Arrhythmias

Normal ECG :

- A normal heartbeat originates in the upper chambers of the heart (atria) – sinusal rrhythm.
- The heart rate (usually between 60 and 100 beats per minute) and rhythm appear regular.
- P waves, QRS complexes, T waves appear normal.
- ST segments are not elevated above or depressed below the baseline of the EKG tracing.

Abnormalities in ECG components

Right Atrial Hypertrophy

- P waves peaked > 2,5 mm in leads II, III, and aVF.

- It is called “P pulmonale”, because it is often met in cor pulmonare.

Left Atrial Hypertrophy

- a notched P wave with a duration > 0,1 seconds (usually 0.12 second).
- It is called P mitrale; it may be two-humpbached (it has two humps) ;
- is best observed in leads II and V1.

Right Ventricular Hypertrophy (RVH)

- Tall R wave in V1 (R wave taller than S wave)
- R wave in V1-V2 > 7 mm
- Right Axis Deviation and deep S wave in left precordial leads.
- Signs of RBBB

Occurs with pulmonary valve disease, primary or secondary pulmonary hypertension or various congenital lesions resulting in either volume or pressure overload of the right ventricle.

Left Ventricular Hypertrophy

- R wave in I and S wave in III > 15 mm, or
- R wave in V_5 or V_6 + S wave in V_1 or V_2 > 35 mm ($RV_{5-6} + SV_{1-2}$ > 35 mm)
- R wave in V_5 or V_6 > 25 mm
- R wave in AVL > 11 mm
- Left Axis Deviation and deep S wave in left precordial leads.
- Also see ST and T wave shift opposite to the R wave (in absence of digitalis effect) and LAE and LAD.

Occurs with systemic hypertension, aortic valve disease , conditions resulting in pressure or volume overload of the left ventricle.

What is an **Arrythmia** anyways?

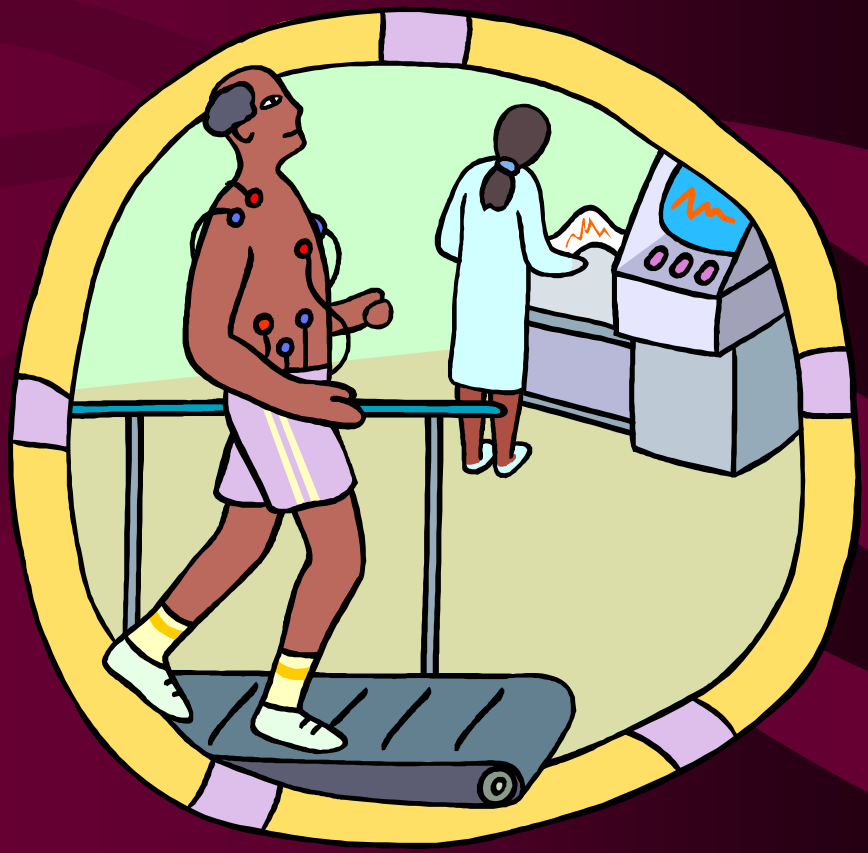
Definition:

- A disorder of impulse formation (automatism).
- An abnormal electrical conduction that changes the heart rate and rhythm (conductiblity).
- A disturbance in the heart's rhythm.

1) Some are mild, asymptomatic – require no treatment
2) Some are catastrophic – require immediate emergency response
3) They can influence cardiac output and blood pressure

“Clinical Significance”

- Thousands of people suffer with arrythmias
- About 15% of strokes occur in patients with atrial arrythmias
- A large majority of sudden cardiac deaths are thought to be caused by ventricular dysrhythmias.



What is The Big Deal?

Why are we so concerned with Arrythmias?

Stroke Volume x Heart Rate = CO

- **SV** dependent on filling time, adequate volume, and myocardial muscle function
- **HR** dependent on electrical stimulus, Autonomic NS, Parasympathetic NS

  - Too Fast
  - Too Slow
  - Too Irregular

NOT GOOD!!!

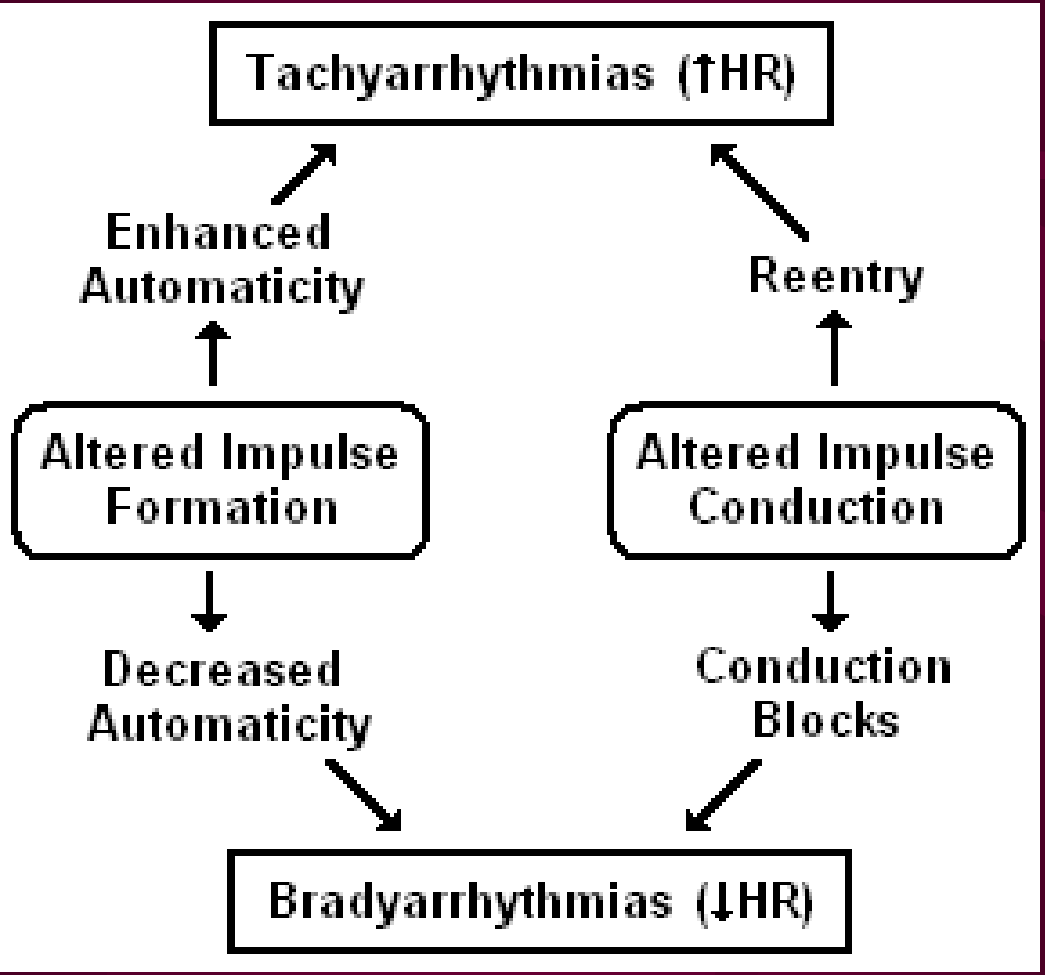
- The oscillations of the cardiac rhythm are dampened but do not entirely disappear with age.

- Exercise and emotion are potent accelerators of sinus rhythm through sympathetic neural and catecholamine drive.

- Normal persons have a marked diurnal variation in heart rate, with lowest rates just before early morning awakening, when sinus acceleration is substantial.

- Absolute regularity of sinus rhythm is pathologic and occurs with autonomic denervation (eg, in advanced diabetes).

Mechanisms of Arrhythmogenesis



- Arrhythmias may cause hemodynamic upset and they may be life threatening.

- Resulting dizziness and syncope are common.

- These arrhythmias require urgent attention and, often, hospitalization.

Diagnosis

- The history of the disease usually provides sufficient information for establishing a working diagnosis.

Complains :

- Fast, irregular palpitations in tachyarrhythmias .

- Syncope or presyncope in AV blocks, SSS (sick sinus sindrome).

Arrhythmias may be:

- Acute, chronic
- Permanent , persistent, paroxysmal

- The history should differentiate brief arrhythmic episodes (eg, ectopic beats) from sustained events.
- The onset and offset characteristics and any other symptoms should be obtained.

- If examined during an arrhythmia, the peripheral pulse and the jugular venous pulse are important for diagnosis and can positively identify VT from other tachycardias (AF, atrial flutter etc.)

*peripheral pulse - reflecting ventricular activation)

*JVP—reflecting atrial and ventricular activation

ECG

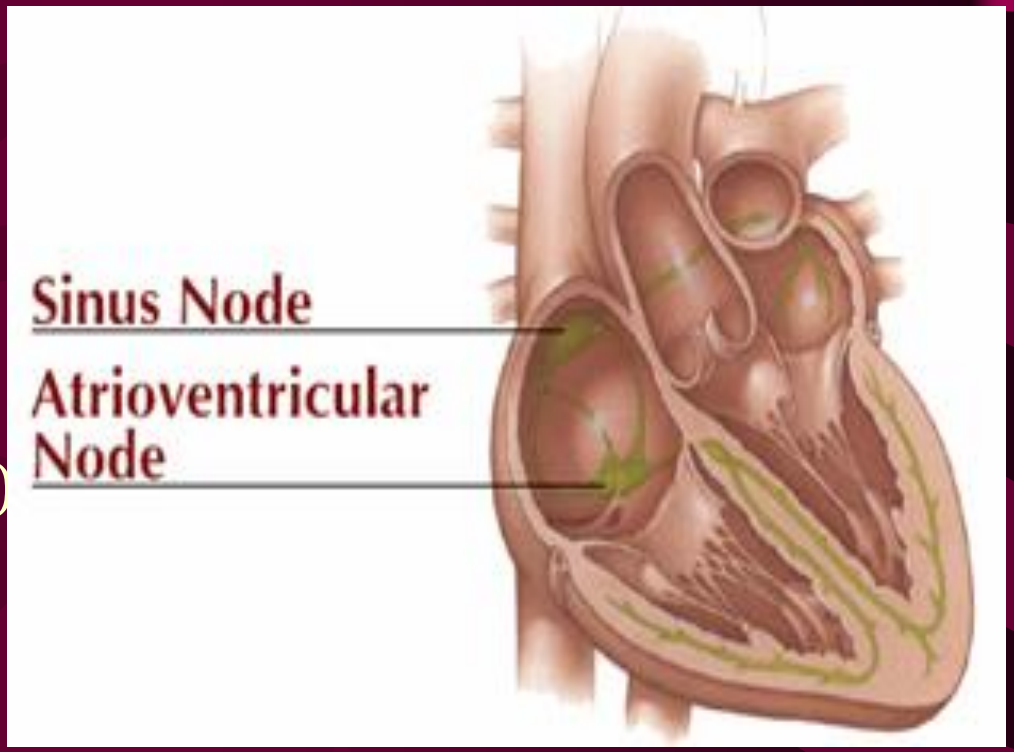
- ECG remains the major diagnostic procedure.
- The surface ECG represents the electrical forces of myocardial depolarization.
- The standard 12-lead ECG is crucial for the characterization and diagnosis of the various tachycardias.

Electrical Conduction Pathway

SA Node – “pacemaker” of the heart (60-100bpm)

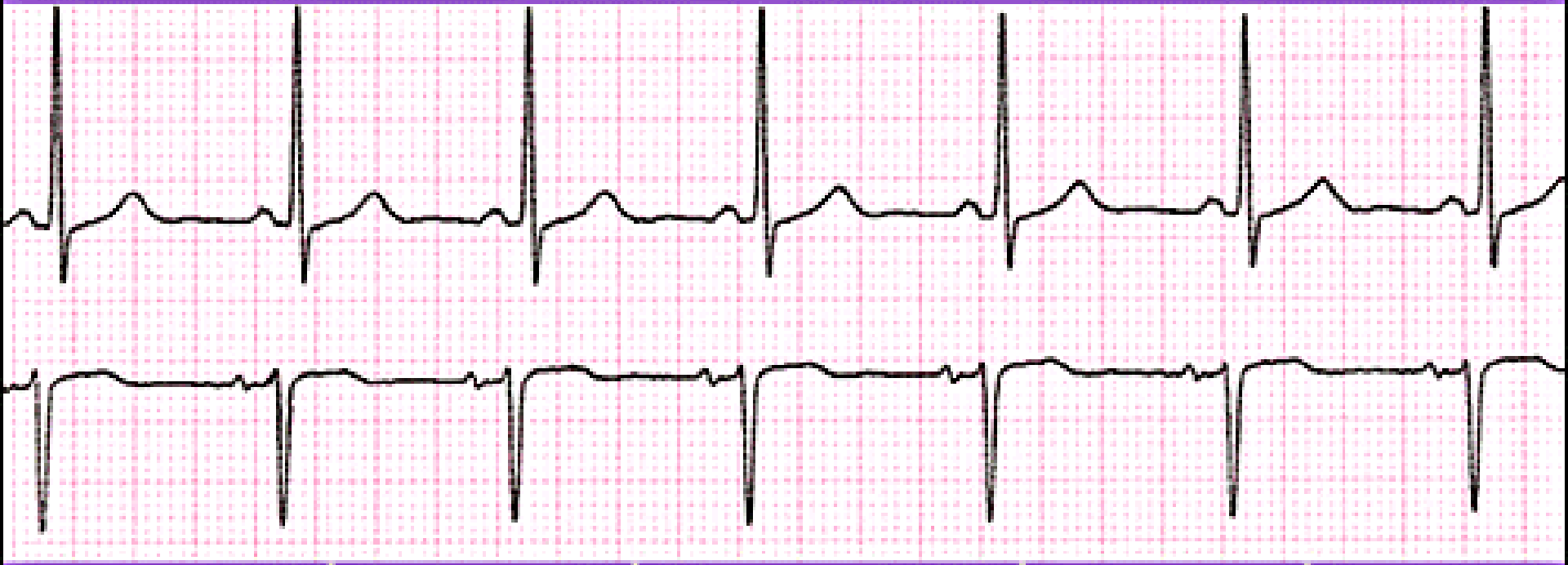
AV Node – junction of the atria and ventricles (40-60bpm)

Bundles – Bundle of His connects the AV node to the bundle branches (20-40bpm)



“Practice Strip”

Normal Sinus Rhythm

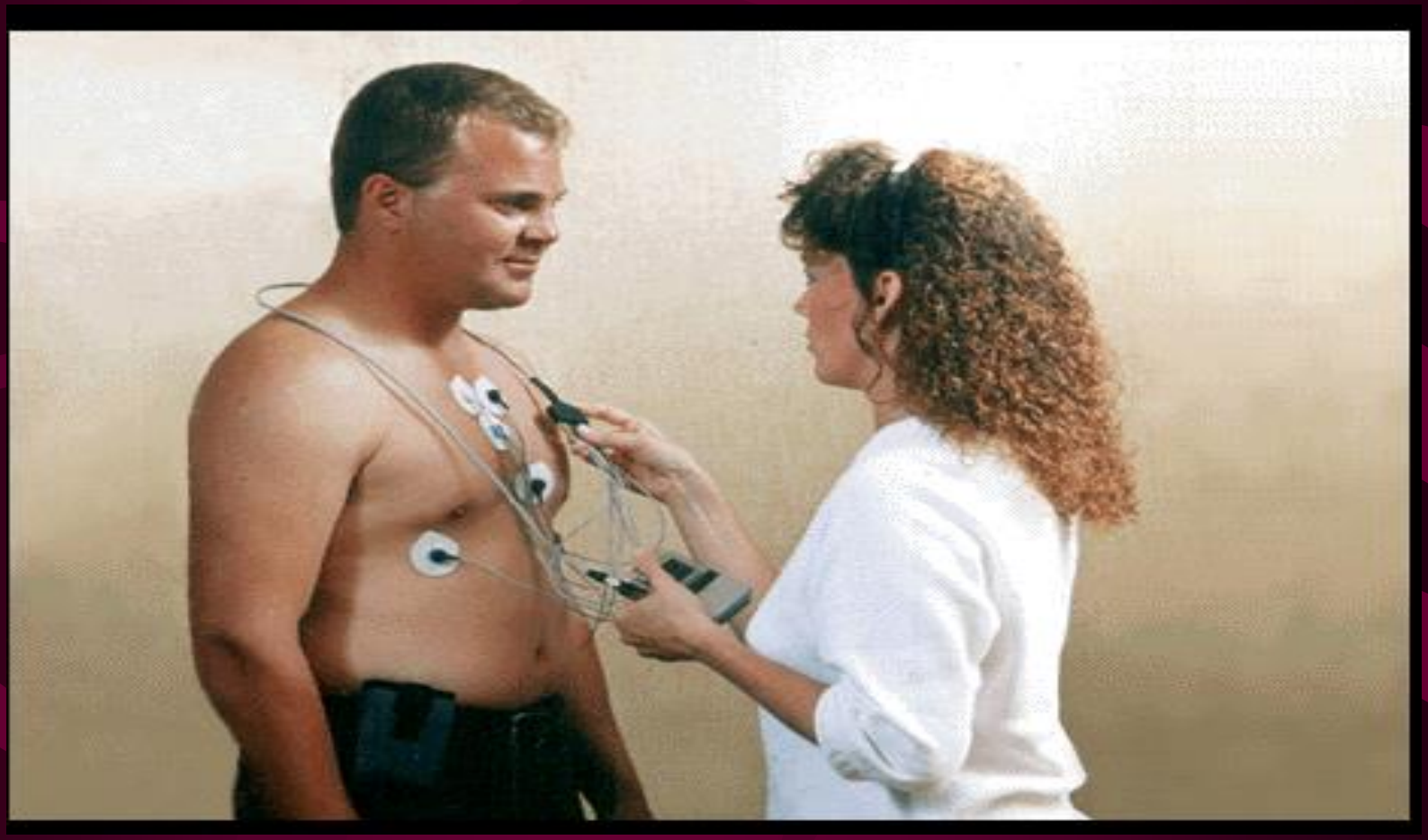


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
60-100 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

ECG monitoring

- Ambulatory ECG monitoring is the most powerful method of capturing arrhythmic events,
- its value is enhanced by keeping a diary of associated symptoms.

A continuous 24 h (Holter 24 h) is helpful for automatic detection of an arrhythmic episode.

