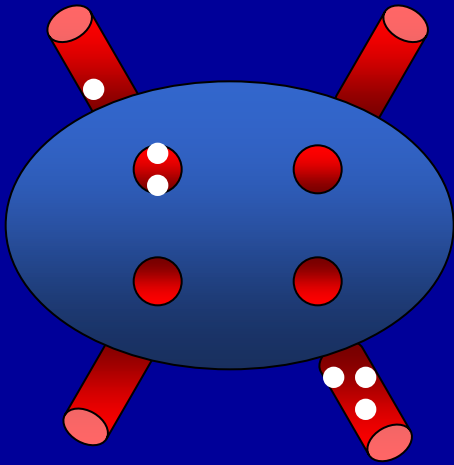
Risk factor	Score
Congestive heart failure/LV dysfunction	1
Hypertension	1
Age ≥ 75	2
Diabetes mellitus	1
Stroke/TIA/thrombo-embolism	2
Vascular disease ^a	1
Age 65–74	1
Sex category (i.e. female sex)	1
Maximum score	9

Approach to thromboprophylaxis in patients with AF

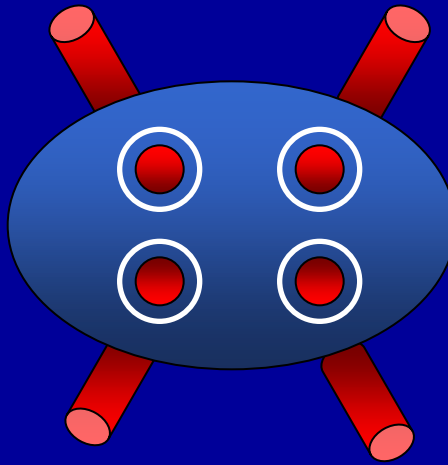
CHA ₂ DS ₂ -VASc score	Recommended antithrombotic therapy
≥ 2	OAC ^a
1	Either OAC ^a or aspirin 75–325 mg daily. Preferred: OAC rather than aspirin.
0	Either aspirin 75–325 mg daily or no antithrombotic therapy. Preferred: no antithrombotic therapy rather than aspirin.

