Cardiac Arrhythmias

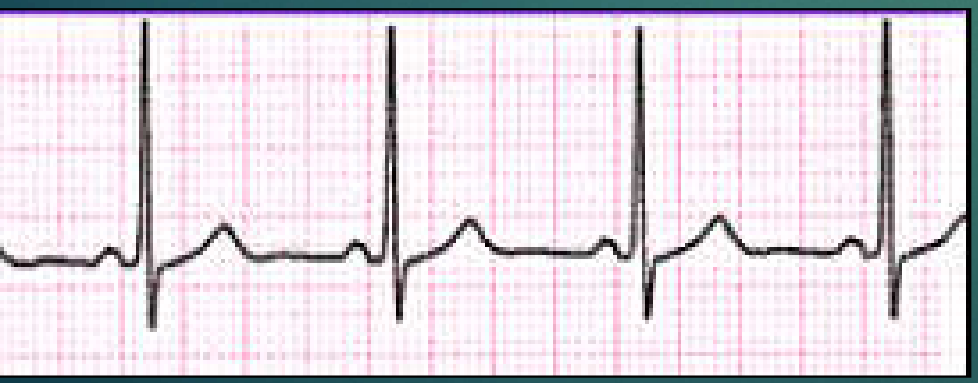
Mohamed Abdel Wahab Ezzat, MD

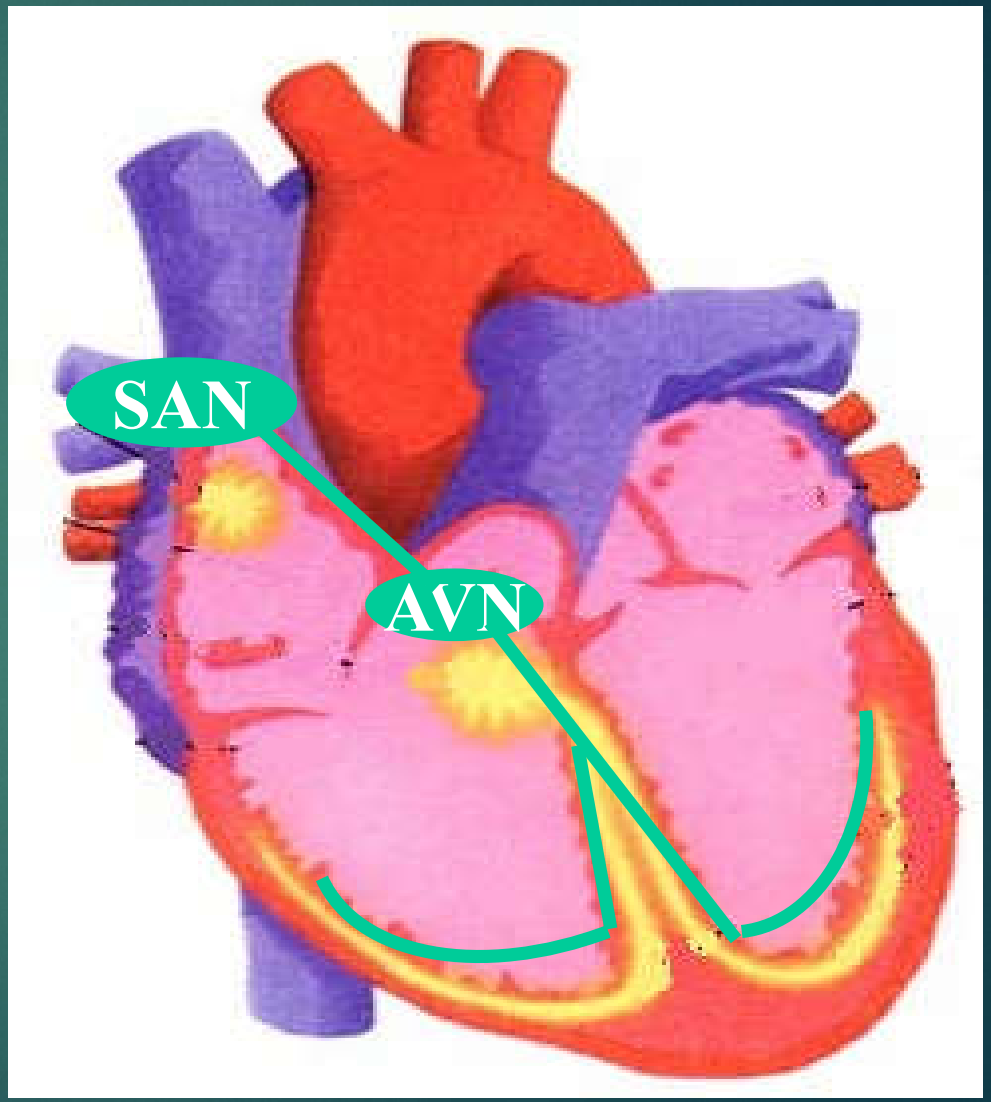
Associate professor of Cardiology

Sohag University

Impulse conduction

Impulses originate regularly at a frequency of **60-100 beat/ min**

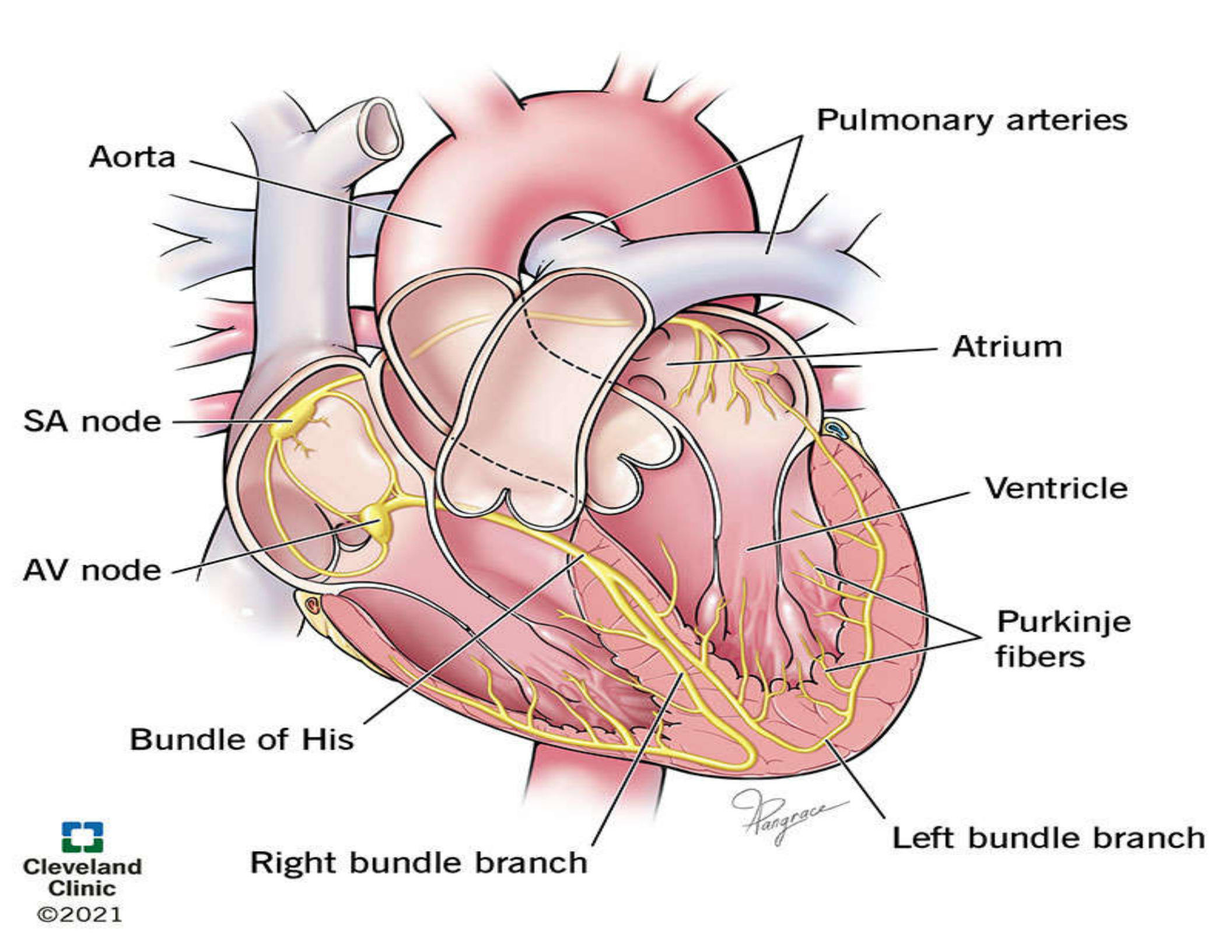


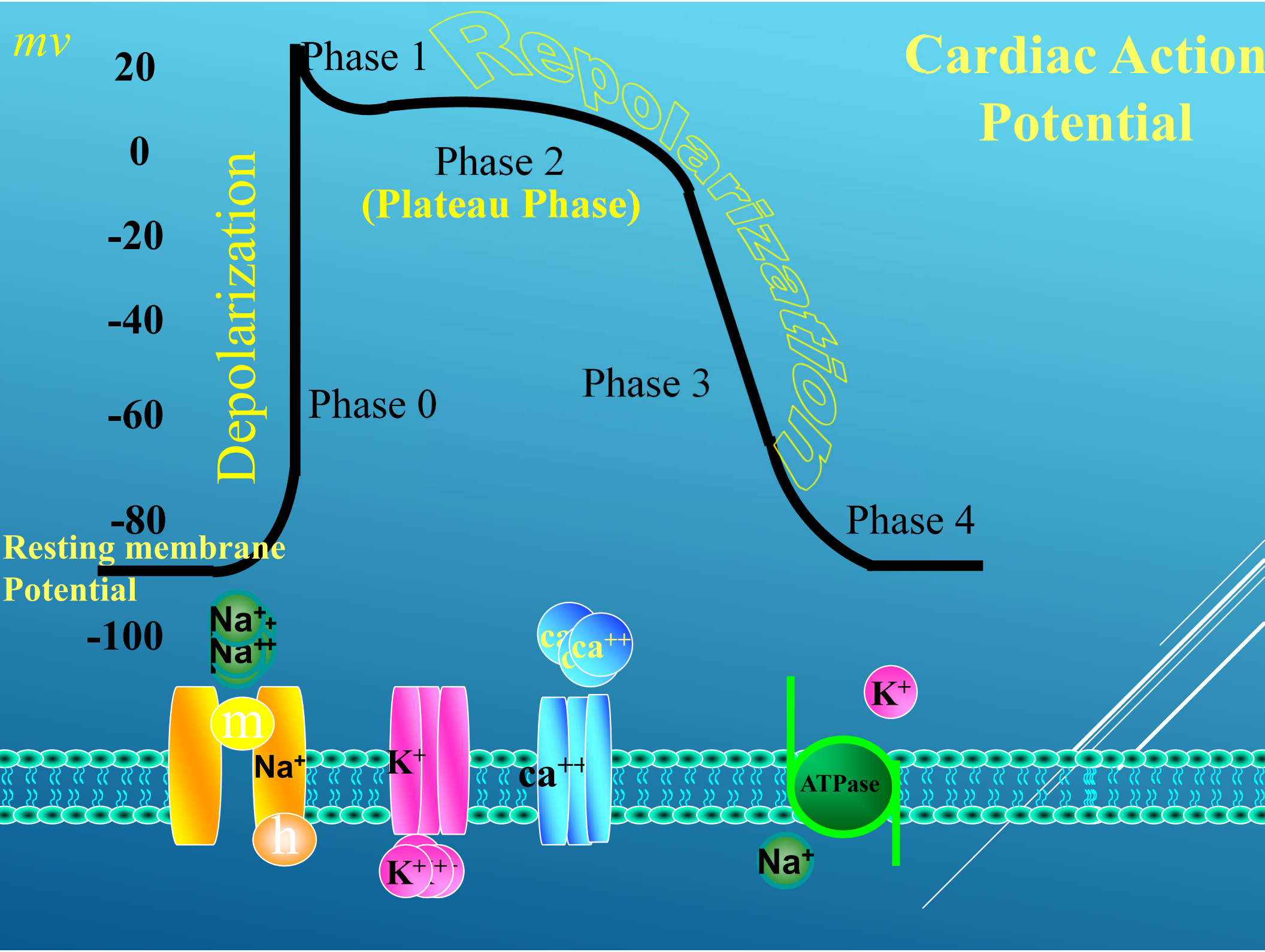


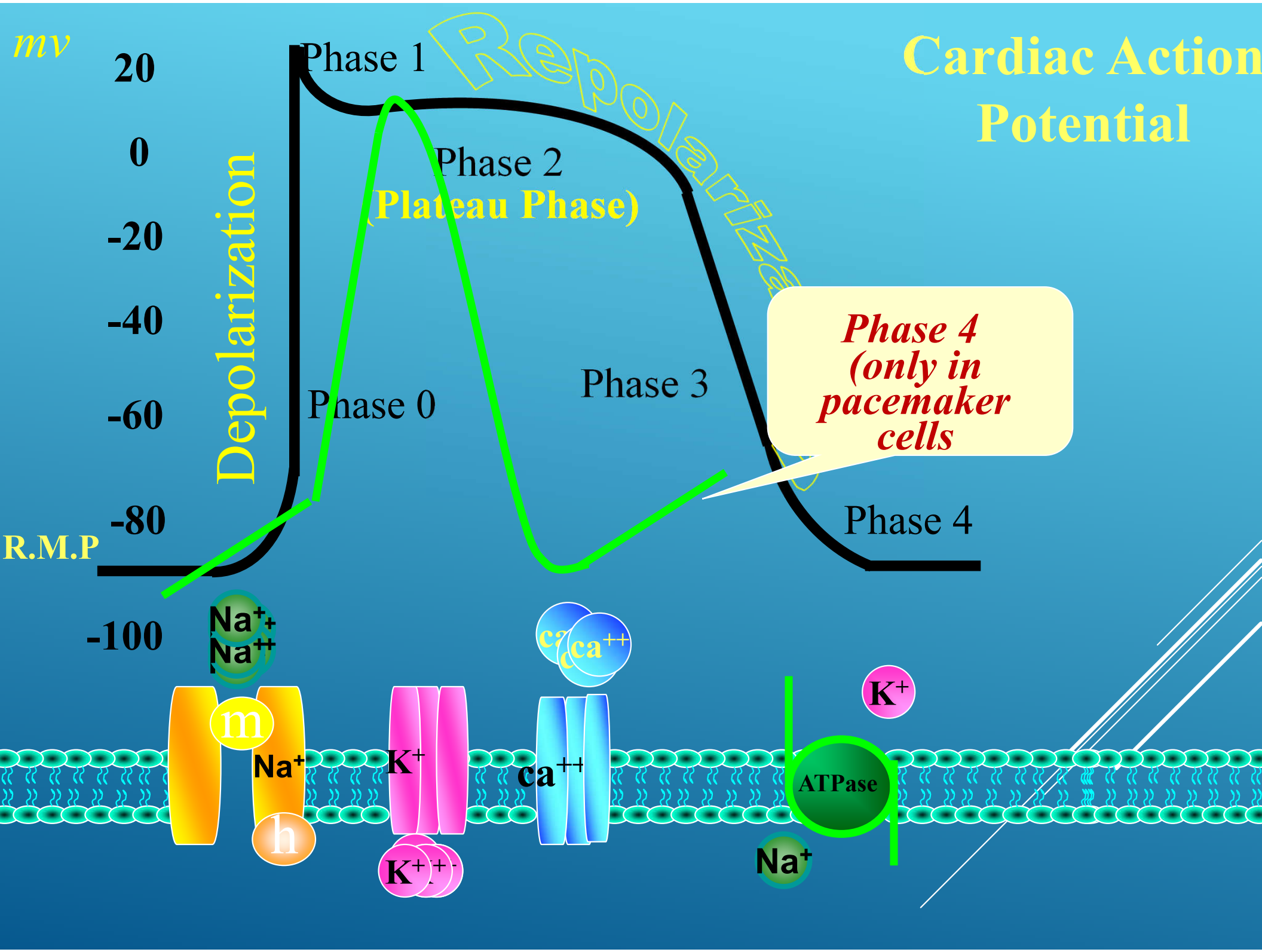
WHAT ARE THE PARTS OF THE CARDIAC CONDUCTION SYSTEM?

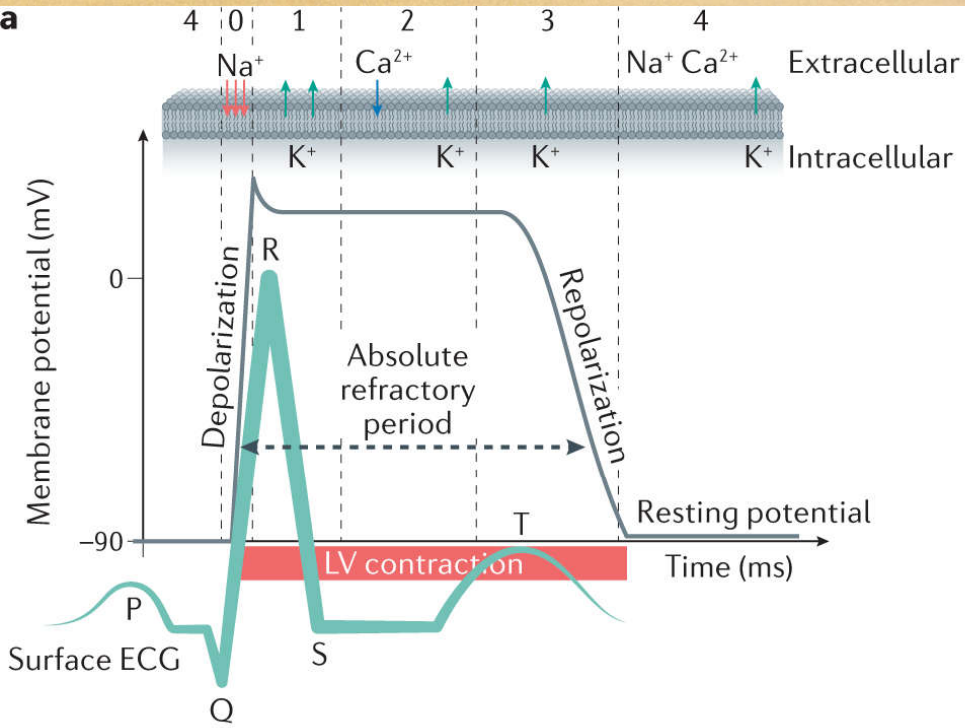
Your cardiac conduction system contains specialized cells and nodes that control your heartbeat. These are the:

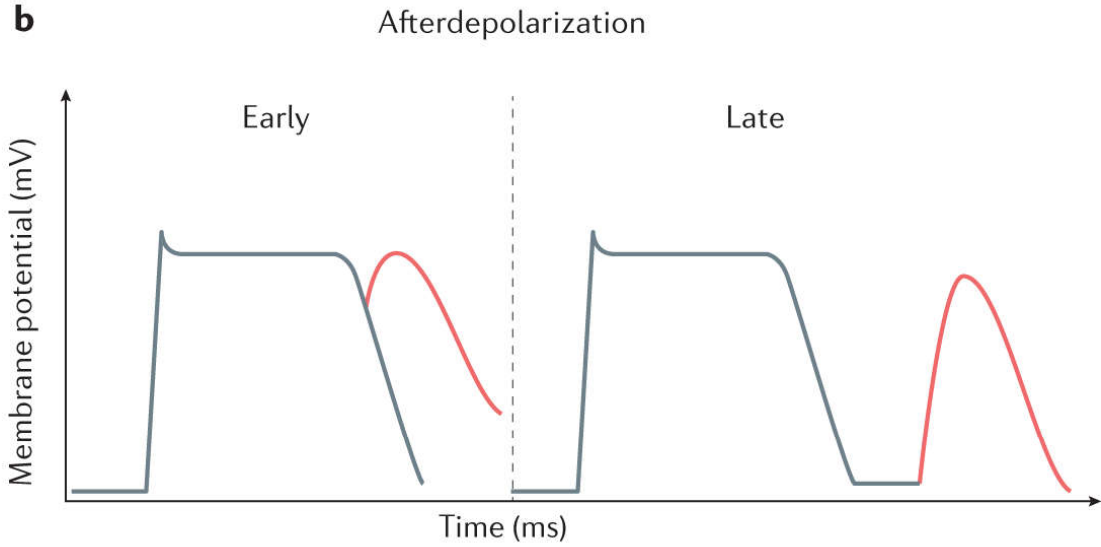
- Sinoatrial node.
- Atrioventricular node.
- Bundle of His (atrioventricular bundle).
