Pediatric Arrhythmias

Arrhythmias are common in the general pediatric population

and frequently are an important clinical problem in patients with structural congenital heart disease

Arrhythmias
have a broad spectrum of clinical presentations, ranging from
no symptoms to sudden death. There is a wide variety of causes
of arrhythmias and an even broader range of treatments.

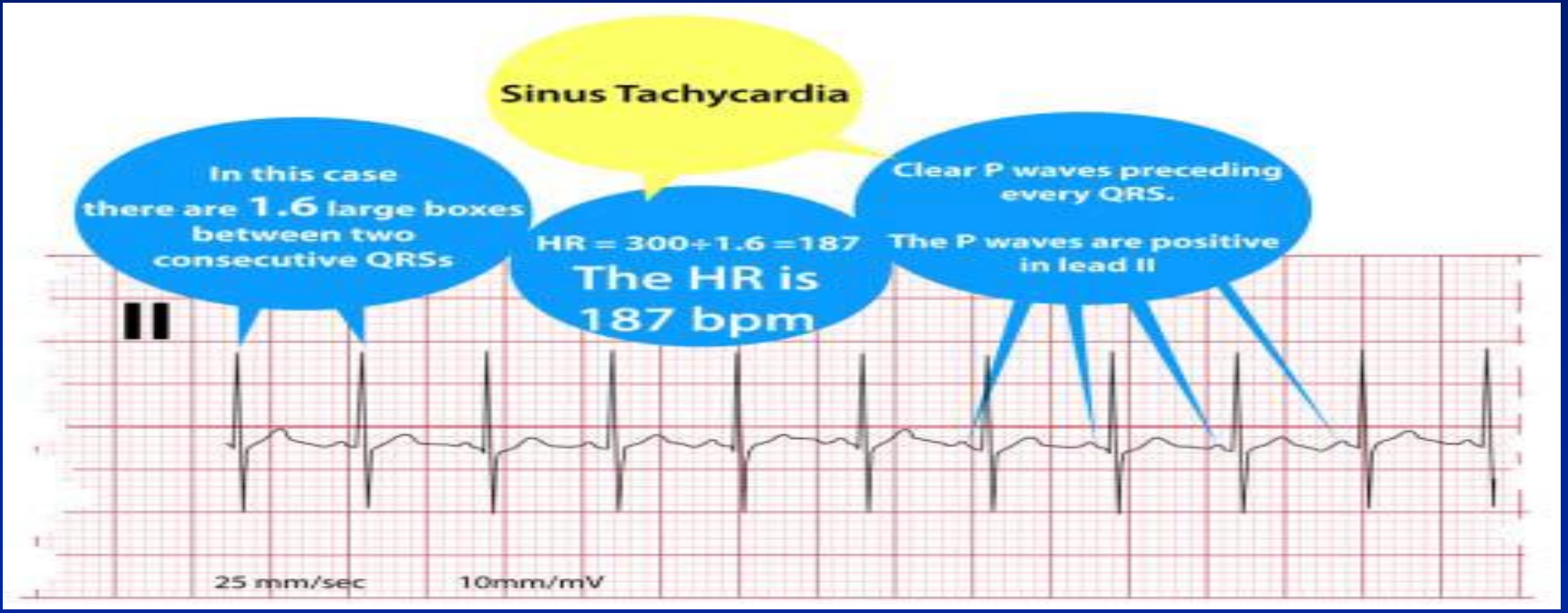
in correct pediatric ECG interpretation is to consider the clinical condition at the time of recording. Many of the non-cardiac diseases in children may have important effects on the normal ECG; therefore, abnormal ECGs do not always equal to the heart disease.

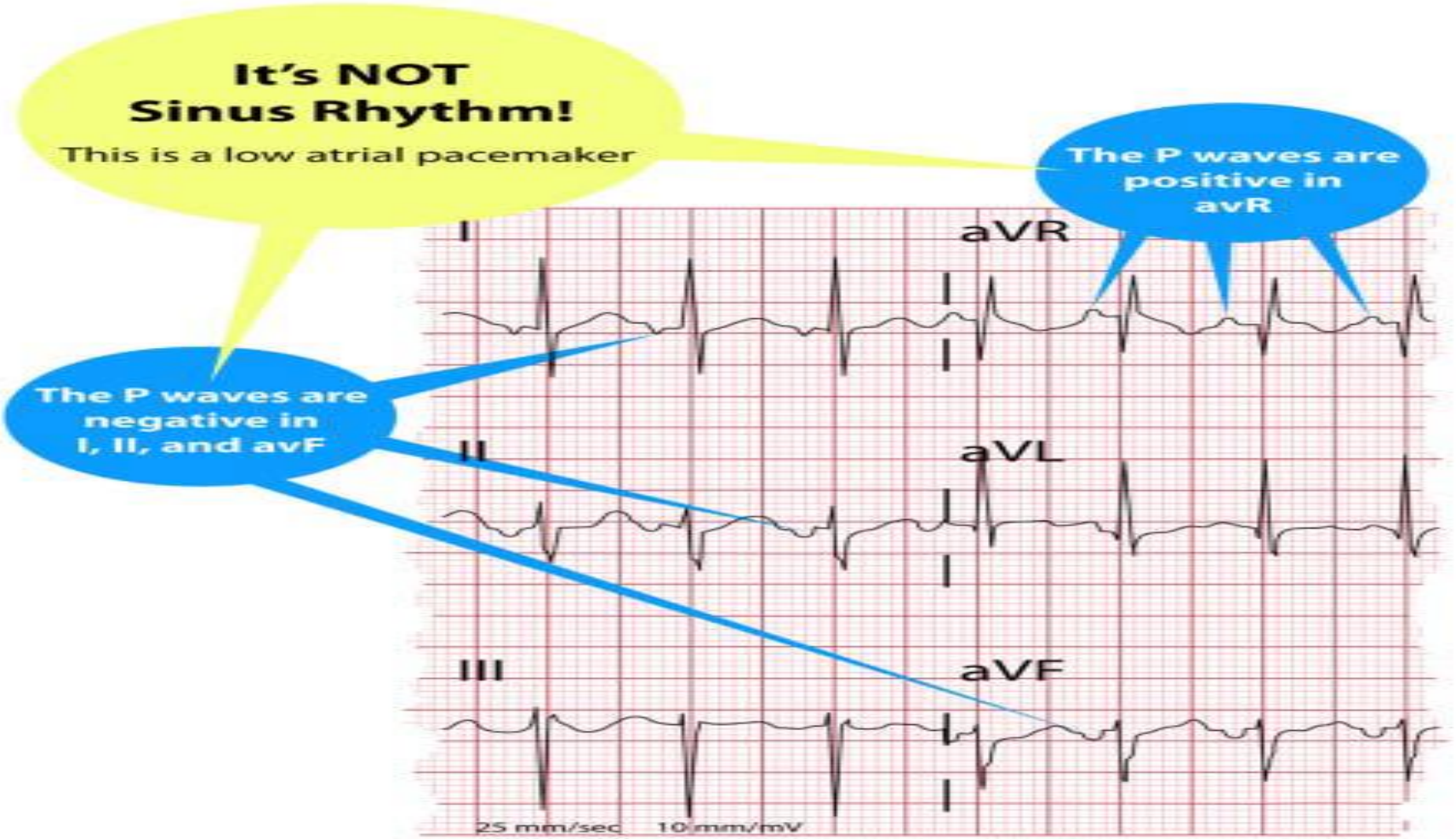
rhythm and during symptoms should be obtained
in children with suspected or
proven arrhythmia

Rhythm

For determination of heart rhythm, it is important to determine the exact origin of cardiac impulses and correlation of each P wave with next QRS copmplex

Sinus tachycardia” is a fast sinus rhythm that is consistent with anxiety, crying, fever, and occasionally hyperthyroidism




It's NOT
Sinus Rhythm!
This is a low atrial pacemaker
The P waves are positive in avR
The P waves are negative in I, II, and avF
I
aVR
II
aVL
III
aVF
25 mm/sec
10 mm/mV

Heart rate

Neonatal heart rate varies between 150 to 230 beats/min especially during crying. The heart rate reaches a peak between one and two months of life and then decreases gradually until six months

Normal resting heart rates

- Newborn: 110 - 150 bpm
- 2 years: 85 - 125 bpm
- 4 years: 75 - 115 bpm
- > 6 years: 60 - 100 bpm
- Adult: 50 - 100 bpm

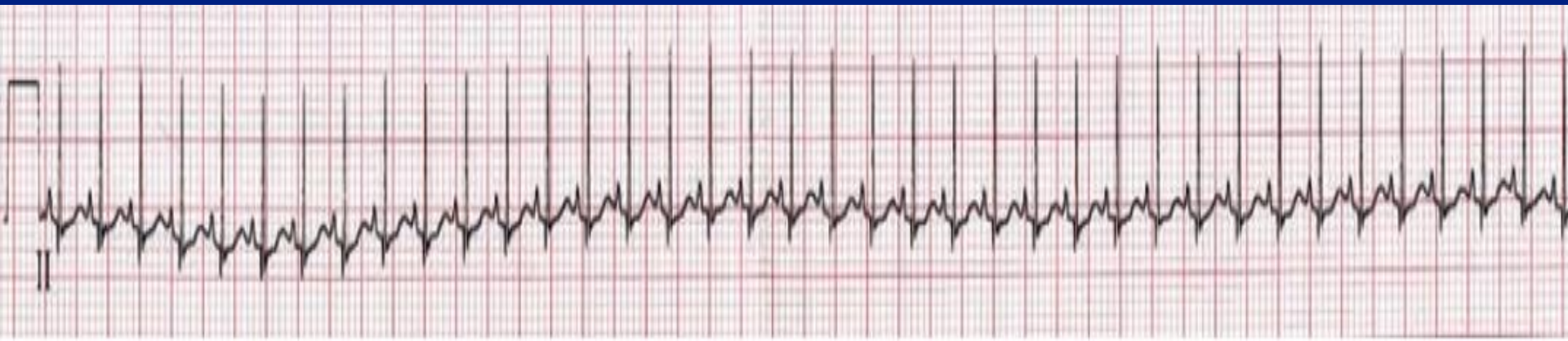
Age-Specific Range of Normal Heart Rates in Children (defined as the 2nd through 98th percentile values

0—1 d	94—155
1—3 d	92—158
3—7 d	90—166
7—30 d	107—182
1—3 mo	120—179
3—6 mo	106—186
6—12 mo	108—168
1—3 yr	90—152
3—5 yr	73—137
5—8 yr	64—133
8—12 yr	63—130

Age	Resting Heart Rate (bpm)
Birth-1 wk	90–160
1 wk–1 yr	100–170
1–2 y	80–150
3–7 y	70–135
7–10 y	65–130
11–15 y	60–120

The third and most rare mechanism of arrhythmogenesis is

This recording in is from a sick neonate (with a normal heart) with septicemia and shows a sinus rhythm at 230/min. This is about the maximum possible
rate for sinus tachycardia, even in the newborn, and rates above 200/min are rare



“Sinus tachycardia” is a fast sinus rhythm that is consistent with anxiety, crying, fever, and occasionally hyperthyroidism

MECHANISMS OF ARRHYTHMIAS

The mechanisms most often responsible for cardiac arrhythmias are (1) failure of impulse formation, (2) conduction block, (3) reentrant excitation, (4) enhanced automaticity, and (5) triggered activity Reentry and block are abnormalities of impulse conduction; failure of impulse formation, enhanced automaticity, and triggered arrhythmias are abnormalities of impulse generation

PEDIATRIC CLINICAL ARRHYTHMIAS

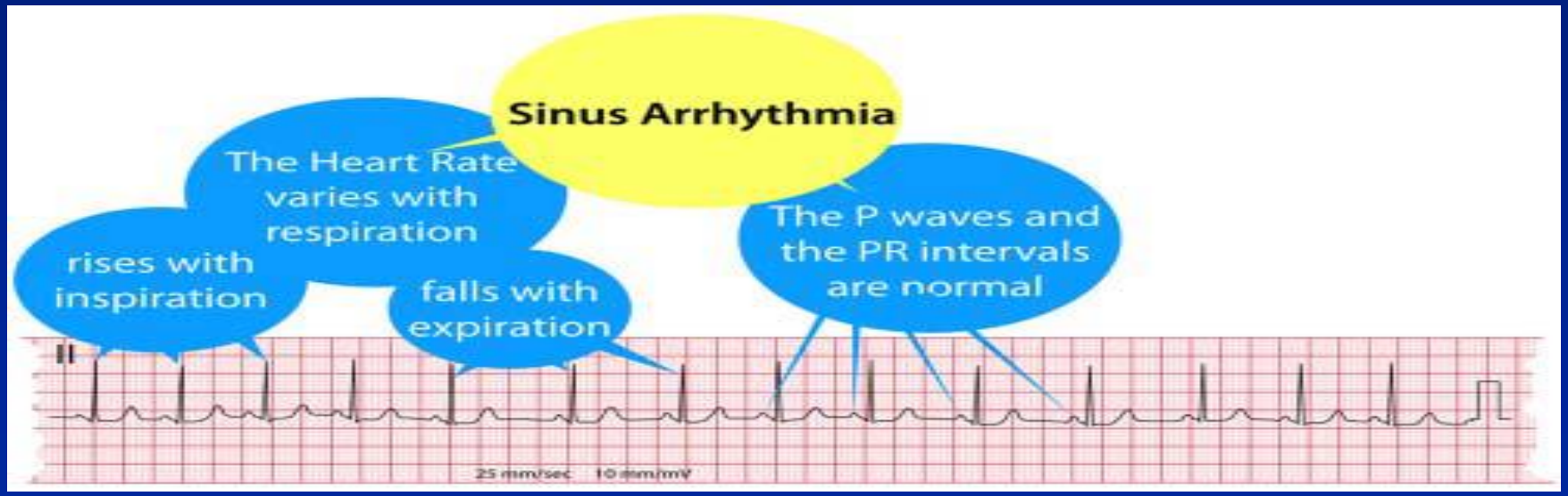
- Bradyarrhythmias
  - *Sinus Bradycardia*
  - *Sinus Arrest*
  - *Sinus Node Exit Block*
  - *Atrioventricular Block*
- Tachyarrhythmias
  - *Supraventricular Tachycardias*
    - Sinus Tachycardia
    - Sinus Node Reentrant Tachycardia
    - Atrial Flutter
    - Atrial Fibrillation
    - Atrial Ectopic Tachycardia
    - Junctional Ectopic Tachycardia
    - AV Nodal Reentrant Tachycardia
    - AV Reciprocating Tachycardia
    - Permanent Junctional Reciprocating Tachycardia
  - *Ventricular Tachyarrhythmias*
    - Premature Ventricular Contractions
    - Ventricular Tachycardia
    - Torsade de Pointes
    - Ventricular Fibrillation

Pediatric dysrhythmias

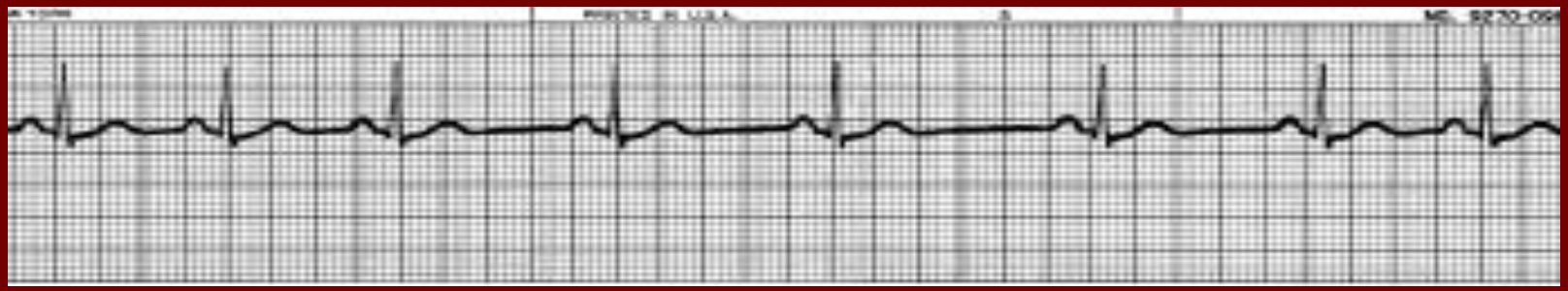
Treatment not required	Treatment is required
Sinus arrhythmia	Supraventricular tachycardia
Wandering atrial pacemaker	
Isolated premature atrial contractions	
Isolated premature ventricular contractions	Ventricular tachycardia
First degree AV block	Third degree AV block with symptoms

Reproduced from Zitelli's Atlas of Pediatric physical diagnosis, 2007, pg 140.

