Cardiac Arrhythmias

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- **Electrical activity of heart (cumulating to electrocardiogram) involves passage of ions (Na^+,K^+,Ca^{++},Cl^- ions) through ionic channels, producing cardiac transmembrane potential, which consists of:-**

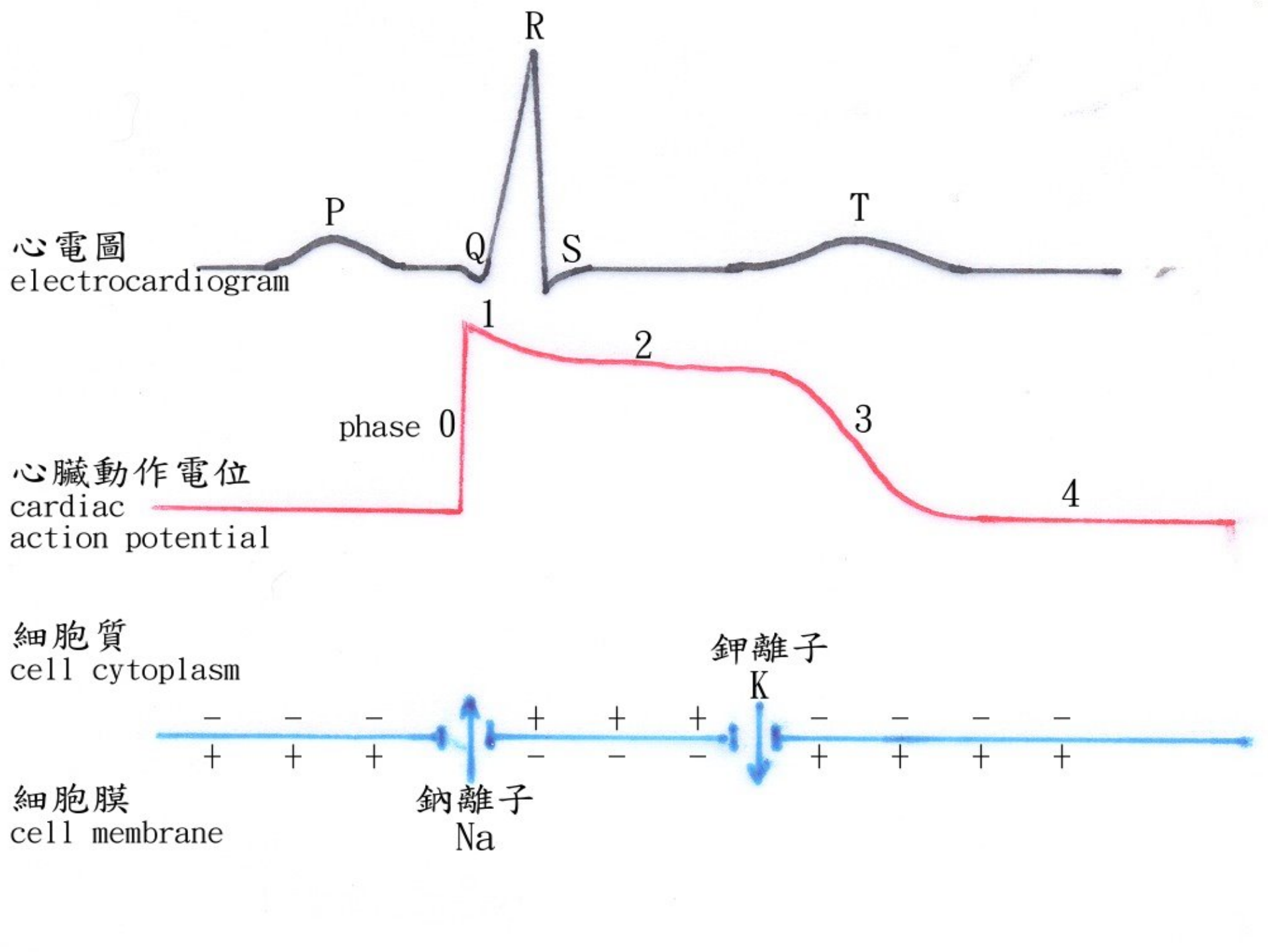
Phase 0 = upstroke or rapid depolarization, due to sudden increase in membrane conductance to Na^+(Na^+ influx)