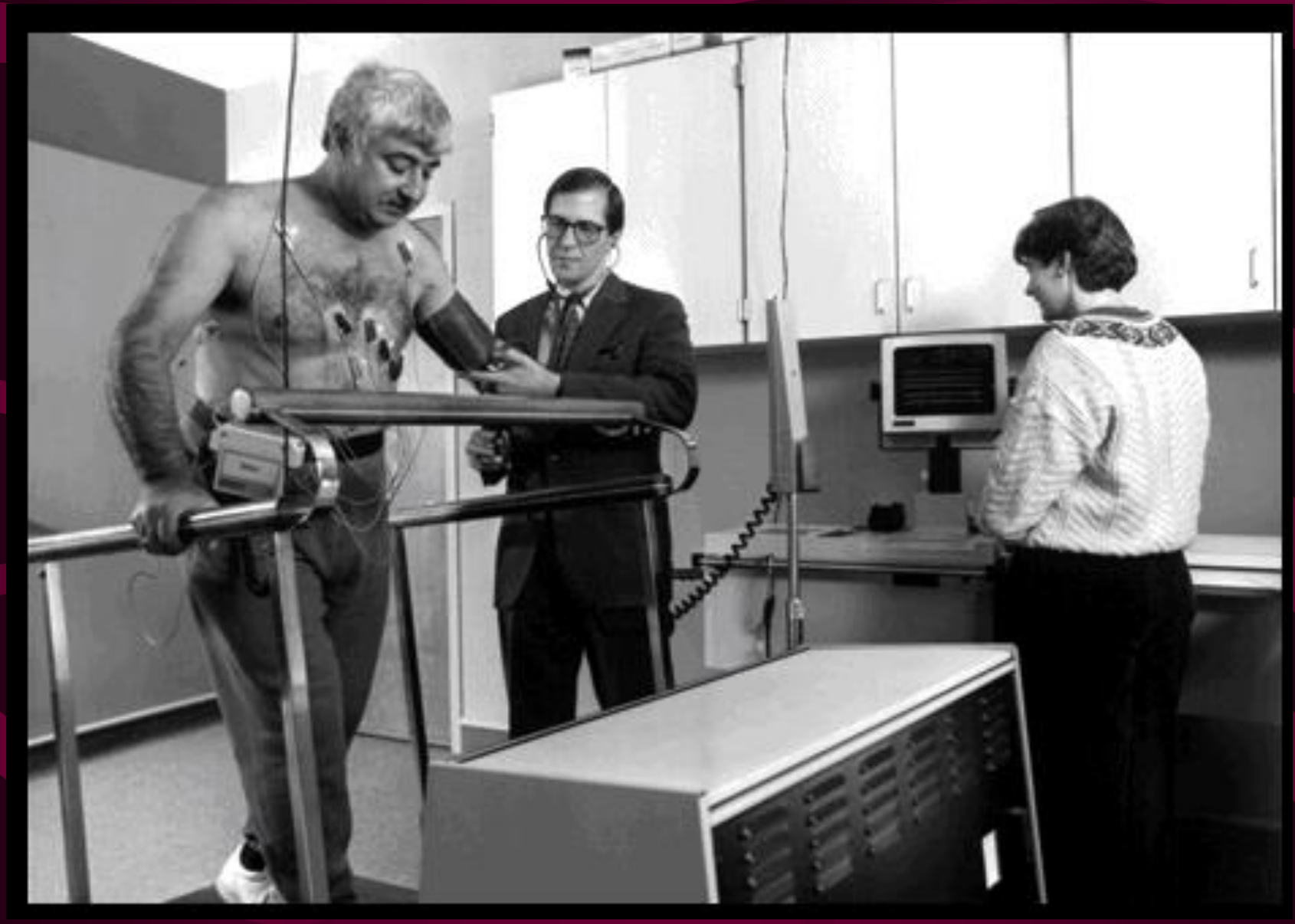


- Ambulatory ECG monitoring is less useful when arrhythmias are infrequent.

- Patients with suspected life-threatening rhythm disturbances should be hospitalized for monitoring to avoid a fatal out-of-hospital event.

Invasive electrophysiological studies

- are indicated when spontaneous arrhythmias are infrequent and when a serious sustained arrhythmia is suspected.



Classification of Arrhythmias

I. Sinus Node Dysfunction (Sick-sinus syndrome-SSS)

- Sinus bradycardia
- Sinus arrhythmia
- Sino-atrial blocks
- Brady-tachy syndrome

II. Extrasystolia (Premature ectopic beats):

- Atrial extrasystolia
- Atrio-ventricle extrasystolia
- Ventricular extrasystolia

III. Tachyarrhythmia

1. Supraventricular Tachyarrhythmia
   - Sinusal tachycardia
   - Atrial paroxysmal tachycardia
   - Atrio-ventricle paroxysmal tachycardia
   - Atrial Fibrillation and Flutter
2. Ventricular Tachyarrhythmia
   - Ventricular paroxysmal tachycardia
   - Ventricular Fibrillation and Flutter

IV. Conductibility disorders

- Sino –atrial block
- Atrioventricular block
- Intraventricular block (bundle brunch blocks)
- Ventricular preexcitation – Wolff-Parkinson-White Syndrome (WPW)

V. Ectopic rhythms

1. Atrial rhythm
2. AV rhythm
3. Idioventricular rhythm

SINO-ATRIAL NODE DYSFUNCTION -SSS

- The SA node is normally the dominant cardiac pacemaker.
- Its responsiveness to alterations in autonomic nervous system tone is responsible for the normal acceleration of heart rate during exercise and the slowing that occurs during rest and sleep.

- **Increases in sinus rate** normally result from an increase in sympathetic tone acting via b-adrenergic receptors and/or a decrease in parasympathetic tone acting via muscarinic receptors.

- **Slowing of the heart rate** is due to increase in sympathetic tone or a decrease in parasympathetic tone.
- In adults, the normal sinus rate is 50-60 to 90-100 beats per minute.

SA node dysfunction ETIOLOGY

- SA node dysfunction is most often found in the elderly as an isolated phenomenon.

Specific diseases associated with SA node dysfunction:

- senile amyloidosis
- interruption of the blood supply to the SA node
- idiopathic degeneration
- secondary to pharmacologic agents - cardiac glycosides, b-adrenergic blocking drugs, calcium channel blockers, amiodarone...

SSS. Clinical picture

refers to a combination of symptoms :

- dizziness, may be paroxysmal
- confusion, fatigue,
- presyncope or syncope,
- congestive heart failure

caused by SA node dysfunction and manifested by marked sinus bradycardia, sinoatrial block, or sinus arrest.

These symptoms usually result from :

- abrupt, prolonged sinus pauses caused by failure of sinus impulse formation , may be >3 sec
- block of conduction of sinus impulses to the surrounding atrial tissue (sinus exit block)
- due to inadequate cardiac output (bradycardia).

Occasionally,

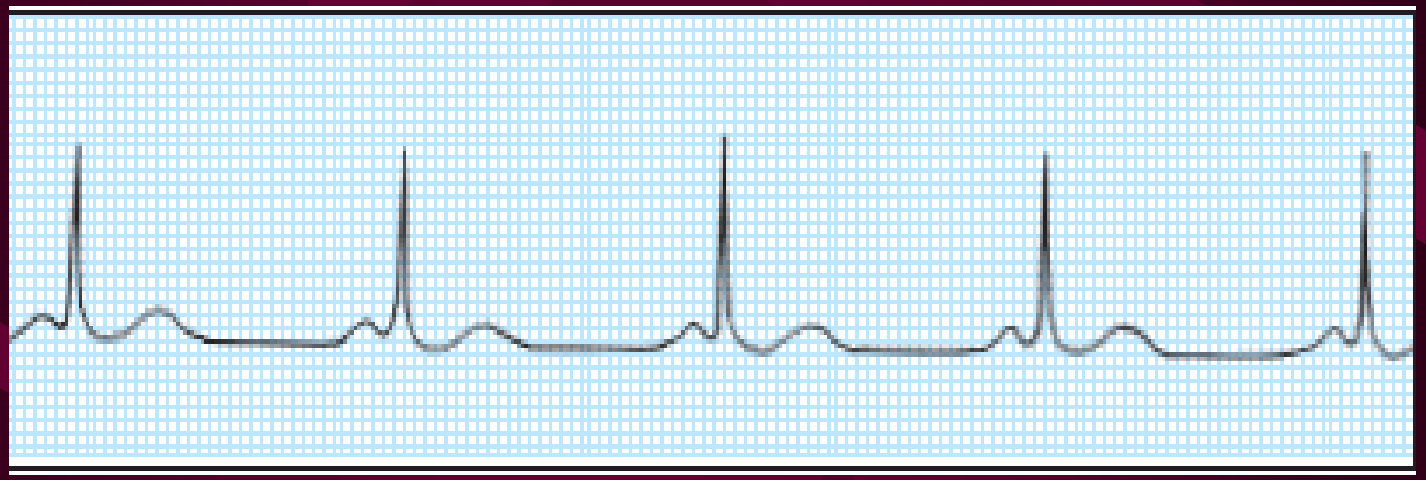
- SA node dysfunction is manifested by an inadequate acceleration in sinus rate in response to a stress such as exercise or fever.
- Because these symptoms are nonspecific, and because ECG manifestations of sinus node dysfunction are often intermittent, it may be difficult to prove that such symptoms are actually caused by SA node dysfunction.

SSS types

- Sinus bradycardia
- Sinus arrhythmia
- Sino-atrial blocks
- Brady-tachy syndrome

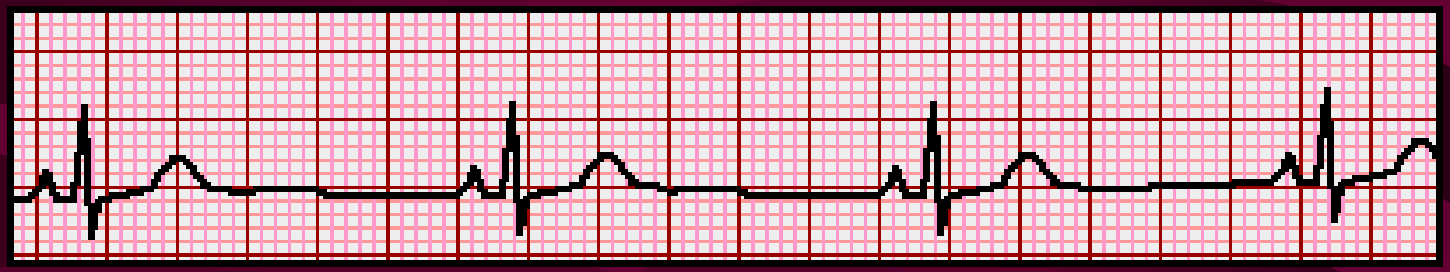
Sinus bradycardia

- Sinus bradycardia is said to exist when the sinus rate is less than 50-60 beats per minute.



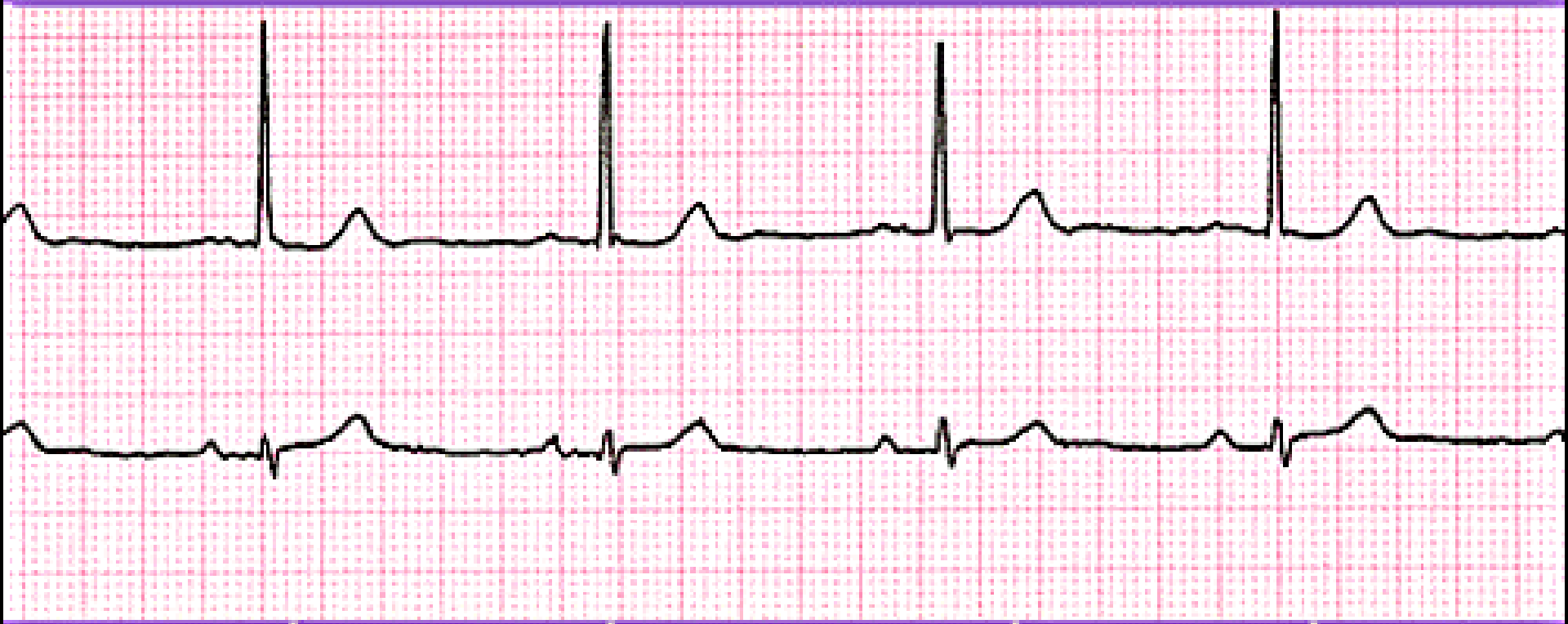
Decreased Automaticity

Sinus Bradycardia



- ECG signs:
- RR intervals equal, but increased

Sinus Bradycardia



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
<60 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

- There is wide variation among individuals, and rates less than 60 beats per minute do not necessarily indicate pathologic states.

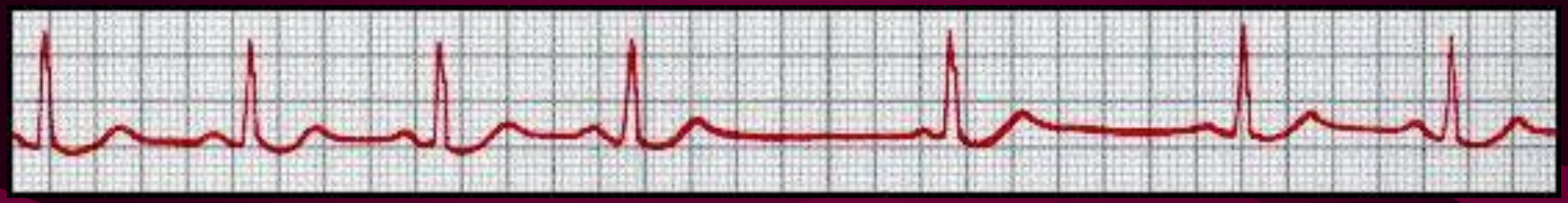
For example, trained athletes often exhibit resting rates under 50 beats per minute due to increases in vagal tone.

Normal elderly individuals may also show marked sinus bradycardia at rest.

Sinus bradycardia is associated with

- hypothyroidism,
- advanced liver disease,
- hypothermia,
- typhoid fever,
- brucellosis;
- episodes of hypervagotonia
- acute hypertension.

Sinus Arrhythmia



Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 7th ed., 2005.

EKG Characteristics: Presence of sinus P waves

Variation of the PP interval which cannot be attributed to either SA nodal block or Premature Atrial Contractions (differences between RR distances are more than 0,1 sec)

When the variations in PP interval occur in phase with respiration, this is considered to be a normal variant. When they are unrelated to respiration, they may be caused by the same etiologies leading to sinus bradycardia.

Tachycardia-Bradycardia Syndrome

Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 7th ed., 2005.

Abrupt termination of atrial flutter with variable AV block, followed by sinus arrest with a junctional escape beat.

The bradycardia-tachycardia syndrome

- refers to atrial tachyarrhythmia that is followed by prolonged sinus pauses or in which there are alternating periods of tachyarrhythmia and bradyarrhythmia.

- Syncope or presyncope may be clinical manifestations of pauses.

Conductibility disorders

- SA blocks
- AV blocks
- Ventricular blocks

Sino – atrial blocks

- First degree I (slowing of the impulse transmitting from SA node to atria)

- Second degree II (gradually slowing of the impulse transmitting with periodically complete blocking of it)

- Third degree III (complete block)

Sino-atrial block, I degree

- denotes a prolonged conduction time from the SA node to the surrounding atrial tissue.
- It cannot be recognized on a standard (surface) ECG but requires invasive intracardiac recordings, which can detect this condition indirectly, by measuring the sinus response to atrial premature beats, or directly, by recording SA node electrograms.

Sino-atrial block, II degree

- denotes the intermittent failure of conduction of sinus impulses to the surrounding atrial tissue;

<u>On ECG</u>

- it is manifested as the intermittent absence of P QRS complexes.

Sino-atrial block, III degree (complete sinoatrial block)

- Usually is characterized by the presence of an ectopic subsidiary atrial or AV pacemaker.
- Only the direct intracardiac recordings of SA node activity allow to put the diagnosis.

On the standard ECG

- it is like sinus arrest – asistolia (long pause) or an ectopic rhythm appear to substitute the function of the SA node.

Diagnosis of SSS

- Ambulatory ECG (Holter) monitoring remains the standard in evaluating sinus node function.

- Single and even multiple 24-h Holter monitor recordings may fail to include a symptomatic episode.

Other noninvasive tests of SA node function

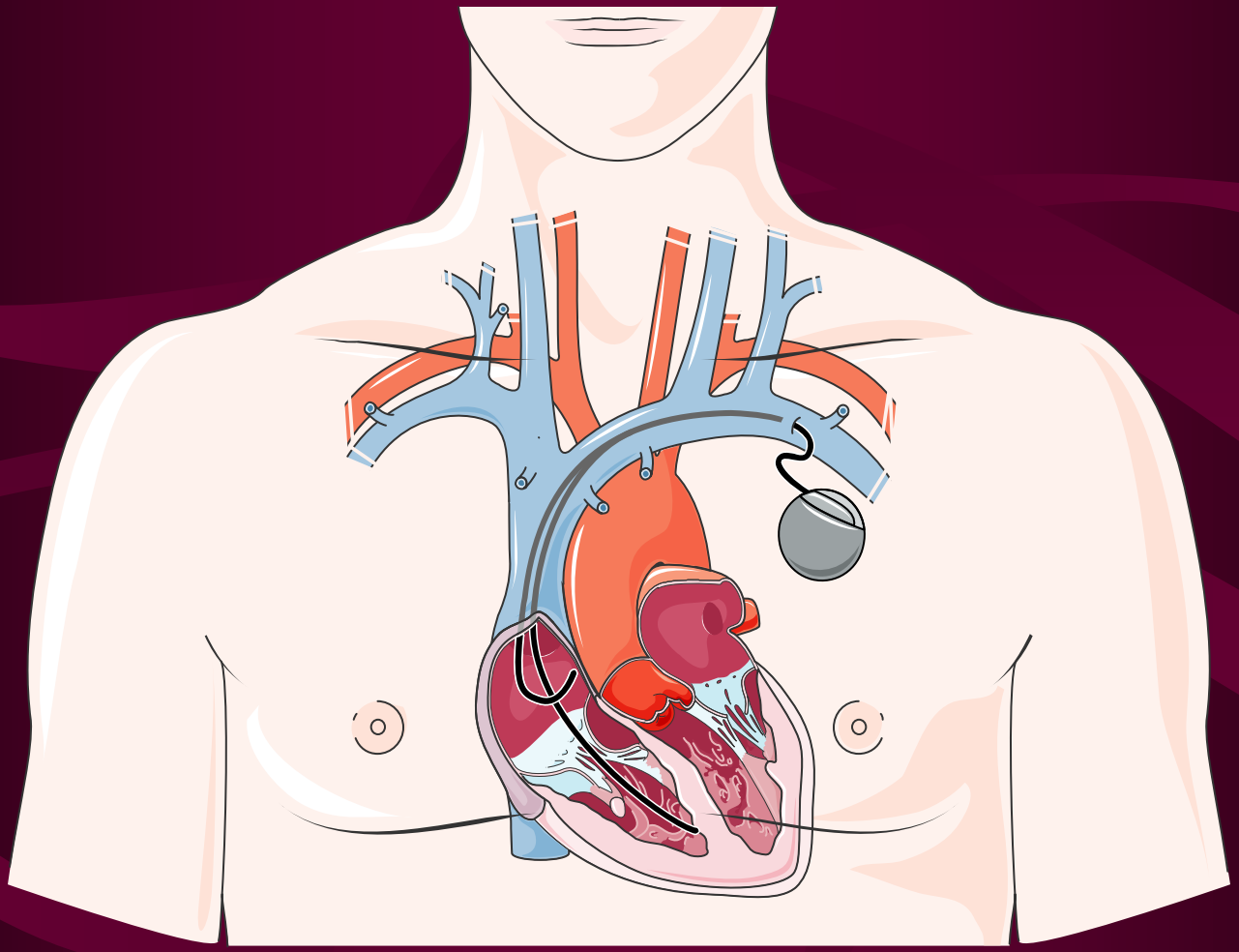
- the use of pharmacologic agents (to assess the balance of parasympathetic and sympathetic activity on the sinus node).

- Physiologic or pharmacologic maneuvers that are vagomimetic (Valsalva maneuver), vagolytic (atropine), sympathomimetic (isoproterenol or hypotension by nitroprusside), or sympatholytic (b-adrenergic blocking agents) can be utilized

TREATMENT

- Permanent pacemakers are the mainstay of therapy for patients with symptomatic SA node dysfunction.

Pacemaker



AV Conduction disturbances – AV blocks

- Are abnormalities of conduction of the sinus impulse from atria to the ventricles
- AV blocks may lead to the development of heart block, which can ultimately lead to cardiac arrest.