- Purkinje fibers.

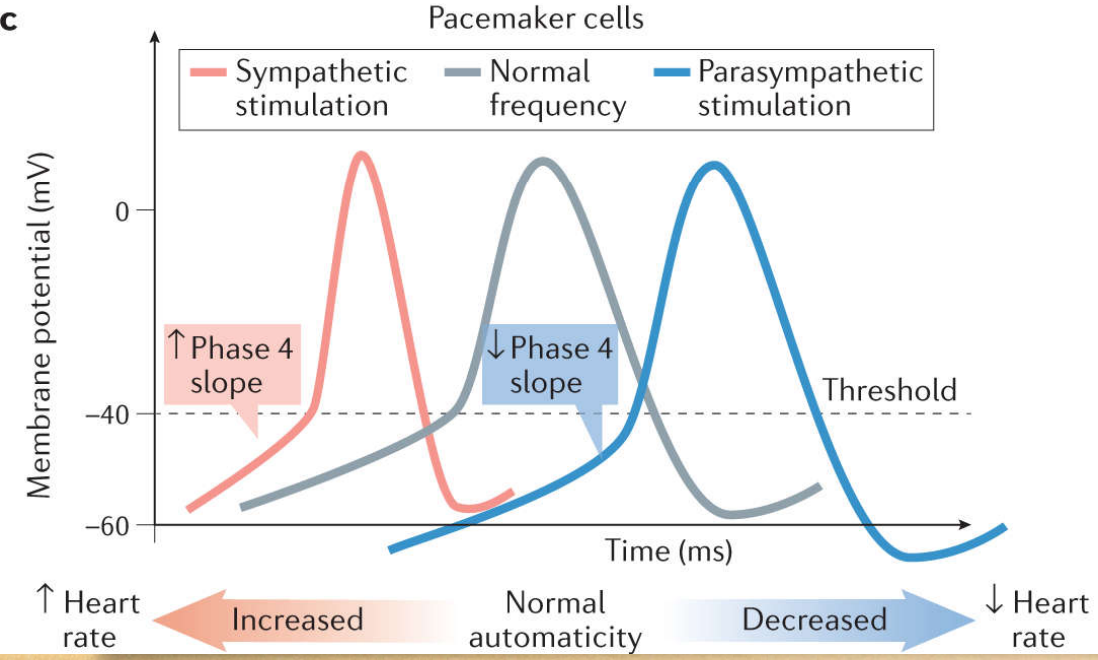

Aorta
Pulmonary arteries
Atrium
SA node
Ventricle
AV node
Purkinje fibers
Bundle of His
Right bundle branch
Left bundle branch
Cleveland Clinic
©2021

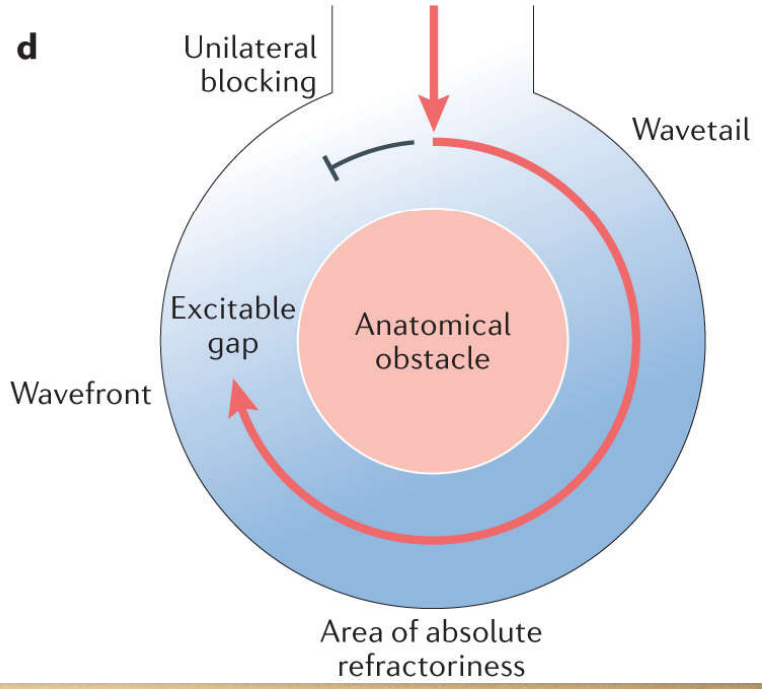

mv
20
0
-20
-40
-60
-80
-100
Phase 1
Phase 2
(Plateau Phase)
Phase 3
Phase 4
Phase 0
Depolarization
Repolarization
Cardiac Action Potential
Resting membrane Potential
Na++
Na++
m
h
Na+
K+
K+
ca++
ca++
K+
ATPase
Na+

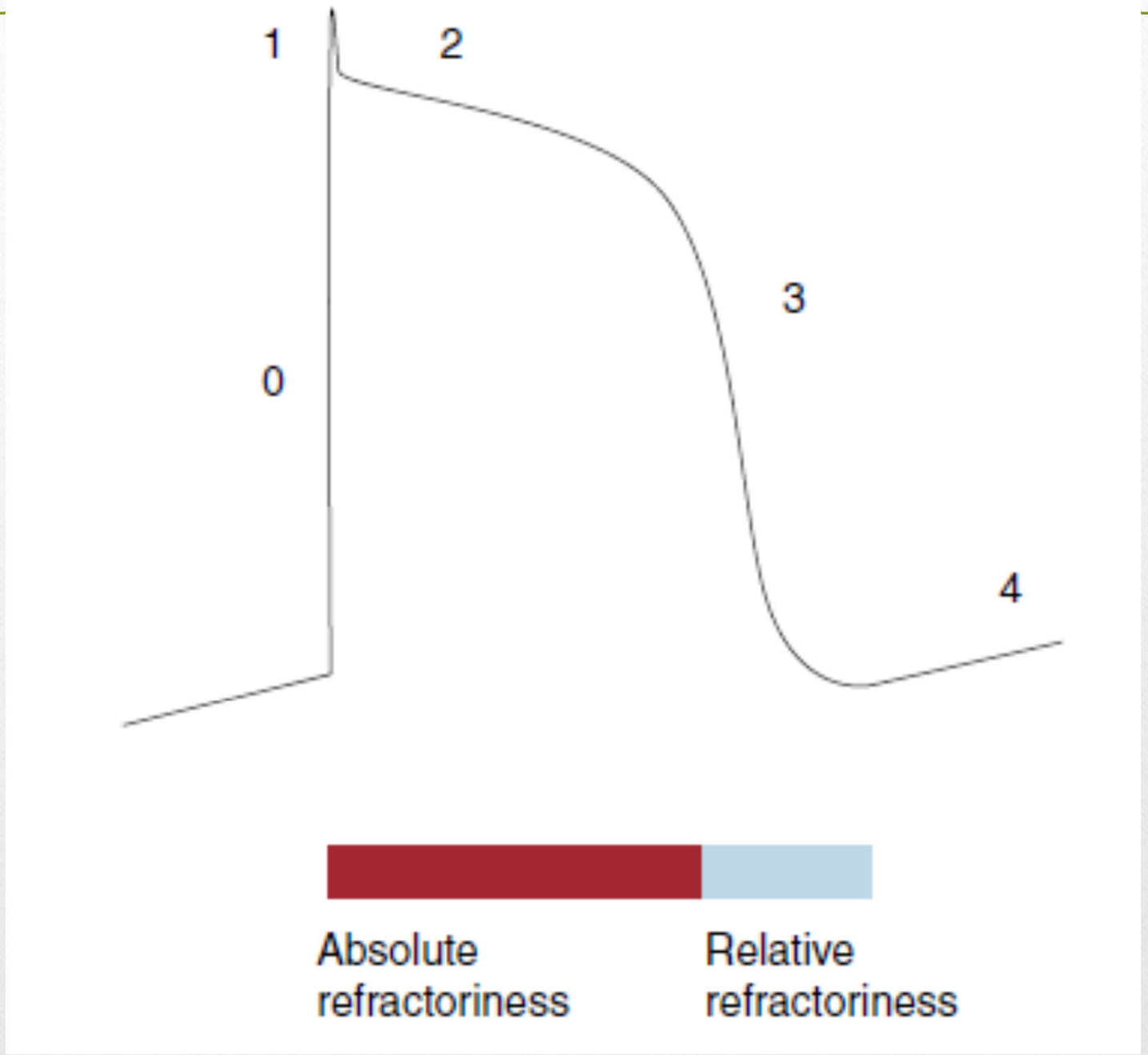

mv
20
0
-20
-40
-60
-80
-100
R.M.P
Depolarization
Repolarization
Phase 1
Phase 2
(Plateau Phase)
Phase 0
Phase 3
Phase 4
Phase 4
(only in
pacemaker
cells
Cardiac Action
Potential
Na+
Na++
m
h
Na+
K+
K++
ca++
ca++
K+
ATPase
Na+