Phase 1 = early rapid repolarization, due to inactivation of Na^+ channel

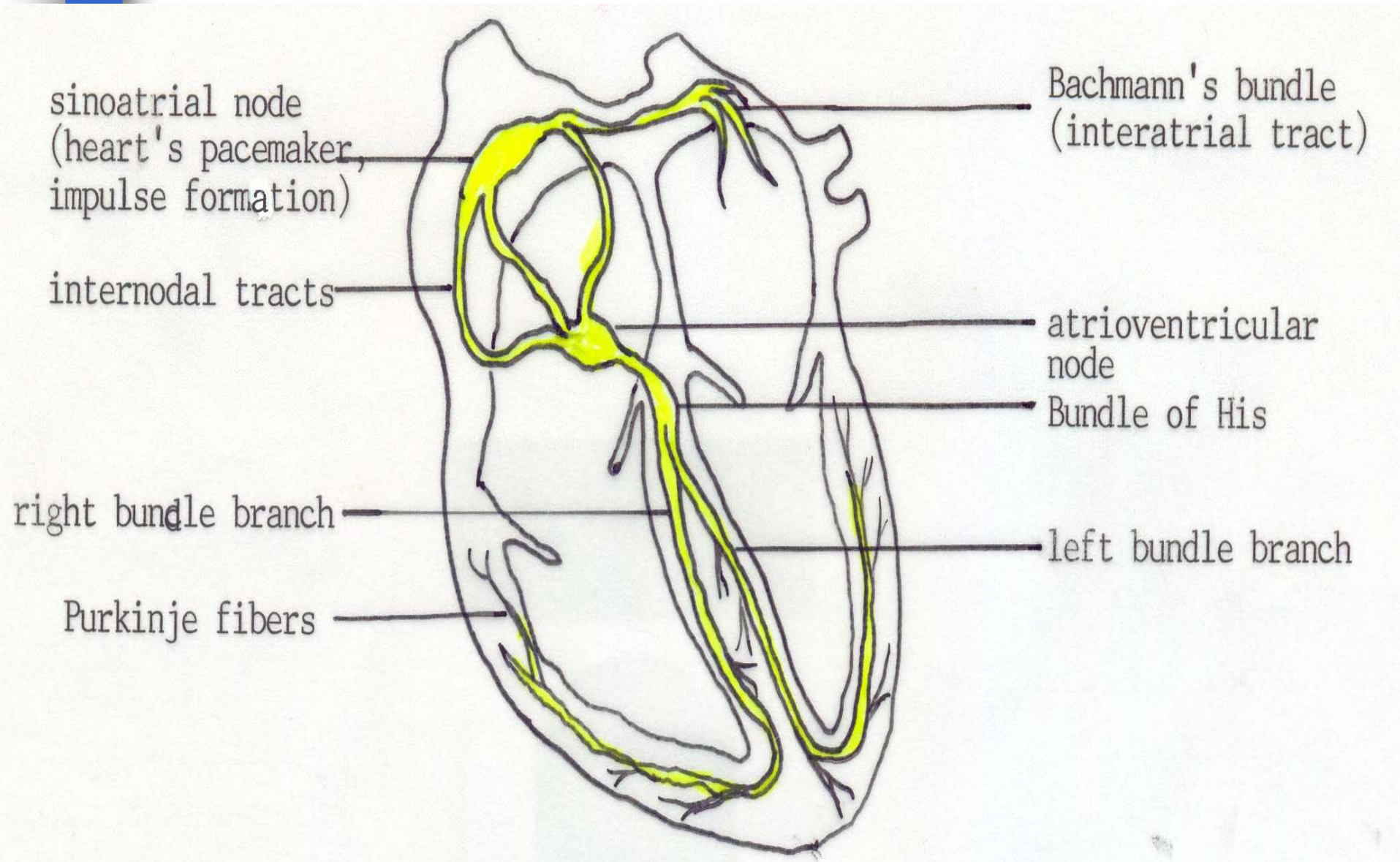
Phase 2 = plateau, due to decrease membrane conductance to all ions

Phase 3 = rapid repolarization, due to K^+ efflux

Phase 4 = resting membrane potential (diastolic depolarization)


R
P
T
心電圖
electrocardiogram
Q
S
1
2
phase 0
3
心臟動作電位
cardiac
action potential
4
細胞質
cell cytoplasm
鉀離子
K
細胞膜
cell membrane
鈉離子
Na