ETIOLOGY

- The AV node is supplied by the parasympathetic and sympathetic nervous systems and is sensitive to variations in autonomic tone.
- Chronic slowing of AV nodal conduction may be seen in highly trained athletes who have hypervagotonia at rest.

A variety of diseases and drugs can also influence AV nodal conduction:

- myocardial infarction, coronary spasm
- Hypertension, aortic or mitral stenosis
- digitalis intoxication, excesses of beta or calcium blockers,
- acute infections such as viral myocarditis, acute rheumatic fever, infectious mononucleosis,
- cardiac amyloidosis and neoplasms,
- AV may be congenital.

Classification of AV blocks

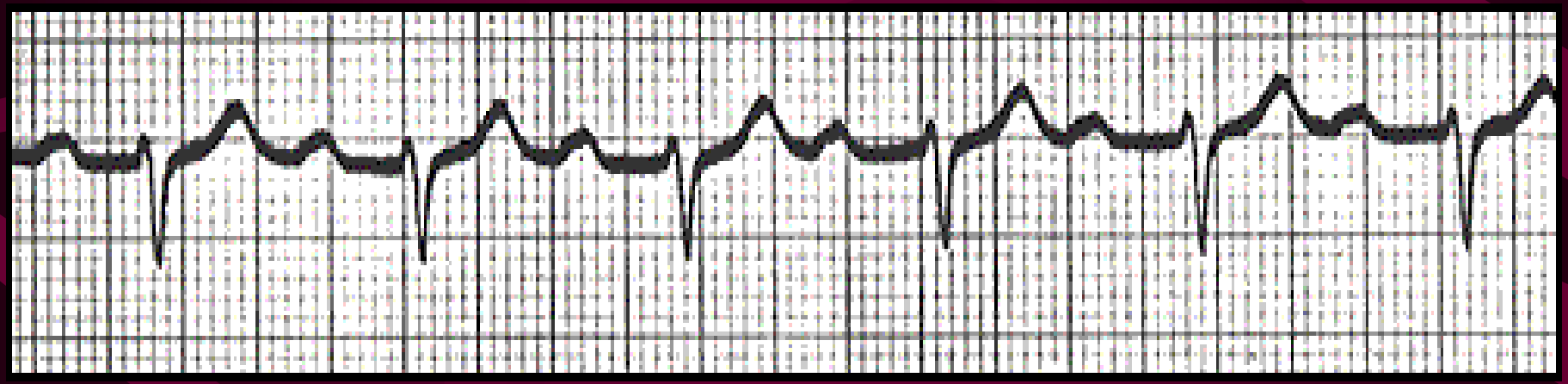
- AV block, I degree

- AV block, II degree
  - Mobitz 1
  - Mobitz 2

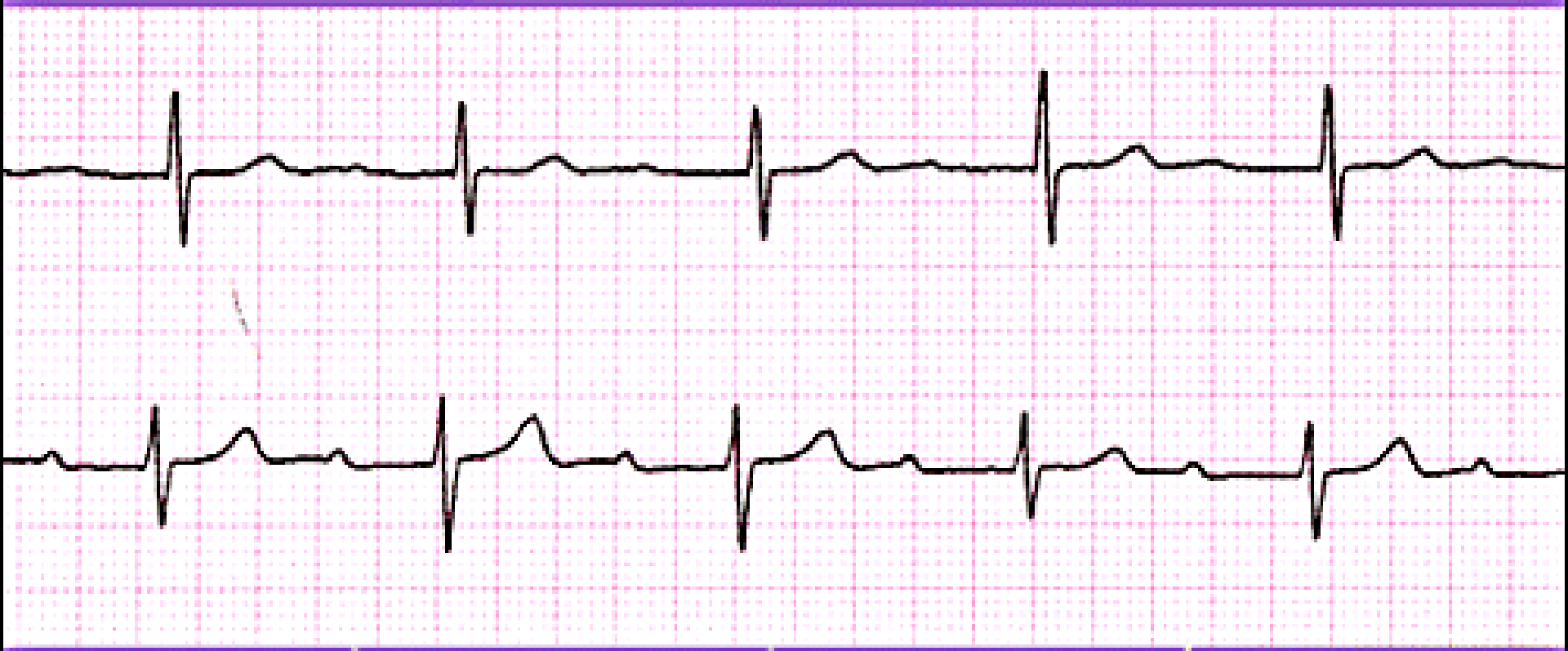
- AV block, III degree

1st degree AV block

- more prolonged AV conduction,
- Prolongation of the PR interval, which is constant > 0.20 sec.
- All P waves are conducted



First Degree AV Block



P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	>.20	>.12	Regular rhythm

- is asymptomatic .
- It is often seen in well-trained athletes, the young with high vagal tone.
- Even when first-degree heart block occurs in the setting of disease, treatment is not mandatory but may suggest further investigation.

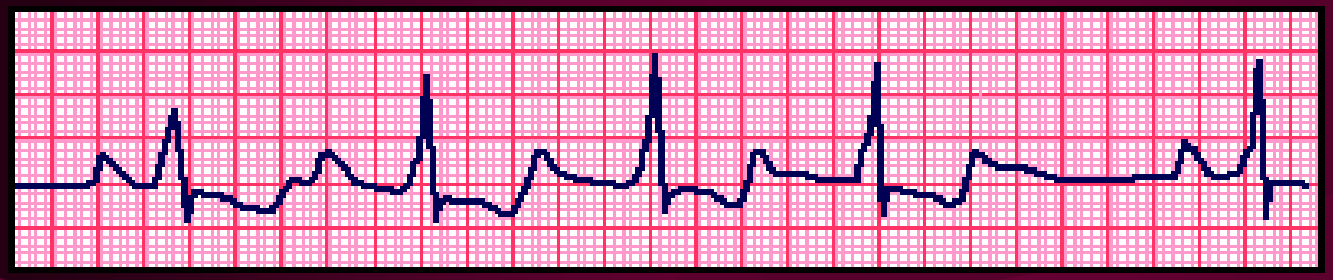
2nd degree AV block (intermittent AV block)

is present when only some atrial impulses fail to conduct to the ventricles.

Two types:

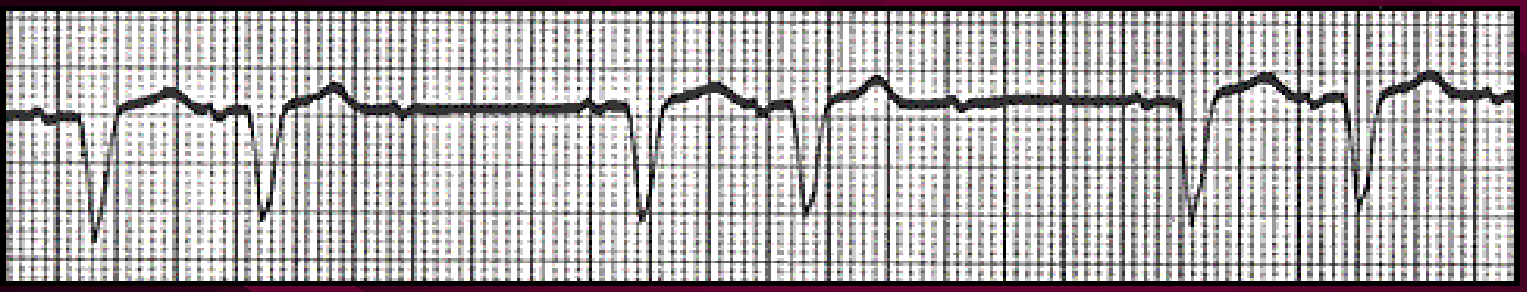
- Type 1 - Mobitz I
- Type 2 - Mobitz II

2nd Degree AV Block



Type 1
(Wenckebach)

EKG Characteristics: Progressive prolongation of the PR interval until a P wave is not conducted.



Type 2

EKG Characteristics: Constant PR interval with intermittent failure to conduct

So, ECG signs of 2nd Degree AV Block Mobitz I (Wenckebach)

- progressive PR interval prolongation until a block of an atrial impulse with a P wave not followed by a QRS complex, after that - repetition of the sequence.
- This sequence is called Wenckebach period

ECG signs of 2nd Degree AV Block Mobitz II

1. the PR interval is constant (normal or prolonged),
2. there is a periodical unexpected dropped QRS complex;

- The high-grade block is the Mobitz II in which there is a mathematical relationship between the P waves and QRS complexes, eg, 2:1, 3:1.
- Symptoms are rare.

3rd degree AV block (complete heart block):

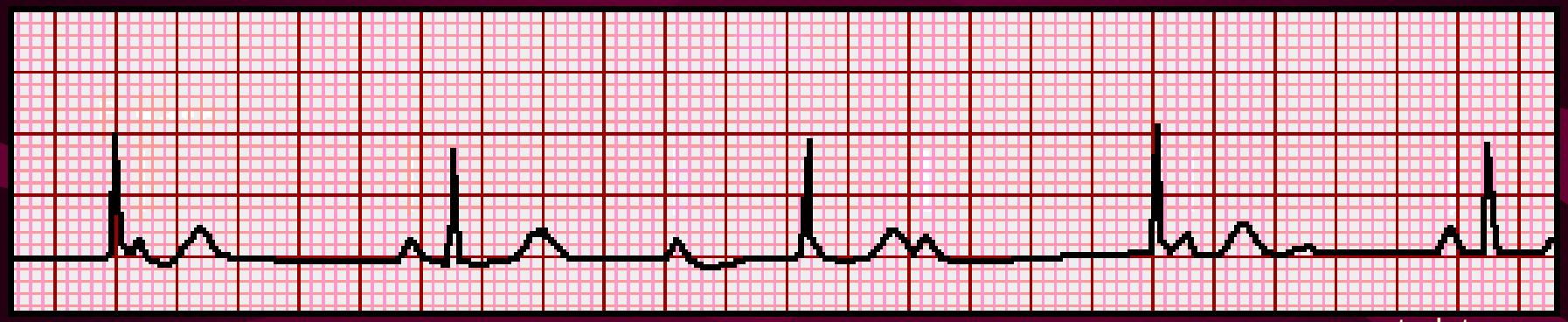
- The impulse is not transmitted at all from atria to the ventricles – complete transversal block.
- Atria are contracting in their own rhythm and the ventricles are working in their own rhythm – no relationship between their activity.
- Ventricular contracting (heart activity and life) is maintained by an escape junctional pacemaker (from Atrioventricular node) or ventricular pacemaker

3rd degree AV block. ECG:

- P waves are seen on the ECG but bear no relationship to the QRS complexes - there is no electrical communication between the atria and the ventricles.

- P waves appear with a frequency of 60-80 per min; QRS complexes – with a frequency of 20-40 per minute.

3rd Degree (Complete) AV Block

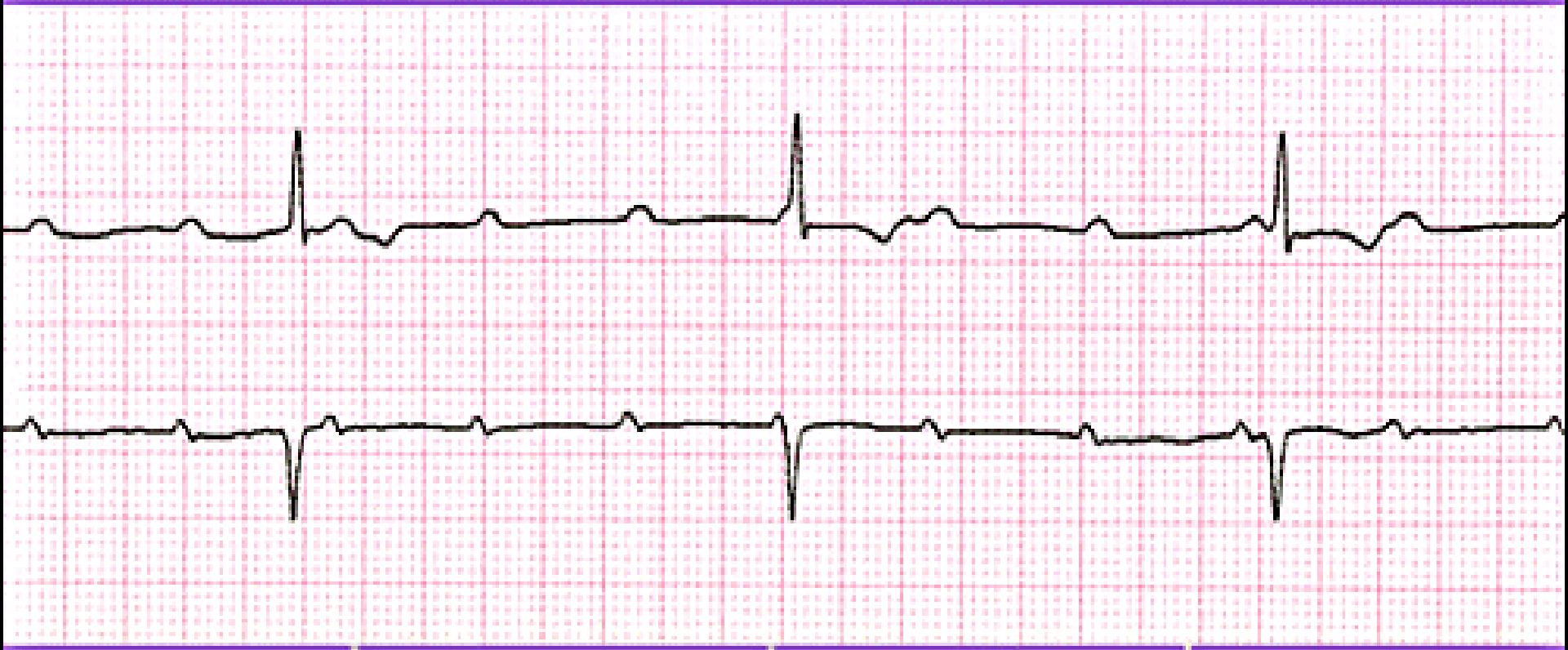


www.uptodate.com

EKG Characteristics:

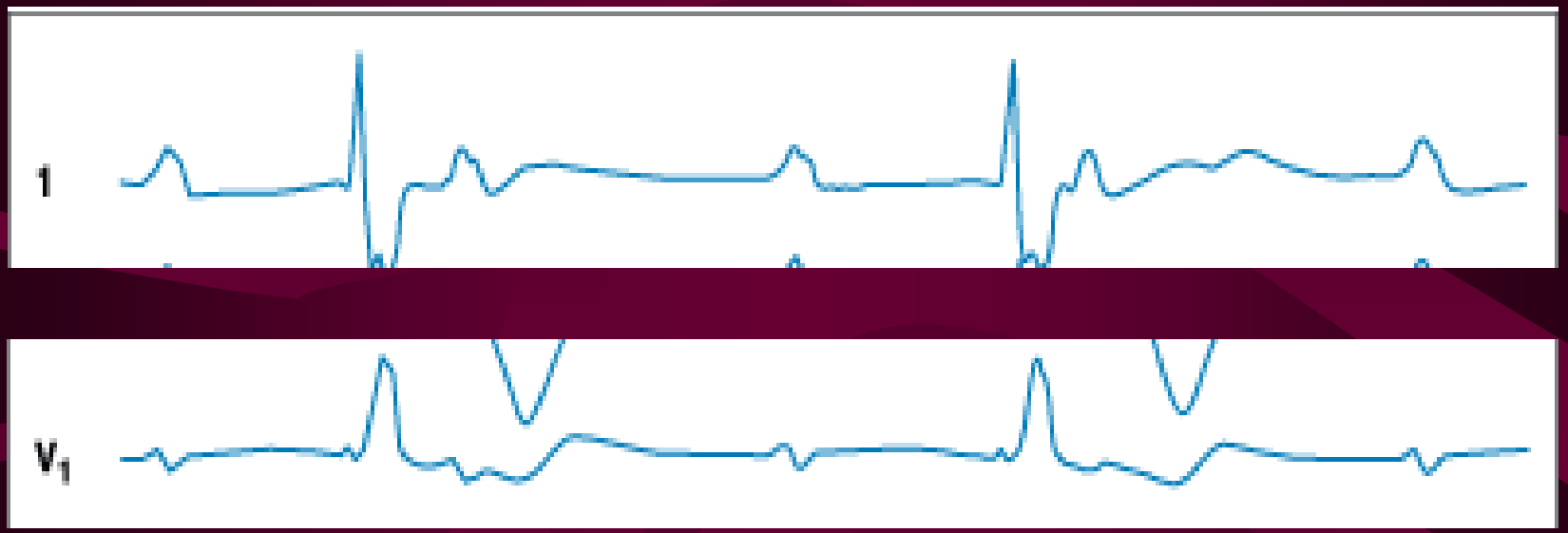
- No relationship between P waves and QRS complexes
- Relatively constant PP intervals and RR intervals
- Greater number of P waves than QRS complexes

Third Degree (complete) AV Block



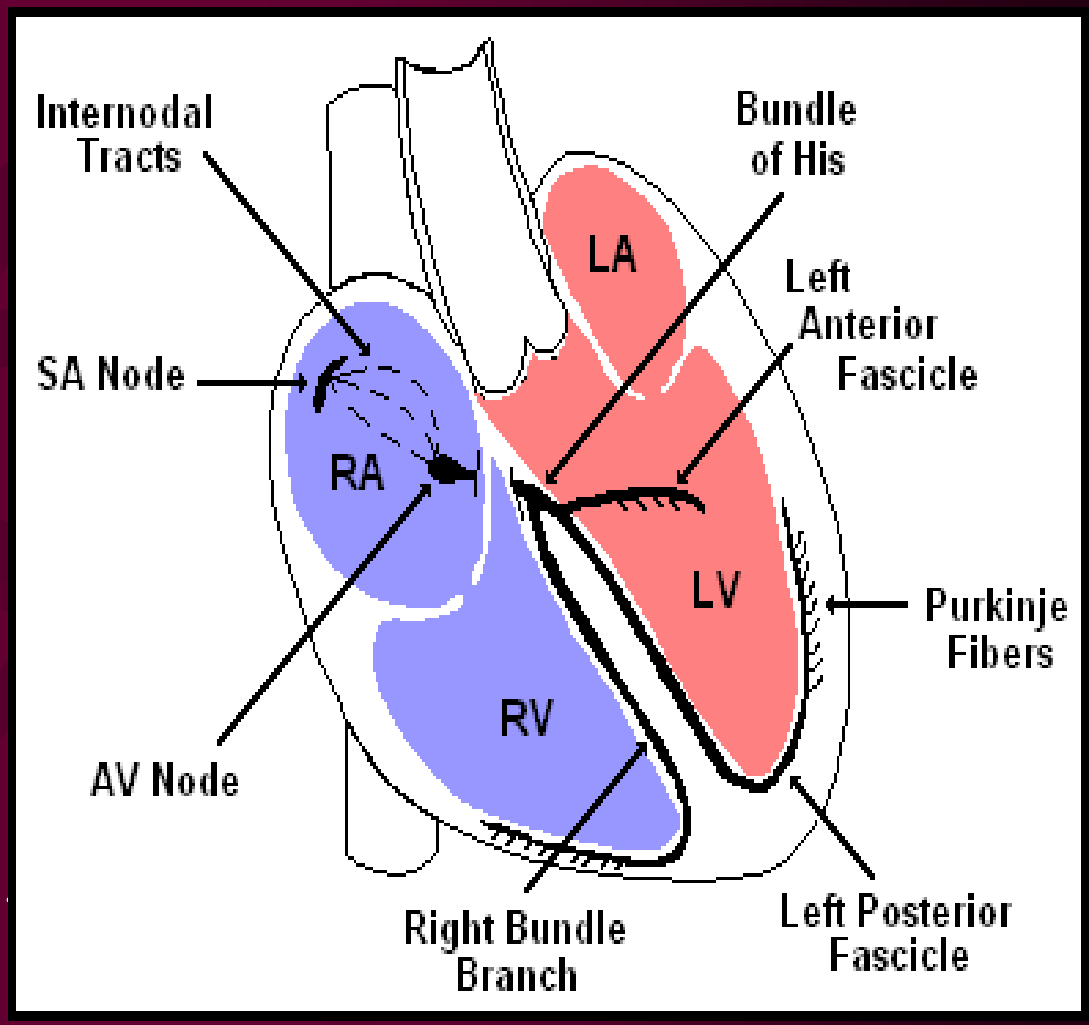
P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Normal but not related to QRS	None	N/A	No relationship between P&RS

AV block III degree



The higher the pacemaker, the narrower the QRS.