a
4
0
1
2
3
4
Na+
Ca2+
Na+ Ca2+
Extracellular
K+
K+
K+
K+
Intracellular
Membrane potential (mV)
0
−90
Depolarization
R
Absolute refractory period
Repolarization
T
Resting potential
LV contraction
Time (ms)
P
Surface ECG
Q
S


b
Afterdepolarization
Early
Late
Membrane potential (mV)
Time (ms)


c
Pacemaker cells
Sympathetic stimulation
Normal frequency
Parasympathetic stimulation
Membrane potential (mV)
0
−40
−60
↑ Phase 4 slope
↓ Phase 4 slope
Threshold
Time (ms)
↑ Heart rate
Increased
Normal automaticity
Decreased
↓ Heart rate


d
Unilateral blocking
Wavetail
Excitable gap
Anatomical obstacle
Wavefront
Area of absolute refractoriness


1
2
3
0
4
Absolute refractoriness
Relative refractoriness

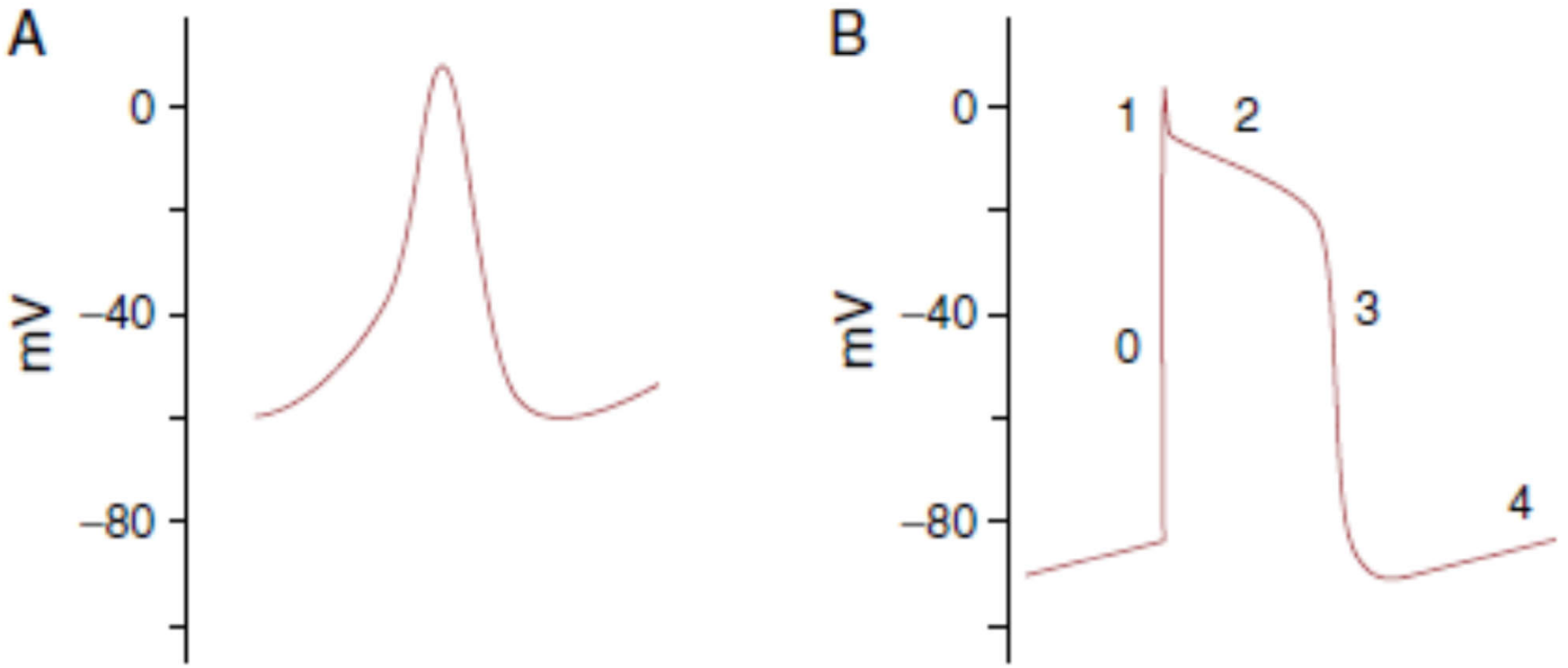


Figure 1. The cardiac action potential. A: sinus node action potential. B: muscle cell action potential.

Cardiac Arrhythmias

- An abnormality of the cardiac rhythm is called a cardiac arrhythmia.
- Arrhythmias may cause sudden death, syncope, heart failure, dizziness, palpitations or no symptoms at all.
- There are two main types of arrhythmia:

bradycardia: the heart rate is slow (< 60 b.p.m).

tachycardia: the heart rate is fast (> 100 b.p.m).

Mechanisms of Cardiac Arrhythmias

Disorders of impulse formation	Disorders of impulse conduction
Automaticity	*Reentry*
Altered normal automaticity	Anatomic reentry
Abnormal automaticity	Functional reentry
Triggered activity	Block
Delayed afterdepolarization	
Early afterdepolarization	

Altered Normal Automaticity

- **Some specialized heart cells, such as sinoatrial nodal cells, the atrioventricular (AV) node, and the His- Purkinje system, as well as some cells in both atria, possess the property of pacemaker activity or automaticity.**

- **Suppression or enhancement of this activity may lead to clinical arrhythmias.**

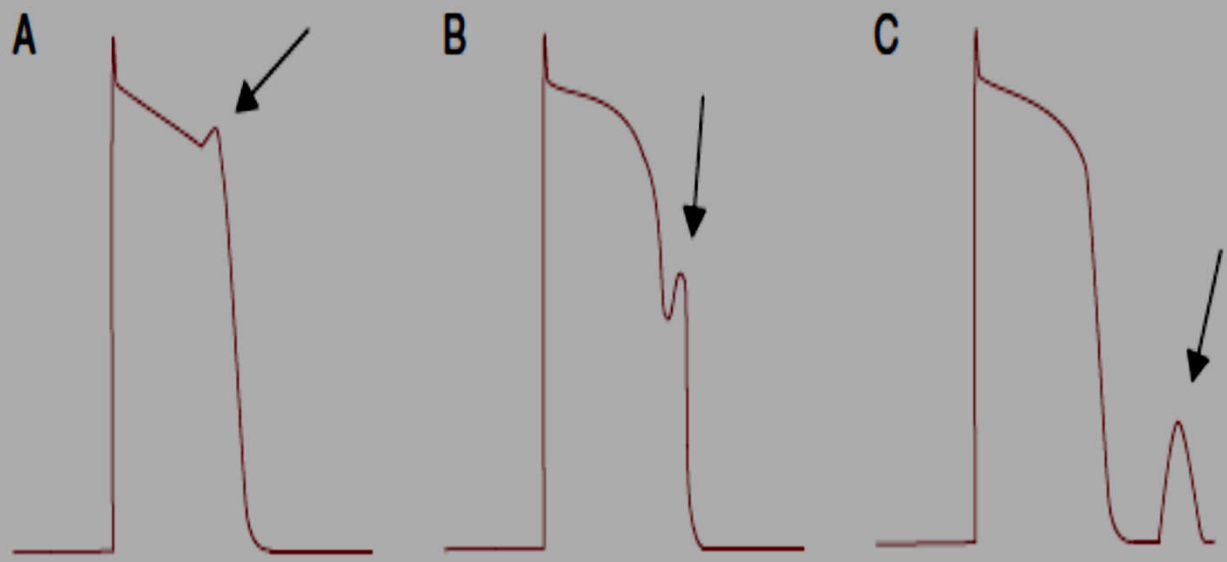
Abnormal Automaticity

- **Atrial and ventricular nonpacemaker myocardial cells, which in the normal heart typically do not exhibit spontaneous activity, may exhibit automaticity properties.**
- **This can happen under conditions that drive the maximum diastolic potential towards the threshold potential, which is explained by the interplay of numerous currents that together result in a net inward depolarizing current associated with a decrease in potassium conductance.**

Triggered Activity

- **Triggered activity (TA) is defined by impulse initiation caused by afterdepolarizations (membrane potential oscillations that occur during or immediately following a preceding action potential {AP}).**

- **Afterdepolarizations occur only in the presence of a previous AP (the trigger), and when they reach the threshold potential, a new AP is generated. This may be the source of a new triggered response, leading to self-sustaining TA.**



ıre 6. Representation of triggered activity. A: phase 2 early afterdepolarization. B: phase 3 early afterdepolarization. C: delayed afterdepolarization.

Re-entry

- During normal electrical activity, the cardiac cycle begins in the sinoatrial node and continues to propagate until the entire heart is activated.
- This impulse dies out when all fibers have been depolarized and are completely refractory.
- However, if a group of isolated fibers is not activated during the initial wave of depolarization, they can recover excitability in time to be depolarized before the impulse dies out.

Re-entry (cont.)

- They may then serve as a link to reexcite areas that were previously depolarized but have already recovered from the initial depolarization.
- Such a process is commonly denoted as reentry, reentrant excitation, circus movement, reciprocal or echo beats, or reciprocating tachycardia (RT), referring to a repetitive propagation of the wave of activation, returning to its site of origin to reactivate that site.

Mechanisms of Cardiac Arrhythmias

Mechanisms of bradycardias:

Sinus bradycardia is a result of abnormally slow automaticity while bradycardia due to AV block is caused by abnormal conduction within the AV node or the distal AV conduction system.

Mechanisms generating tachycardias include:

- Accelerated automaticity.
- Triggered activity
- Re-entry (or circus movements)

ACCELERATED AUTOMATICITY

- It occurs due to increasing the rate of diastolic depolarization or changing the threshold potential.
- Abnormal automaticity can occur in virtually all cardiac tissues and may initiate arrhythmias.
- Such changes are thought to produce sinus tachycardia, escape rhythms and accelerated AV nodal (junctional) rhythms.

TRIGGERED ACTIVITY

- Myocardial damage can result in oscillations of the transmembrane potential at the end of the action potential. These oscillations, which are called 'after depolarizations', may reach threshold potential and produce an arrhythmia.

- The abnormal oscillations can be exaggerated by pacing, catecholamines, electrolyte disturbances, and some medications.

- Examples as atrial tachycardias produced by digoxin toxicity and the initiation of ventricular arrhythmia in the long QT syndrome.

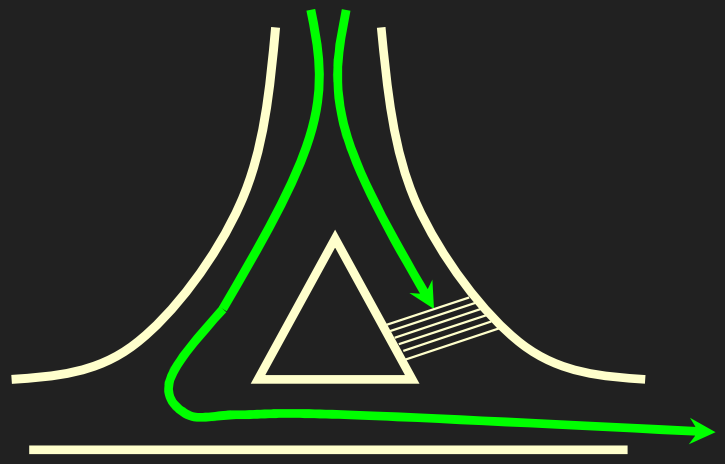
Re-entry (or circus movement)

- The mechanism of re-entry occurs when a 'ring' of cardiac tissue surrounds an inexcitable core (e.g. in a region of scarred myocardium).
- Tachycardia is initiated if an ectopic beat finds one limb refractory (α) resulting in unidirectional block and the other limb excitable.
- Provided conduction through the excitable limb (β) is slow enough, the other limb (α) will have recovered and will allow retrograde activation to complete the re-entry loop.