Sinus arrhythmia” is a normal variation in sinus rhythm that occurs with respiration. The heart rate rises and falls with inspiration and expiration. The variation is more pronounced in young children and less pronounced in infants and adolescents

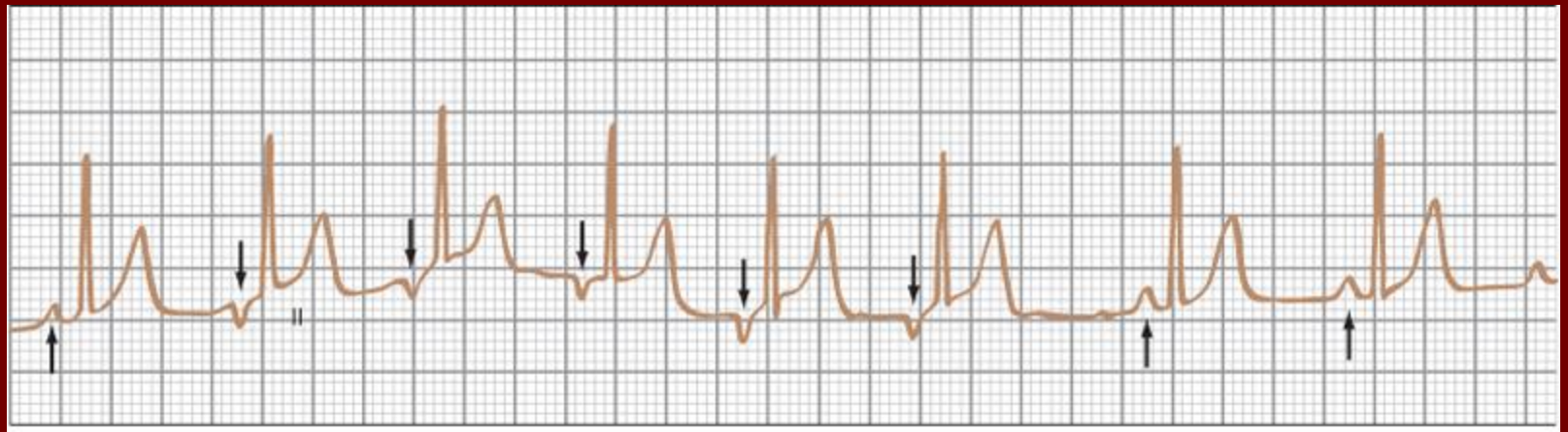


Sinus arrhythmia



Wandering atrial pacemaker

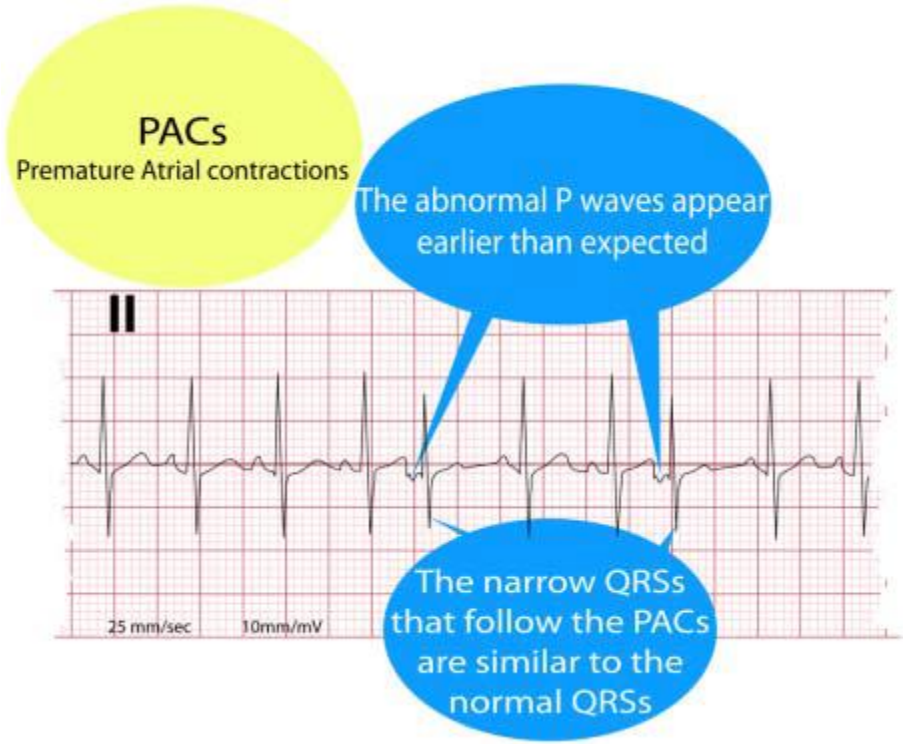
- Atrial pacemaker shifts from sinus node to another atrial site
- Normal variant, irregular rhythm



Isolated PAC's

- Premature atrial contractions
- Benign in absence of underlying heart dz
- Common in newborn period
- Early p wave, sometimes with different morphology than a sinus p wave
- Can be either:
  - Not conducted to ventricle, apparent pause
  - Conducted to ventricle with aberrant or widened QRS complex (careful not to mix up with PVC's)

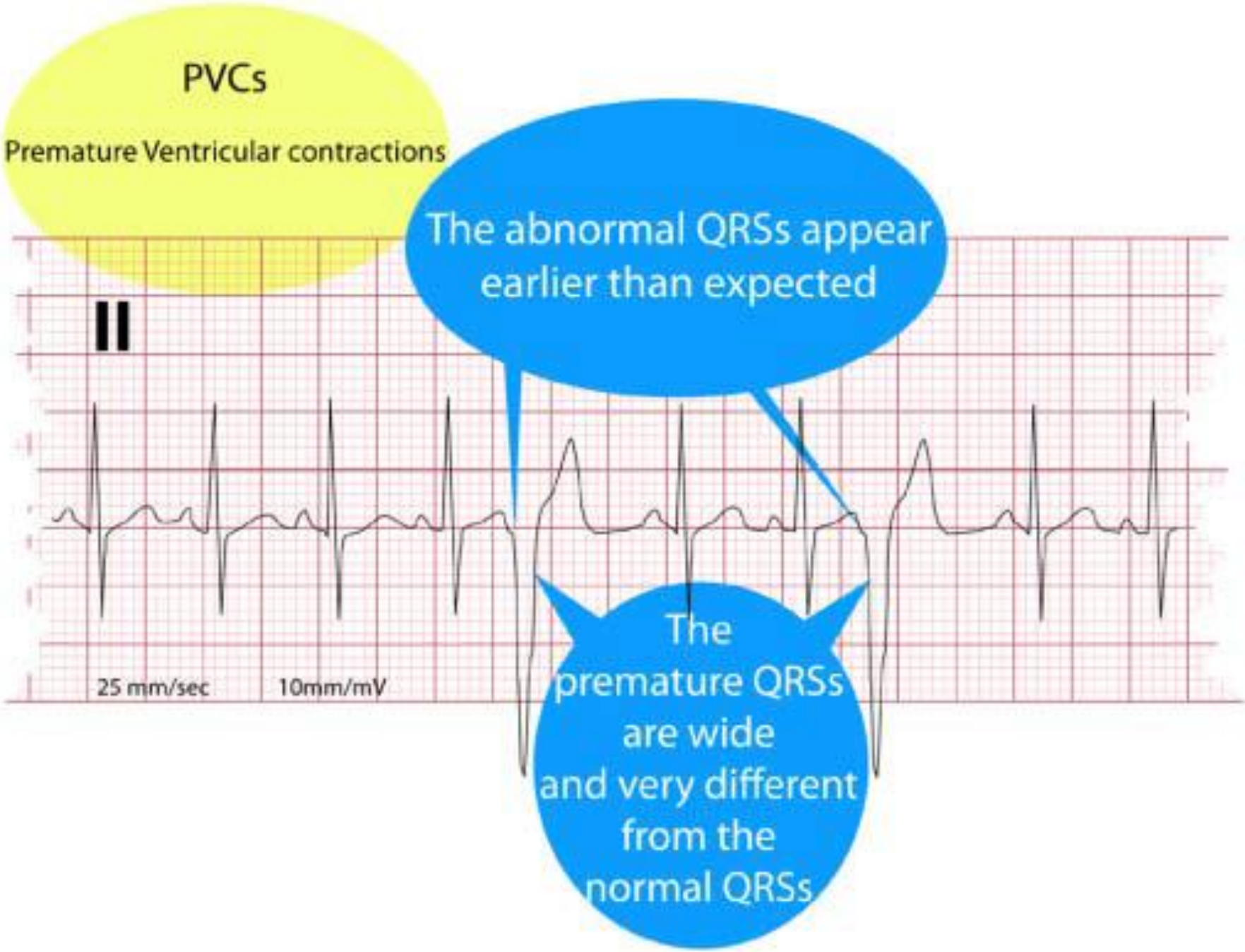
If there are ectopics, describe them:
a. Frequent or infrequent narrow QRS ectopics usually premature atrial contractions
(PACs).


PACs
Premature Atrial contractions
The abnormal P waves appear earlier than expected
II
25 mm/sec
10mm/mV
The narrow QRSs that follow the PACs are similar to the normal QRSs

Isolated PAC's

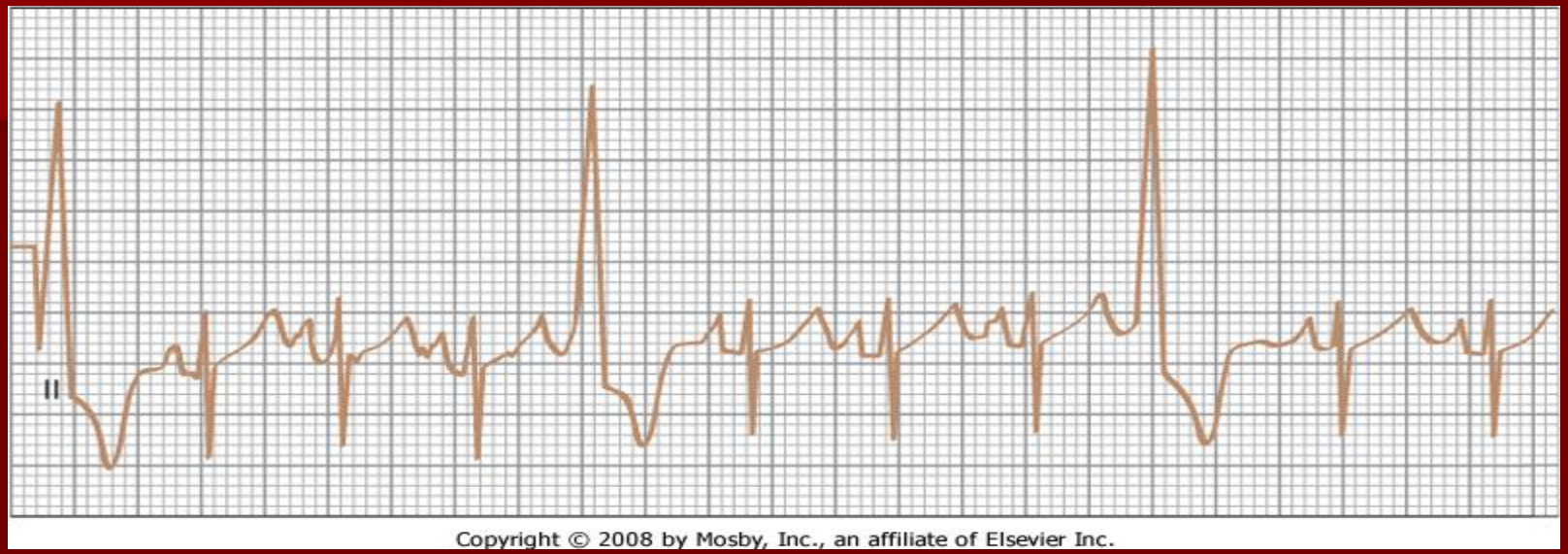
Premature Ventricular Contractions (PVC's)

- Not very commonly seen in children
- Incidence of 0.3 to 2.2 %
- Early, wide QRS complexes
- T waves in opposite direction of QRS
- Unifocal PVC's are most encountered type
- Bigeminy, sinus beat followed by PVC, repeating as a pattern, also frequently seen


PVCs
Premature Ventricular contractions
The abnormal QRSs appear earlier than expected
II
25 mm/sec
10mm/mV
The premature QRSs are wide and very different from the normal QRSs

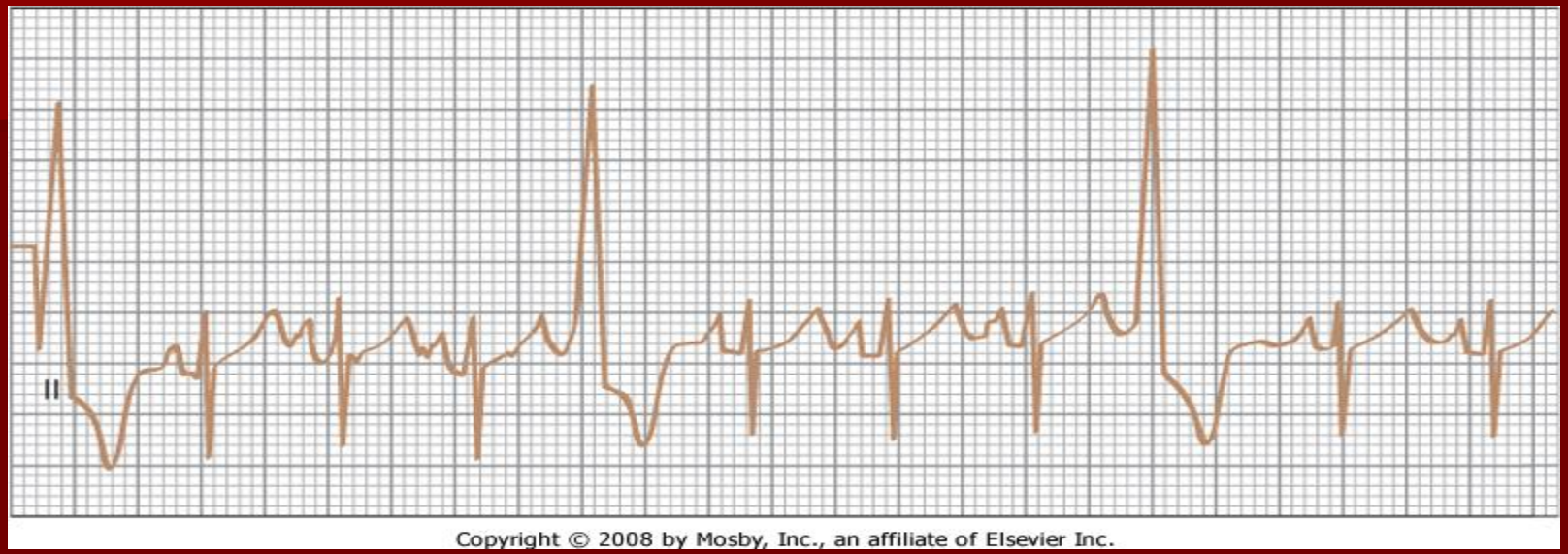
Frequent or infrequent wide QRS ectopics
usually premature ventricular contractions (PVCs).