- High pacemakers (from lower part of AV node) produce impulses 40-50 beats/min, and narrow QRS complexes.
- Low pacemakers (brunches) are slow, produce impulses 20-40 per min, and broad QRS complexes.



Third-degree heart block has important hemodynamic consequences :

- Syncope, dizziness, and acute heart failure are common.
- When the escape pacemaker rate is > 40 beats/min, symptoms are less acute and include lethargy, hypotension, and dyspnea.
- Asystole is a constant threat.

Bundle Branch BLOCK

- Partial or complete interruptions of electrical conduction in the bundle branches.
- may arise from any cardiac disease process, including intrinsic degeneration
- They usually cause no symptoms and require no direct treatment but are often of prognostic significance.

Right bundle branch block (RBBB)

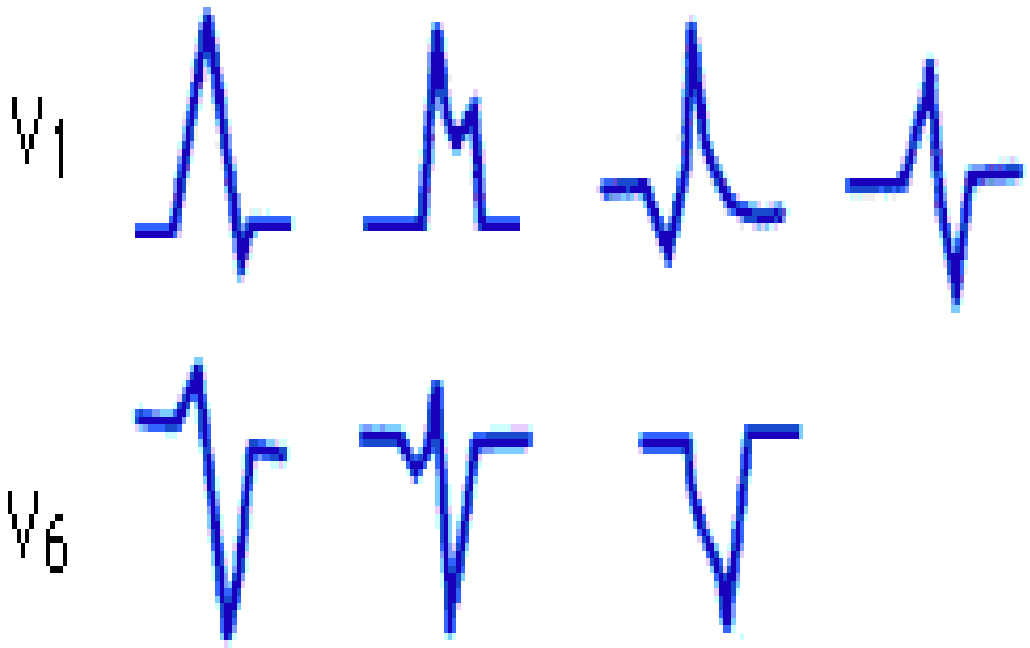
- can occur in apparently normal persons.
- Its most important relationship is with anterior MI.
- Transient RBBB may occur after pulmonary embolism.

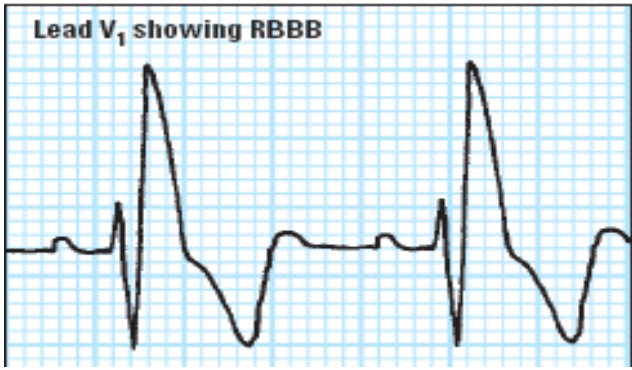
QRS complex behavior in RBBB:

In V1,2 – usually RsR complexes; in V5,6 – rS complexes

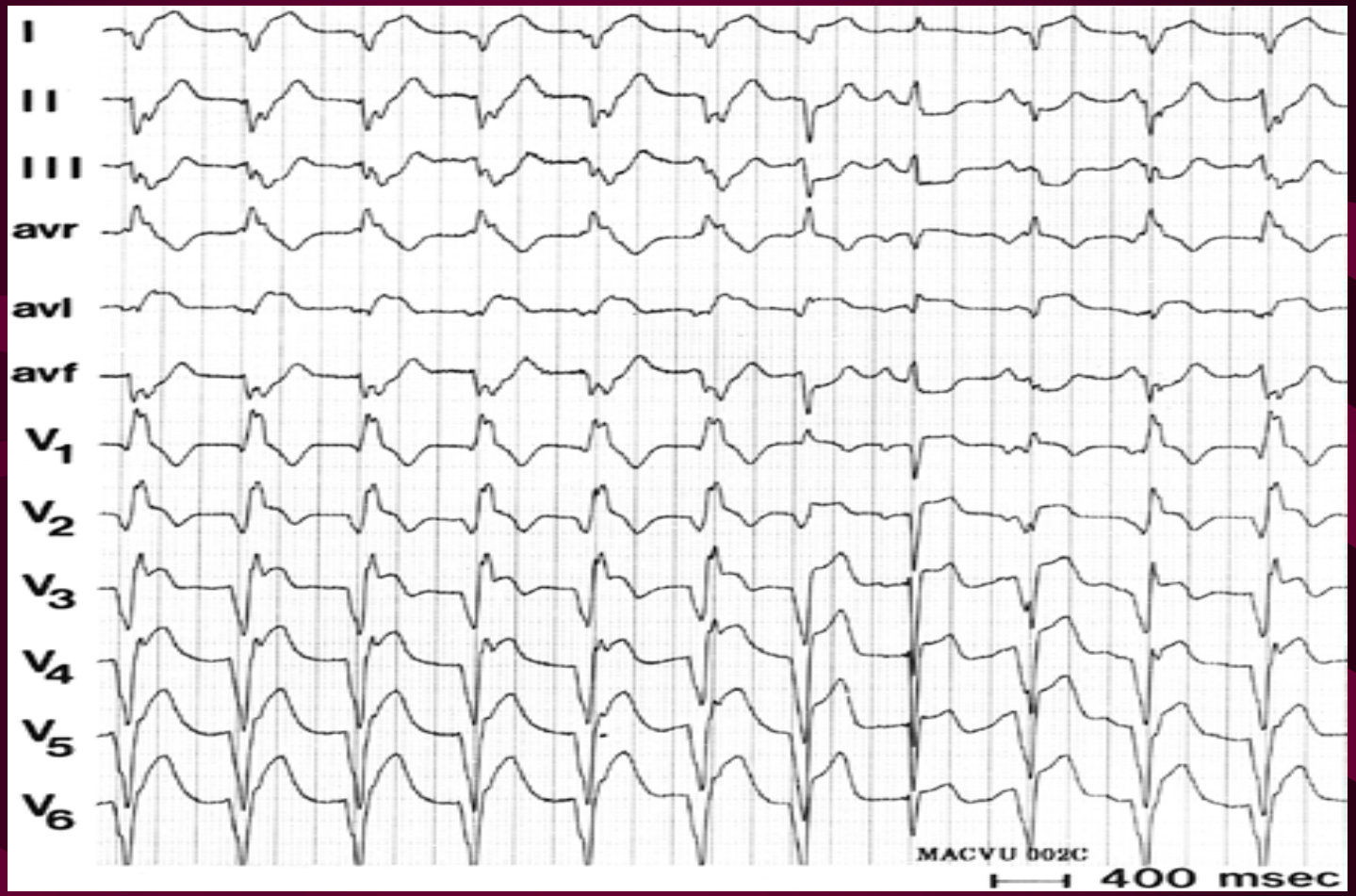
RBBB: Mono- or biphasic complex in V_1

RS (*only with left axis deviation*) or QS in V_6





RBBB



Left bundle branch block (LBBB)

- was once thought of as always pathologic with a bad prognosis, but population studies suggest that benign LBBB may occur.

- LBBB may cower a myocardial infarction.

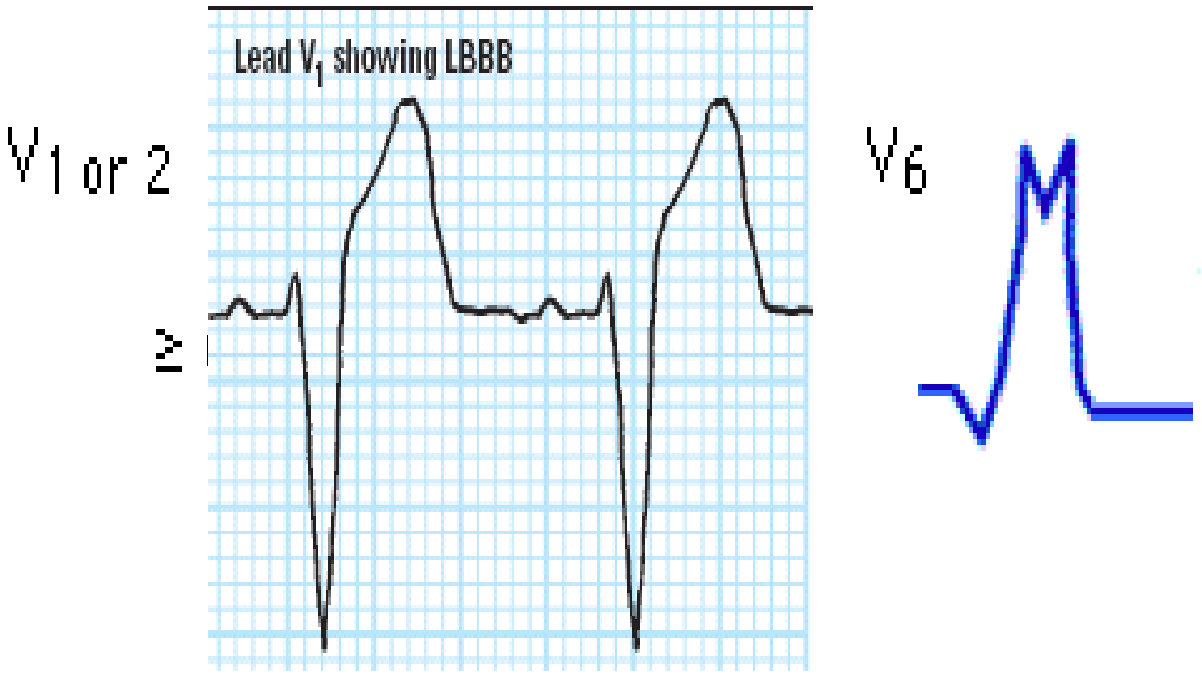
QRS complex behavior in LBBB:

in V1,2 – rS complexes;

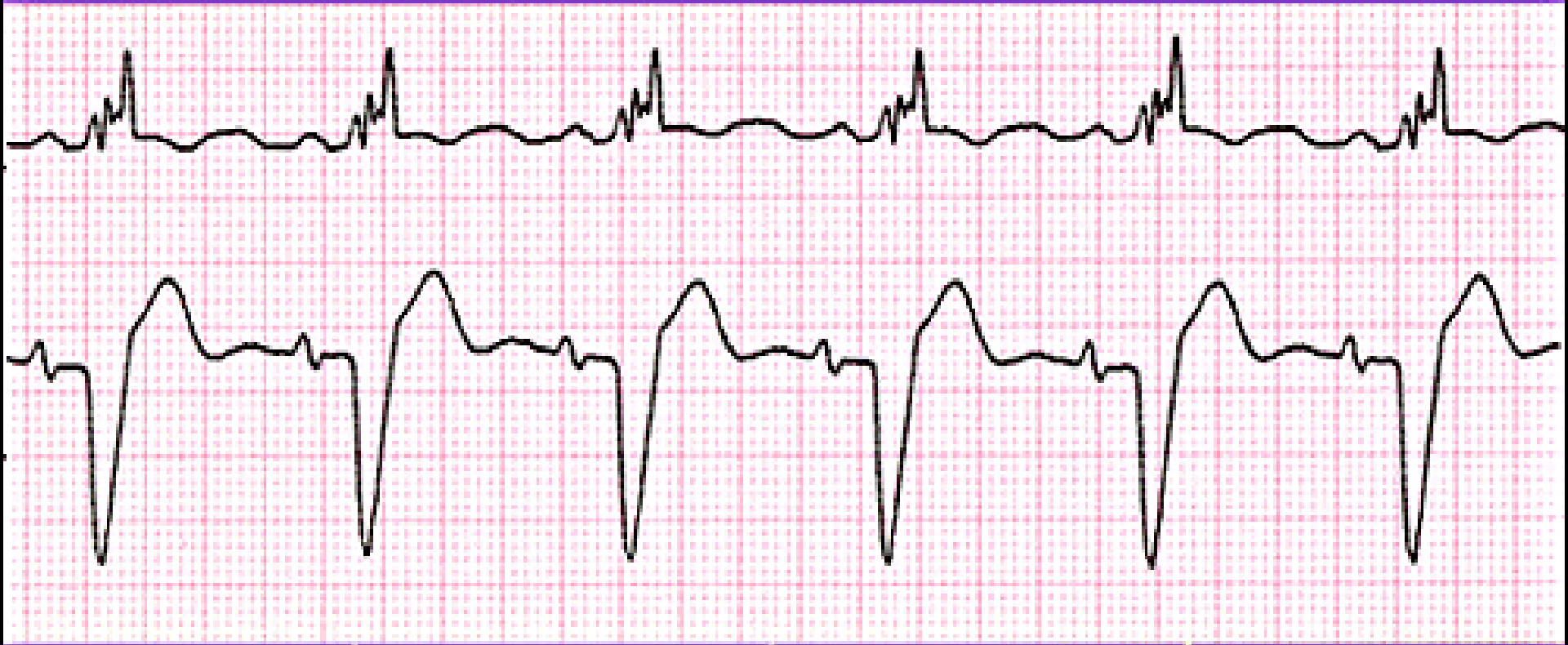
in V5,6 – R wave is deformed or rounded or double-humped.

Notched downslope of S wave in V_1 or V_2

Q wave in V_6



Left Bundle Branch Block



P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	.12 to .20	≥.12	RR' in V5

HEMIBLOCK (Fascicular Block)

- Partial or complete interruptions of electrical conduction in the fascicles.
- modest QRS prolongation (< 0, 12 sec)

Some of you might be feeling a bit overwhelmed at this time ….

EXTRASYSTOLIA

Premature beats resulting from either an abnormal electrical focus or reentry mechanism.

Atrial Extrasystolia (APC)

Premature beats resulting from either an abnormal electrical focus in the atria or intra- atrial reentry.

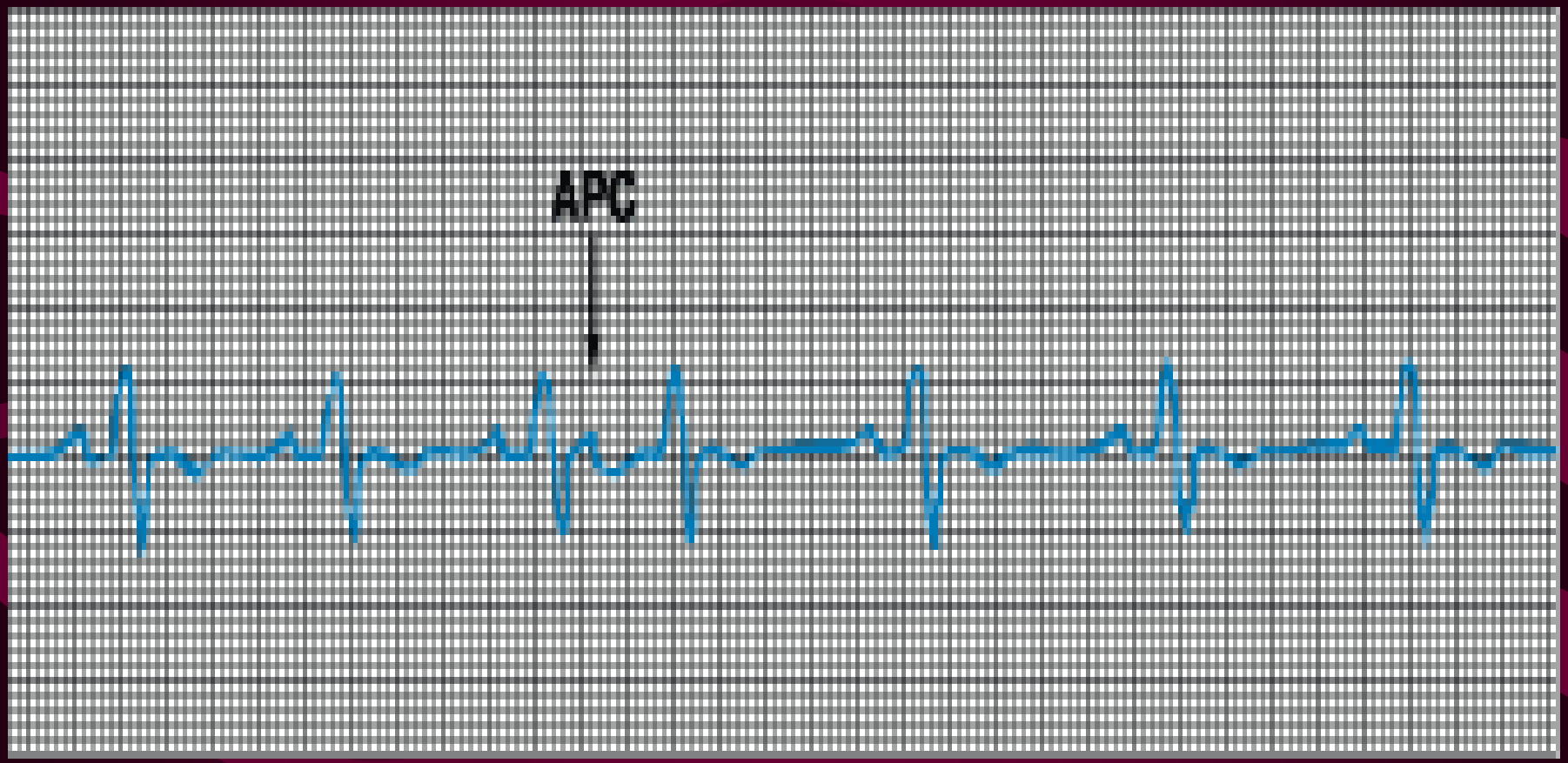
Are frequent in normal persons and rarely cause symptoms.