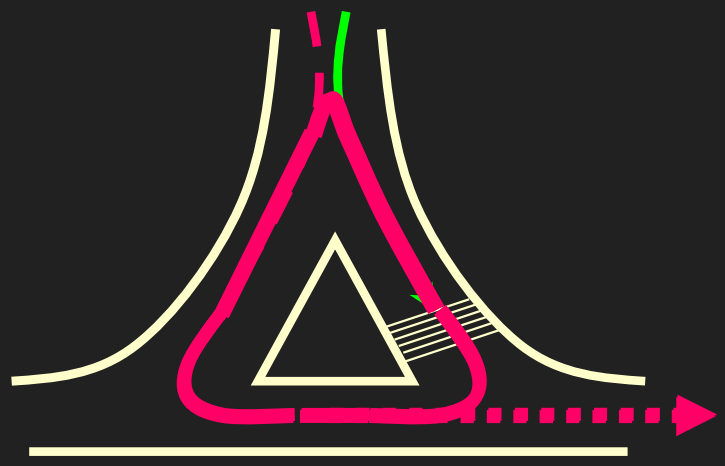
Re-entry (or circus movement), cont.

- If the time to conduct around the ring is longer than the recovery times (refractory periods) of the tissue within the ring, circus movement will be maintained, producing a run of tachycardia.
- The majority of regular paroxysmal tachycardias are produced by this mechanism.

Reentry Arrhythmias



Normal



Re-enterant Tachycardia

Atrial Arrhythmias

Sinus arrhythmia:

- A condition in which the heart rate varies with breathing.
- This is usually a benign condition

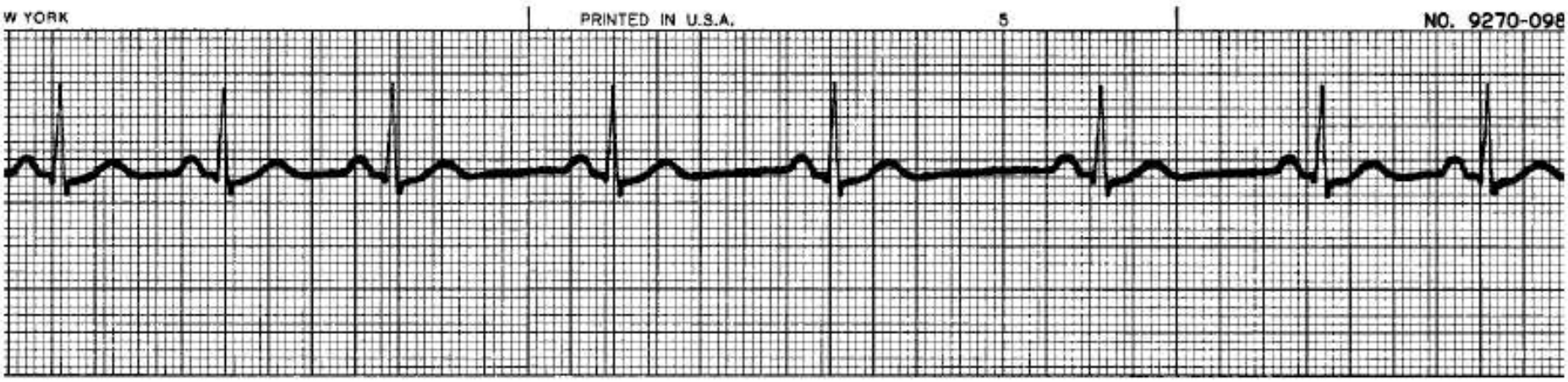


FIGURE 27-8 Sinus arrhythmia in lead II.

SUPRAVENTRICULAR TACHYCARDIAS

- Supraventricular tachycardias (SVTs) arise from the atrium or the atrioventricular junction.
- Conduction is via the His-Purkinje system; therefore the QRS shape during tachycardia is usually similar to that seen in the same patient during baseline rhythm.

Causes of SVT

Tachycardia	ECG features	Comment
Sinus tachycardia	P wave morphology similar to sinus rhythm	Need to determine underlying cause
AV nodal re-entry tachycardia (AVNRT)	No visible P wave, or inverted P wave immediately before or after QRS complex	Commonest cause of palpitations in patients with normal hearts
AV reciprocating tachycardia (AVRT)	P wave visible between QRS and T wave complexes	Due to an accessory pathway. If pathway conducts in both directions, ECG during sinus rhythm may be pre-excited
Atrial fibrillation	Irregularly irregular RR intervals and absence of organized atrial activity	Commonest tachycardia in patients over 65 years
Atrial flutter	Visible flutter waves at 300/min (saw-tooth appearance) usually with 2 : 1 AV conduction	Suspect in any patient with regular SVT at 150/min
Atrial tachycardia	Organized atrial activity with P wave morphology different from sinus rhythm	Usually occurs in patients with structural heart disease
Multifocal atrial tachycardia	Multiple P wave morphologies (≥3) and irregular RR intervals	Rare arrhythmia; most commonly associated with significant chronic lung disease
Accelerated junctional tachycardia	ECG similar to AVNRT	Rare in adults

SVT

Sinus tachycardia

- A condition in which the heart rate is 100-160/min
- Symptoms may occur with rapid heart rates including; weakness, fatigue, dizziness, or palpitations.
- Sinus tachycardia is often temporary, occurring under stresses from exercise, strong emotions, fever, dehydration, thyrotoxicosis, anemia and heart failure.
- If necessary, beta-blockers may be used to slow the sinus rate, e.g. in hyperthyroidism

SINUS TACHYCARDIA

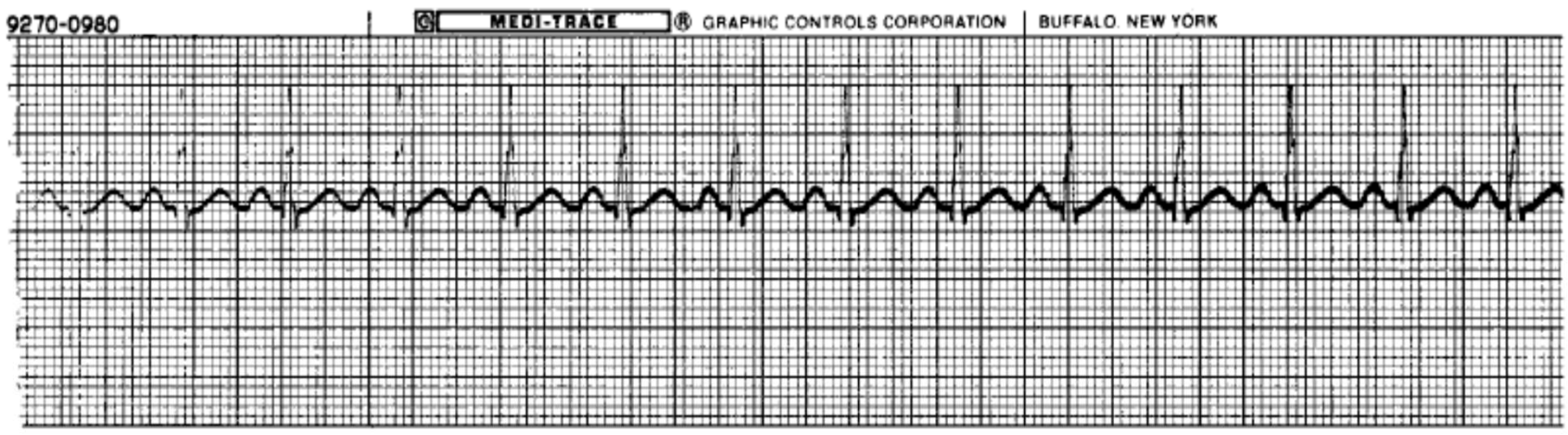
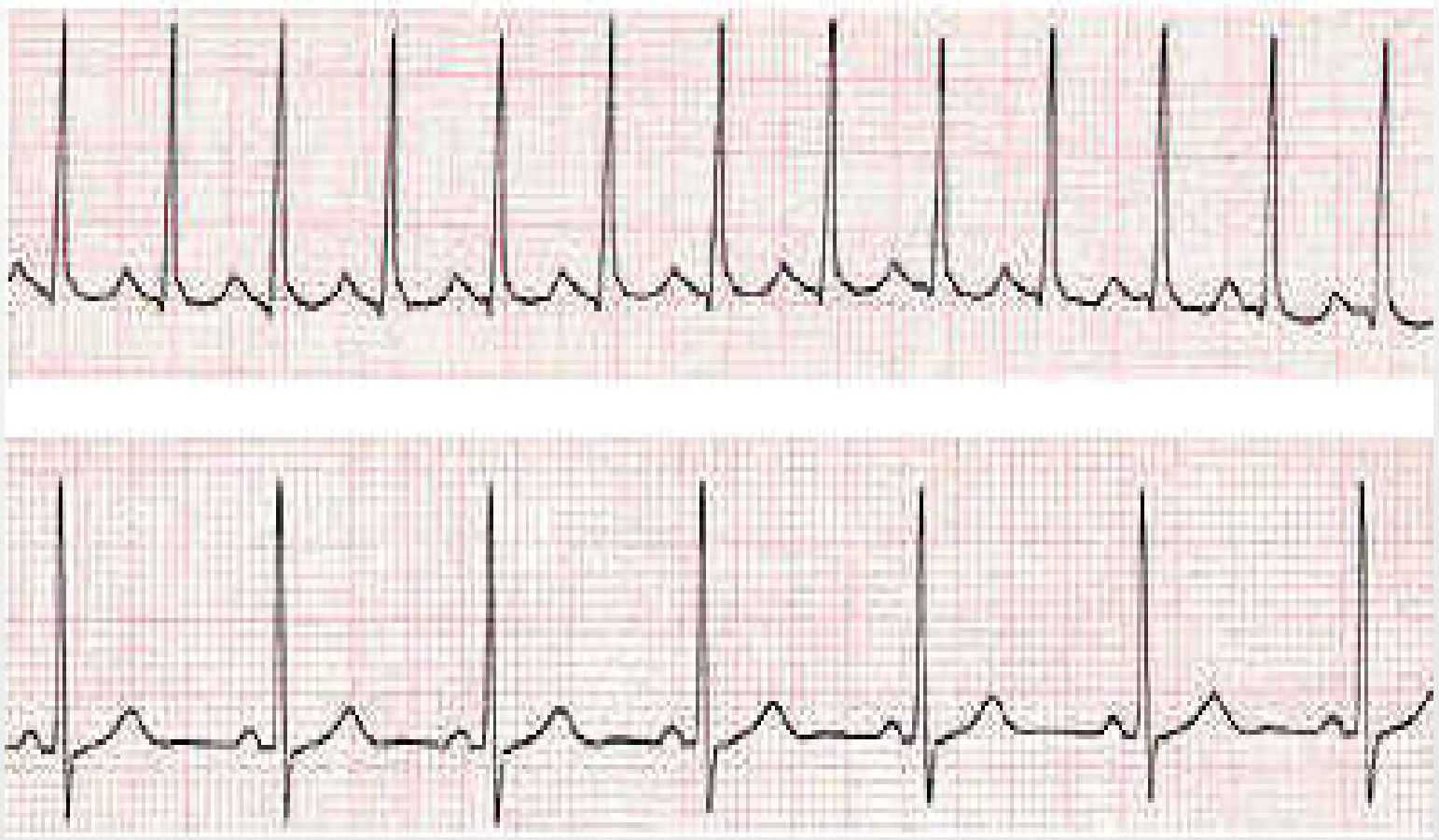


FIGURE 27-7 Sinus tachycardia in lead II.

Sinus tachycardia converted to NSR



Atrial Arrhythmias

Premature supraventricular contractions or premature atrial contractions (PAC)

- A condition in which an atrial pacemaker site above the ventricles sends out an electrical signal early. The ventricles are usually able to respond to this signal, but the result is an irregular heart rhythm.
- PACs are common and may occur as the result of stimulants such as coffee, tea, alcohol, cigarettes, or medications.
- Treatment is rarely necessary.

PAC

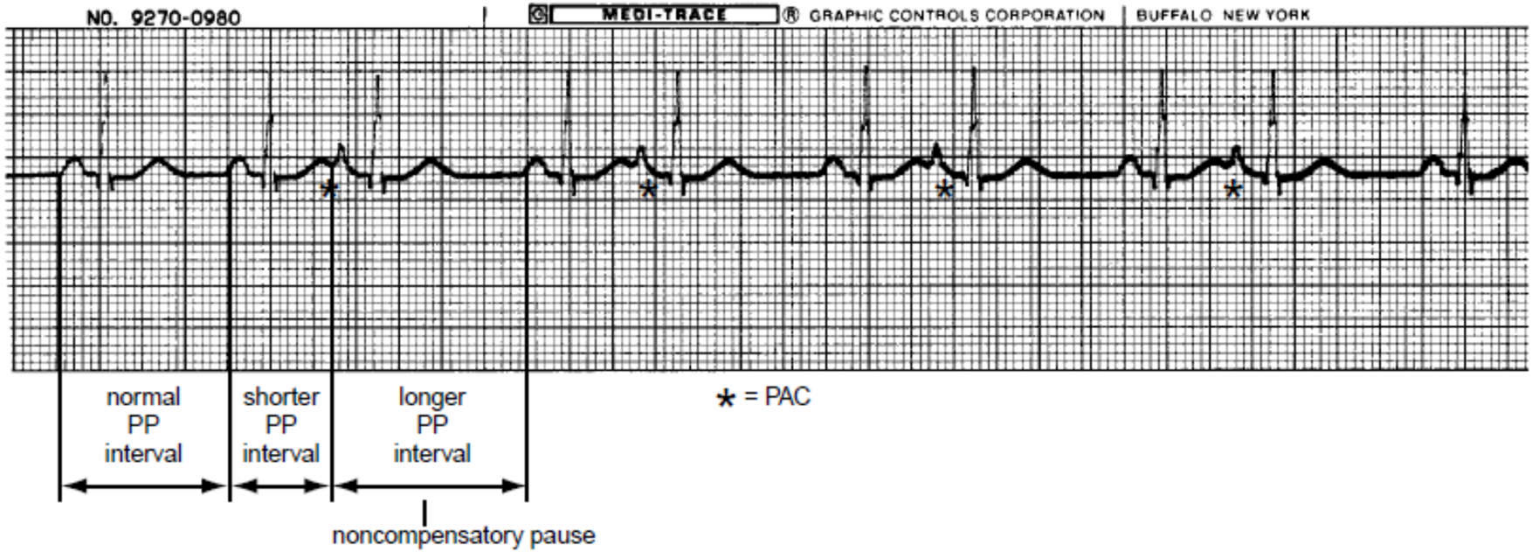


FIGURE 27-9 Premature atrial complexes (PACs) in lead II. Note pause following PAC that is longer than the normal PP interval, but shorter than twice the normal PP interval.