PVC's



- If unifocal, disappear with exercise, and associated with structurally and functionally normal heart, then considered benign, no therapy needed

PVC's

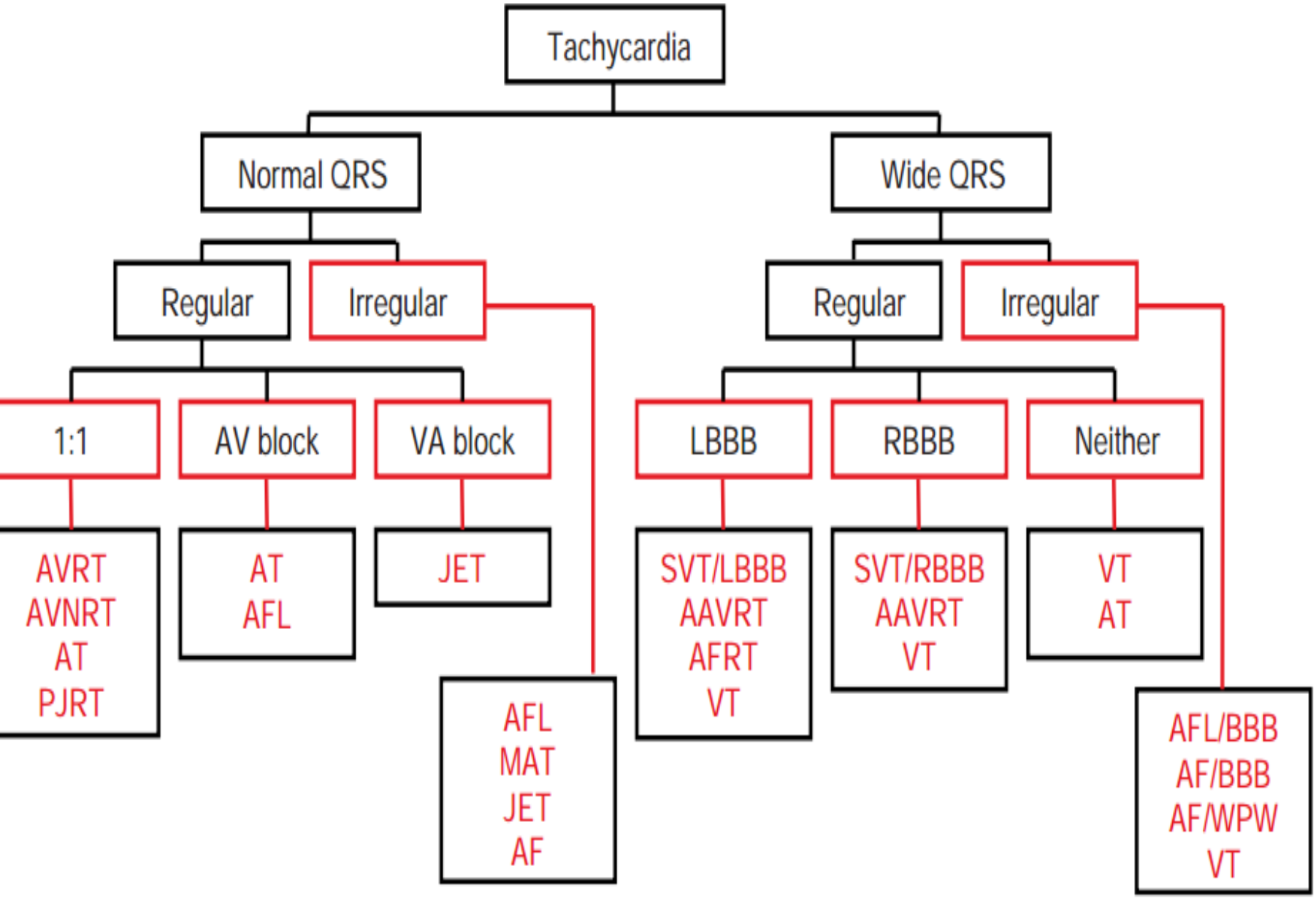


- If unifocal, disappear with exercise, and associated with structurally and functionally normal heart, then considered benign, no therapy needed

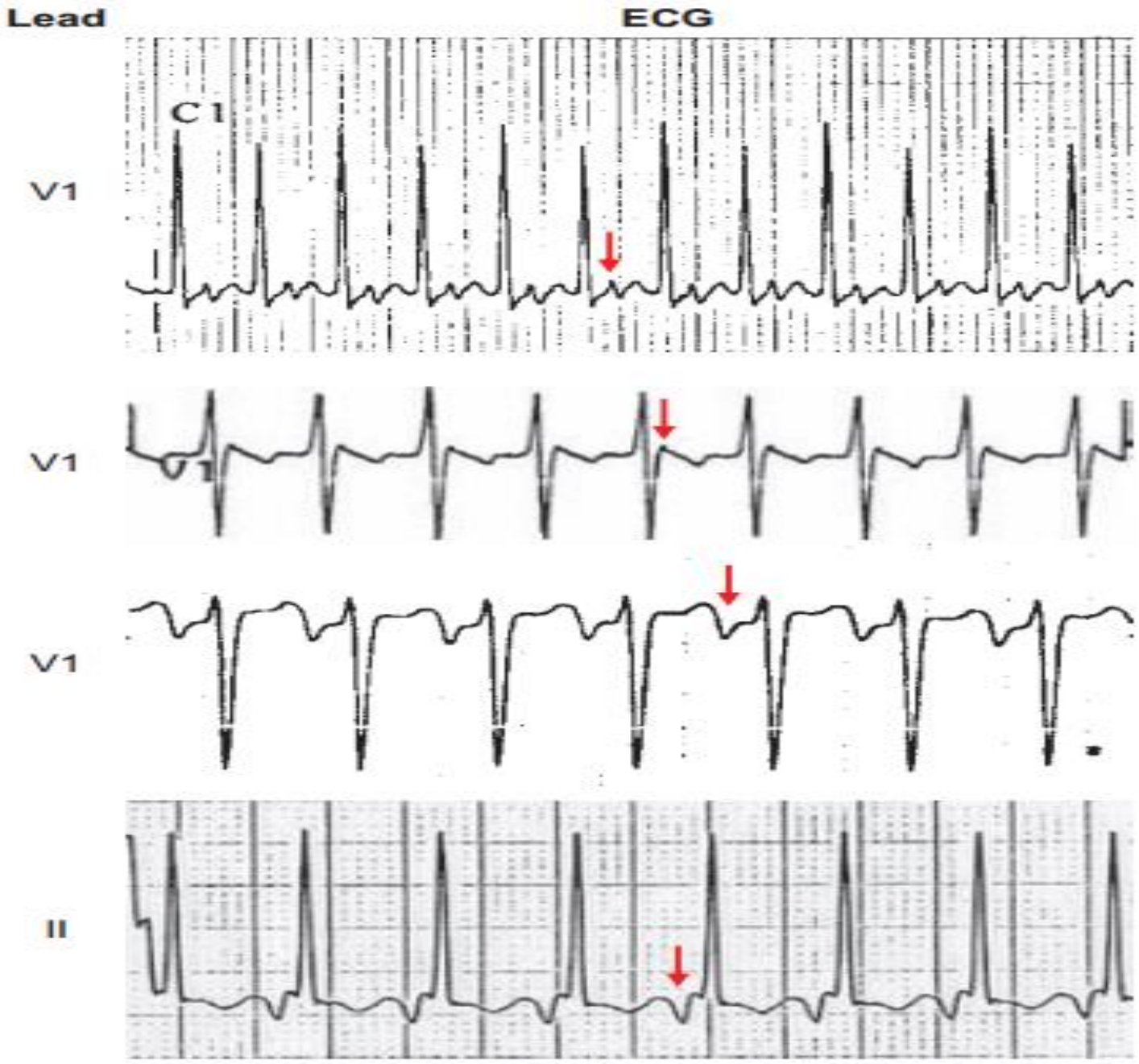
PVC's evaluation

- 12 lead EKG, Echocardiogram
- Perhaps Holter monitoring
- Brief exercise in office to see if ectopy suppressed or more frequent
- Multifocal or paired PVC's more worrisome
- Medications usually not needed
- Advise patients to avoid caffeine and other stimulants