Atrial Fibrillation

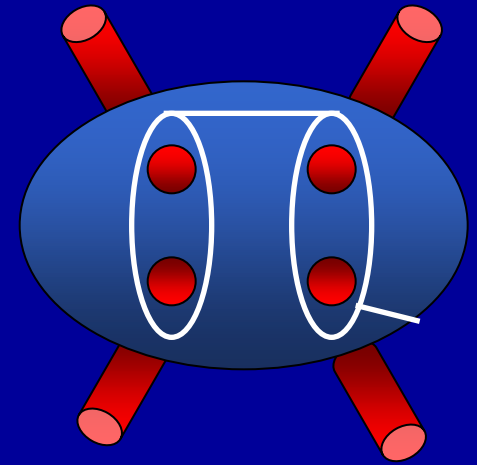
Catheter Ablation



Ablate PV potentials



PV Isolation

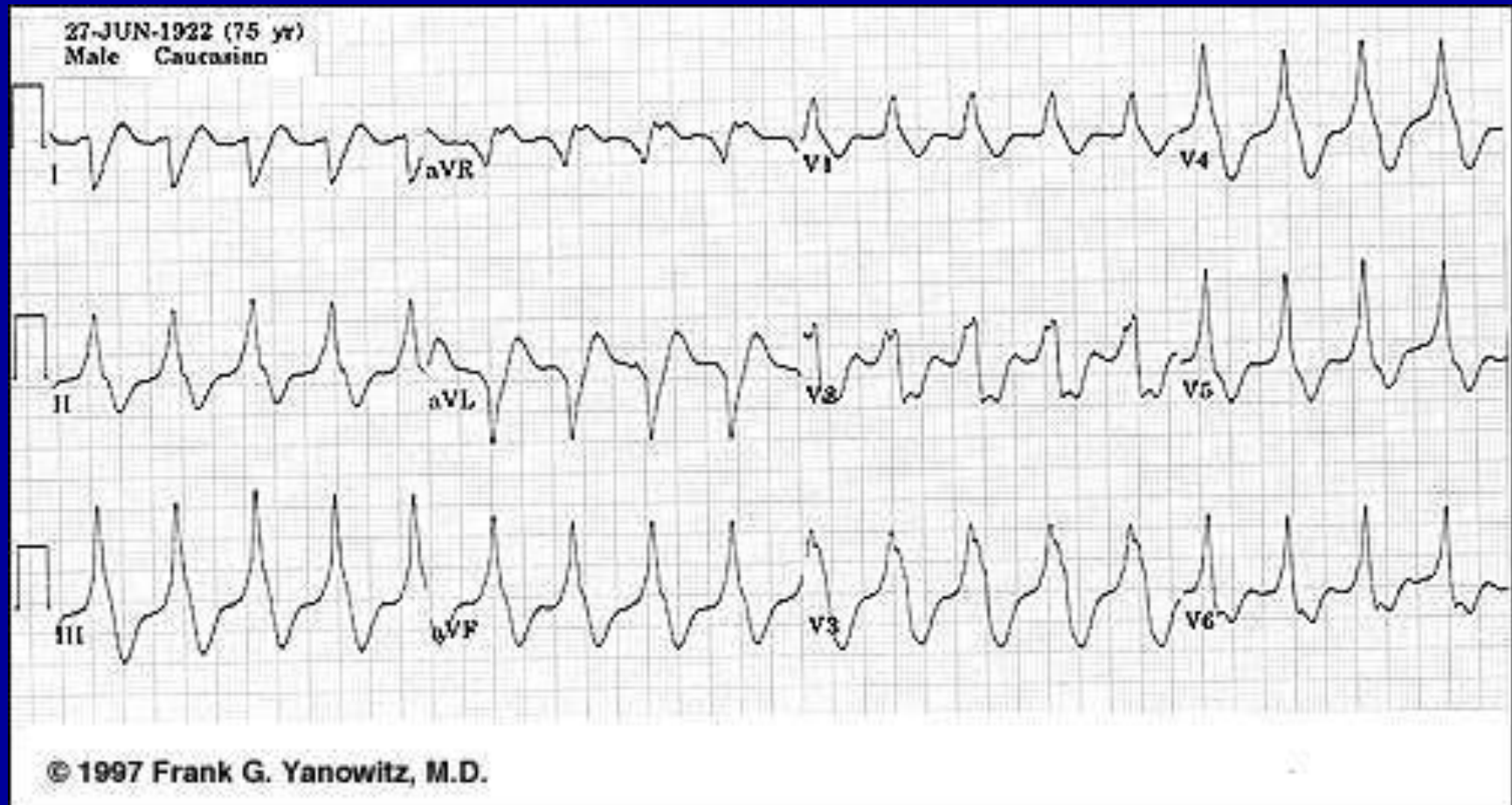


Pappone
(circumferential LA ablation)

Ventricular tachycardia

- **Etiology:** often in organic heart disease
CAD, MI, DCM, HCM, HF, long QT syndrome
- Sustained VT (>30s), Nonsustained VT
- Monomorphic VT, Polymorphic VT

ECG in ventricular tachycardia:
ventricular rate 150-250, regular or irregular
no P waves
QRS>0.12 msec



VT

Manifestation:

- 1. Nonsustained VT with no symptom**
- 2. Sustained VT : with symptom and unstable hemodynamic, patient may feel palpitation, short of breathness, presyncope, syncope, angina, hypotension and shock.**

Treatment of VT

1. Treat underlying disease
2. Cardioversion: Hemodynamic unstable VT (hypotension, shock, angina, CHF) or hemodynamic stable but drug was no effect
3. Pharmacological therapy: β -blockers, lidocain or amiodarone
4. RFCA, ICD or surgical therapy