SVT

Paroxysmal Supraventricular tachycardia [HR 160-250/min]

- **Atrioventricular nodal re-entry tachycardia (AVNRT)**
- It usually begins and ends rapidly, occurring in repeated periods. This condition can cause symptoms such as weakness, fatigue, dizziness, fainting, or palpitations if the heart rate becomes too fast.
- In AVNRT, there are two functionally and anatomically different pathways within the AV node: one is characterized by a short effective refractory period and slow conduction, and the other has a longer effective refractory period and conducts faster.

Atrioventricular nodal re-entry tachycardia (AVNRT) (cont.)

- In sinus rhythm, the atrial impulse that depolarizes the ventricles usually conducts through the fast pathway.

- If the atrial impulse (e.g. an atrial premature beat) occurs early when the fast pathway is still refractory, the slow pathway takes over in propagating the atrial impulse to the ventricles. It then travels back through the fast pathway which has already recovered its excitability, thus initiating the most common 'slow-fast', or typical, AVNRT.

AVNRT (continue)

The rhythm is recognized on ECG by normal regular QRS complexes, usually at a rate of 140-240 per minute. Sometimes the QRS complexes will show typical bundle branch block. P waves are either not visible or are seen immediately before or after the QRS complex because of simultaneous atrial and ventricular activation.

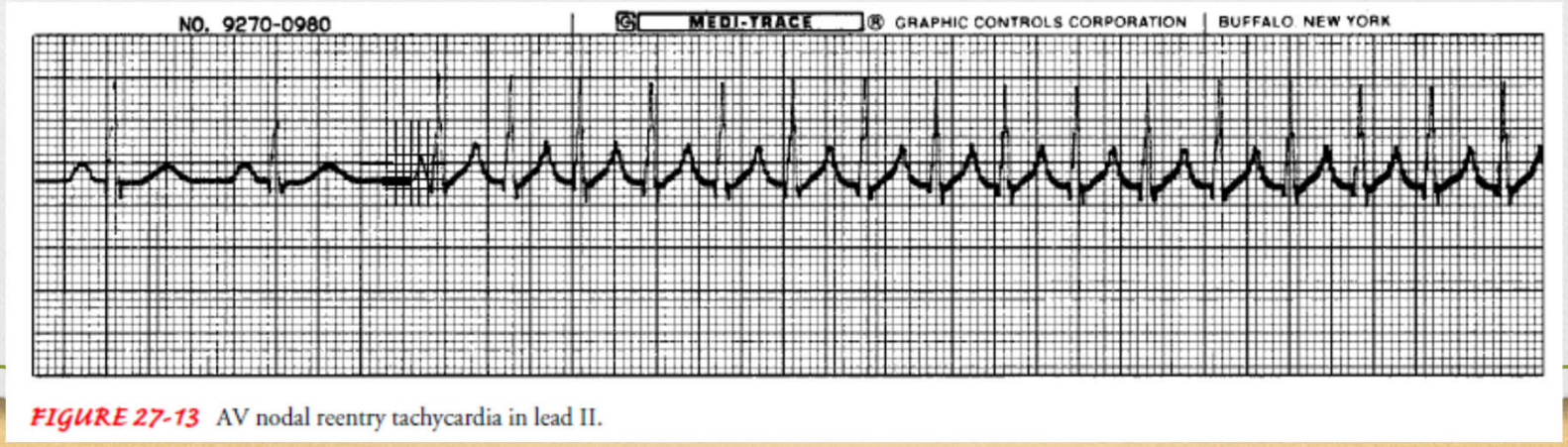


FIGURE 27-13 AV nodal reentry tachycardia in lead II.

SVT

Atrioventricular reciprocating tachycardia (AVRT)

- In AVRT there is a large circuit comprising the AV node, the His bundle, the ventricle and an abnormal connection from the ventricle back to the atrium. This abnormal connection is called an accessory pathway or bypass tract.
- Bypass tracts result from incomplete separation of the atria and the ventricles during fetal development.
- Atrial activation occurs after ventricular activation and the P wave is usually clearly seen between the QRS and T complexes

PSVT

Acute Management

- Patients presenting with SVTs and haemodynamic instability require emergency cardioversion.
- If the patient is haemodynamically stable, vagal manoeuvres, including right carotid massage, Valsalva manoeuvre and facial immersion in cold water can be successfully employed.
- If not successful, intravenous adenosine (up to 0.25 mg/kg) , verapamil 5-10 mg i.v. over 5-10 minutes, i.v. diltiazem, or beta-blockers should be tried.

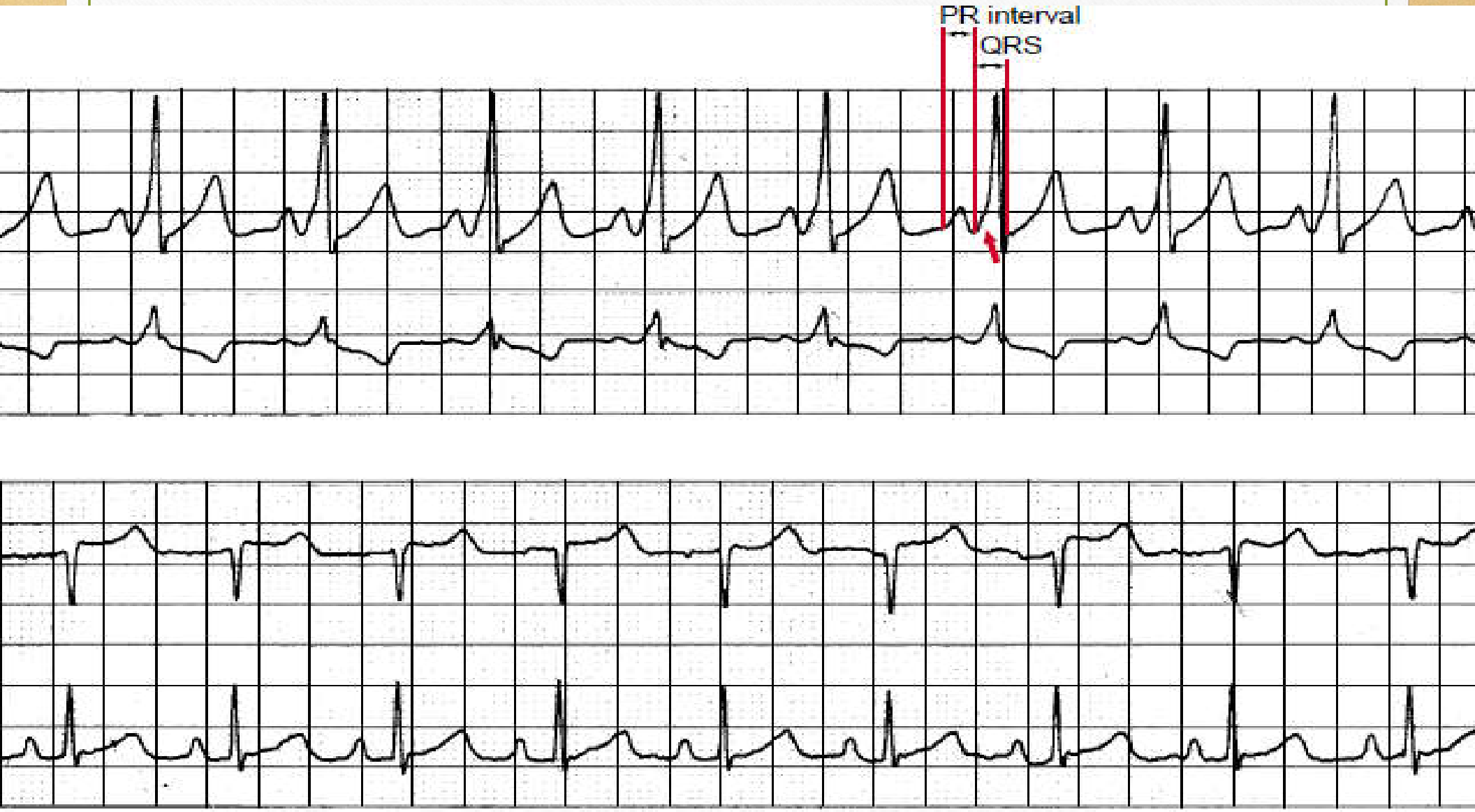
Long-term management

- It includes ablation of an accessory pathway. Also, verapamil, diltiazem & β-blockers; are effective in 60-80% of patients.

Wolf Parkinson White Syndrome (WPW)

- An abnormal band of atrial tissue connects the atria and ventricles and can electrically bypass the normal pathways of conduction; a re-entry circuit can develop causing paroxysms of tachycardia.
- ECG shows:
  - Short PR interval
  - Delta wave on the upstroke of the QRS complex
- Drug treatment includes flecainamide, amiodarone or disopyramide.
- Digoxin and verapamil are contraindicated.
- Transvenous catheter radiofrequency ablation is the treatment of choice.

WPW syndrome



IGURE 27-33 Wolff-Parkinson-White syndrome. (**A**) Sinus rhythm. Note the short PR interval, slurred initial stroke of the QRS complex (delta wave, at the arrow), and prolonged QRS duration, upper lead II, lower lead V_1.) Rhythm strip of same patient following ablation, upper lead V_1, lower lead II. ECG strips courtesy of Linda Ardini

Atrial Arrhythmias

Atrial flutter (HR200-350/min)

- A condition in which the electrical signals come from the atria at a fast but even rate, often causing the ventricles to contract faster and increase the heart rate.
- When the signals from the atria are coming at a faster rate than the ventricles can respond to, the ECG pattern develops a signature "sawtooth" pattern, showing two or more flutter waves between each QRS complex.

Atrial Arrhythmias

Atrial flutter (TREATMENT)

- **Treatment of the symptomatic acute paroxysm is electrical *cardioversion*.**
- Patients who have been in atrial flutter more than 1-2 days should be treated in a similar manner to patients with atrial fibrillation and *anticoagulated* for 4 weeks prior to cardioversion.
- Recurrent paroxysms may be prevented by *class Ic* and *class III* agents
- The treatment of choice for patients with recurrent atrial flutter is radiofrequency catheter *ablation*

ATRIAL FLUTTER

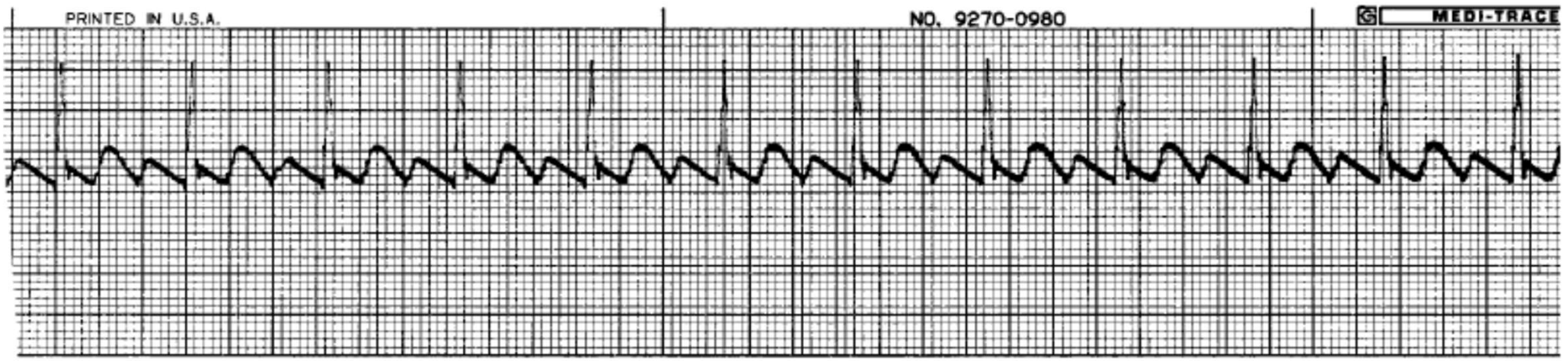


FIGURE 27-10 Atrial flutter in lead II.

Atrial Arrhythmias

Atrial fibrillation (AF) :

- A condition in which the electrical signals come from the atria at a very fast and erratic rate. The ventricles contract in an irregular manner because of the erratic signals coming from the atria.
- The ECG shows normal but irregular QRS complexes, fine oscillations of the baseline (so-called fibrillation or f waves) and no P waves.
- Common causes include CAD, valvular heart disease, hypertension, hyperthyroidism and others. In some patients no cause can be found 'lone' atrial fibrillation.