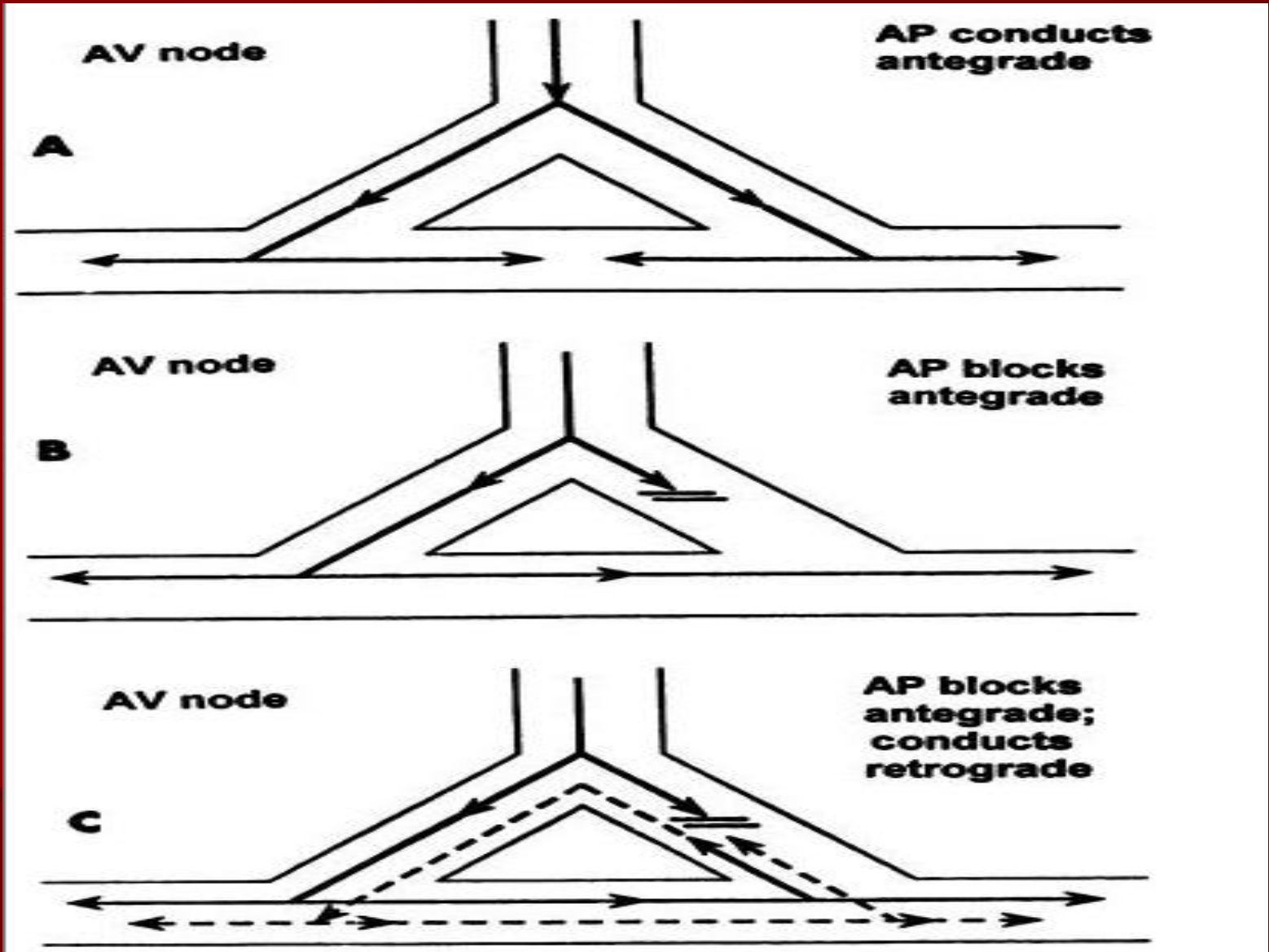
tachyarrhythmias

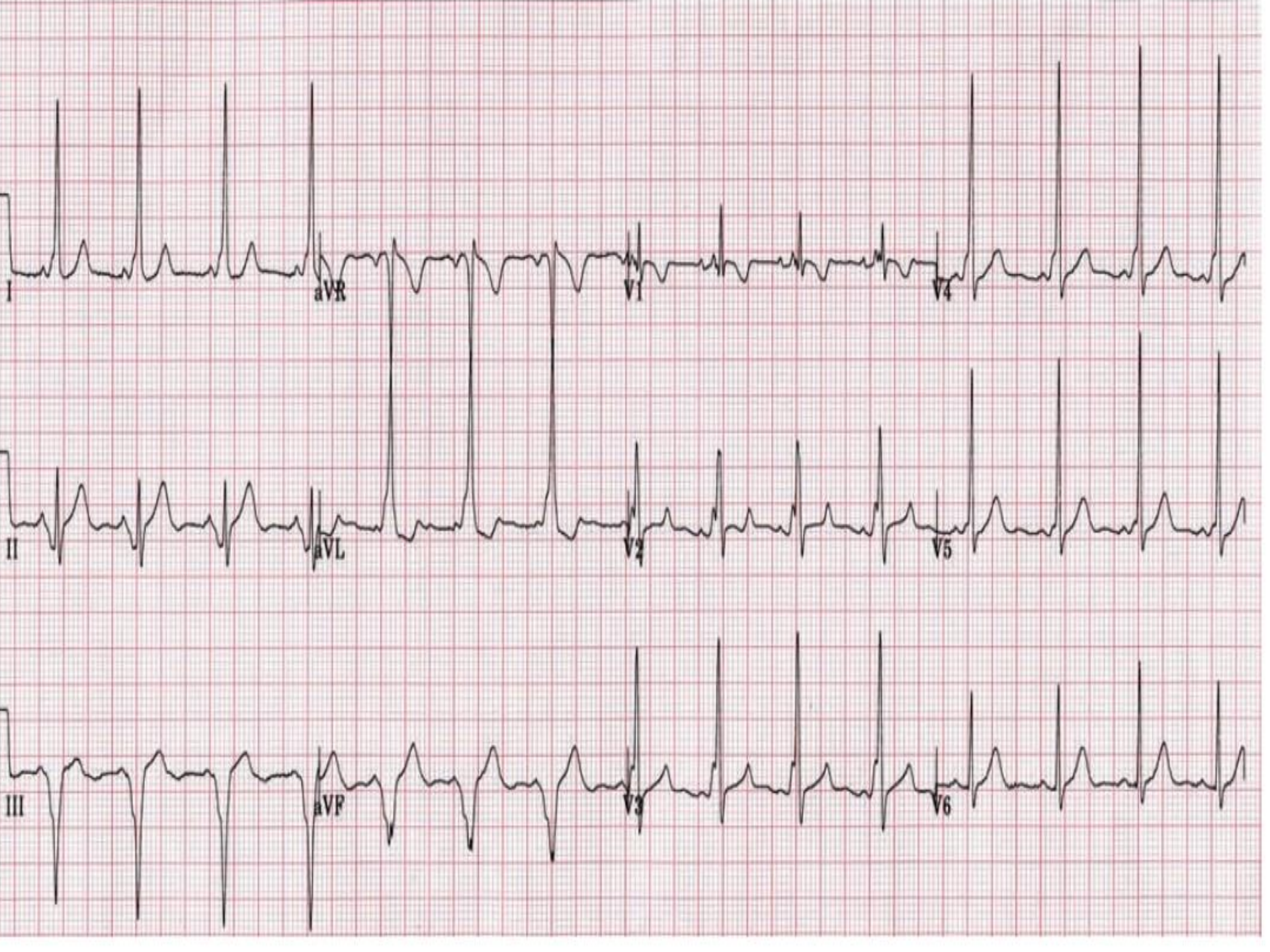

Tachycardia
Normal QRS
Wide QRS
Regular
Irregular
Regular
Irregular
1:1
AV block
VA block
LBBB
RBBB
Neither
AVRT
AVNRT
AT
PJRT
AT
AFL
JET
AFL
MAT
JET
AF
SVT/LBBB
AAVRT
AFRT
VT
SVT/RBBB
AAVRT
VT
VT
AT
AFL/BBB
AF/BBB
AF/WPW
VT

Regular tachycardia with a normal QRS and 1:1 AV relationship


Lead
ECG
V1
C1
V1
V1
II

Reentrant excitation is the most common cause of tachyarrhythmias



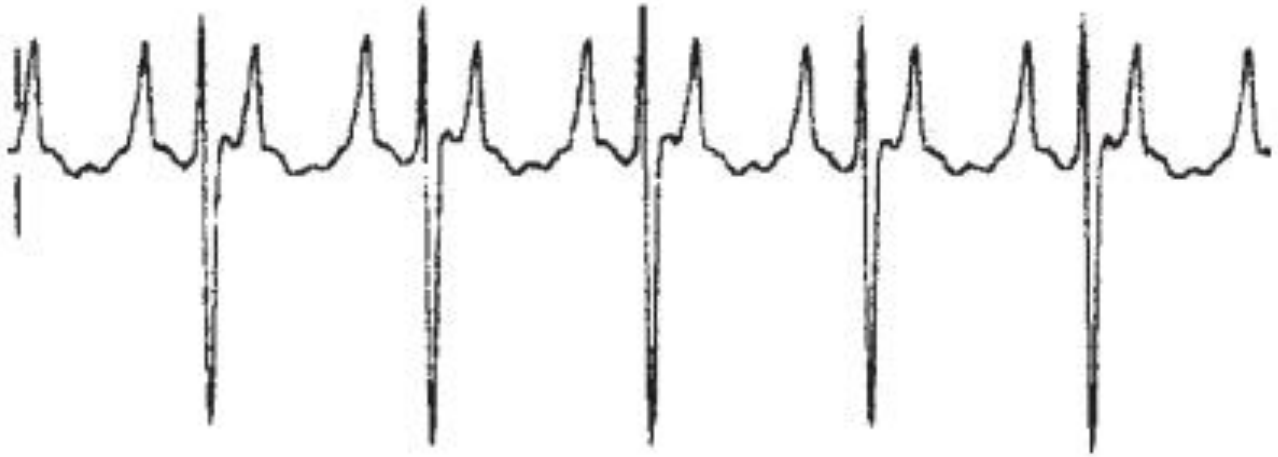

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Regular tachycardia with a normal QRS and AV block

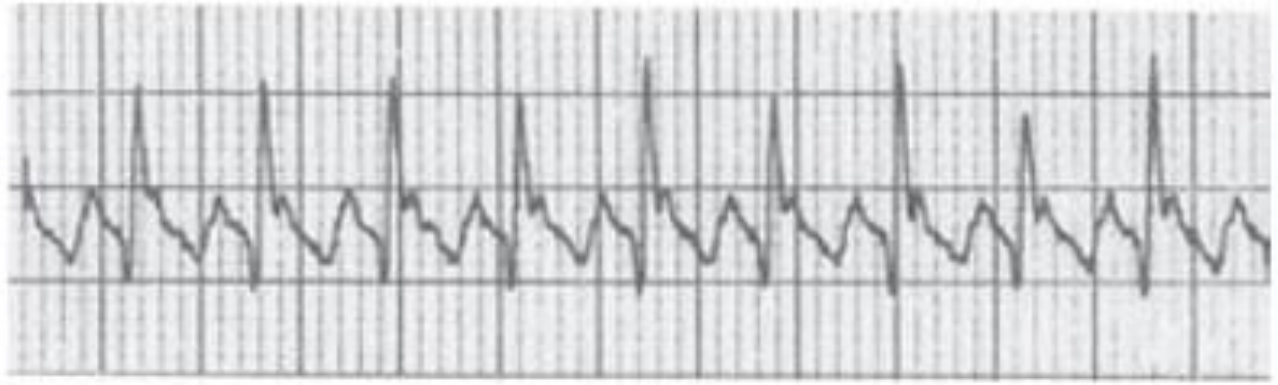
Lead

ECG

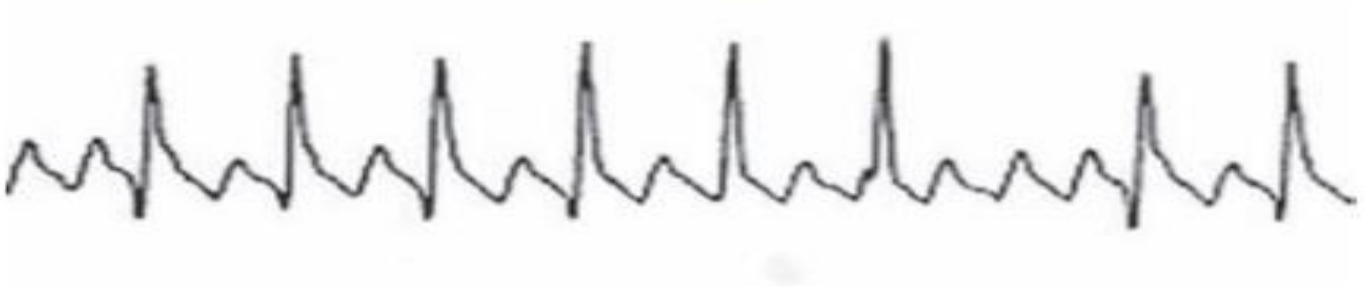
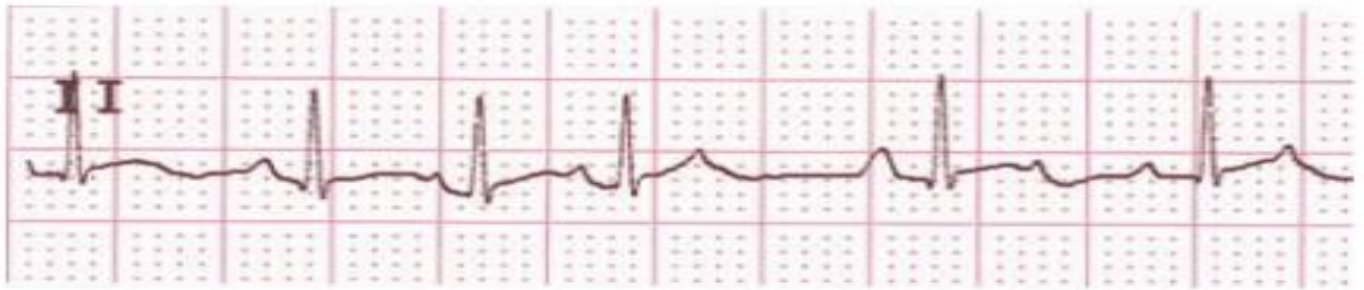
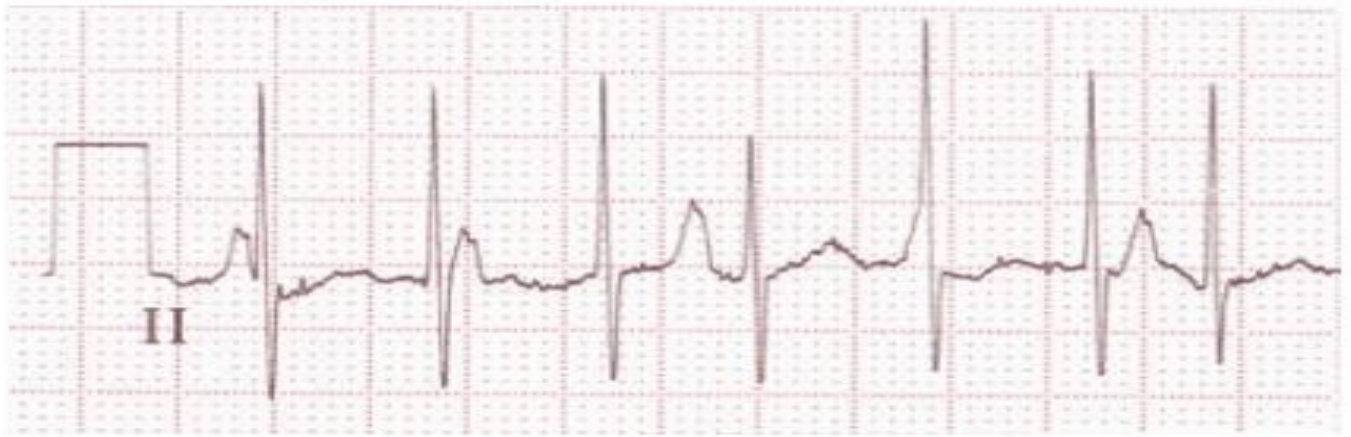
V1



III



Irregular tachycardia with a normal QRS

Lead	ECG
III	
II	
III	
II	

Wide QRS complex tachycardia

It is best to consider the morphology and regularity of the QRS complexes before looking for P waves

if dissociated P waves are obvious, the diagnosis is clearly ventricular tachycardia

Wide QRS complex tachycardia

QRS analysis in wide complex tachycardia assesses whether the pattern more

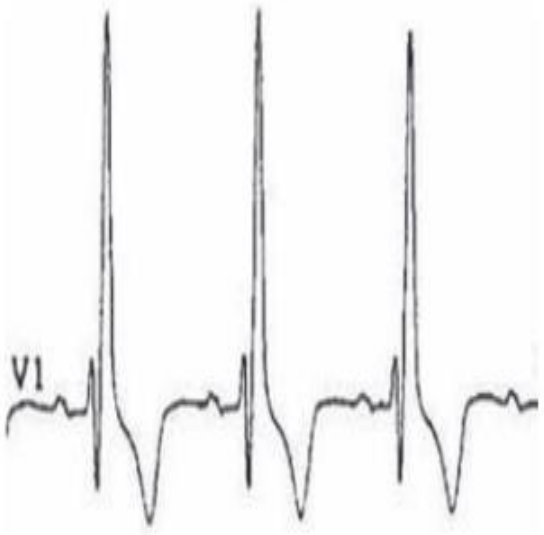
closely resembles right or left bundle branch block or neither