Implanatable defibrillators

Medtronic Implantable Defibrillators (1989-1997)



209 cc



113 cc



80 cc



80 cc



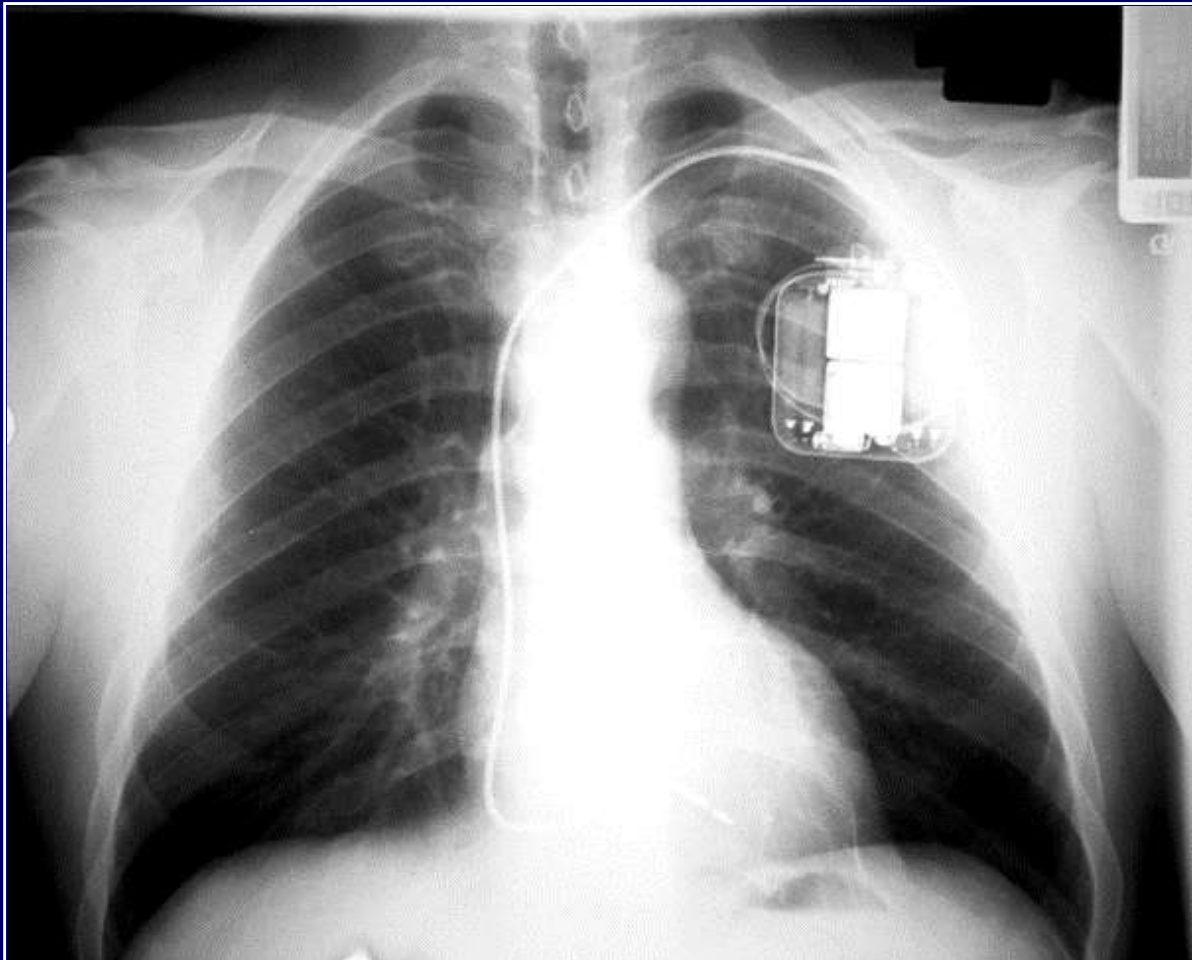
72 cc



54 cc

71 mm x 58 mm x 16 mm
2 4/5 in x 2 1/3 in x 2/3 in

Implanatable defibrillator in-situ



Ventricular tachycardia

Torsades de points (Tdp): A special type of polymorphic VT

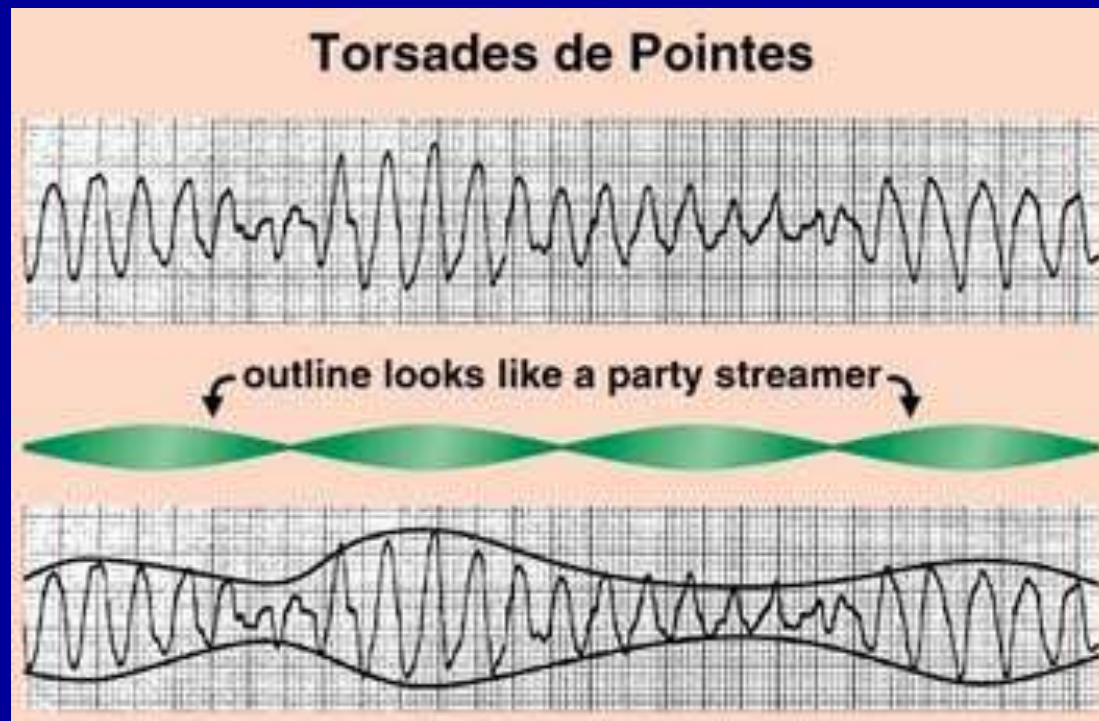
Etiology:

1. congenital (Long QT),
2. electrolyte disturbance: hypo/hyperkalemia, *HYPOMAGNESEMIA*
3. antiarrhythmia drug proarrhythmia (IA or IC), antianxiety drug, antimicrobial drugs (acquired long QT-syndrome),
4. brain disease

Treatment - includes treating cause(s), medications (magnesium) , and defibrillation or cardioversion.

VT- Torsades de Pointes

French for twisting of the points



Ventricular flutter and fibrillation

- Often occur in severe organic heart disease: AMI, ischemia heart disease
- Proarrhythmia (especially produce long QT and Tdp), electrolyte disturbance
- Anaesthesia, electric shock, heart operation
- It's a fatal arrhythmia

Ventricular Fibrillation



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
300-600	Extremely irregular	Absent	N/A	Fibrillatory baseline

Ventricular flutter and fibrillation

Manifestation:

Unconsciousness, twitch, no blood pressure and pulse, going to die

Therapy:

Cardio-Pulmonary Resuscitate (CPR)

ICD