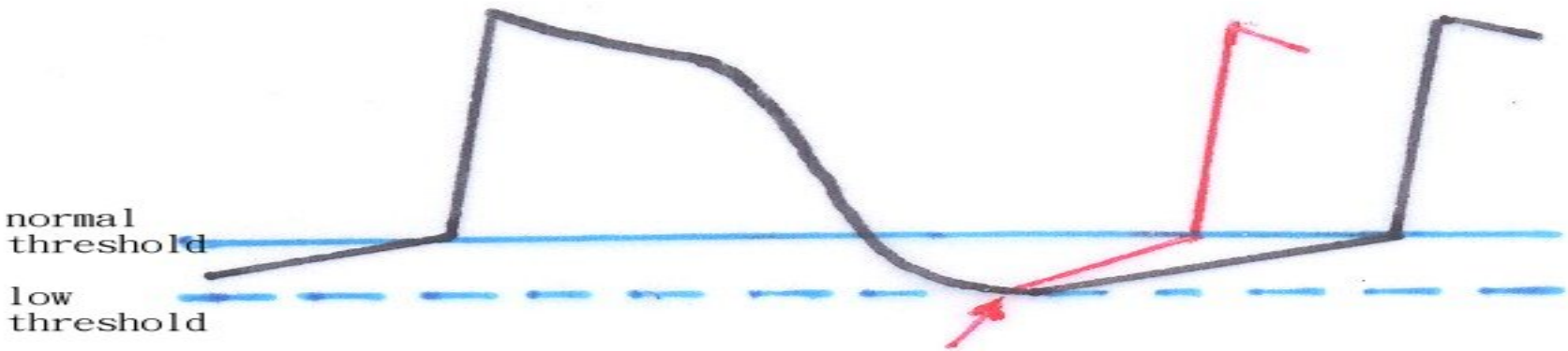
Conduction system of heart



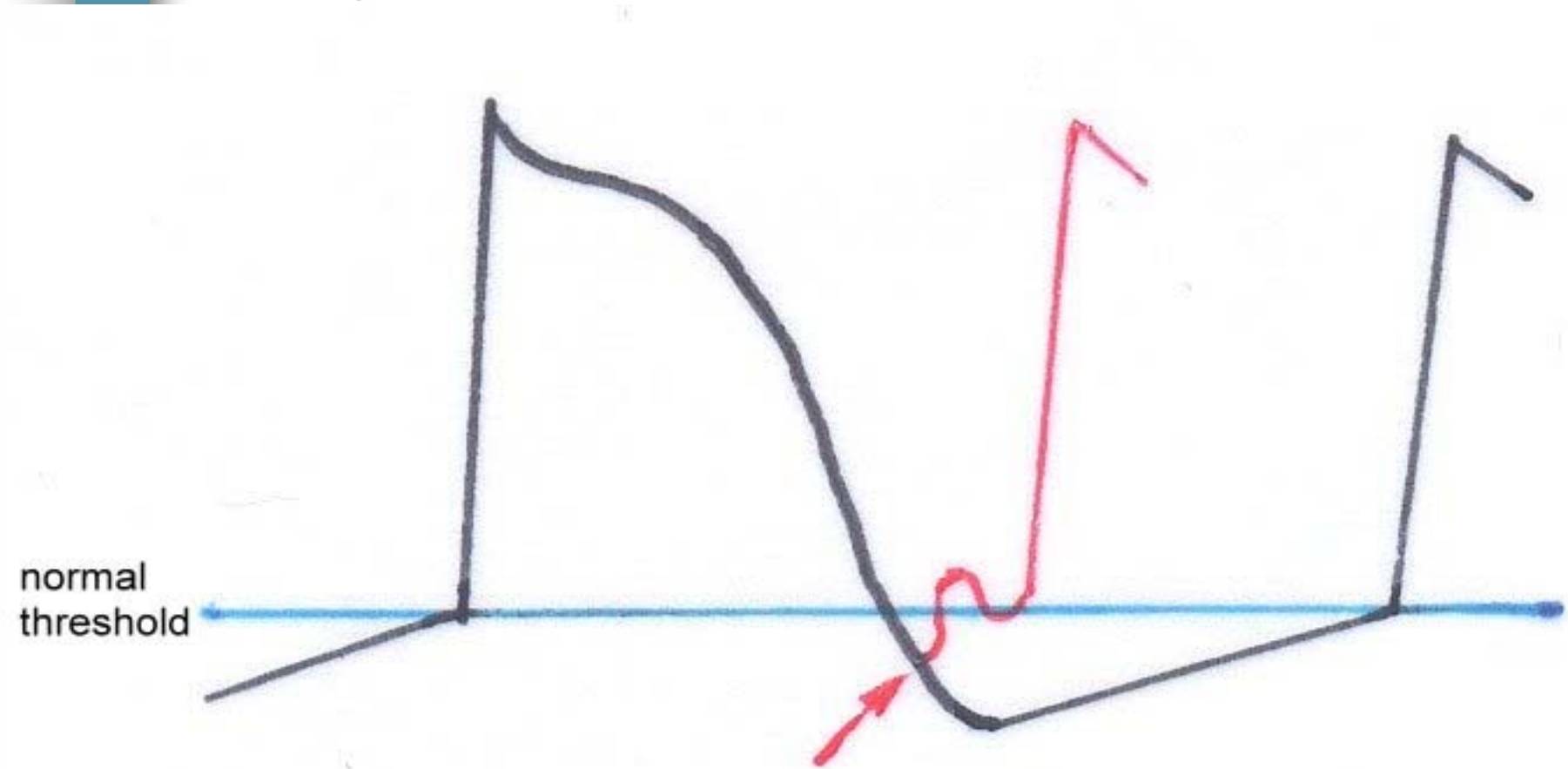
Mechanisms of arrhythmogenesis

- **Due to abnormal impulse formation, conduction, or combination**
- ***ABNORMAL IMPULSE FORMATION:***

  1. *Accelerated automaticity:* due to spontaneous decline or increasing rate of phase 4 diastolic depolarization (= pacemaker potential) → sinus tachycardia, escape beat and accelerated AV nodal rhythm

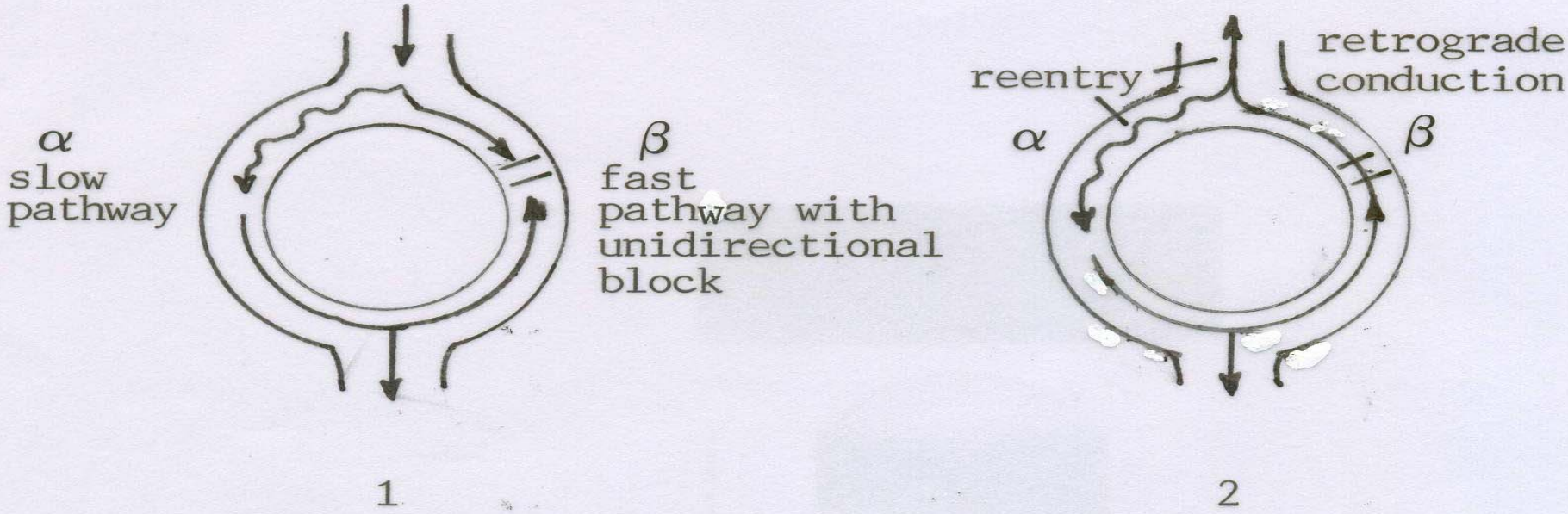


2. *Triggered activity:* due to afterdepolarizations reaching threshold potential that follow the upstroke of an action potential (the trigger), usually occurring in digitalis intoxication, catecholamines, hypoxia etc.