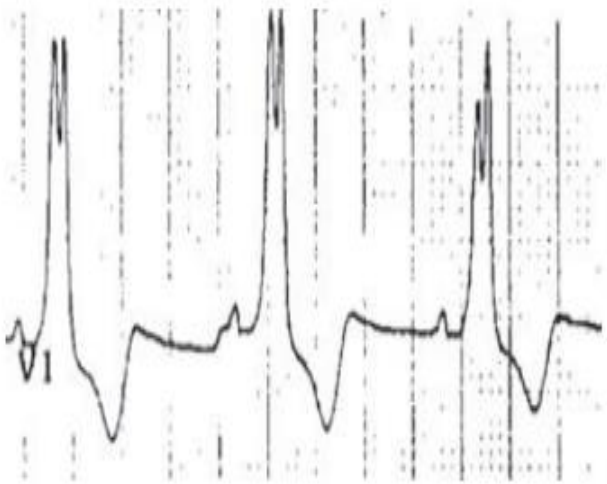
RBBB

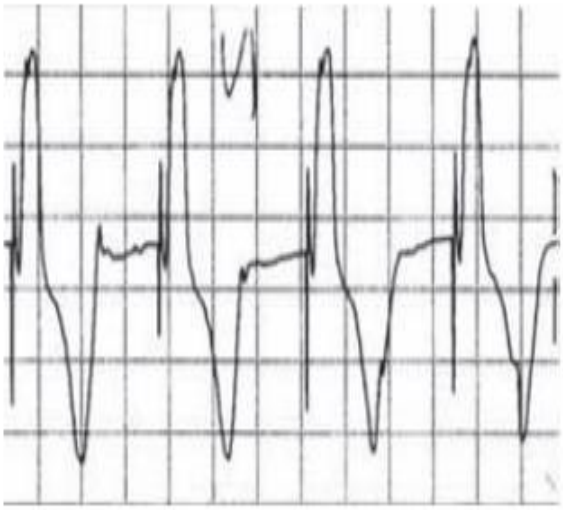
Left sided WPW

LV pacing

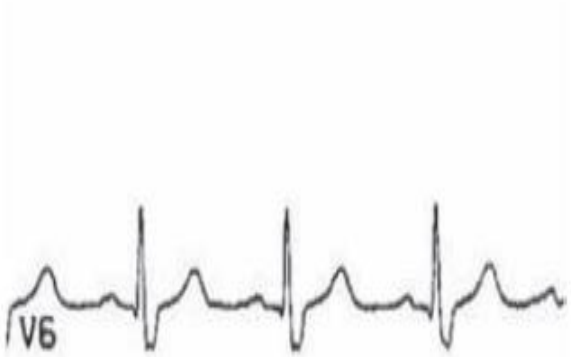
V1

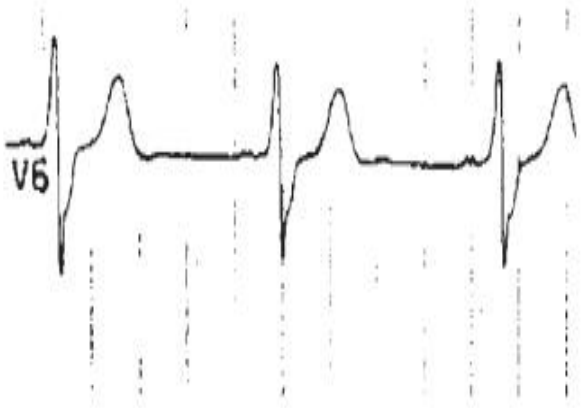


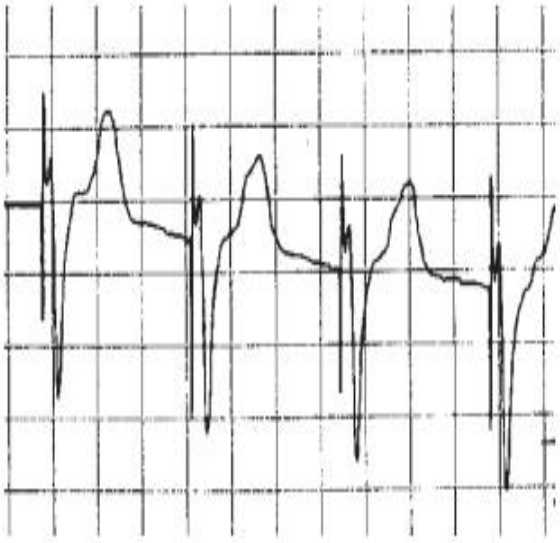




V6





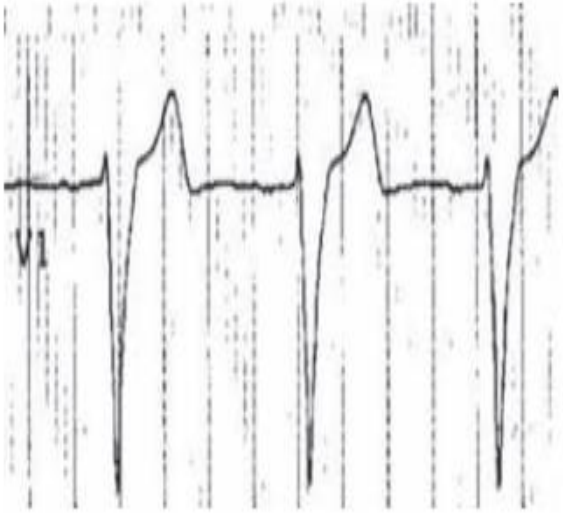


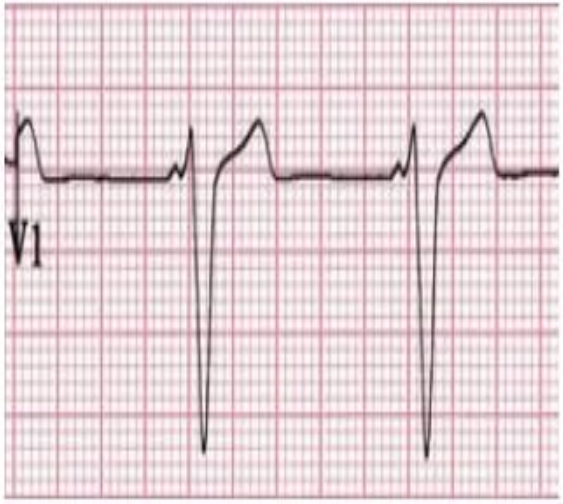
LBBB

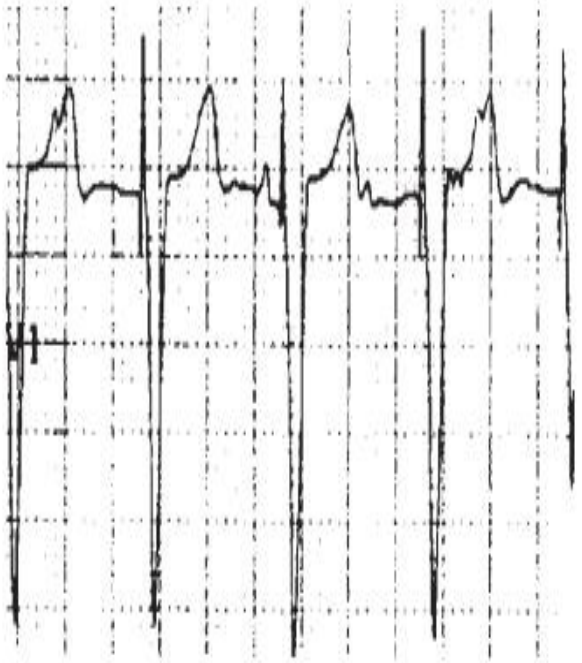
Right sided WPW

RV pacing

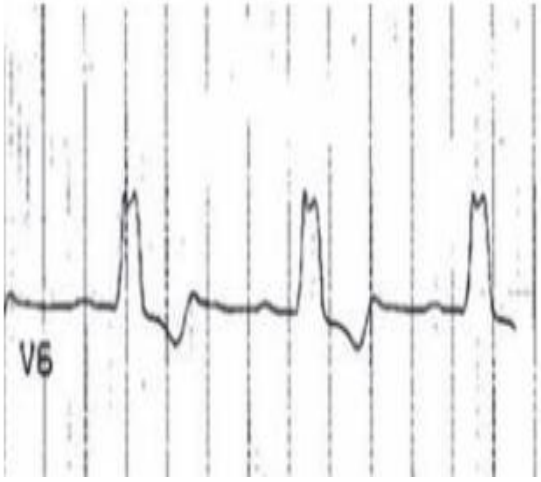
V1

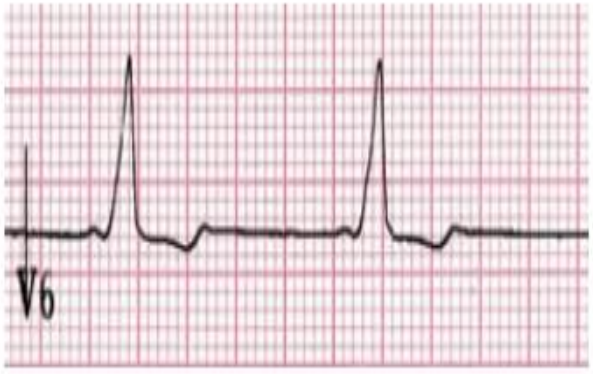


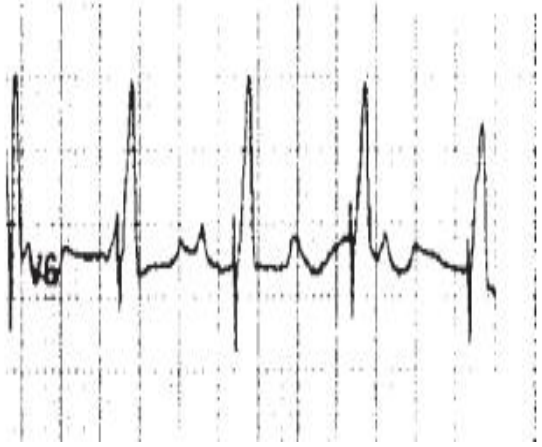




V6

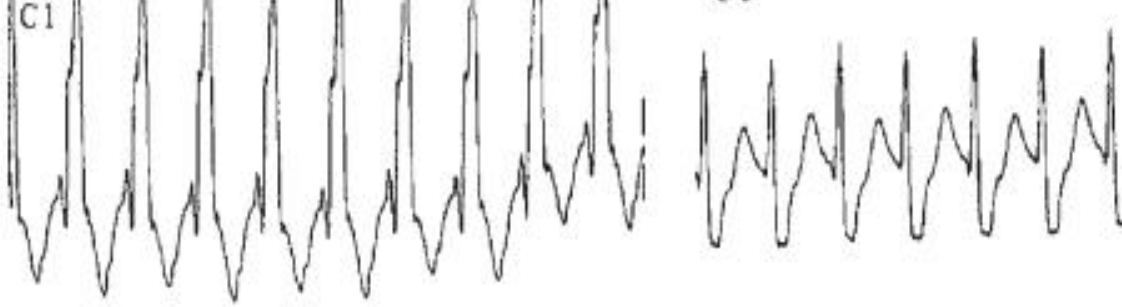




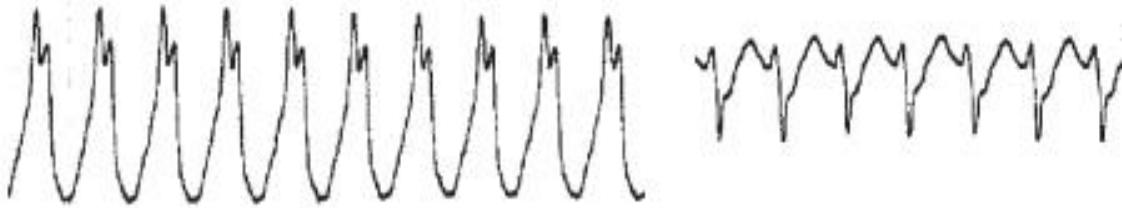


Regular tachycardia with a wide QRS with right bundle
branch block morphology

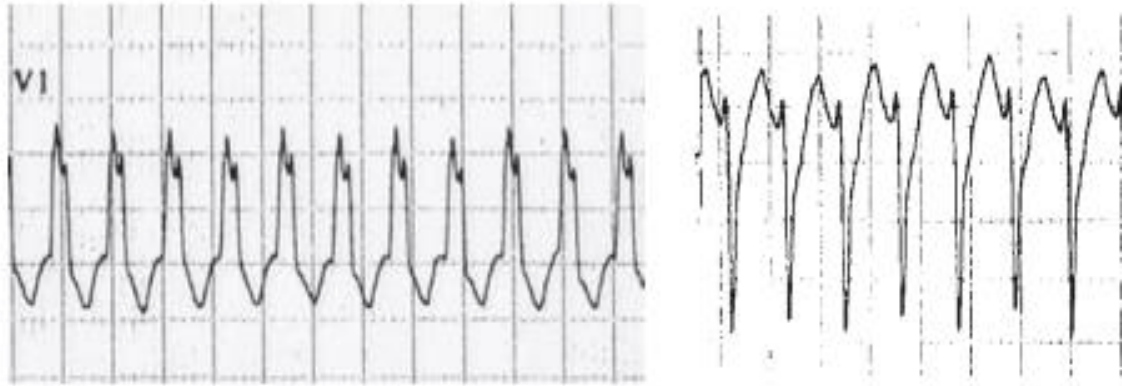
"SVT" with RBBB



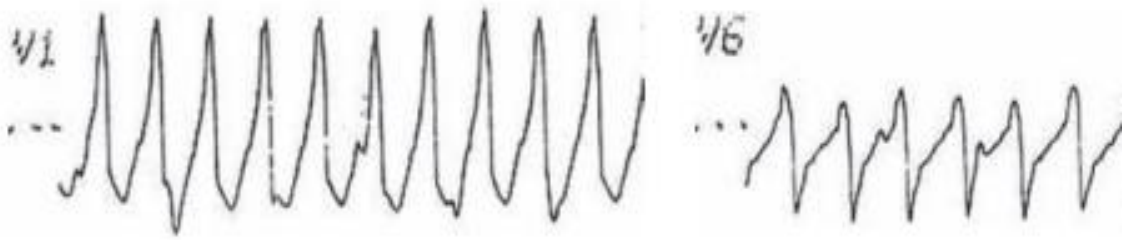
Antidromic AV re-entry tachycardia



Ventricular tachycardia (from LV)

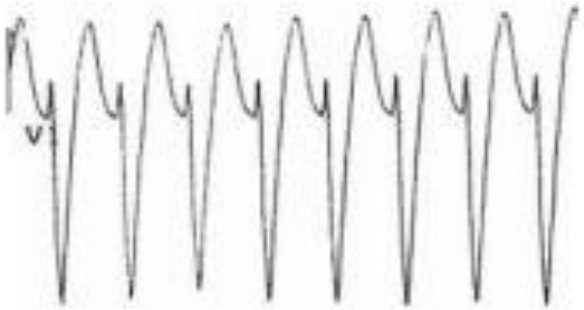


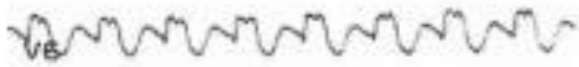
Ventricular tachycardia (from LV)



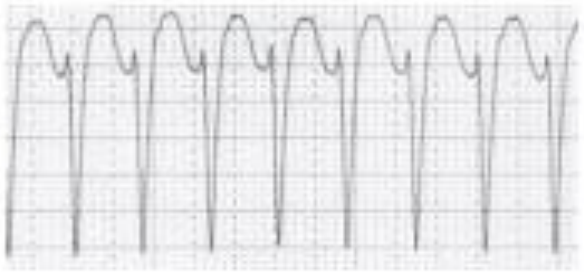
Regular tachycardia with a wide QRS with left bundle branch block morphology

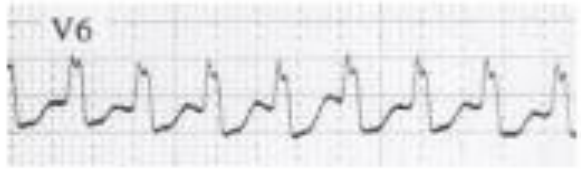
"SVT" with LBBB



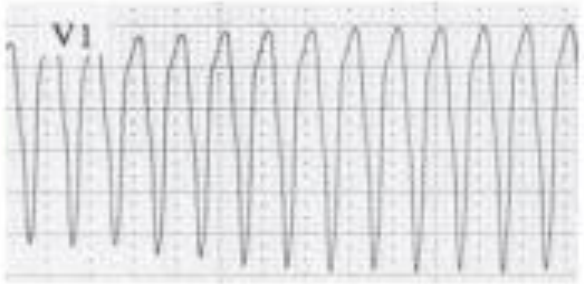


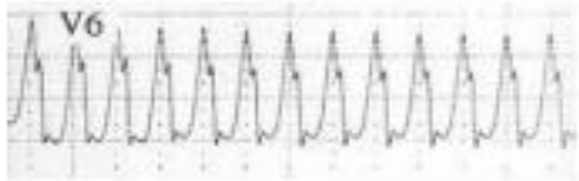
Atriofascicular re-entry tachycardia



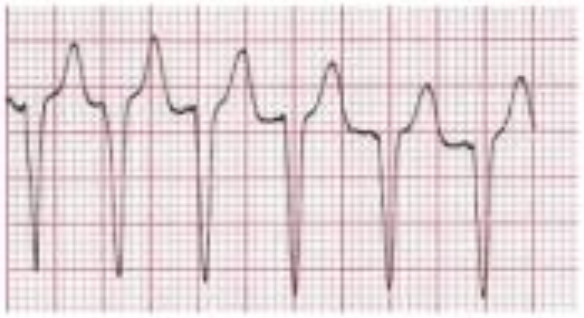


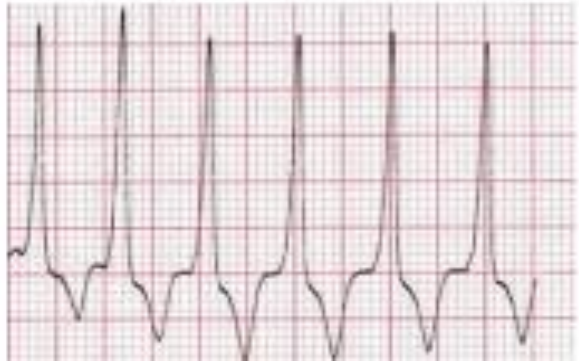
Antidromic AV re-entry tachycardia (right sided pathway)