- Precipitating factors - coffee, tea, alcohol, sympathomimetic cold remedies
- Could be associated with respiratory, cardiovascular diseases,
- If treatment is necessary, b-blocker usually is effective, safe, and well tolerated

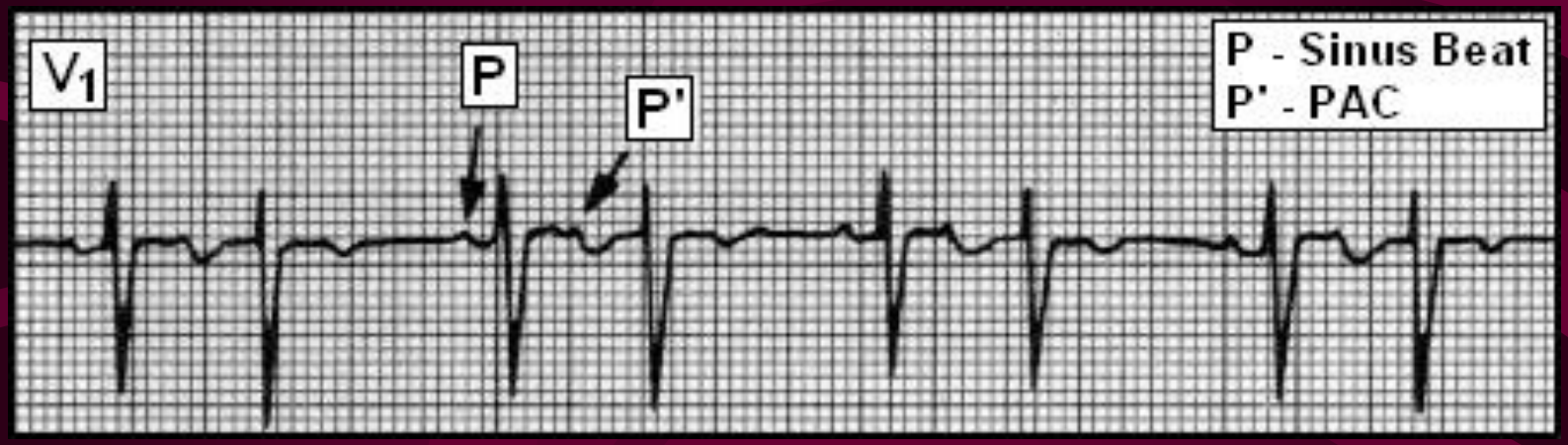
Atrial extrasystolia
Signs on ECG :

- a premature PQRS complex
- P wave if different from the usual one (humped, negative)
- QRS complex is unchanged (comparing with the previous, normal one)
- The compensatory pause is uncompleted

Atrial extrasystolia



Atrial Bigeminy



ATRIO- VENTRICULAR Extrasystolia (AVE)

- Premature beats resulting from an abnormal electrical focus in the AV node.
- are more often associated with cardiac disease or digitalis intoxication.

AVE may or may not cause symptoms or be of prognostic significance.

When symptomatic, they should be treated like AE.

Signs on ECG of AV extrasystolia:

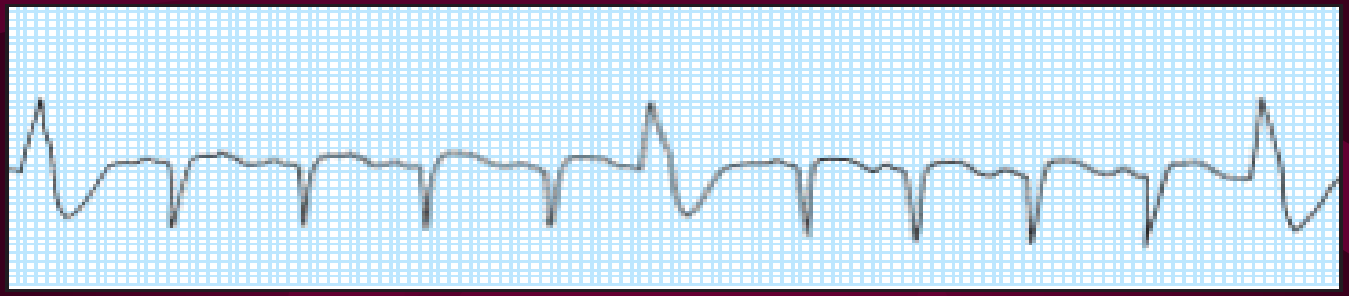
- a premature PQRS complex
- P wave is absent or may be negative after the QRS complex (retrograde)
- normal-appearing QRS complexes
- The compensatory pause is uncompleted

VENTRICULAR extrasystolia (VE)

- VE are a manifestation of abnormal automaticity of a protected ventricular focus.
-
- Because this focus is not penetrated by sinus impulses, it is not reset by them.

ECG signs of VE

- Wide, bizarre QRS complexes (usually >0.14 s), that are not preceded by P waves.
- The compensatory pause is complete



Symptomatic VE commonly are perceived as a missed beat.

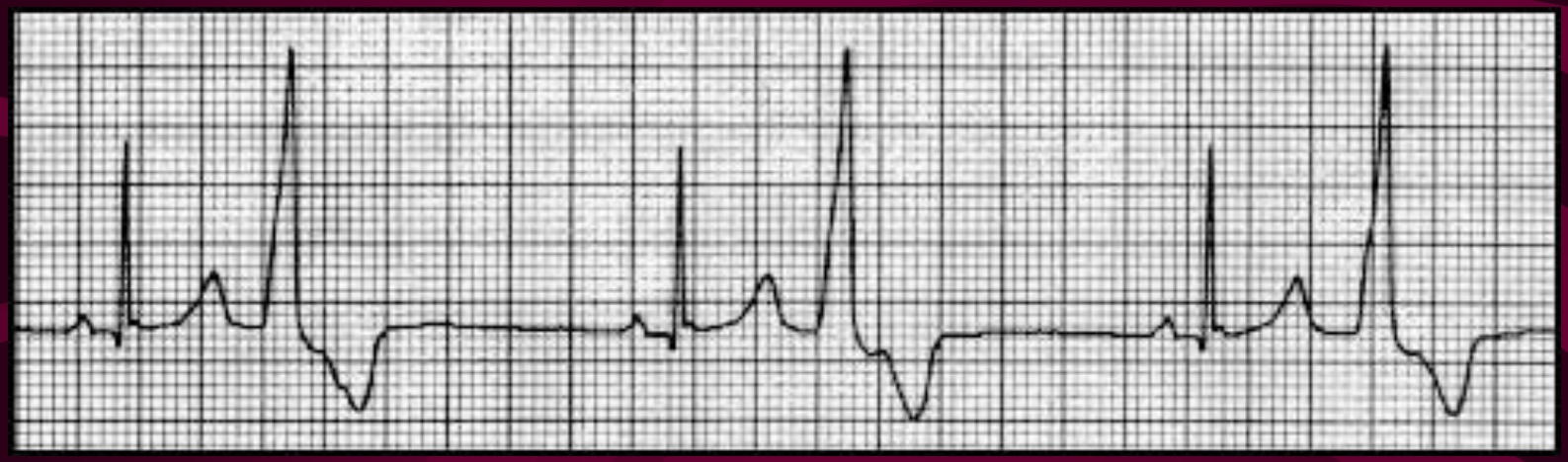
- Of adult males, 60% will exhibit VE during a 24-h Holter monitoring.
- These are among the most common arrhythmias and occur in patients with and without heart disease.
- VE may occur in up to 80% of patients with previous myocardial infarction

- In this setting, if frequent (>10 per hour) or occurring in couplets, they have been associated with increased mortality.

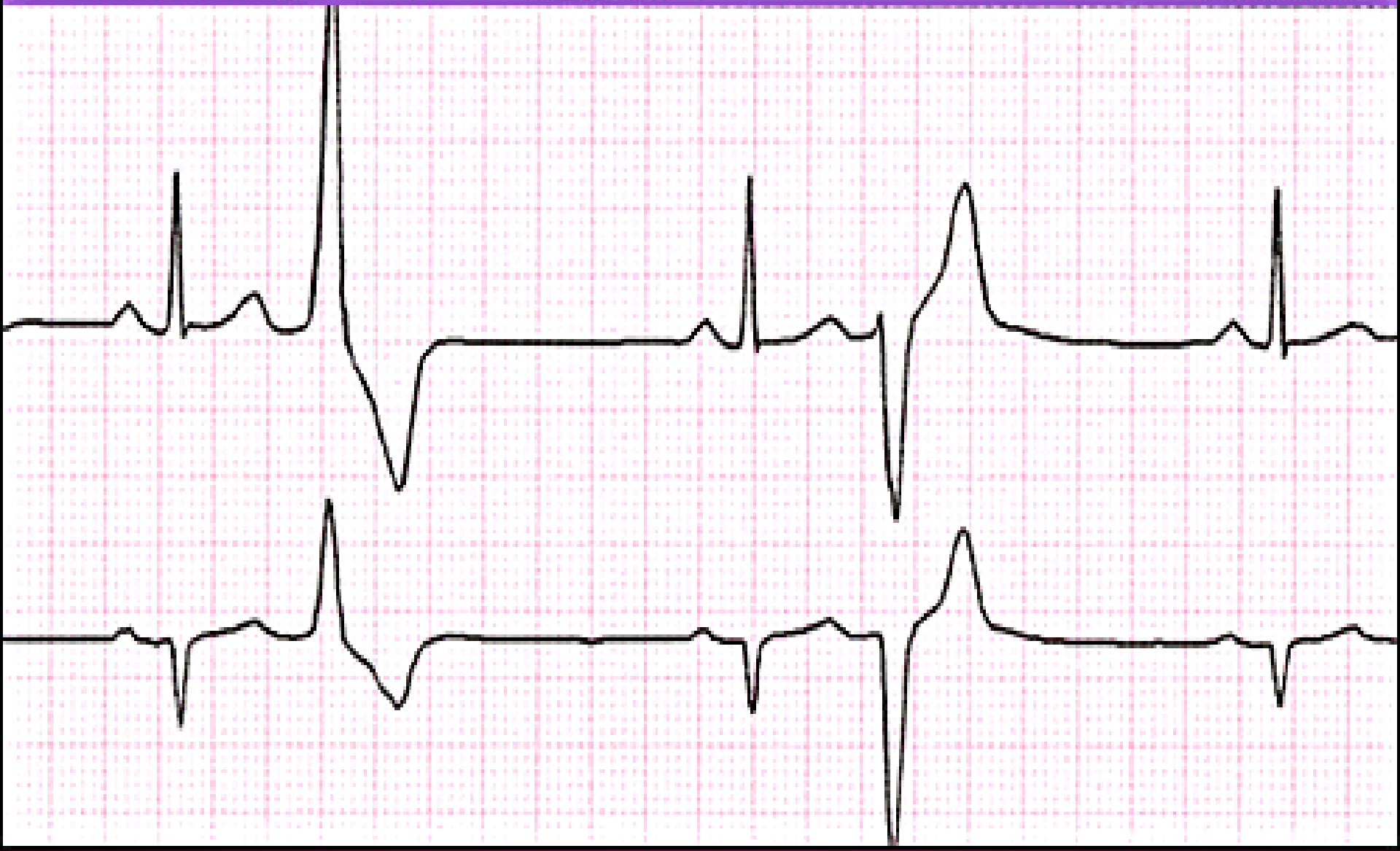
- Very early cycle (R-on-T) VE have been stated to increase the risk of VT or fibrillation and of sudden death.

- VE may occur singly;
- If every sinus beat is followed by a VPC -bigeminy;
- If two sinus beats are followed by a VPC -trigeminy;
- Quadrigeminy - three sinus beats are followed by a VPC

Ventricular Bigeminy



Multifocal PVC's: more than one shape



TACHYARRHYTHMIAS

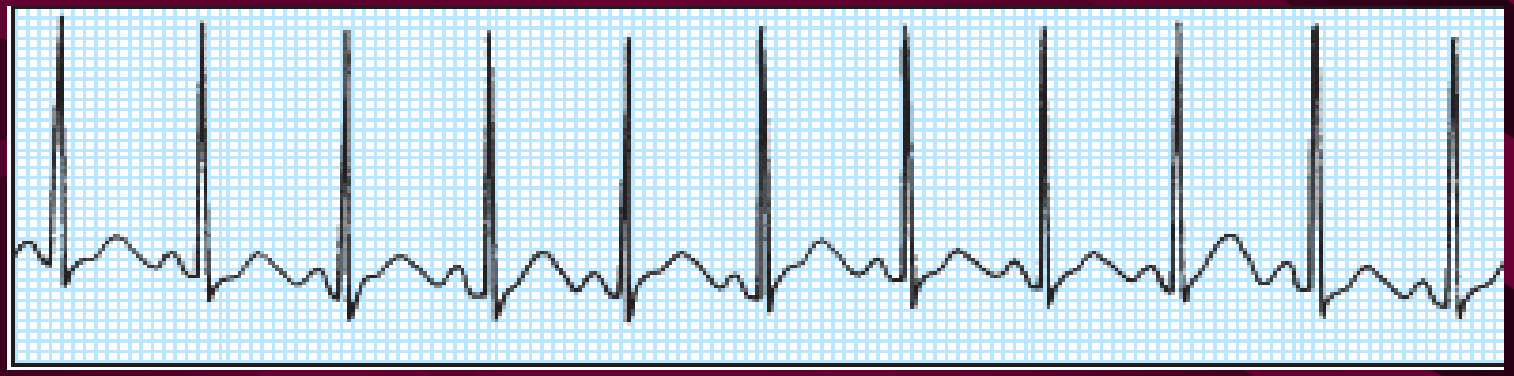
- Sinus tachycardia
- Paroxysmal tachycardia
- Atrial fibrillation
- Atrial flutter

SINUS TACHYCARDIA

- In the adult, sinus tachycardia is said to be present when the heart rate exceeds **90- 100** beats per minute (bpm),
- it represents a physiologic response to a variety of stresses, such as fever, volume depletion, anxiety, exercise, thyrotoxicosis, hypoxemia, hypotension, or congestive heart failure.
- Sinus tachycardia has a gradual onset and offset.

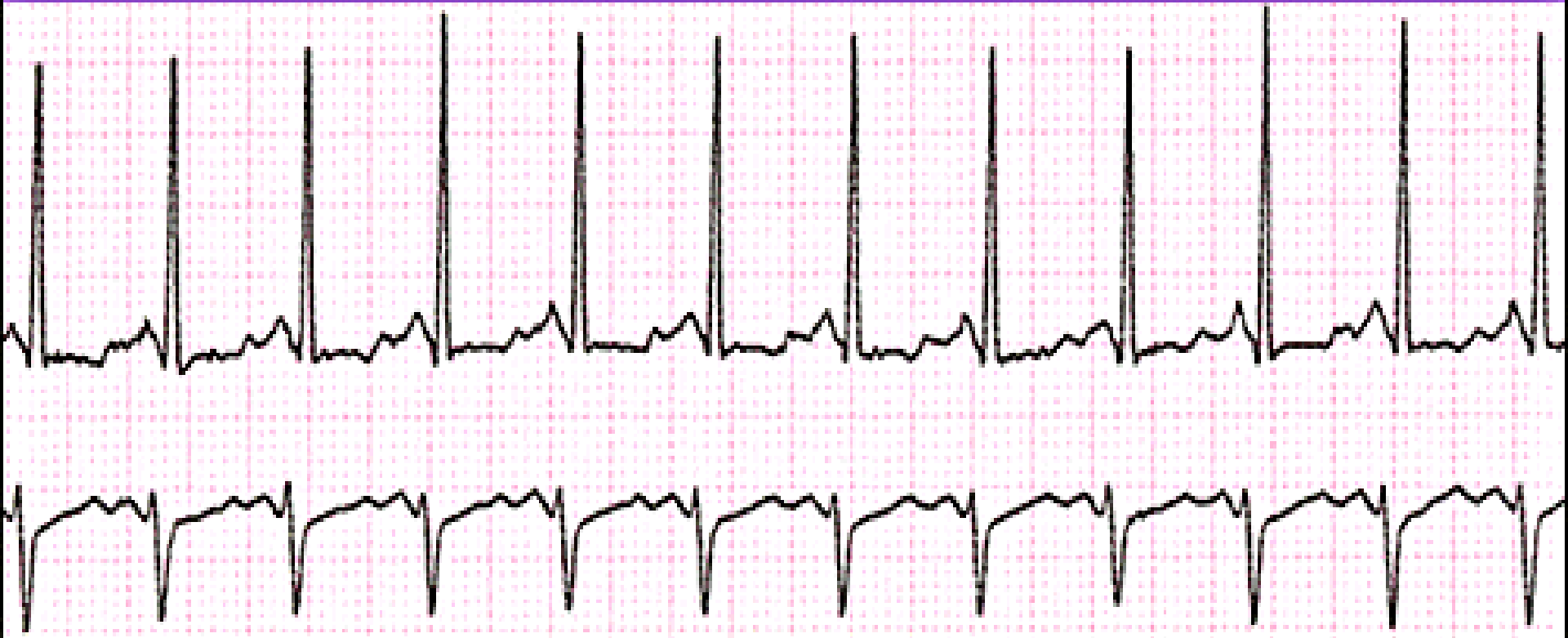
The ECG in ST

- demonstrates P waves with sinus contour preceding each QRS complex.
- heart rate exceeds 90- 100 beats per minute (R-R intevals are shorter)



Paroxysmal supraventricular tachycardias may slow slightly and terminate abruptly.

Sinus Tachycardia



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
> 100 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

ATRIAL FIBRILLATION

- Intermittent rapid irregular atrial rhythm due to multiple reentrant wavelets.
- AFB is characterized by an atrial rate between 400 and 600 bpm.
- The ventricular rate may be normal – 60-90 bpm, may be tachyform - > 100 bpm; may be bradyform - < 60 bpm (it depedes on the ability of AV node to block th impulse

- The symptoms of paroxysmal AFB are often devastating because of the sporadic dramatic changes of heart rate and regularity.

- AF is a common arrhythmia that may occur in paroxysmal, persistent or permanent forms.
- May be seen **paroxysmal AFB** in normal subjects, particularly during emotional stress or following surgery, exercise, acute alcoholic intoxication, or a prominent surge of vagal tone (i.e., vasovagal response).
- **Persistent AFB** may occur in patients with heart or lung disease who develop metabolic or hemodynamic derangements.

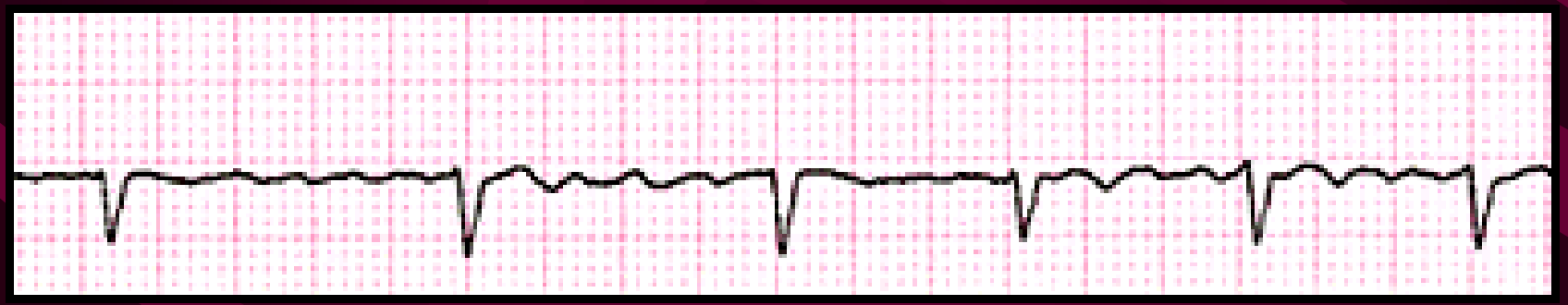
- **Persistent and permanent** AF usually occurs in patients with cardiovascular disease, most commonly rheumatic heart disease, nonrheumatic mitral valve disease, hypertensive cardiovascular disease, chronic lung disease, atrial septal defect, in thyrotoxicosis.

Clinical picture in AFB

- The first heart sound usually varies in intensity.
- On EchoCG : the left atrium is frequently enlarged, and in patients in whom the left atrial diameter exceeds 4.5 cm, it may be difficult to convert AF to sinus rhythm and/or maintain the latter, despite therapy.