- ***ABNORMAL IMPULSE CONDUCTION:***

Reentry: **presuppose 2 conduction pathways (1. slow conduction, 2. unidirectional block) so that impulse propagates through slow conduction pathway (β) and backward through the undepolarized unidirectional block pathway (α), that is to reenter the conduction circuit producing majority of reentrant arrhythmias eg. atrial fibrillation, PSVT, VT, Vf etc.**



Sinus node disturbance

1. ***SINUS TACHYCARDIA:***

   >100 beats/min, due to accelerated phase 4 diastolic depolarization

   - normal reaction to physiological or pathophysiological causes eg. fever, hypotension, anemia, anxiety, exertion, myocardial ischemia, pulmonary emboli, heart failure etc.

2. ***SINUS BRADYCARDIA:***

   <60 beats/min, due to increased vagal or decreased sympathetic tone eg. myocardial ischemia, hypoxia, hypothermia, infection, sick sinus syndrome (fibrosis of sinus node)

3. ***SINUS ARRHYTHMIA:***

   = phasic variation in sinus cycle length (p-p interval), usually normal event.

Disturbances of atrial rhythm

1. ***PREMATURE ATRIAL CONTRACTION:***

   = premature p wave (usually different morphology)

   - causes: infection, inflammation, myocardial ischemia, anxiety, drugs etc.

2. ***ATRIAL FLUTTER:***

   = sawtooth flutter waves

   - causes: rheumatic heart disease, coronary artery disease, cardiomyopathy, atrial dilatation, pulmonary emboli, heart failure, valvular heart disease

- ***ATRIAL FIBRILLATION:***

  = irregular f waves with no p waves

  - causes: heart failure, valvular heart disease, stroke, atrial dilatation, pericarditis, etc

  - risk of embolization and anticoagulation needed for atrial fibrillation patients with risk factors for stroke eg. prior stroke, valvular heart disease, diabetes, coronary artery disease, heart failure etc.

4. ***ATRIAL TACHYCARDIA:***

- rapid atrial rate with different p wave morphology

- causes: coronary artery disease, valvular heart disease, cor pulmonale, digitalis intoxication, hypokalemia, cardiomyopathy, pulmonary disease

AV junctional rhythm disturbances

- ***Causes:*** **congenital heart disease, heart failure, cardiac surgery, coronary artery disease, cardiomyopathy, mitral valve prolapse etc.**
- ***Precipitating factors:*** **drugs, infection, anxiety etc.**

1. ***AV NODAL REENTRANT TACHYCARDIA (AVNRT):***

   = reentrant tachycardia within dual pathways (fast and slow pathways) in AV node characterized by QRS complex of supraventricular origin with sudden onset and termination at regular rates 150-250 beats/min

   - **p wave usually buried in QRS complex, or seen just prior to or just after QRS**
   - **anterograde conduction to ventricle occurs over the slow (alpha) pathway and retrograde conduction over the fast (beta) pathway**

- ***AV RECIPROCATING TACHYCARDIA (AVRT):***

  eg. Preexcitation syndrome (WPW syndrome)

  - characterized by short PR interval and slurred QRS (delta wave)
  - reentrant tachycardia within dual pathways in accessory pathway at left free wall, posteroseptal, right free wall, anteroseptal

Ventricular rhythm disturbances

1. ***PREMATURE VENTRICULAR CONTRACTION:***

   - causes: age, coronary artery disease, hypotension, heart failure, cardiomyopathy, valvular heart disease, infection,inflammation, hypoxia, surgery, electrolyte imbalance,drugs

2. ***VENTRICULAR TACHYCARDIA:***

   ≥ 3 consecutive premature ventricular contractions

3. ***VENTRICULAR FLUTTER AND FIBRILLATION:***

   = severe derangements of heartbeat usually fatal within 3-5 minutes without treatment.

   - causes: coronary artery disease, metabolic alterations, autonomic modulation, drugs, hypoxia, heart failure

Heart block

- **Due to idiopathic fibrosis of conduction system (mostly), coronary artery disease, congenital heart disease, valvular heart disease, infection, cardiomyopathy, cardiac surgery, drugs etc.**

1. ***FIRST DEGREE HEART BLOCK:***
   - **prolonged conduction time but all impulses are conducted (PR interval > 0.2 sec), due to conduction delay in AV node (A-H interval), His-Purkinje system (H-V interval), or both.**

2. ***SECOND DEGREE HEART BLOCK :***
   = blocking of some atrial impulses conducted to ventricle

   a) Mobitz I heart block: **characterized by progressive lengthening of conduction time until an impulse is not conducted.**

   b) Mobitz II heart block: **characterized by sudden block of an impulse without prior lengthening of conduction time**

- ***COMPLETE HEART BLOCK:***

  - when no impulses are conducted, and therefore the atria and ventricles are controlled by independent pacemakers, due to conduction block in AV node, His-Purkinje system.