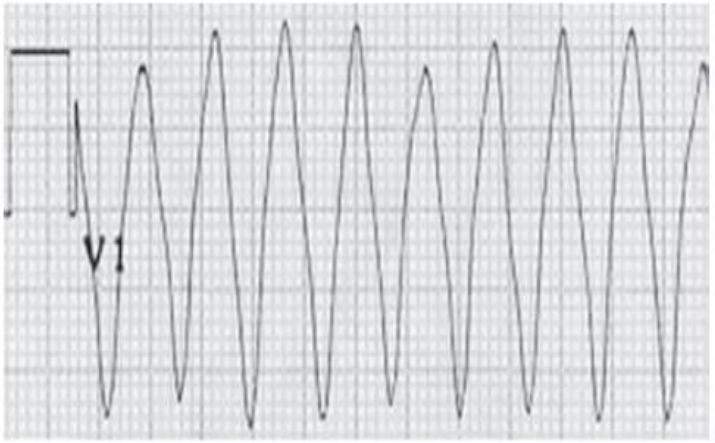
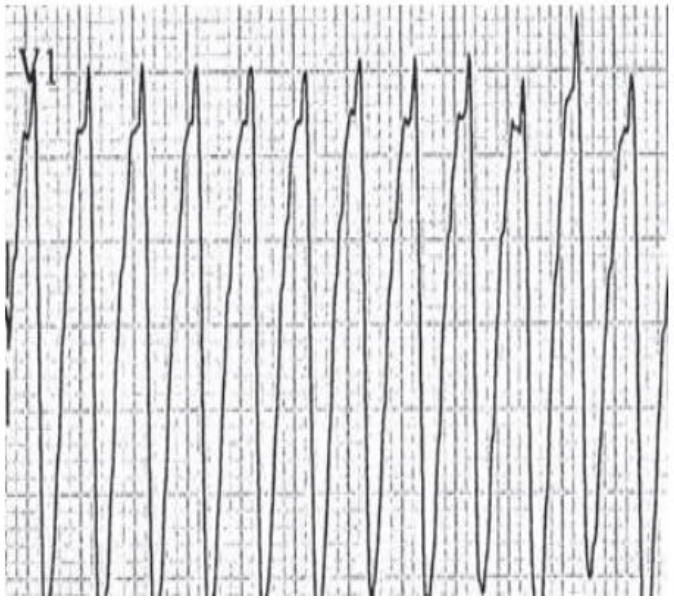


Ventricular tachycardia (from RV)

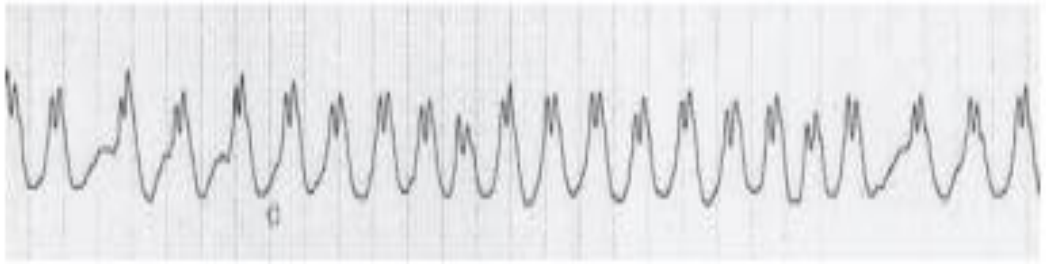
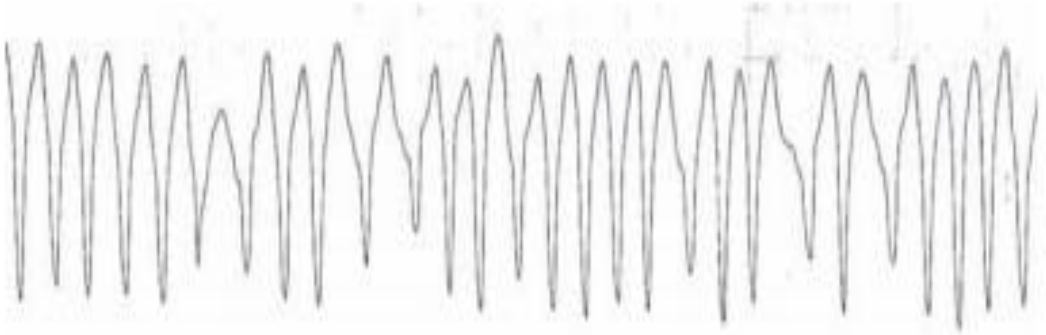
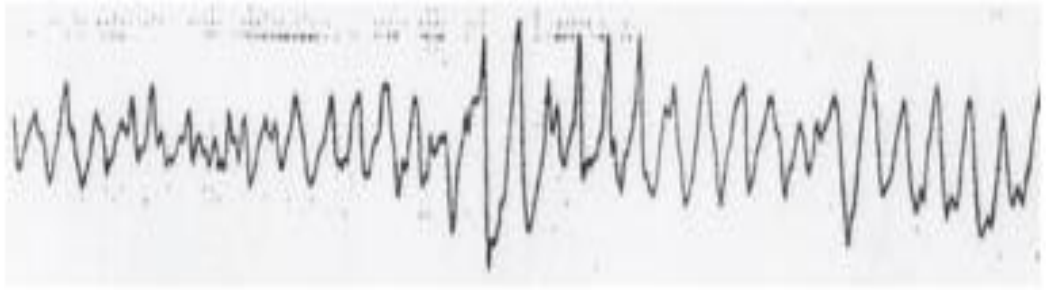




Regular tachycardia with a wide QRS and neither
RBBB
nor LBBB morphology

Diagnosis	Lead	ECG
Ventricular tachycardia	V1	
Atrial flutter with BBB (especially drug effect)	V1	

Irregular tachycardia with a wide QRS

Diagnosis	Lead	ECG
Atrial fibrillation with BBB	II	
Atrial fibrillation in WPW syndrome	aVF	
Polymorphic ventricular tachycardia (torsades de pointes)	Holter	

Changing QRS morphology in tachycardia

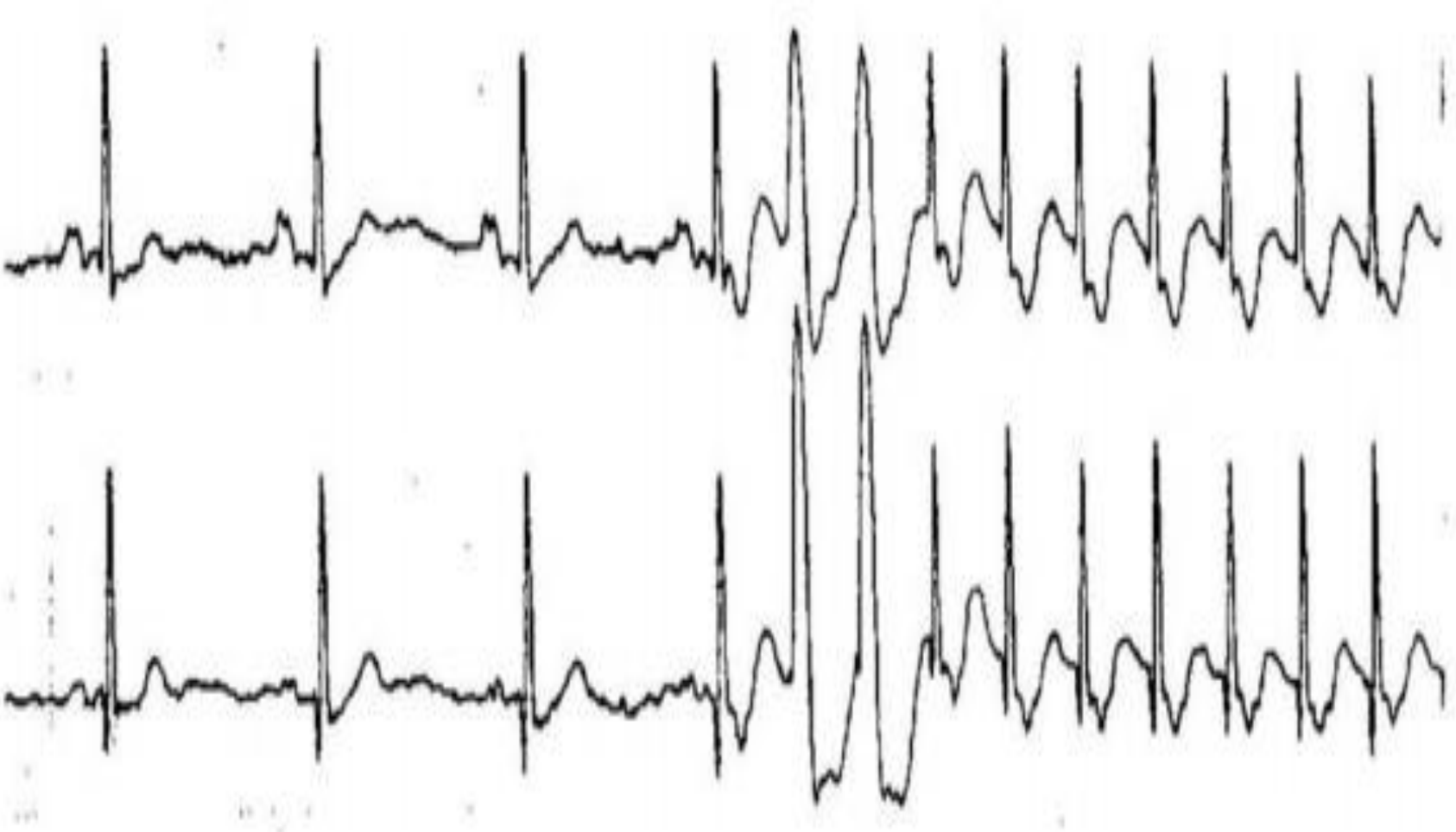


Figure 5.11

Adenosine in the diagnosis of tachycardias

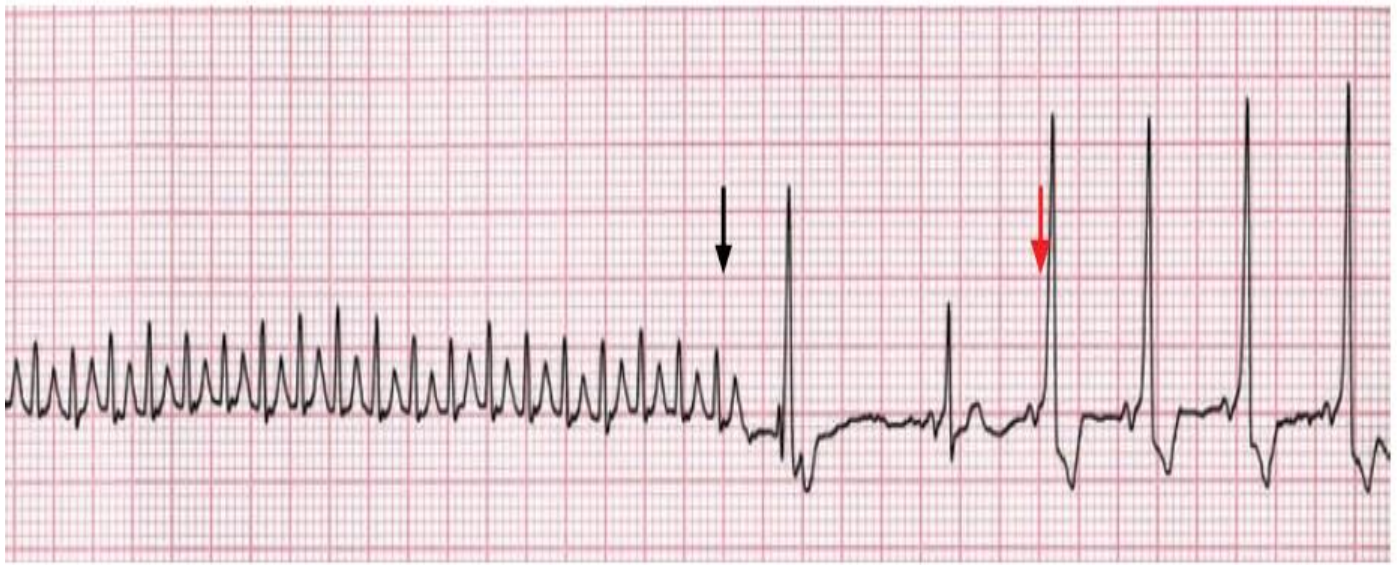
Intravenous adenosine is the first-line treatment for any sustained regular tachycardia in infancy or childhood, with either a normal or a wide QRS. The main aim is

termination of tachycardia but any change in the ECG, even if only transient, may