ATRIAL FIBRILLATION

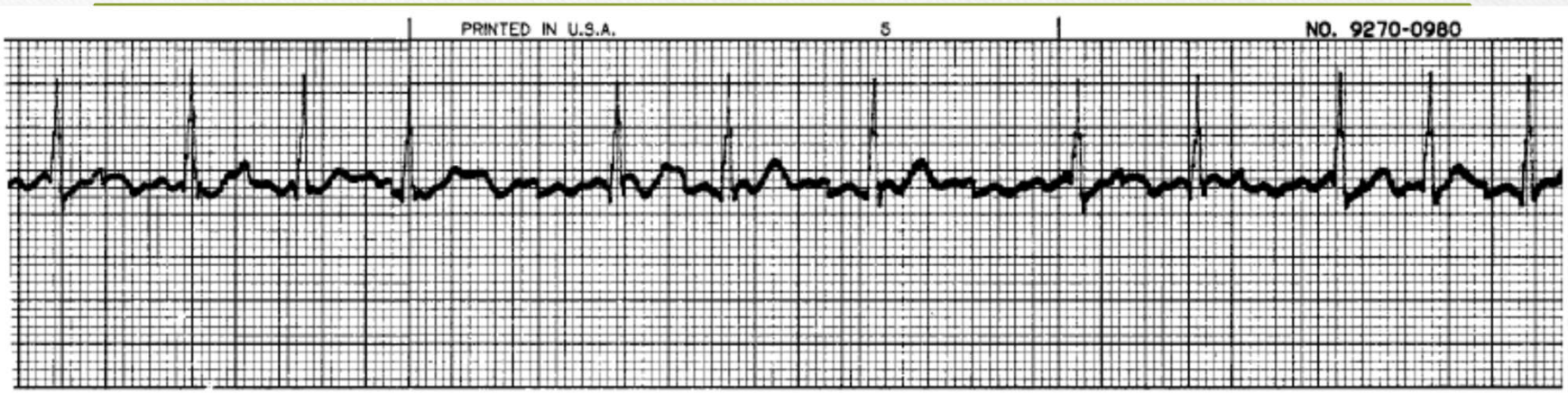


FIGURE 27-11 Atrial fibrillation in lead II.

Atrial Arrhythmias

Management

- When atrial fibrillation is due to an acute precipitating event such as alcohol toxicity, chest infection or hyperthyroidism, the provoking cause should be treated.
- Strategies for the acute management of AF are ventricular rate control or cardioversion (± anticoagulation).
  - Ventricular rate control is achieved by drugs which block the AV node
  - Cardioversion is achieved electrically by DC shock or medically either by IV infusion of an anti-arrhythmic drug such as a class Ic or a class III agent

AF

The choice depends upon:

- **How well the arrhythmia is tolerated (is cardioversion urgent?)**
- **Whether anticoagulation is required before considering elective cardioversion**
- **Whether spontaneous cardioversion is likely (previous history? reversible cause?).**

Atrial Arrhythmias

Management (continue)

- Patients are anticoagulated with warfarin for 4 weeks before cardioversion.
- Anticoagulants are used to minimize the risk of thromboembolism associated with cardioversion unless atrial fibrillation is of less than 1-2 days' duration.
- Transoesophageal echocardiography is being used to document the presence or absence of atrial thrombus as a guide to the necessity for long-term anticoagulation.

Atrial Arrhythmias

Management

- Long-term management of atrial fibrillation include two strategies:
  - Rhythm control: antiarrhythmic drugs plus DC cardioversion plus warfarin
  - Rate control: AV nodal slowing agents plus warfarin
- Recurrent paroxysms may be prevented by oral medication; class Ic agents are employed in patients with no significant heart disease and class III agents are preferred in patients with structural heart disease.
- Rate control is usually achieved by a combination of digoxin beta-blockers or calcium channel blockers (diltiazem or verapamil).

AF

- **Anticoagulation (target INR 2.0-3.0) This is indicated in patients with atrial fibrillation and one of the following major or two of the moderate risk factors:**
- **Major risk factors: Prosthetic heart valve, Rheumatic mitral valve disease, Prior history of CVA/TIA, Age > 75 years, Hypertension, Coronary artery disease with poor LV function**
- **Moderate risk factors: Age 65-75 years, Coronary artery disease but normal LV function, Diabetes mellitus.**

Ventricular Tachyarrhythmias

Ventricular tachyarrhythmias can be considered under the following headings:

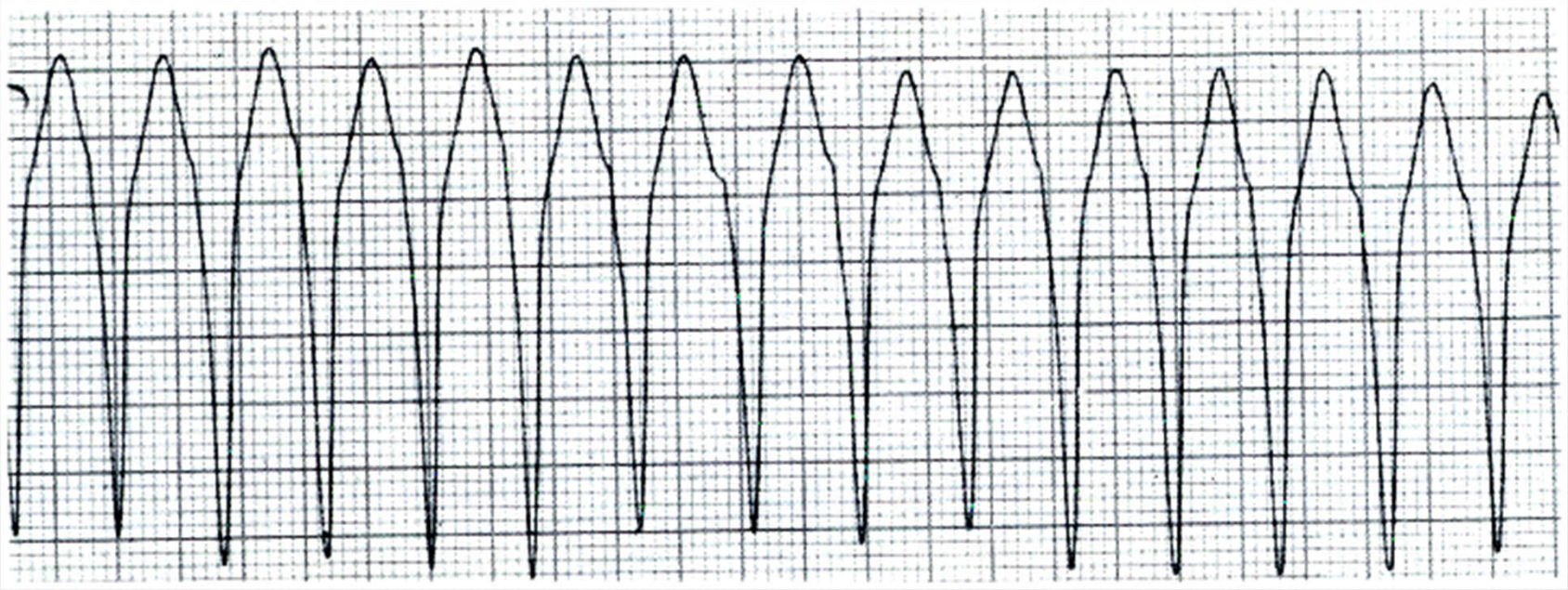
- life-threatening ventricular tachyarrhythmias (Sustained ventricular tachycardia and ventricular fibrillation)
- torsades de pointes
- normal heart ventricular tachycardia
- non-sustained ventricular tachycardia
- ventricular premature beats

Ventricular Arrhythmias

Ventricular tachycardia (VT)

- A condition in which an electrical signal is sent from the ventricles at a very fast but often regular rate.
- The ECG shows a rapid ventricular rhythm with broad (often 0.14 s or more), abnormal QRS complexes. AV dissociation may result in visible P waves
- *Treatment:* in haemodynamically compromised patients, emergency DC cardioversion may be required. If the blood pressure and cardiac output are well maintained, intravenous therapy with class I drugs or amiodarone is usually used. First-line drug treatment consists of lidocaine (50-100 mg i.v. over 5 minutes) followed by a lidocaine infusion (2-4 mg i.v. per minute). DC cardioversion is necessary if medical therapy is unsuccessful.

Ventricular Tachycardia



Ventricular Arrhythmias

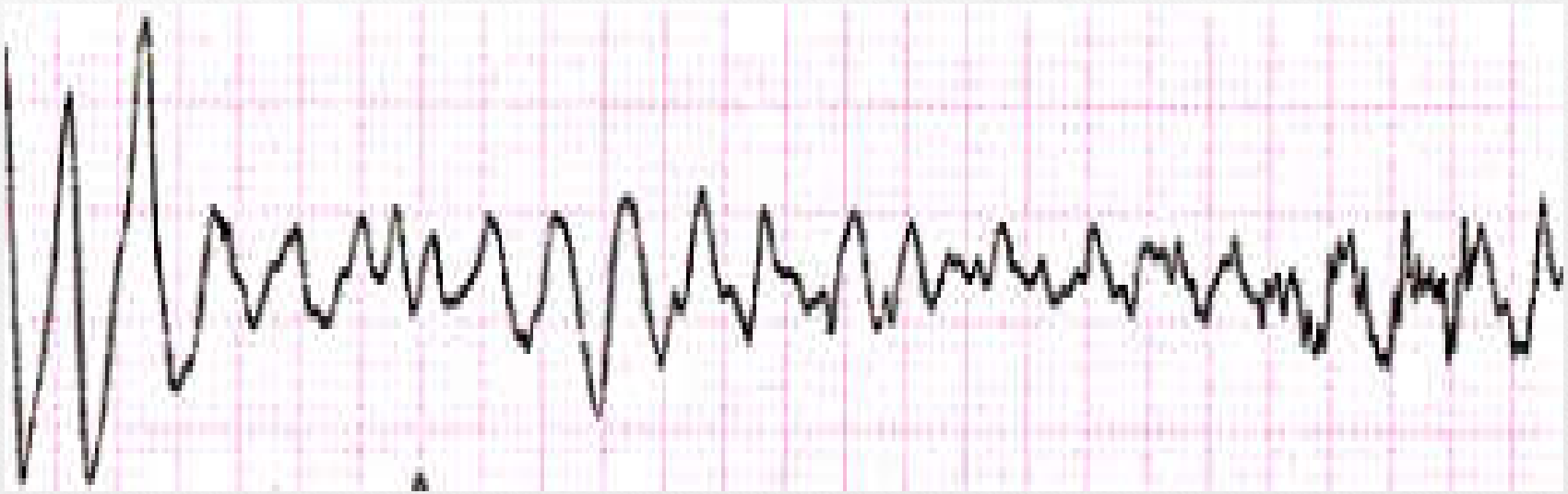
Ventricular fibrillation (VF)

- A condition in which many electrical signals are sent from the ventricles at a very fast and erratic rate. As a result, the ventricles are unable to fill with blood and pump.
- This rhythm is life-threatening because there is no pulse and complete loss of consciousness.
- The ECG shows shapeless, rapid oscillations and there is no hint of organized complexes

VF

- A person in VF requires prompt defibrillation to restore the normal rhythm and function of the heart. It may cause sudden cardiac death. Basic and advanced cardiac life support is needed
- Survivors of these ventricular tachyarrhythmias are, in the absence of an identifiable reversible cause (e.g. acute myocardial infarction, severe metabolic disturbance), at high risk of sudden death. Implantable cardioverter-defibrillators (ICDs) are first-line therapy in the management of these patients

Ventricular Fibrillation



Ventricular Arrhythmias

Torsades de pointes :

- This is a type of short duration tachycardia that reverts to sinus rhythm spontaneously.
- It may be due to:

  - Congenital

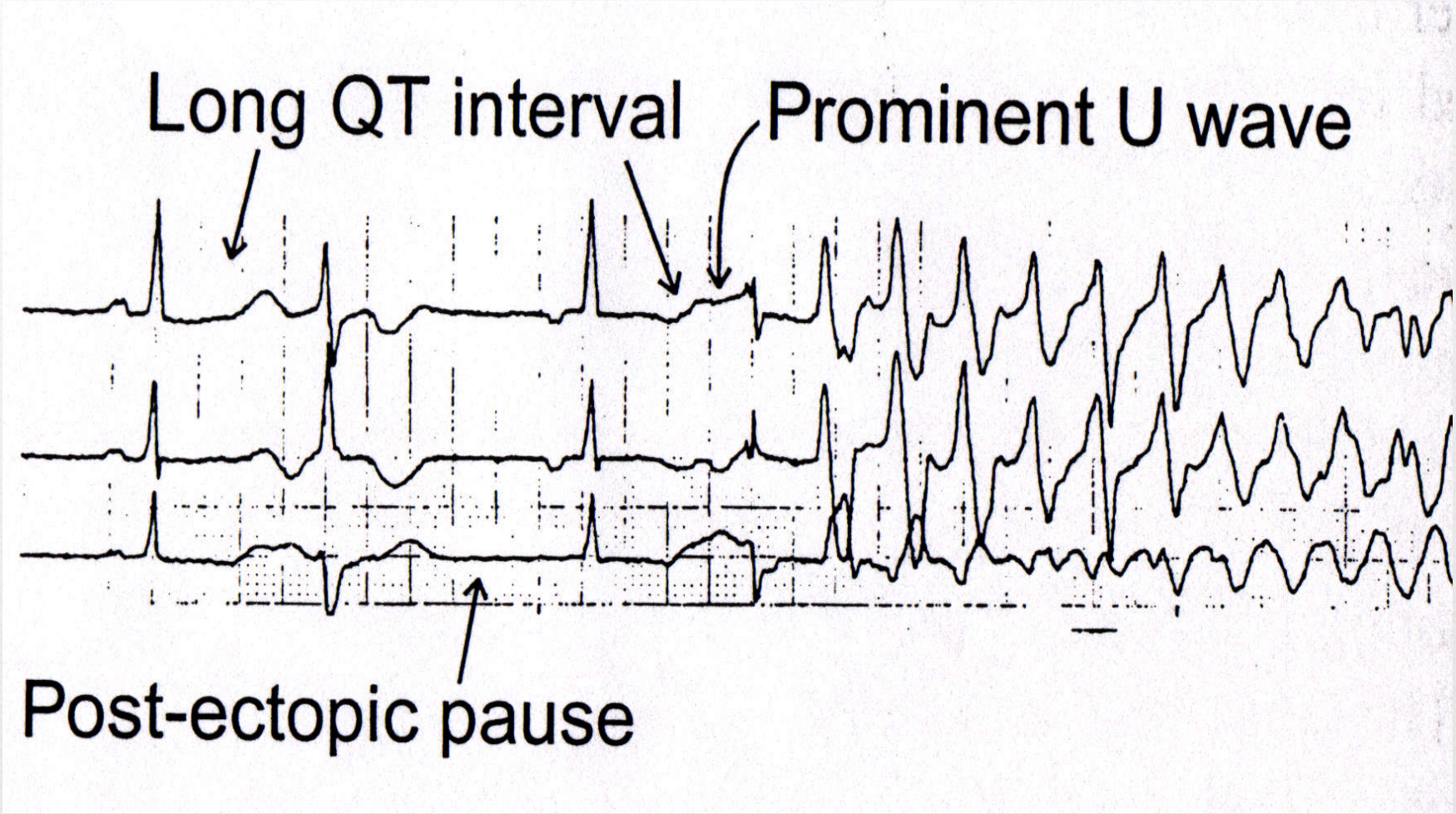
  - Electrolyte disorders e.g. hypokalemia, hypomagnesemia, hypocalcemia.