Signs on ECG:

- P waves are absent
- R-R intervals are different
- Instead of P wave appear small f-waves (> 600/min)



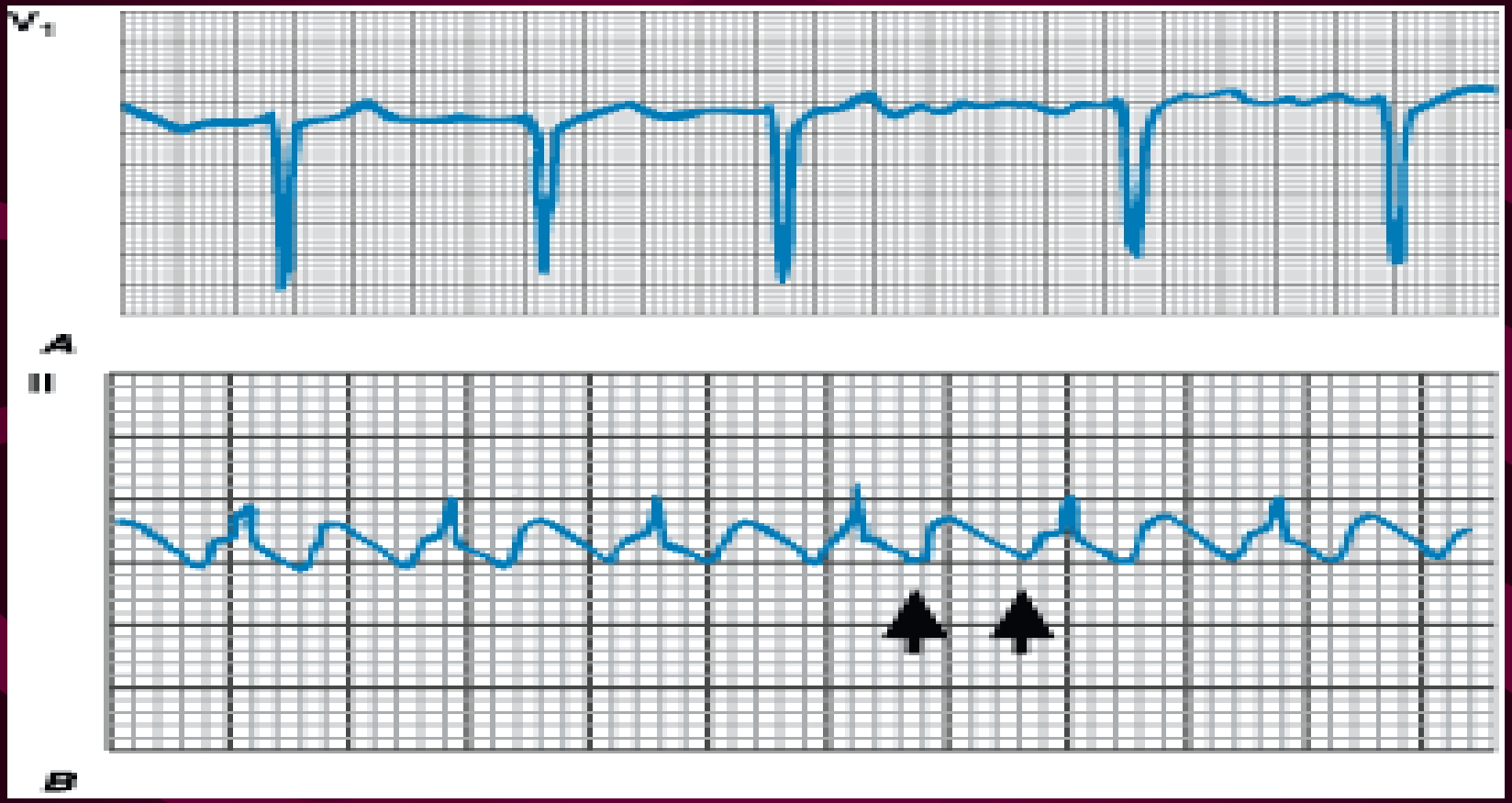
ATRIAL FLUTTER

- This arrhythmia occurs most often in patients with organic heart disease.
- Flutter may be paroxysmal (pericarditis or acute respiratory failure) or it may be persistent.
- Atrial flutter is usually less long-lived than is AF, although on occasion it may persist for months to years.

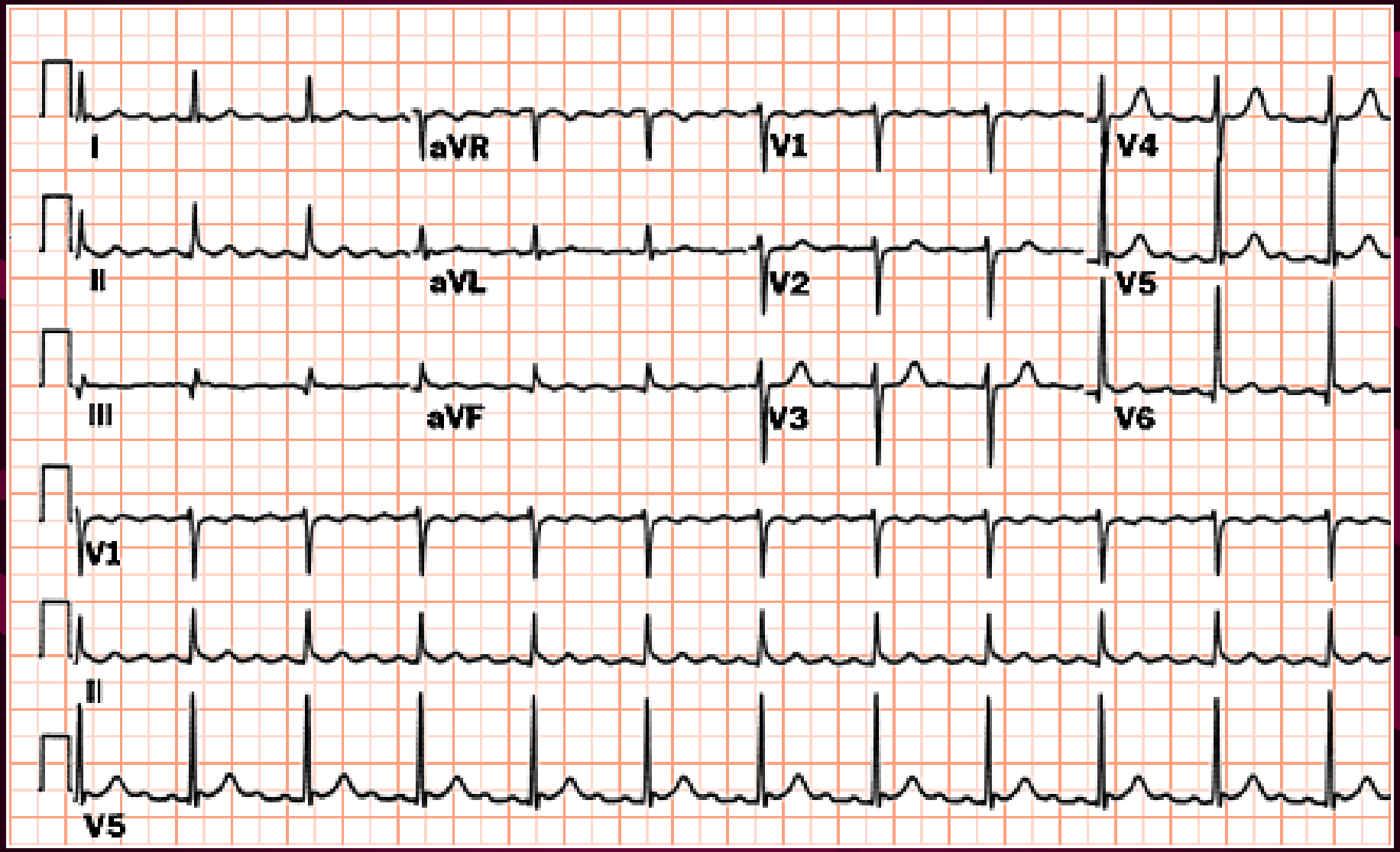
ECG

- Classically, flutter waves are seen as regular sawtooth-like waves (instead of P wave), most prominent in the inferior leads
- P wave - absent
- QRS is not changed

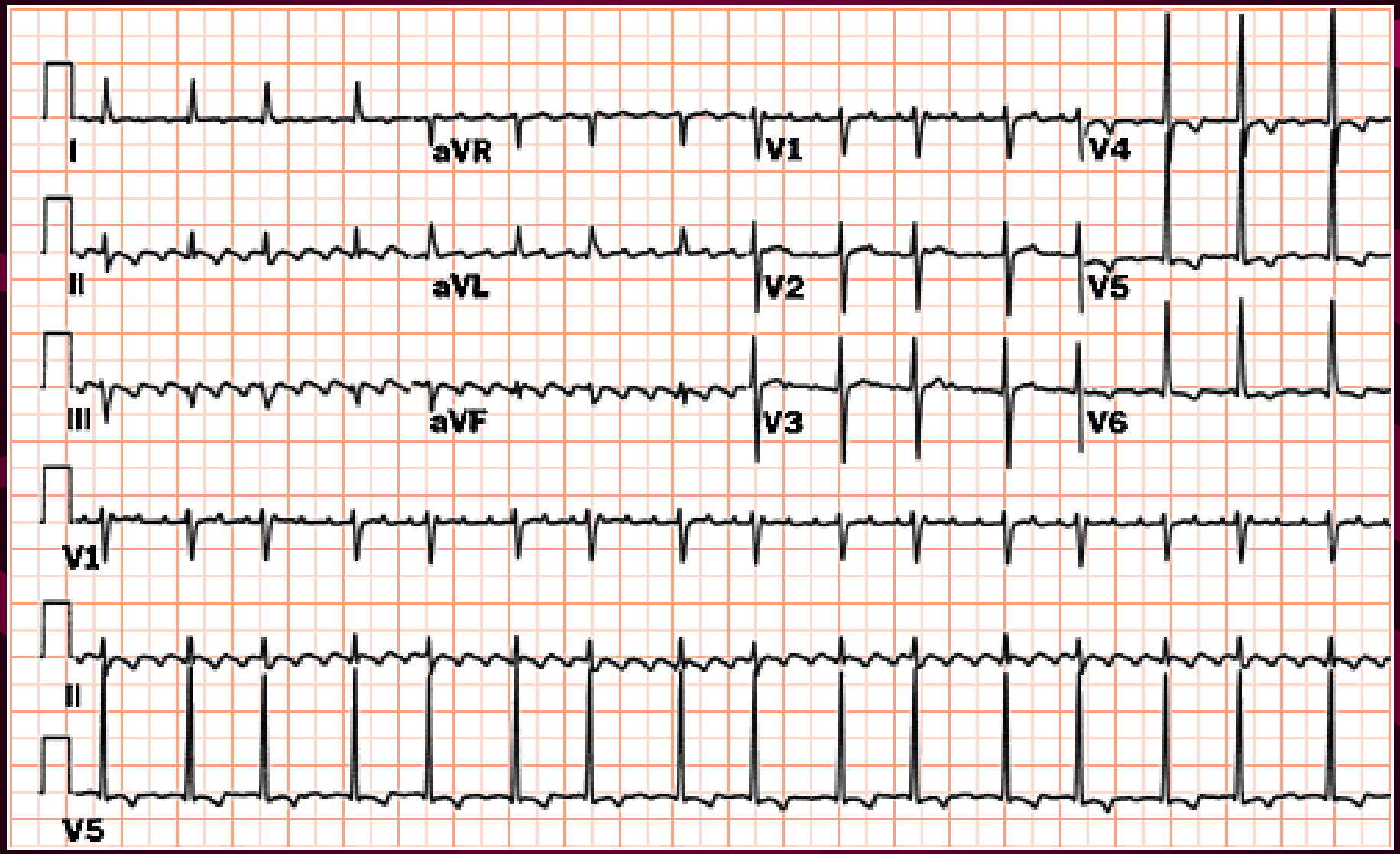
Atrial flutter



Atrial flutter



Atrial flutter



- Most commonly, if it lasts for more than a week, atrial flutter will convert to AF.
- Systemic embolization is less common in atrial flutter than in AF
- Atrial flutter is characterized by an atrial rate between 250 and 350 bpm. Typically, the ventricular rate is half the atrial rate, i.e., approximately 150 bpm.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIAS (PSVT)

- In most cases, functional differences in conduction and refractoriness in the AV node or the presence of an AV tract provide the substrate for the development of PSVT .
- Electrophysiological studies have demonstrated that reentry is responsible for the majority of PSVT

Paroxysmal supraventricular tachycardia

- **Atrial Paroxysmal tachycardia**
- **Junctional (AV) Paroxysmal tachycardia**

Sometimes is difficult to make difference between them.

- palpitations are universal in PSVT but variably tolerated.

These arrhythmias can complicate most forms of heart disease, severe chronic lung disease, may be exacerbated by theophylline.

The algorithm of their treating and managing is the same.

ATRIAL TACHYCARDIA

Signs on ECG:

- sudden onset and sudden finishing
- Abnormal P waves precede QRS complexes
- heart rate (ventricular rate) will be 160-200 b/min.

  It appears like a line of atrial extrasystoles coming with a high frequency (160-200 per minute)

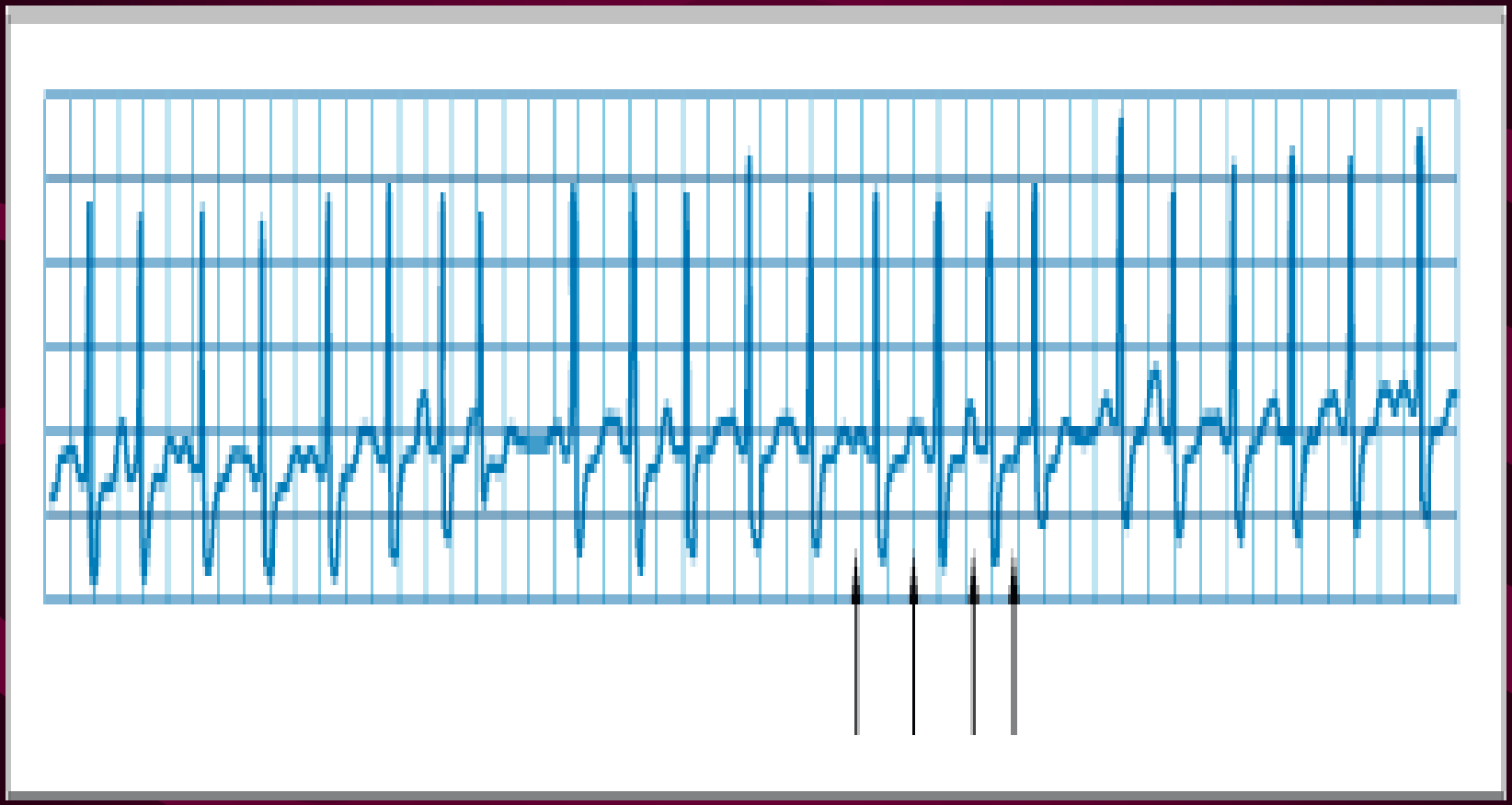
ATRIOVENTRICULAR TACHYCARDIA

Signs on ECG:

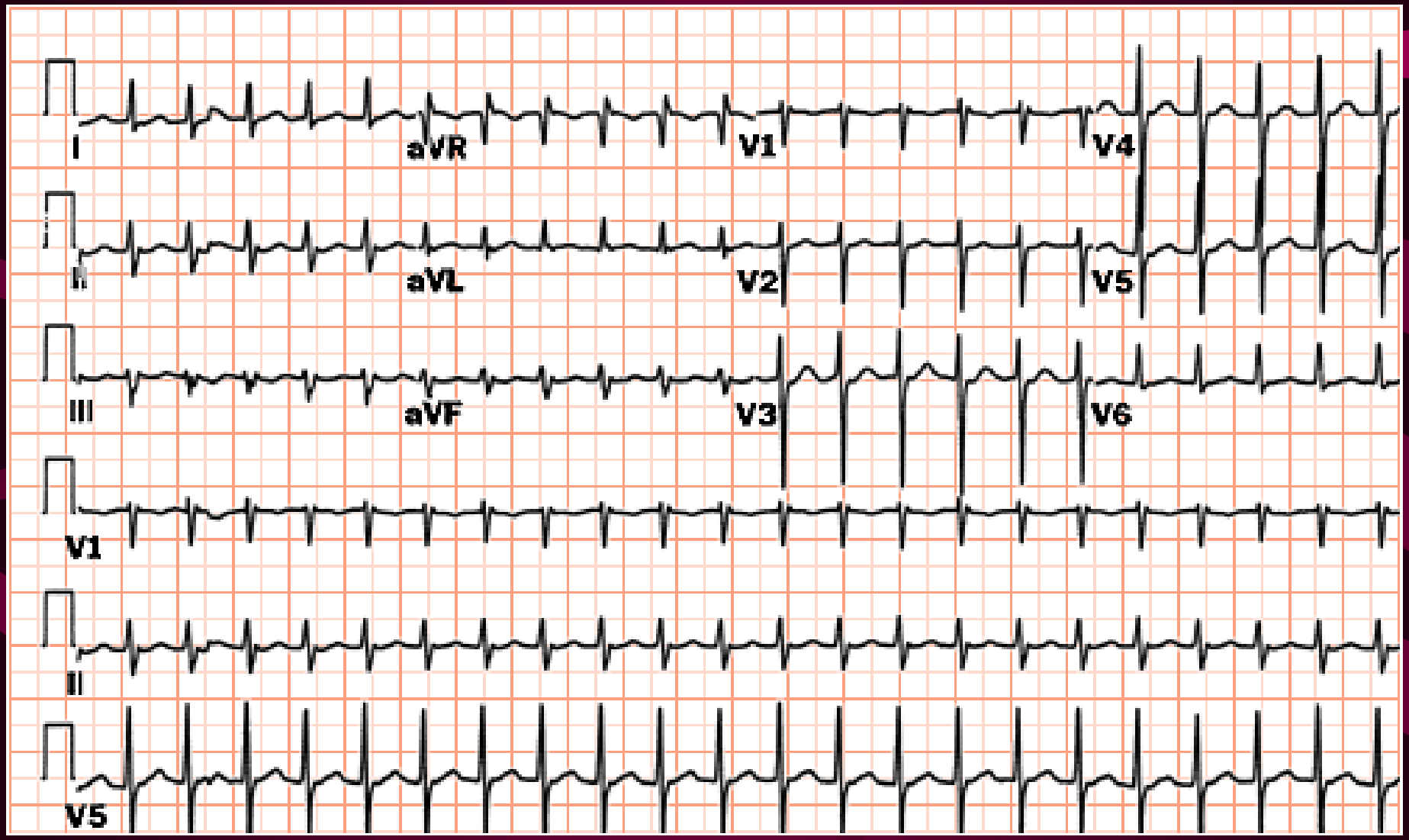
- sudden onset and sudden finishing
- heart rate will be 160-200 /min.
- P' waves absent or negative after the QRS.
- QRS – unchanged

Appears like many frequent AV extrasystoles (160-200 per min)