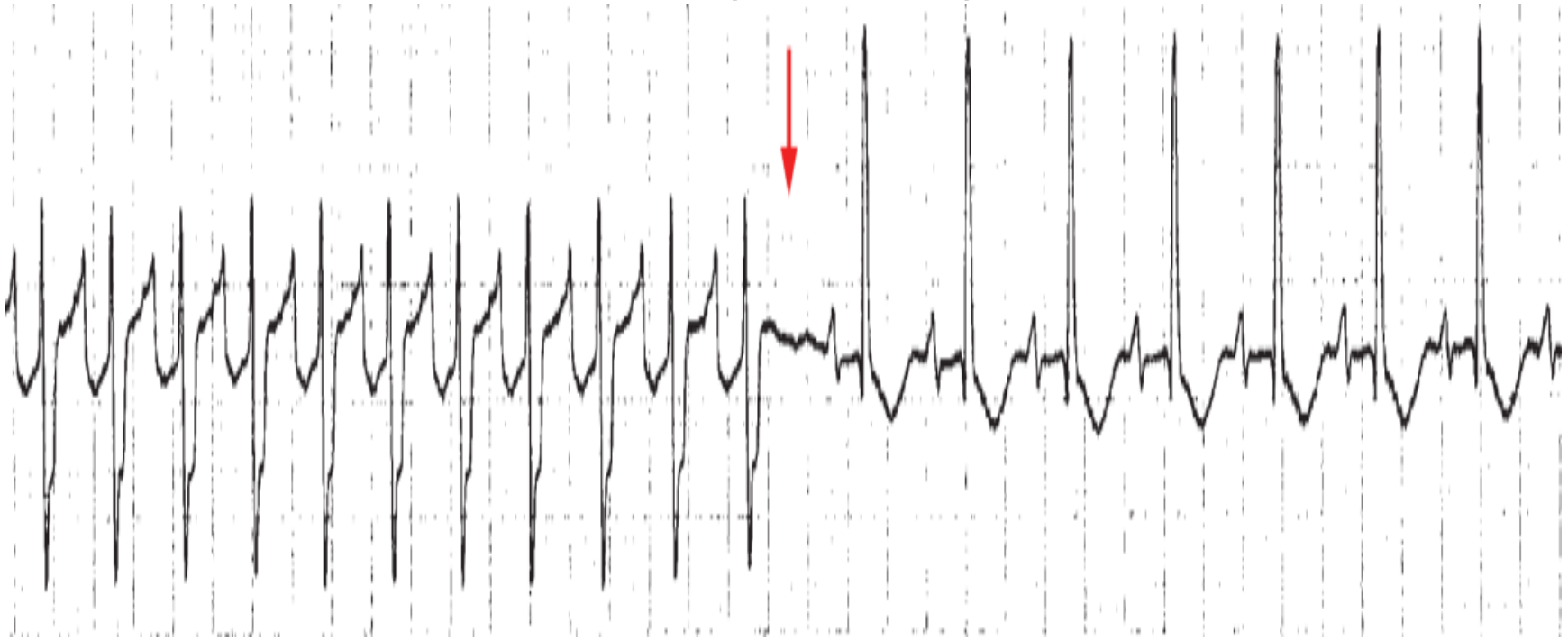
give useful diagnostic information

AV re-entry tachycardia

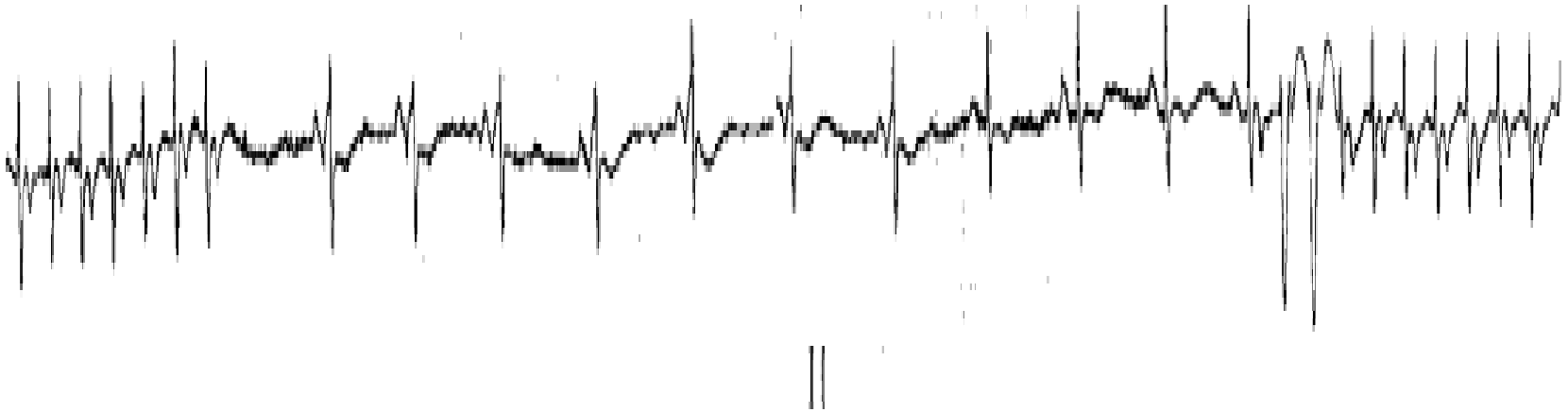
pre-excitation in sinus rhythm (red arrow), confirming the presence of an accessory pathway



retrograde block – shown by a missing P wave-
antidromic
AV re-entry tachycardia

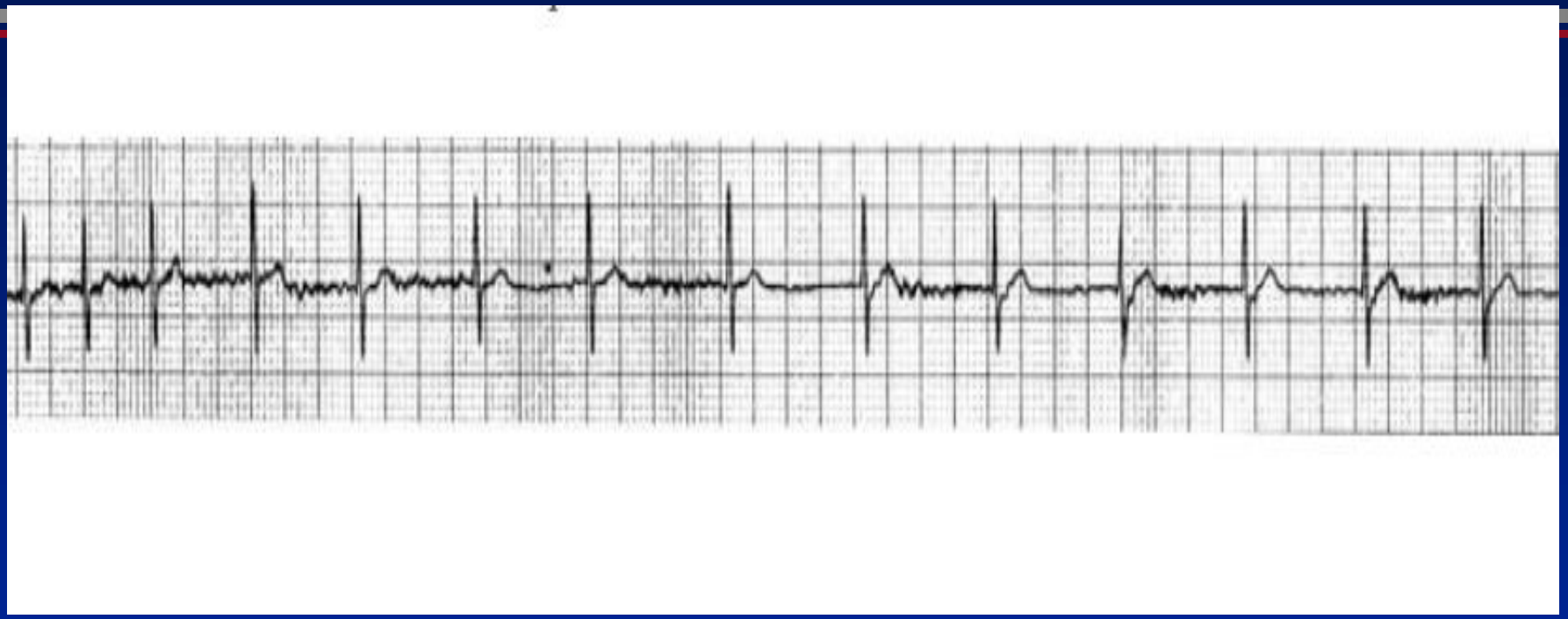


Tachycardia stops transiently and then restarts



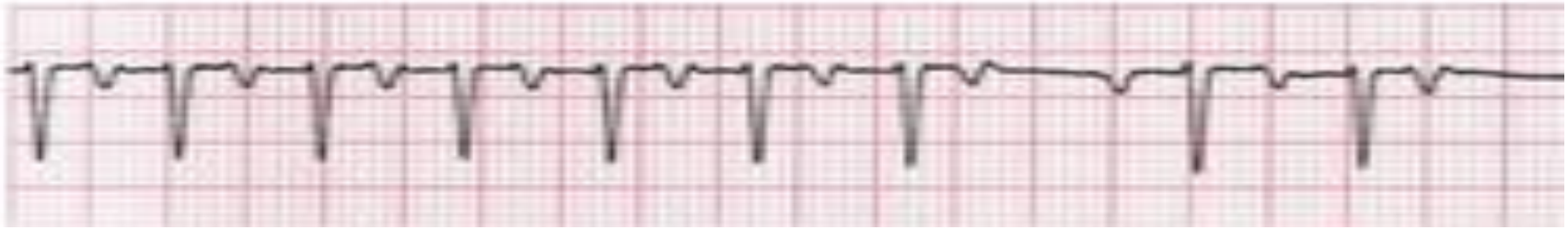
sinus tachycardia

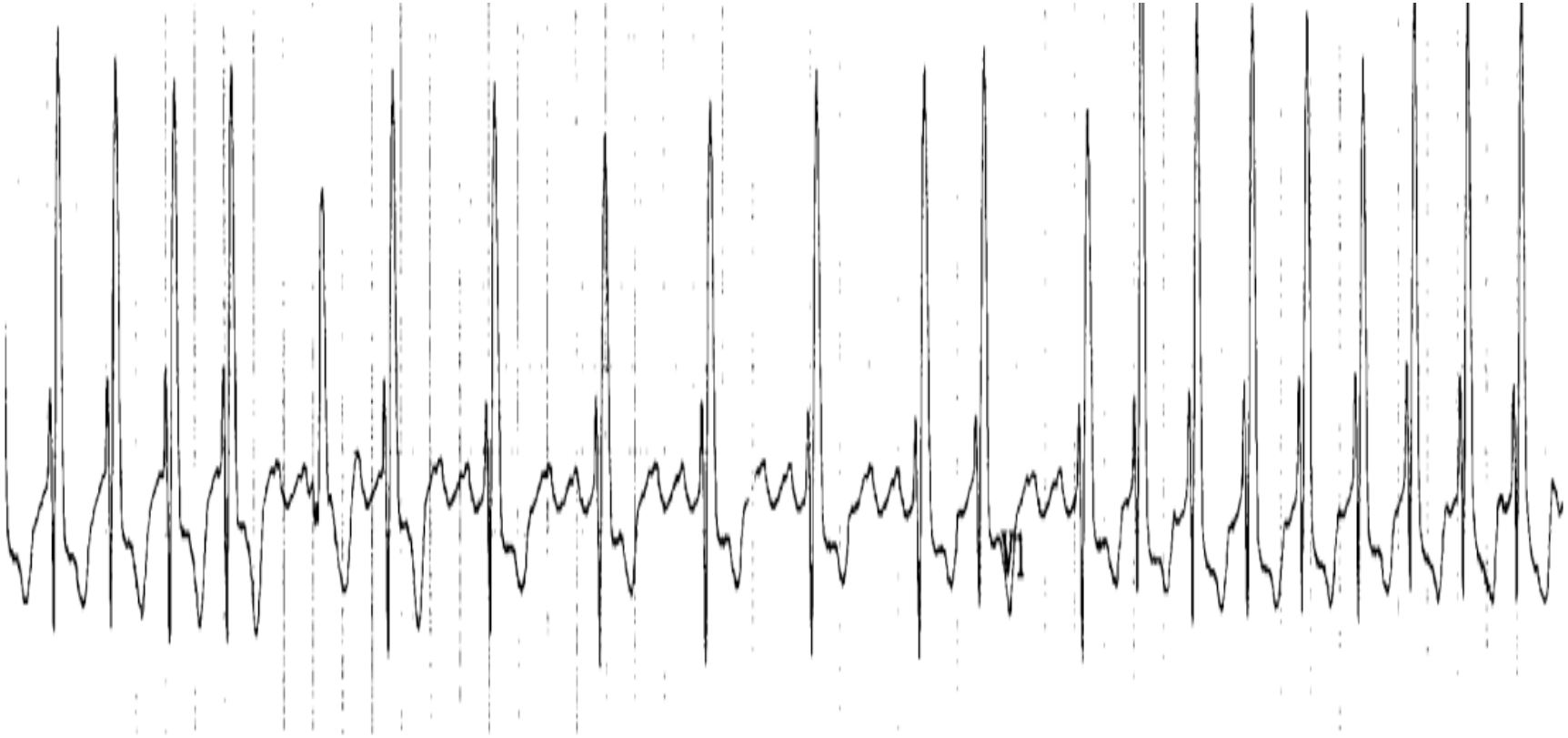
Tachycardia slows transiently and then speeds up



atrial tachycardia or atrial flutter

Tachycardia continues in the presence of AV block

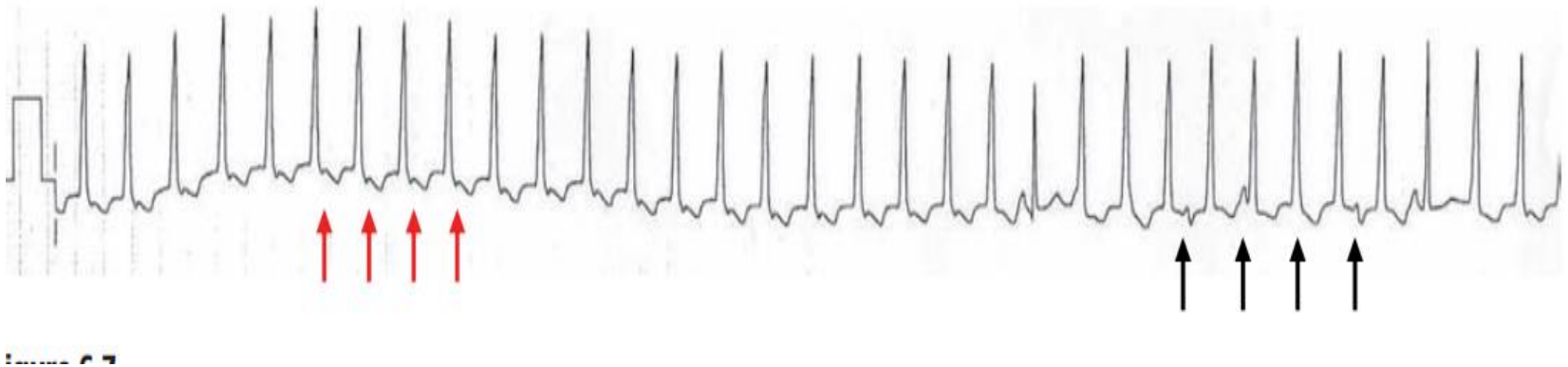




ventricular tachycardia

Adenosine causes retrograde block, which allows a sinus capture beat (a sinus P wave produces a conducted sinus beat with a normal QRS) followed by obviously dissociated P waves (black arrows).