  - Drugs e.g. tricyclic antidepressant, class IA and III antiarrhythmics.
- It may present with syncopal attacks and occasionally ventricular fibrillation.
- QRS complexes are irregular and rapid that twist around the baseline. In between the spells of tachycardia the ECG show prolonged QT interval.

Torsades de pointes

- Treatment includes; correction of any electrolyte disturbances, stopping of causative drug, atrial or ventricular pacing, Magnesium sulphate 8 mmol (mg2+) over 10-15 min for acquired long QT, IV isoprenaline in acquired cases and B blockers in congenital types
- Long-term management of acquired long QT syndrome involves avoidance of all drugs known to prolong the QT interval. Congenital long QT syndrome is generally treated by beta-blockade, left cardiac sympathetic denervation, and pacemaker therapy. Patients who remain symptomatic despite conventional therapy and those with a strong family history of sudden death usually need ICD therapy.

Torsade de Pointes in patient on Sotalol

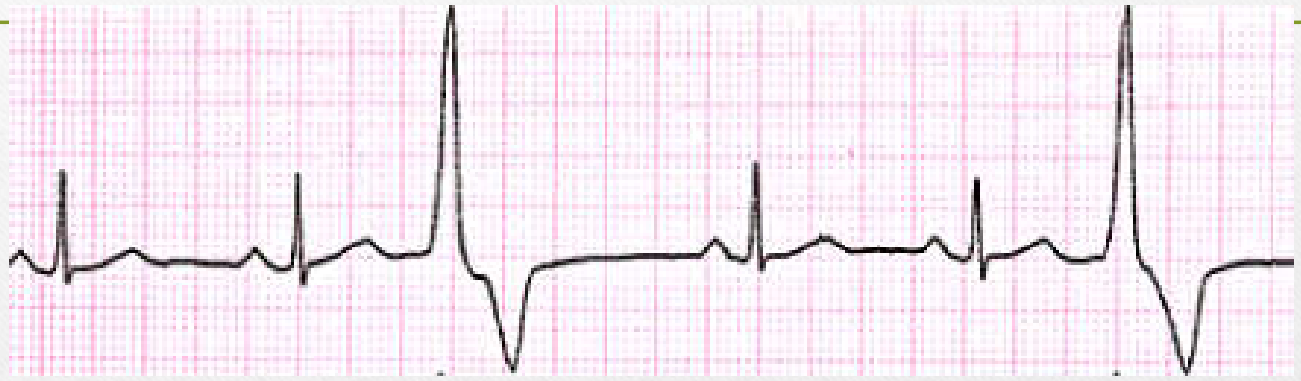


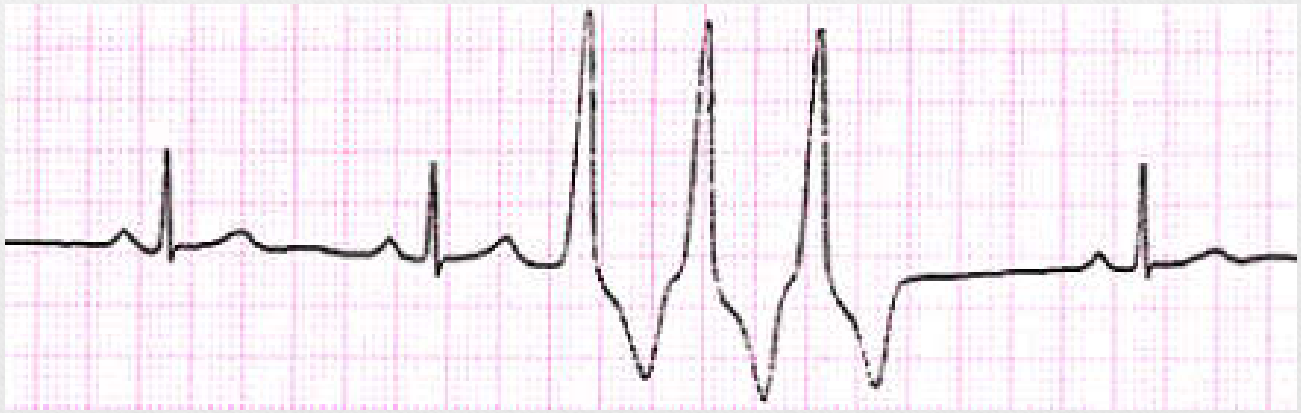
Ventricular Arrhythmias

Premature ventricular contactions (PVCs)

- A condition in which an electrical signal originates in the ventricles and causes the ventricles to contract before receiving the electrical signal from the atria.
- ECG shows wide and bizarre QRS complex
- Early 'R-on-T' ventricular premature beats may induce ventricular fibrillation
- PVCs are not uncommon and often do not cause symptoms or problems.
- Treated only if symptomatic with beta-blockers.

Premature ventricular contractions (PVCs)





Bradycardias

Sinus Bradycardia

- Physiological variant due to strong vagal tone or atheletic training.
- Rate as low as 50 at rest and 40 during sleep.
- Common causes of sinus bradycardia include:
- Extrinsic causes ;Hypothermia, hypothyroidism, cholestatic jaundice and raised intracranial pressure. Drug therapy with beta-blockers, digitalis and other antiarrhythmic drugs.
- Intrinsic causes; Acute ischaemia and infarction of the sinus node (as a complication of acute myocardial infarction). Chronic degenerative changes such as fibrosis of the atrium and sinus node (sick sinus syndrome).

SINUS BRADYCARDIA

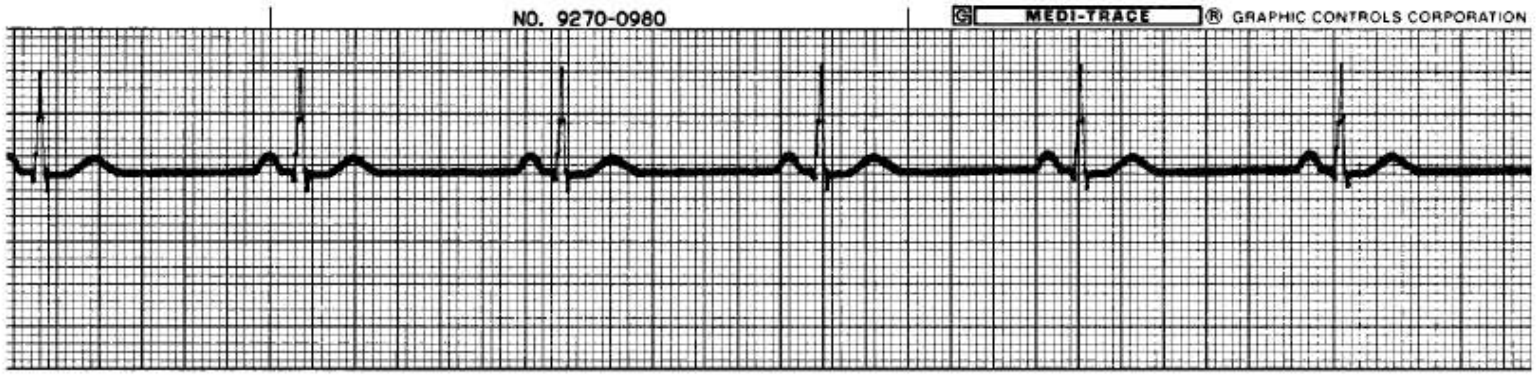


FIGURE 27-6 Sinus bradycardia in lead II.

Bradycardias

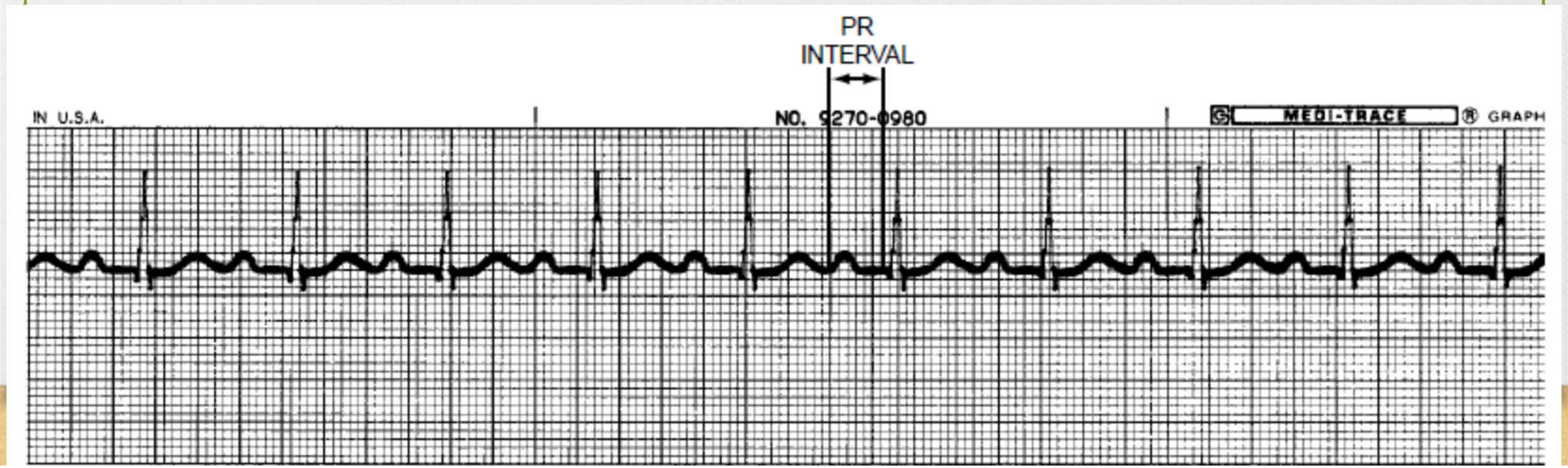
Sick sinus syndrome

- A condition in which the sinus node sends out electrical signals either too slowly or too fast. There may be alternation between too-fast and too-slow rates.
- This condition may cause symptoms if the rate becomes too slow or too fast for the body to tolerate.
- Chronic symptomatic sick sinus syndrome requires permanent pacing (AAI), with additional antiarrhythmic drugs (or ablation therapy) to manage any tachycardia element.
- Thromboembolism is common in tachy-brady syndrome and patients should be anticoagulated unless there is a contraindication.

Atrioventricular (AV) Block

First degree A-V Block

- Seldom of clinical significance, and unlikely to progress.
- ECG shows prolonged PR interval.
- May be associated with acute rheumatic fever, diphtheria, myocardial infarction or drugs as digoxin



Atrioventricular (AV) Block

Second degree A-V Block

Mobitz type I (Wenchebach phenomenon):

- Gradually increasing P-R intervals culminating in an omission.
- When isolated, usually physiological and due to increased vagal tone and abolished by exercise and atropine.

Second degree A-V Block

Mobitz type II

- The P wave is sporadically not conducted. Occurs when a dropped QRS complex is not preceded by progressive PR interval prolongation.
- Pacing is usually indicated in Mobitz II block, whereas patients with Wenckebach AV block are usually monitored.

Second Degree AV Block

Acute myocardial infarction may produce second-degree heart block. In inferior myocardial infarction, close monitoring and transcutaneous temporary back-up pacing are all that is required.

In anterior myocardial infarction, second-degree heart block is associated with a high risk of progression to complete heart block, and temporary pacing followed by permanent pacemaker implantation is usually indicated.