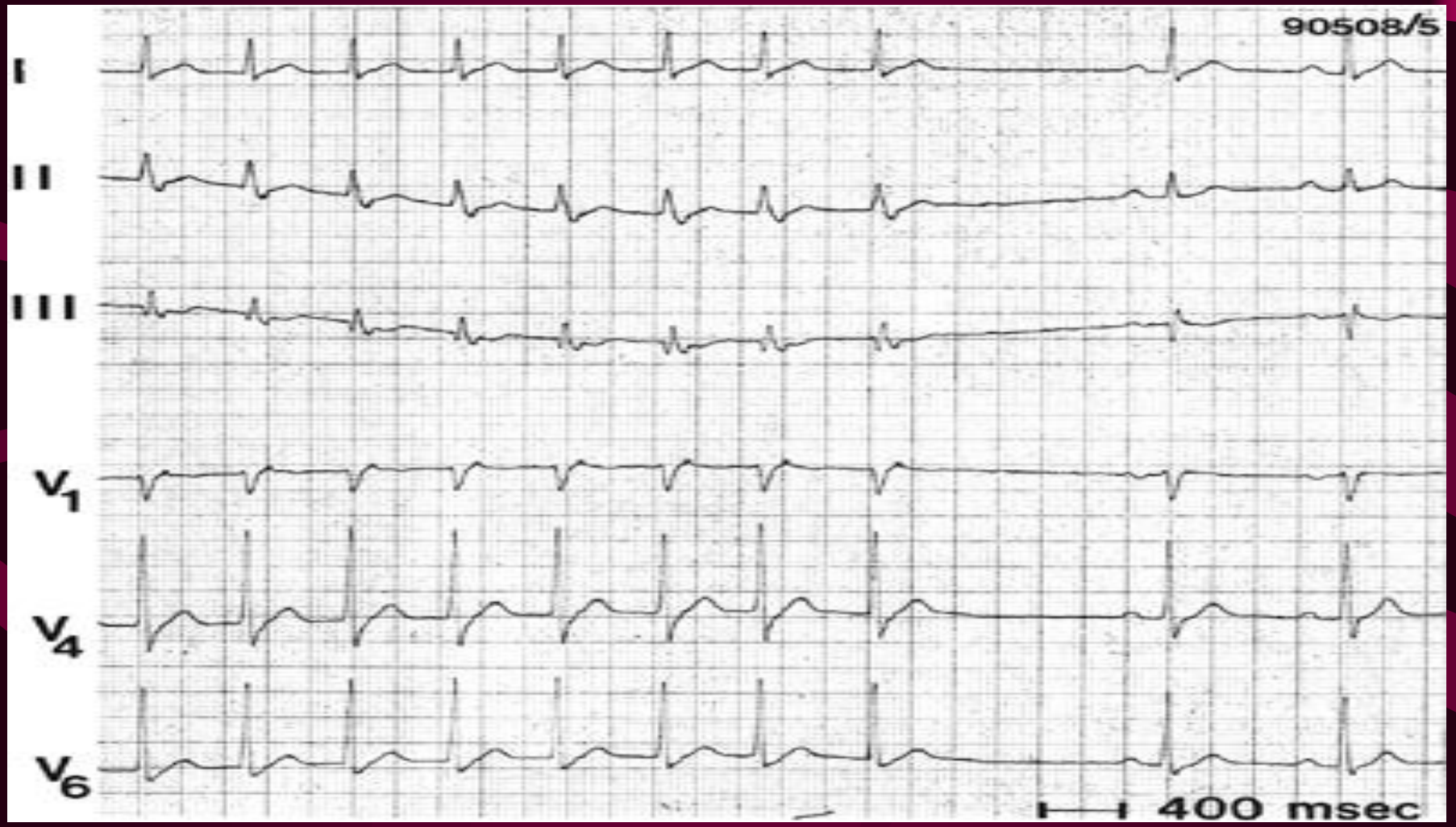
AT



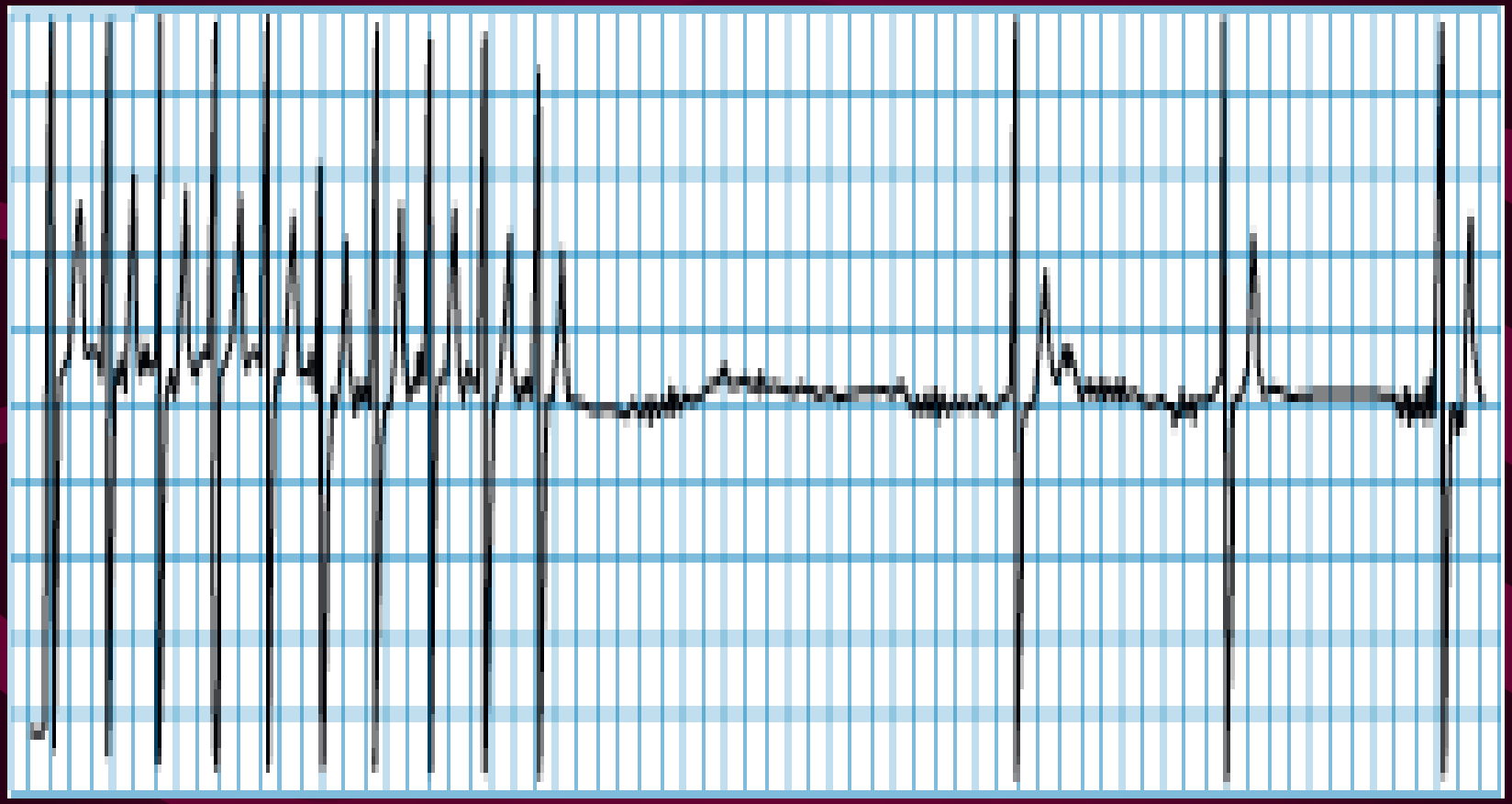
AV tachycardia



AV tachycardia



AVT



VENTRICULAR TACHYCARDIA

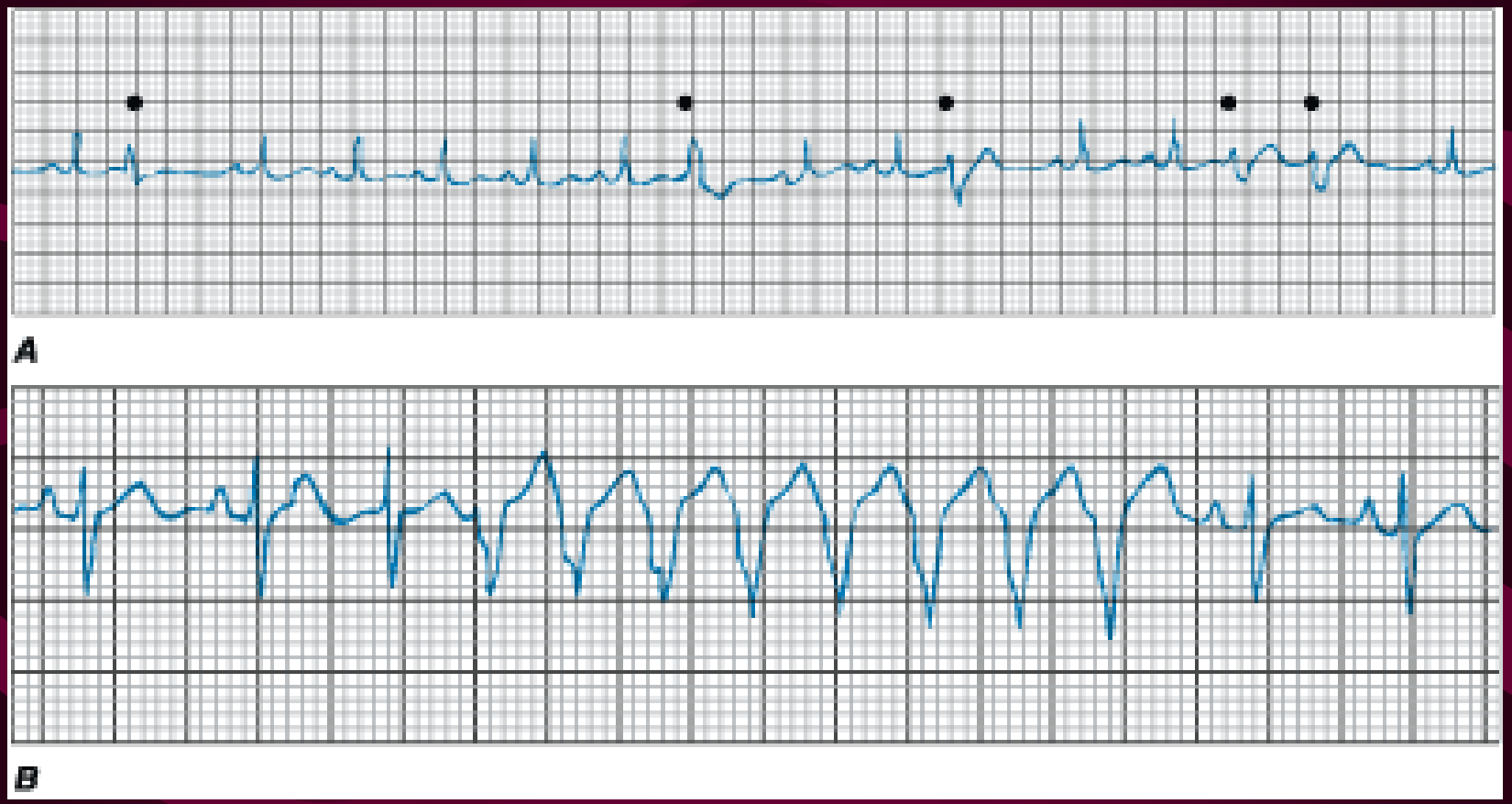
- A rapid regular rhythm due to abnormal automaticity within ventricular cells , a salvo of three or more consecutive ventricular beats at a rate > 120 beats/min.
- Unless producing hemodynamic upset, it usually is not treated.
- It occurs in patients after thrombolytic therapy, in heart diseases, hypokaliemia.

Signs on ECG:

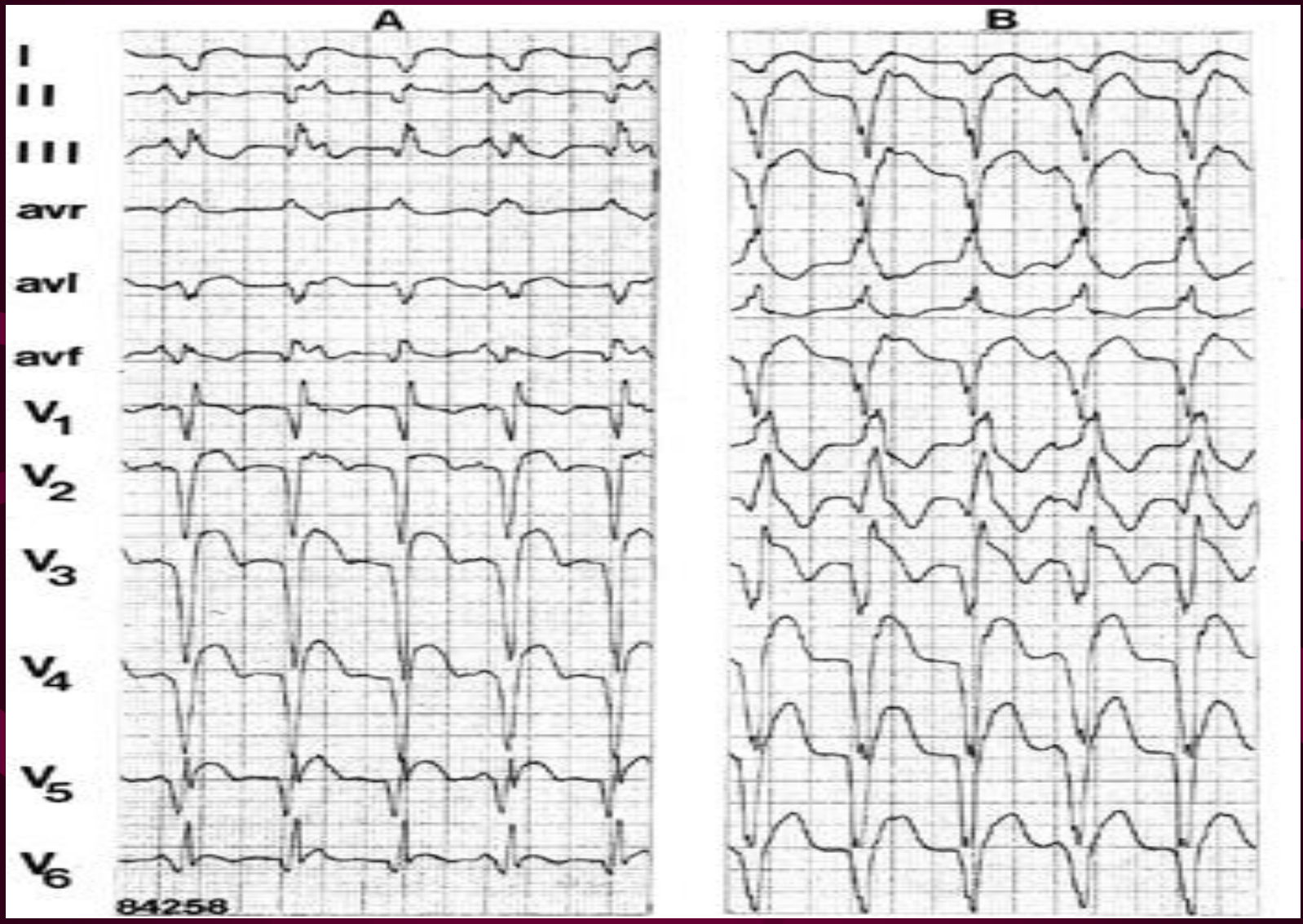
- **Abrupt** sudden onset and sudden finishing
- P waves are not appreciated
- Broad wide-complexes QRS (QRS > 0,12 sec) at a rate exceeding 100 bpm.

Any Broad QRS tachycardia should be considered VT until proven otherwise.