4. ***BUNDLE BRANCH BLOCK:***

   conduction block at His bundle

Consequences of cardiac arrhythmias:-

1. e1ectrophysiological consequences: development of serious arrhythmias (=proarrhythmia)
2. hemodynamic consequences: decrease blood pressure, coronary blood flow, causing cardiac dilatation and heart failure

Management of cardiac arrhythmias:-

1. **treat underlying causes eg. coronary artery disease, heart failure, hypotension, myocarditis, etc. and precipitating factors eg. Infection, electrolyte imbalance, anemia, thyroid function etc.**
2. **antiarrhythmic drugs**
3. **ablation therapy**
4. **device therapy eg. Implantable cardioverter-defibrillator (ICD)**

Antiarrhythmic drugs

Vaughan William's classification of antiarrhythmic drugs based on their effects on cardiac action potentials:-

Class I (block sodium channel):

Ia : lengthen action potential eg. quinidine, procainamide, disopyramide

Ib : shorten action potential eg. lidocaine, mexiletine

Ic : no effect on action potential duration eg. flecainide, propafenone

Class 2 : beta-blocking agent acting on sinus node eg. propranolol, metoprolol

Class 3 (block potassium channel) : increase action potential duration eg. amiodarone, sotalol

Class 4 (block calcium channel) : calcium blockers acting on AV node eg. verapamil, diltiezem,

Quinidine

Indications: **premature supraventricular and ventricular contractions, substained tachyarrhythmias (AVNRT, WPW, Af etc.)**

Mechanism of action: **suppress automaticity by decreasing slope of phase 4 diastolic depolarization**

- prolong AV node and His-Purkinje conduction times and refractoriness in AV accessory pathway.

Side effects: **hypotension**

gastrointestinal: nausea, vomiting, diarrhea, anorexia

neurological: tinnitus, hearing loss, confusion, psychosis

allergy: rash, fever, thrombocytopenia

syncope, torsades, hypokalemia

Procainamide

Indications: **supraventricular and ventricular arrhythmias**

Mechanism of action: **similar to quinidine**

Side effects: **rash, myalgia, vasculitis, Raynaud's phenomenon, fever, agranulocytosis, giddiness, psychosis, hallucination, depression, hypotension, torsades, SLE-like syndrome**

Lidocaine

Indications: **ventricular arrhythmias**

Mechanism of action: **block sodium channel → decrease action potential duration and refractory period**

Side effects: **neurologic: dizziness, confusion, delirium, coma, seizure, hyperthermia**

- Mexiletine

Indications: **ventricular arrhythmias**

Mechanism of action: **similar to lidocaine**

Side effects: **tremor, dysarthria, dizziness, anxiety, vomiting, hypotension, bradycardia, proarrhythmias**

- **Propafenone**

Indications: **paroxysmal supraventricular tachycardia and atrial fibrillation, ventricular tachyarrhythmias**

Mechanism of action: **block sodium channel**

- suppress automaticity and triggered activity

Side effects: **dizziness, blur vision, heart block, worsening of heart failure, proarrhythmias**

- Amiodarone

Indications: **supraventricular and ventricular arrhythmias**

Mechanism of action: **increase action potential duration and refractory period**

- inhibit automaticity

- exhibit class 1(block sodium channel), 2 (antiadrenergic) and 4 (block calcium channel) effects

Side effects **(uncommon at ≤ 200 mg/day):**

pulmonary toxicity (probably hypersensitivity reaction)

neurologic dysfunction, gastrointestinal disturbance, hyperthyroidism, poor liver function, cardiac: bradycardia, proarrhythmias, worsening of heart failure

- **Sotalol**

  Indications: **ventricular tachyarrhythmias, supraventricular tachycardias, atrial flutter/fibrillation**

  Mechanism of action: **beta blocker**

  - **increase action potential duration and refractory period**

  Side effects: **proarrhythmias, other adverse effects of beta blockers**

- **Adenosine**

  Indications: **supraventricular tachycardia**

  Mechanism of action: **activate potassium channel**

  Side effects: **flushing, dyspnea, chest discomfort, transient bradycardia, heart block**