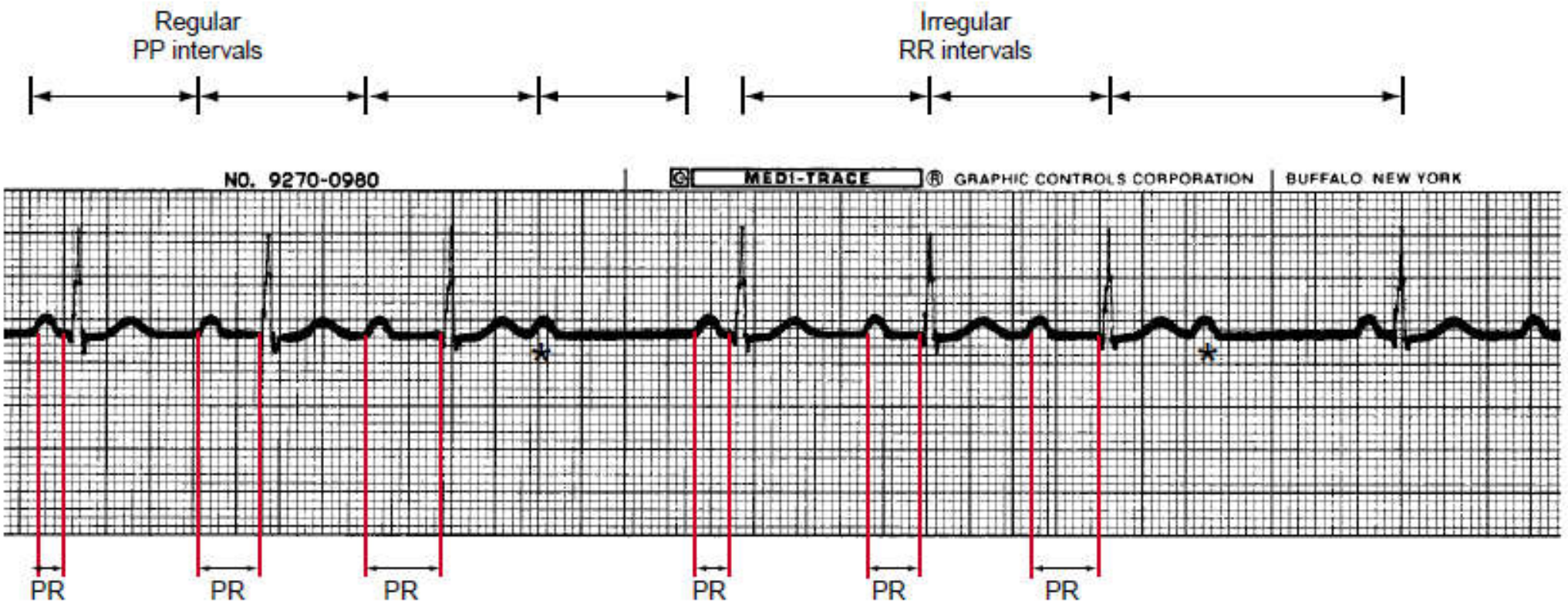
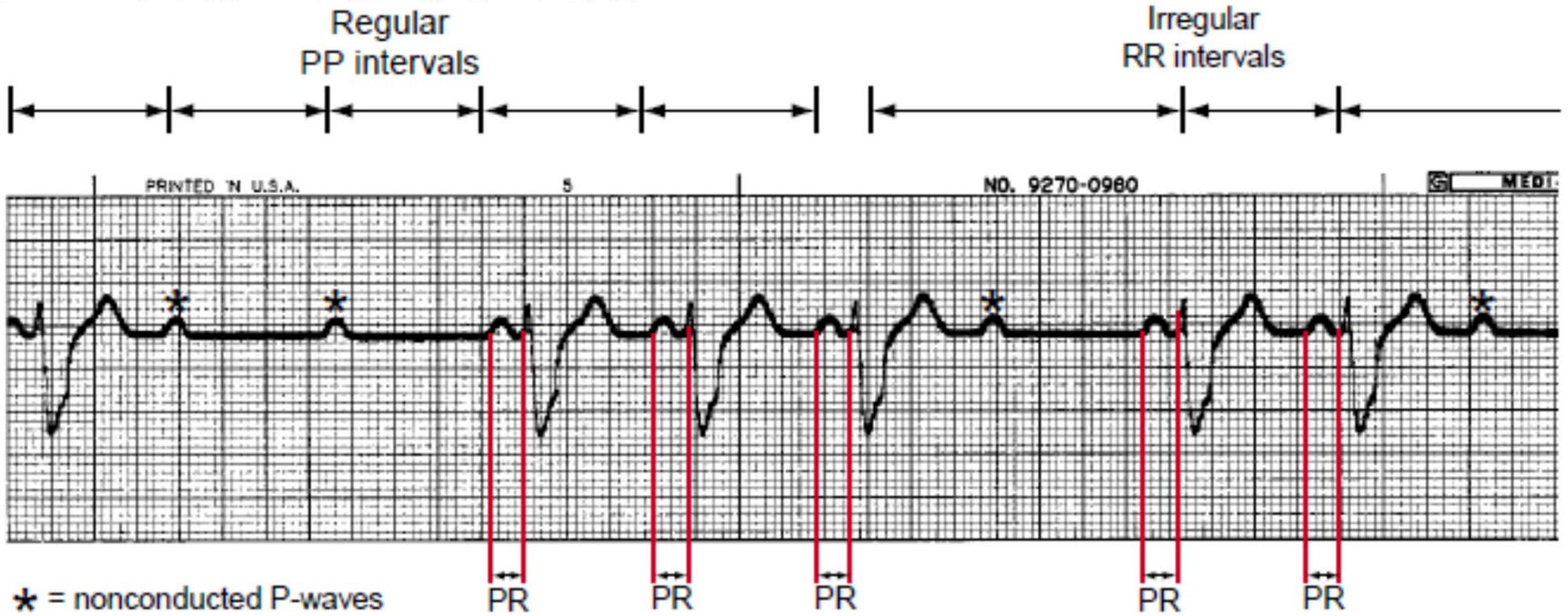


FIGURE 27-21 Sinus rhythm with second-degree AV block, type I in lead II. Note progressively longer PR durations until the



Atrioventricular (AV) Block

Third degree A-V Block

- Common in elderly age groups due to idiopathic bundle branch fibrosis.
- Other causes include coronary heart disease, calcification from aortic valve, sarcoidosis or congenital.
- ECG shows bradycardia, P wave continue, unrelated to regular slow idioventricular rhythm.
- Treatment is permanent pacing.

Third Degree A-V block

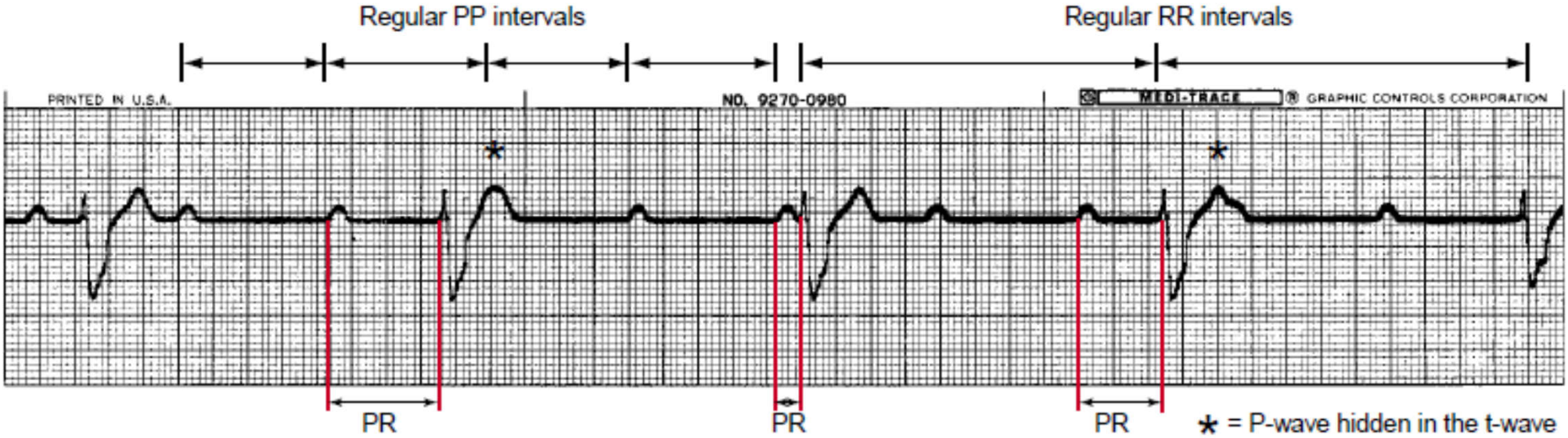


FIGURE 27-23 Sinus rhythm with third-degree AV block and idioventricular rhythm in lead V_1; note irregular PR intervals.

Atrioventricular (AV) Block

Bundle Branch Block (BBB):

- Interruption of the right or left branch of the bundle of Hiss delays activation of the corresponding ventricle leading to broadening of the QRS complex
- Unlike right BBB, left BBB is always associated with an underlying heart disease.
- Both RT and LT BBB show wide deformed QRS complex. In RBBB there is rSR pattern in lead V1, while in LBBB there is a broad monophasic (or notched) R wave in leads V5 and V6.

Atrioventricular (AV) Block

Bundle Branch Block (BBB):

Hemiblock

Delay or block in the divisions of the left bundle branch produces a swing in the direction of depolarization (electrical axis) of the heart. When the anterior division is blocked (left anterior hemiblock), there is left axis deviation. Delay or block in the postero-inferior division causes(right axis deviation).

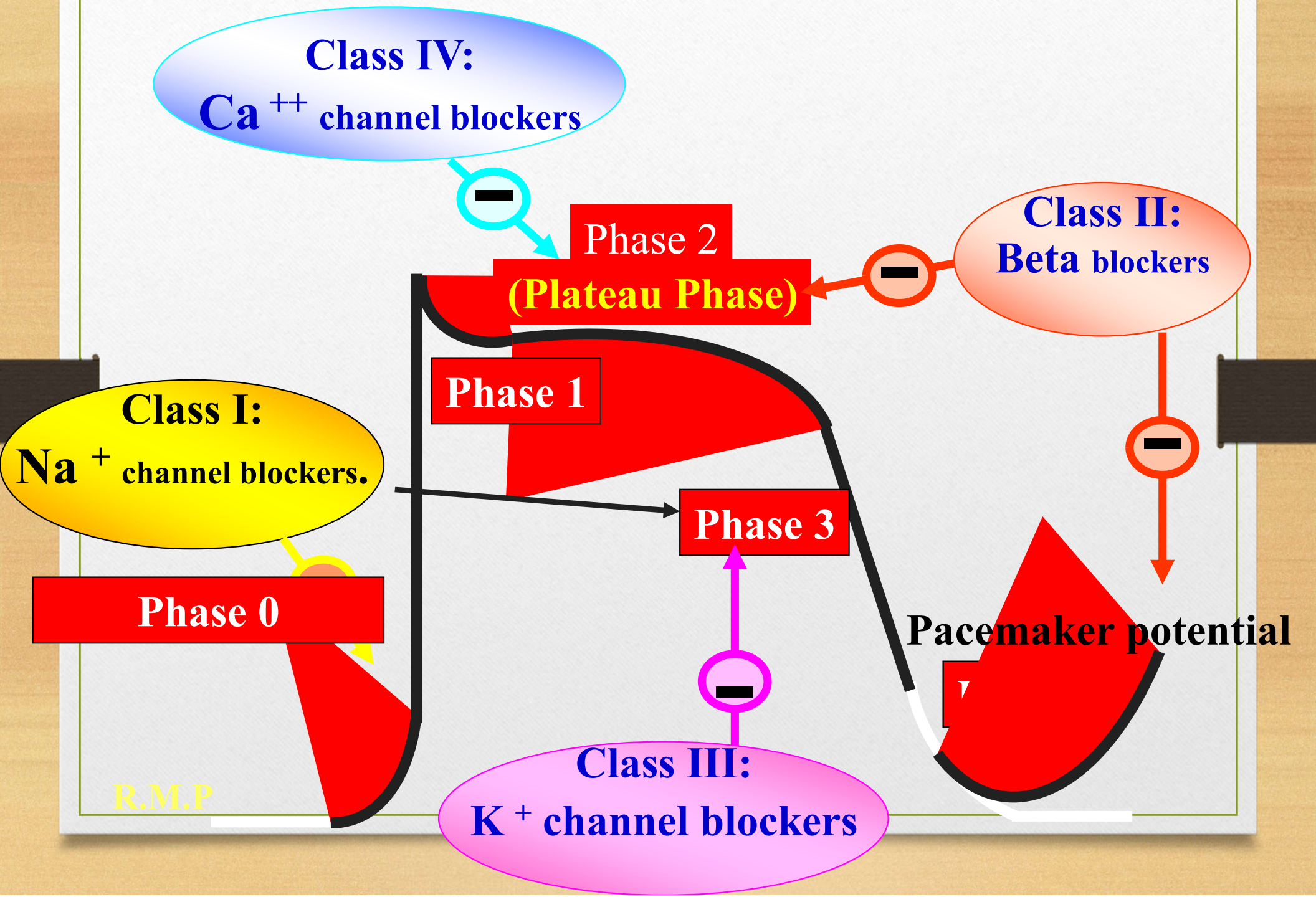
Bifascicular block

This is a combination of a block of any two of the following: the right bundle branch, the left antero-superior division and the left postero-inferior division. Block of the remaining fascicle will result in complete AV block.

MANAGEMENT OF ARRHYTHMIAS

- Pharmacological therapy.
- Cardioversion.
- Pacemaker therapy.
- Surgical therapy e.g. aneurysmal excision.
- Interventional therapy “ablation”.

Classification of Anti-Arrhythmic Drugs



Classification of Antiarrhythmic Drugs based on Drug Action

CLASS	ACTION	DRUGS
I.	Sodium Channel Blockers	
1A.	Moderate phase 0 depression and slowed conduction (2+); prolong repolarization	Quinidine, Procainamide, Disopyramide
1B.	Minimal phase 0 depression and slow conduction (0-1+); shorten repolarization	Lidocaine
1C.	Marked phase 0 depression and slow conduction (4+); little effect on repolarization	Flecainide
II.	Beta-Adrenergic Blockers	Propranolol, esmolol
III.	K^+ Channel Blockers (prolong repolarization)	Amiodarone, Sotalol, Ibutilide
IV.	Calcium Channel Blockade	Verapamil, Diltiazem


THANK
YOU