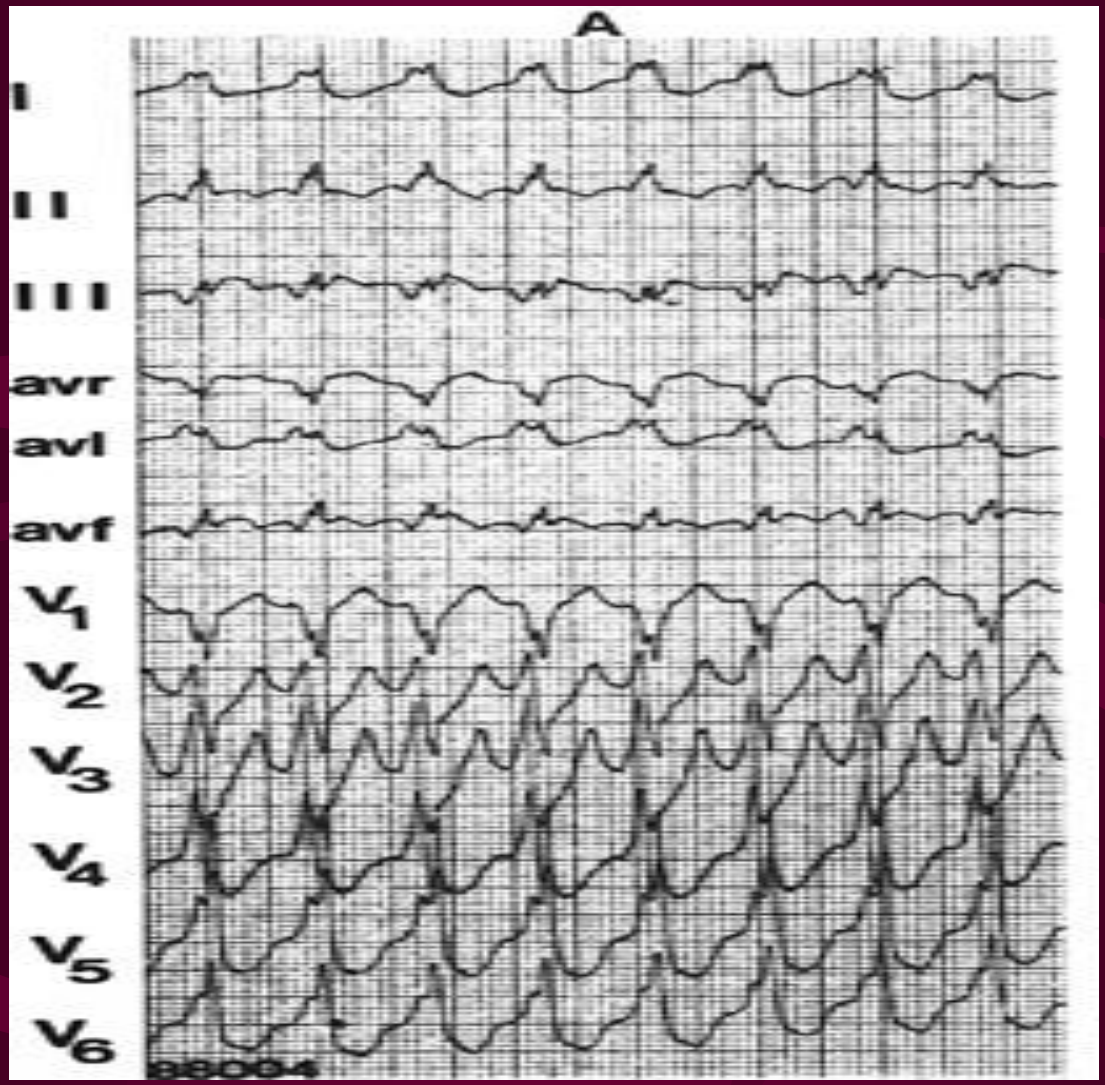
Ventricular tachycardia



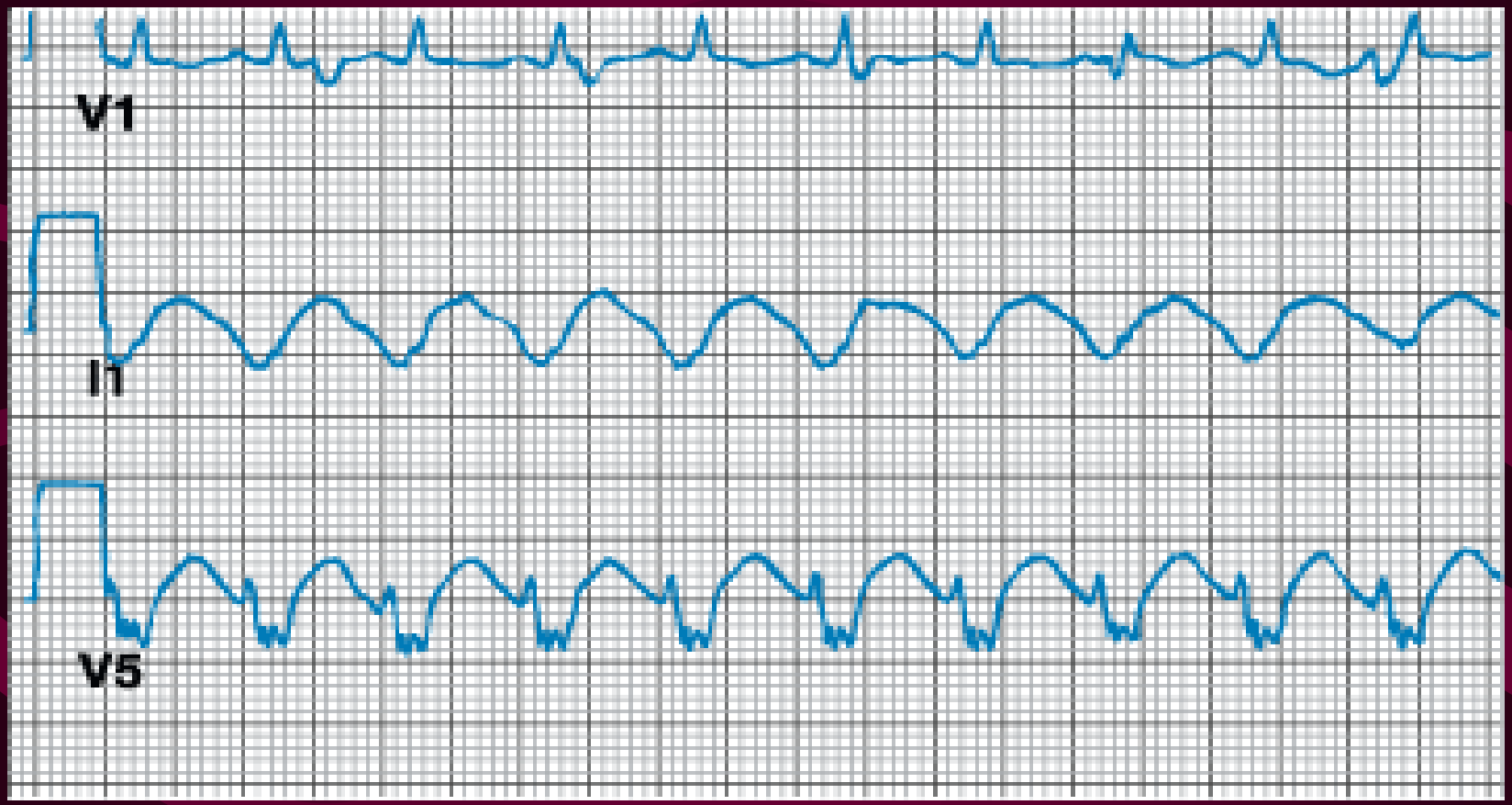
Ventricular tachycardia



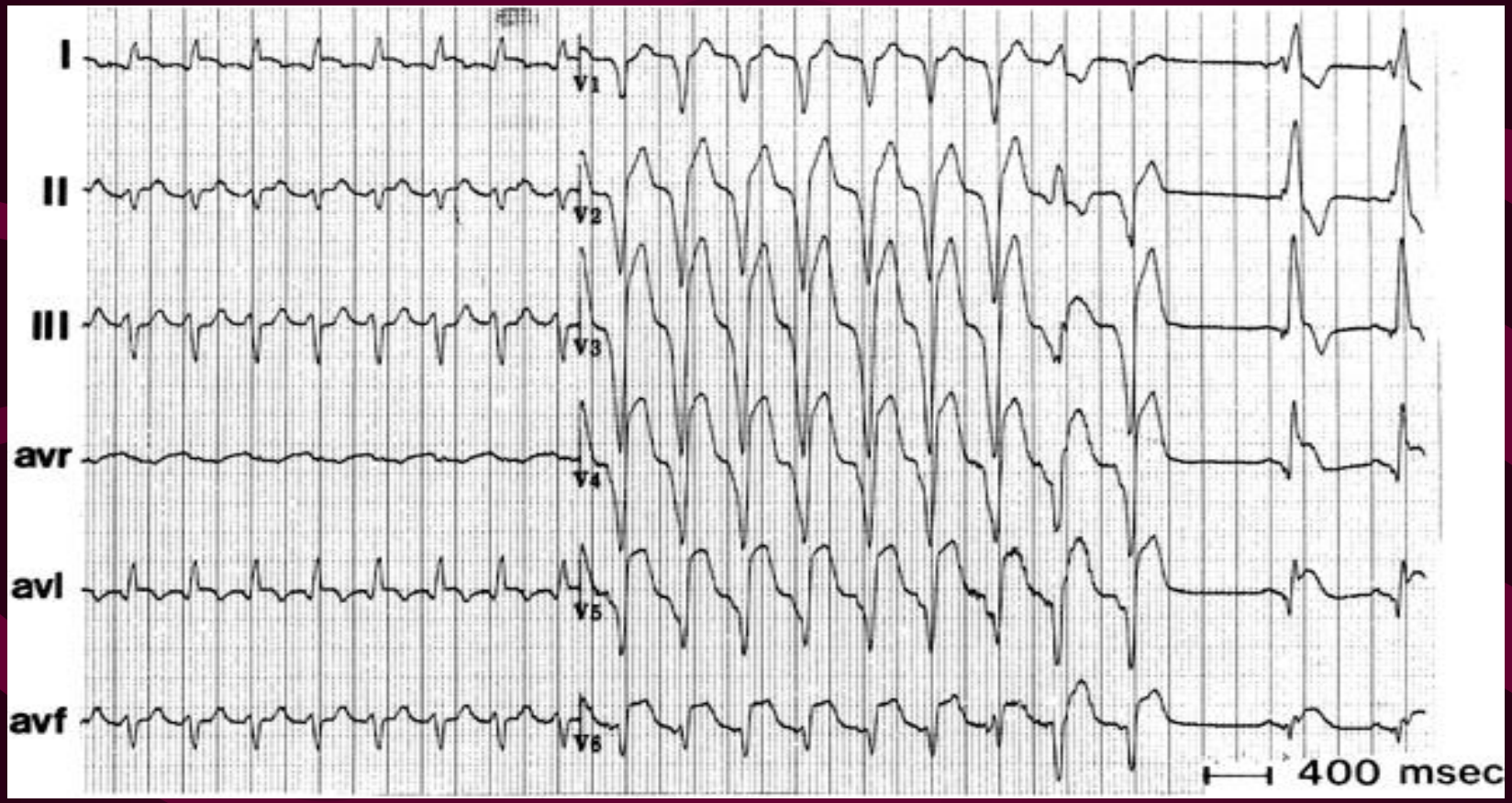
Ventricular tachycardia



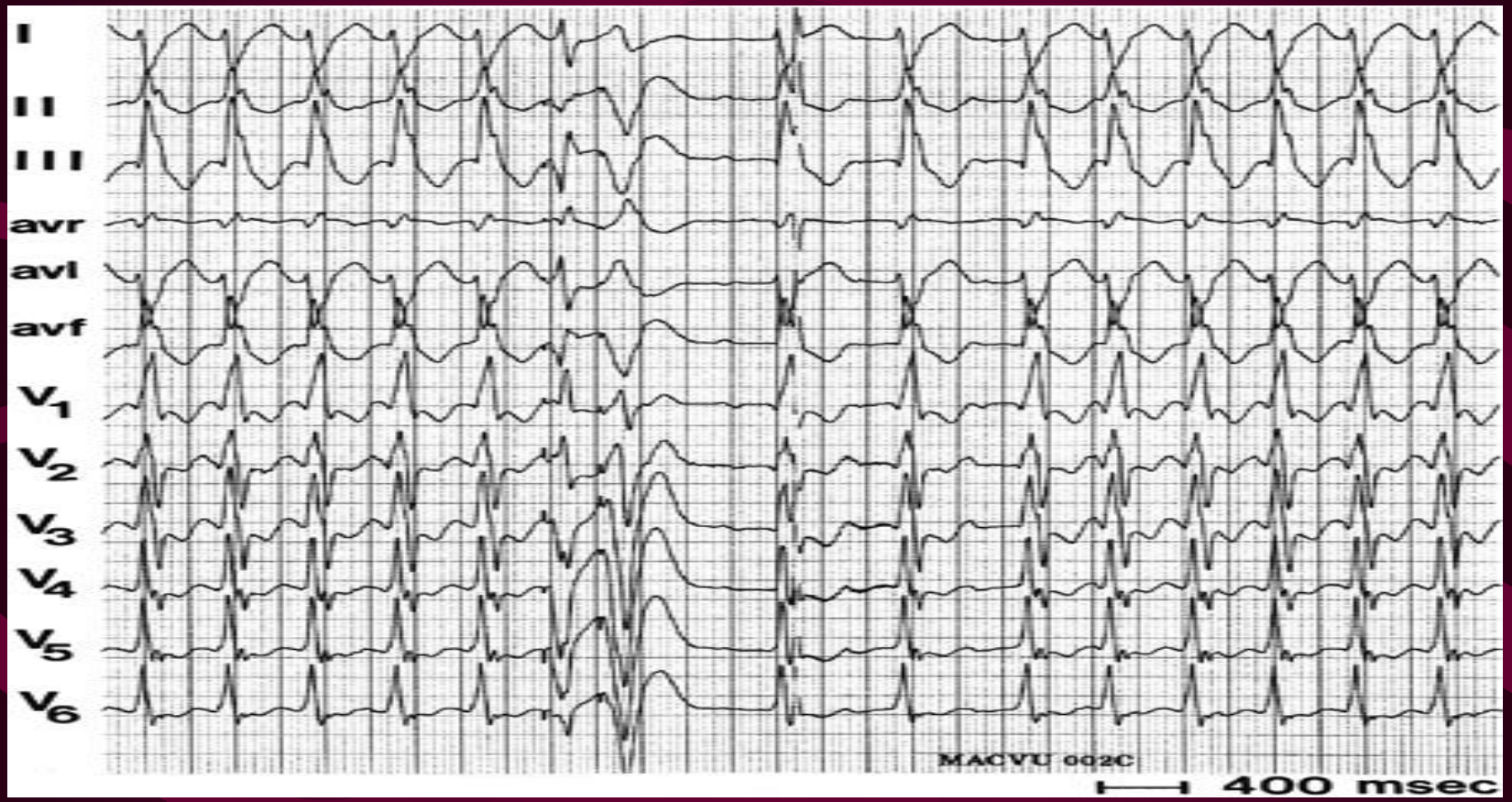
Ventricular tachycardia



Ventricular tachycardia



Ventricular tachycardia



VENTRICULAR FIBRILLATION and flutter

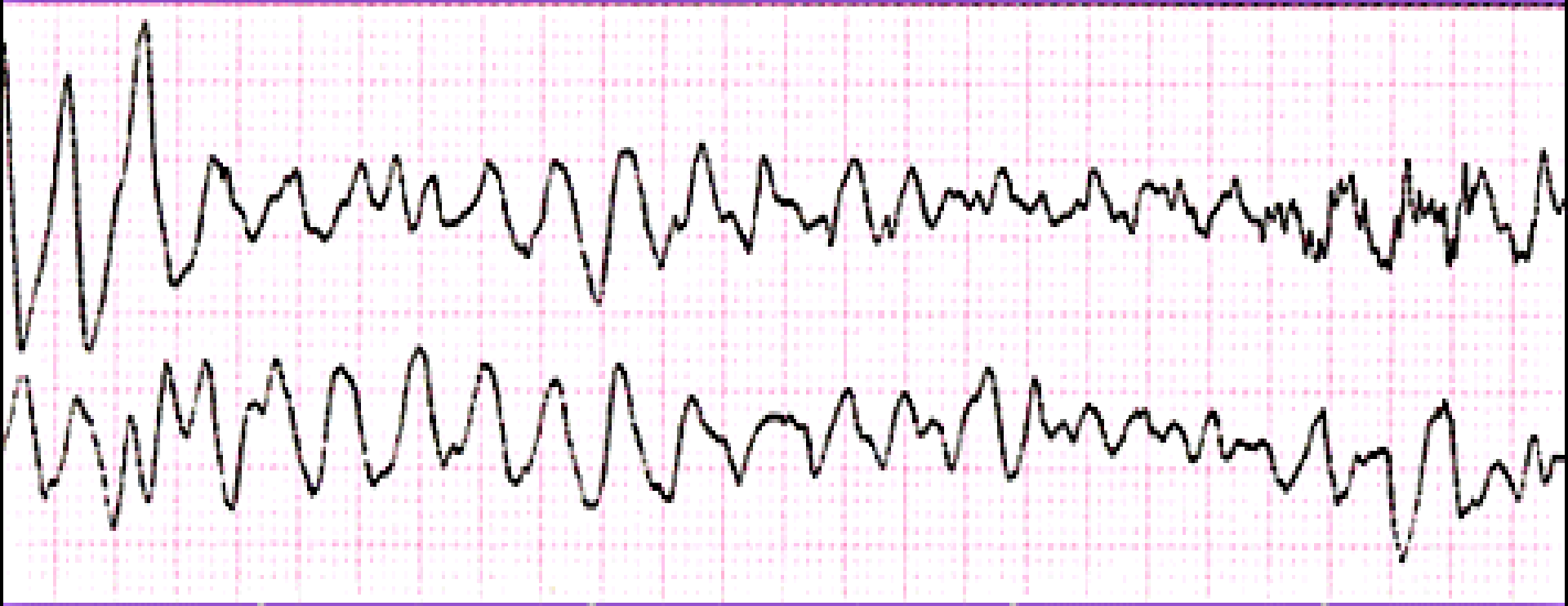
- A rapid irregular ventricular rhythm due to multiple ectopic or reentrant activities
- is associated with zero cardiac output – clinical death.
- VF is complicating acute myocardial infarction (MI); is the more often cause of sudden death.
- VF is fatal unless reversed by defibrillation or antiarrhythmics.

ECG signs

There are no recognizable waves and complexes and segments.

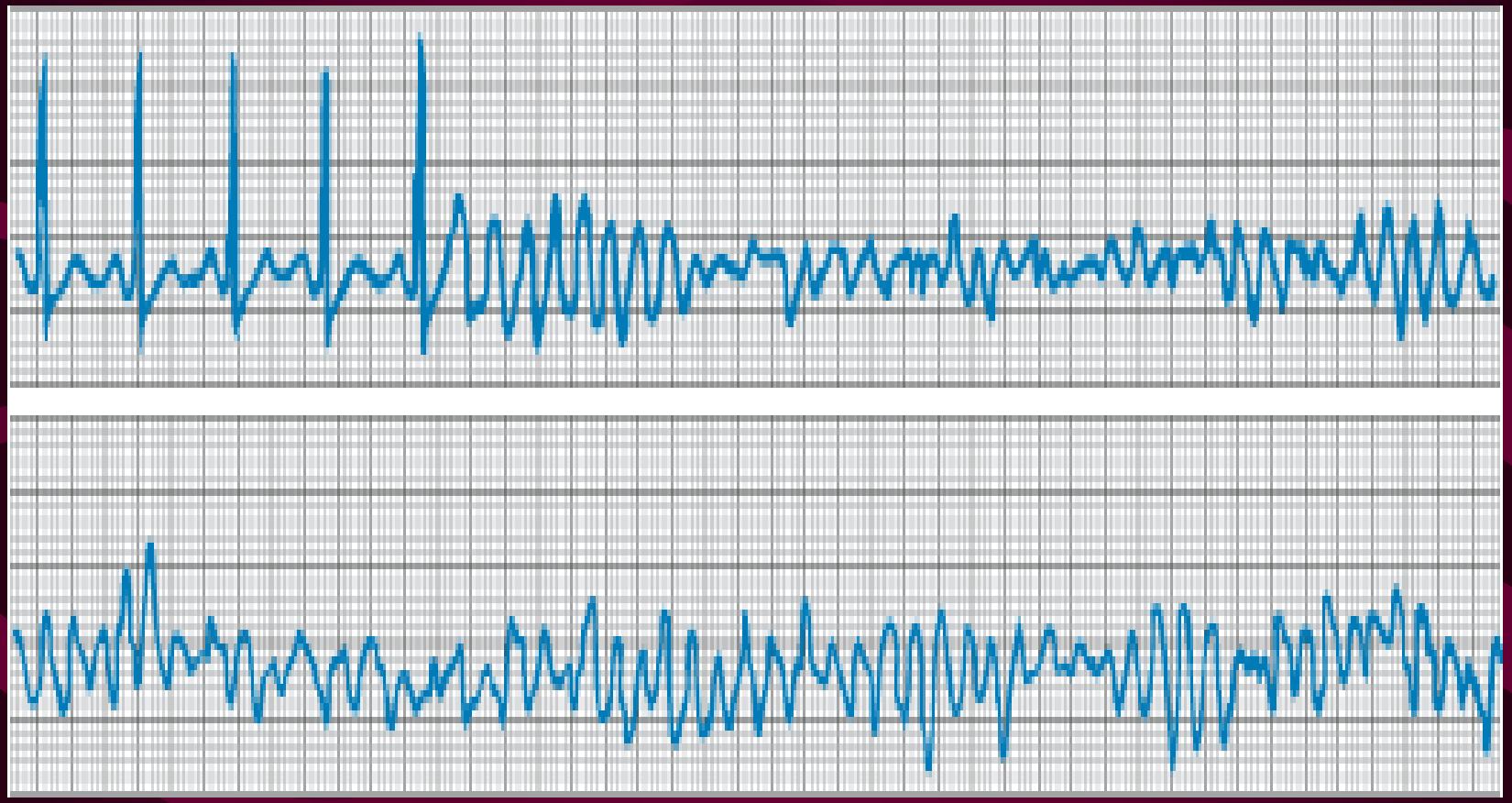
Results from the ventricle depolarizing at a rapid and erratic uncontrolled manner.

Ventricular Fibrillation



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

Ventricular fibrillation



PREEXCITATION (WPW) SYNDROME

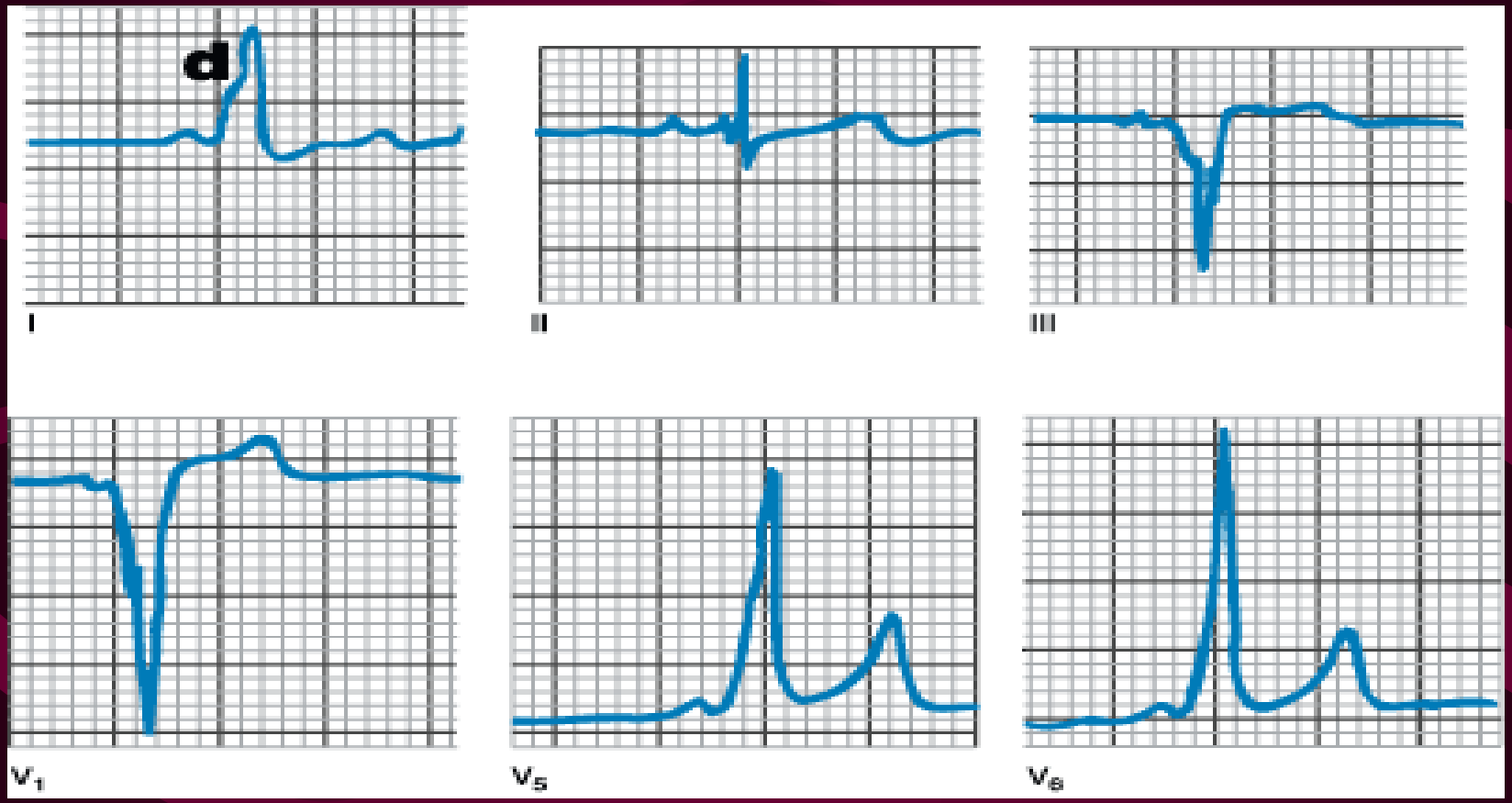
- Preexcitation is a condition characterized by an accessory pathway of conduction (called Kent, Jeims), which allows the heart to depolarize in an atypical sequence.
- It is congenital.
- The most common form of preexcitation is called Wolfe-Parkinson-White (WPW) syndrome, in which a direct atrioventricular connection (avoiding AV node) allows the ventricles to begin depolarization while the standard action potential is still traveling through the AV node.

WPW. ECG signs:

- short PR interval (<0.12 s),
- a slurred upstroke of the QRS complex (delta wave),
- a wide QRS complex.

This pattern results from an earlier and abnormal activation of the intraventricle septum.

WPW



Further Study on Your Own

Taking it a step further …..
Challenge yourself