Tachycardia continues in the presence of ventriculoatrial block



CLINICAL ENTITIES FREQUENTLY ASSOCIATED WITH ARRHYTHMIAS & Sudden Cardiac Death

Myocarditis-Cardiomyopathies

Cardiac chanellopathy & Long QT Syndrome

Arrhythmias in congenital heart defects

Electrolyte Imbalance

Cardiac Tumors

Cardiomyopathy		
Hypertrophic cardiomyopathy	?1:10000	1
Arrhythmogenic right ventricular cardiomyopathy	Rare	?0.8
Cardiovascular malformation		
Anomalous origin of a coronary artery	?1:2000	0.5
Primary arrhythmia		
Congenital long QT syndrome	?1:5000	?
Catecholaminergic polymorphic ventricular tachycardia	Rare	Rare
Wolff–Parkinson–White syndrome	1:700	Rare
Brugada syndrome	Rare	Rare
Other		
Myocarditis	–	0.5
Commotio cordis	–	Rare

Myocarditis usually results in flattened or inverted T waves

and low-voltage QRS patterns. The QT interval may be prolonged. AV block and intraventricular conduction delay also

can occur

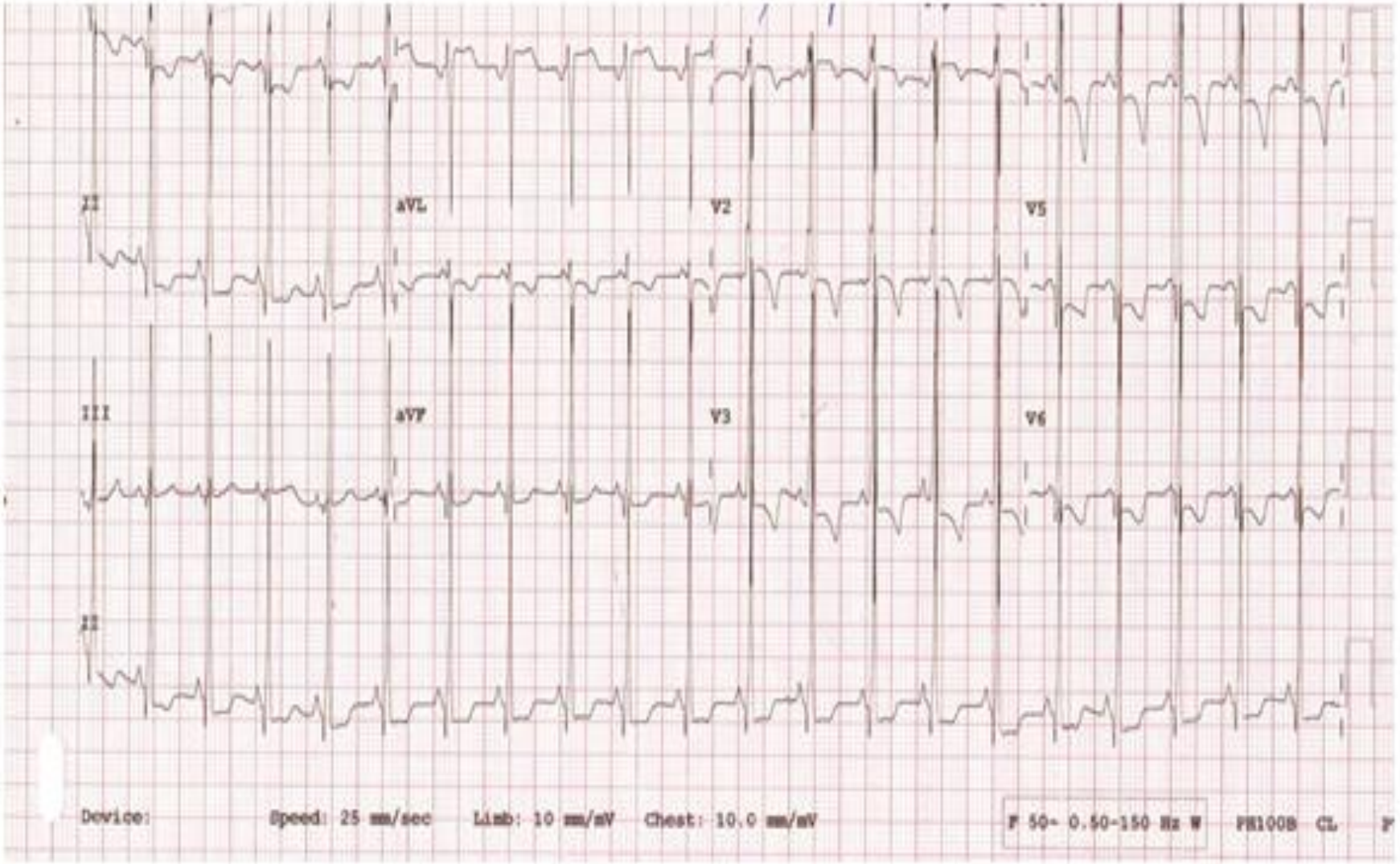


Figure.the ST –T change and left ventricular hyperthrophy in infants withPompe disease.

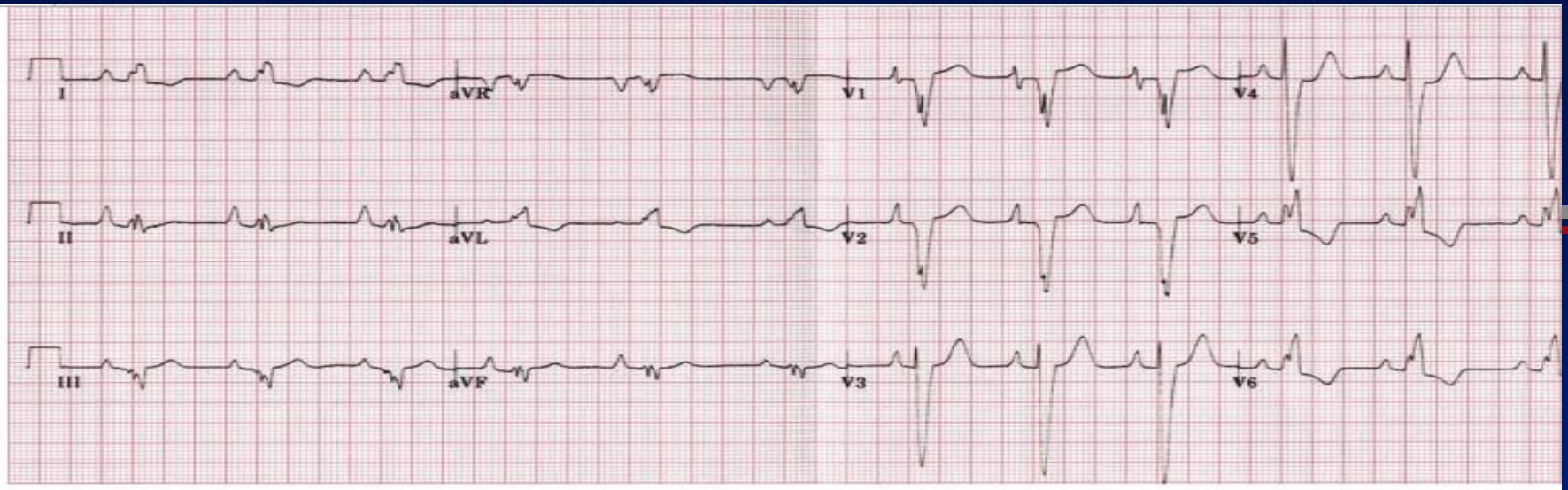


Figure 34.1

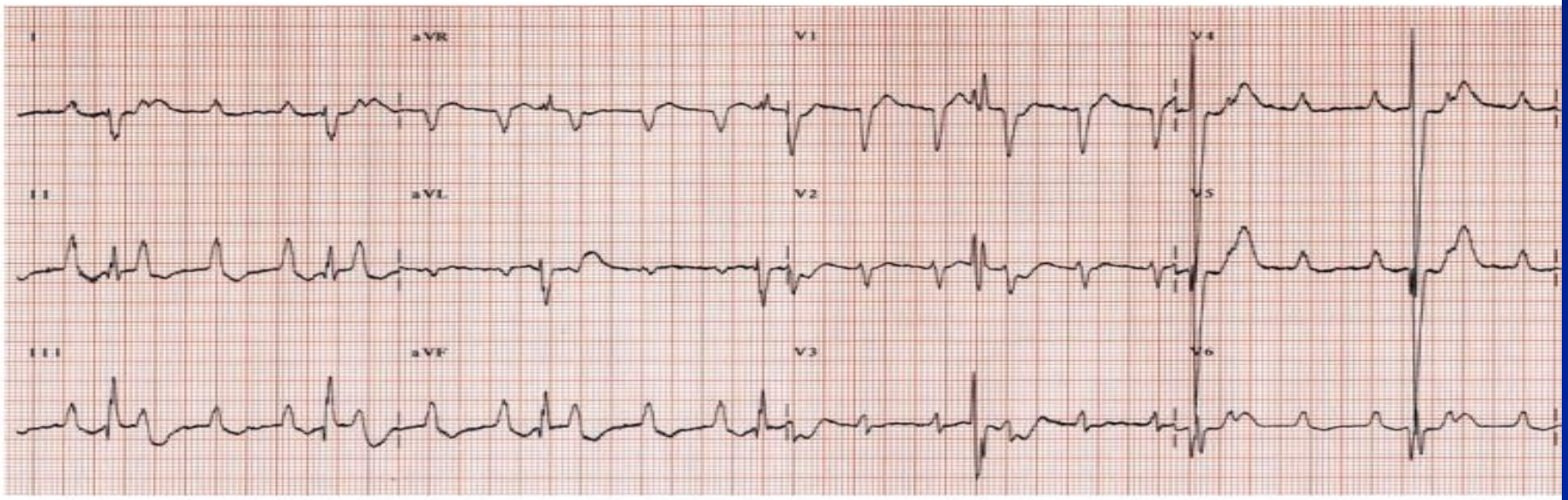


Figure 34.2

Long QT syndrome

Congenital long QT syndrome (LQTS) was first described as an association of syncope, sudden death, QT prolongation, and deafness with autosomal recessive inheritance by Jervell and Lange-Nielsen in 1957. A more common form with autosomal

dominant inheritance and normal hearing was described independently by Romano

$$QTc = QT \div \sqrt{RR}$$

A QTc measurement of440 ms (0.44 s) is generally regarded as normal and values over 460–470 ms (0.46–0.47 s) are abnormal. A measurement of 440–460/470 is borderline and cannot, by itself, confirm or exclude an abnormality. The machinemeasured QTc interval is generally reliable but should always be checked by manual

measurement when it gives a borderline result or when the diagnosis of LQTS is being considered seriousl

Risk stratification

Various predictors of increased risk in LQTS have been described but few of them

have a major impact on management. The main predictors of increased risk are a

QTc duration of500 ms and a history of syncope. A family history of premature

sudden cardiac death seems not to be independent risk factor

Acquired long QT syndrome

Antiarrhythmics, e.g. quinidine, sotalolol, disopyramide
Antihistamines, e.g. terfenidine, astemizole
Macrolide antibiotics, e.g. erythromycin, clarithromycin
Fluoroquinolones, e.g. sparfloxacin, ciprofloxacin
Antimalarials, e.g. chloroquine, halofantine
Imidazole antifungals, e.g. ketoconazole
Tricyclic antidepressants, e.g. imipramine
Antianginals, e.g. bepridil
Prokinetics, e.g. cisapride
Antiemetics, e.g. domperidone, droperidol
Antipsychotics, e.g. haloperidol, thioridazine, pimozide
Anti-infective agents, e.g. pentamidine
Anti-cancer drugs, e.g. arsenic trioxide, tamox

The precise underlying cause of acquired LQTS is not as well understood as for congenital LQTS

A small number of acquired LQTS patients can be shown to have mutations associated with congenital LQTS and may just have a subclinical variant. Female sex is also a risk factor.

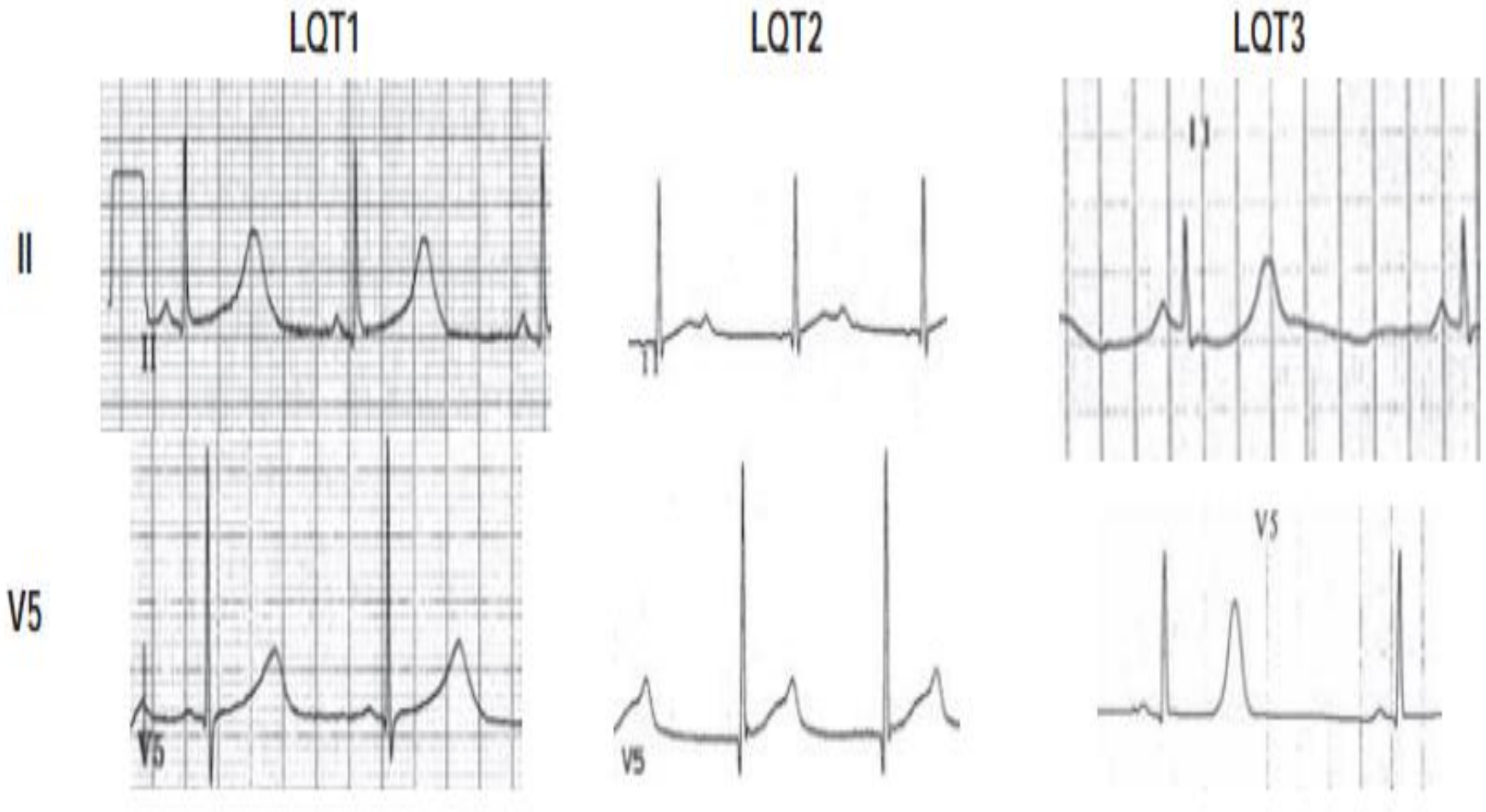
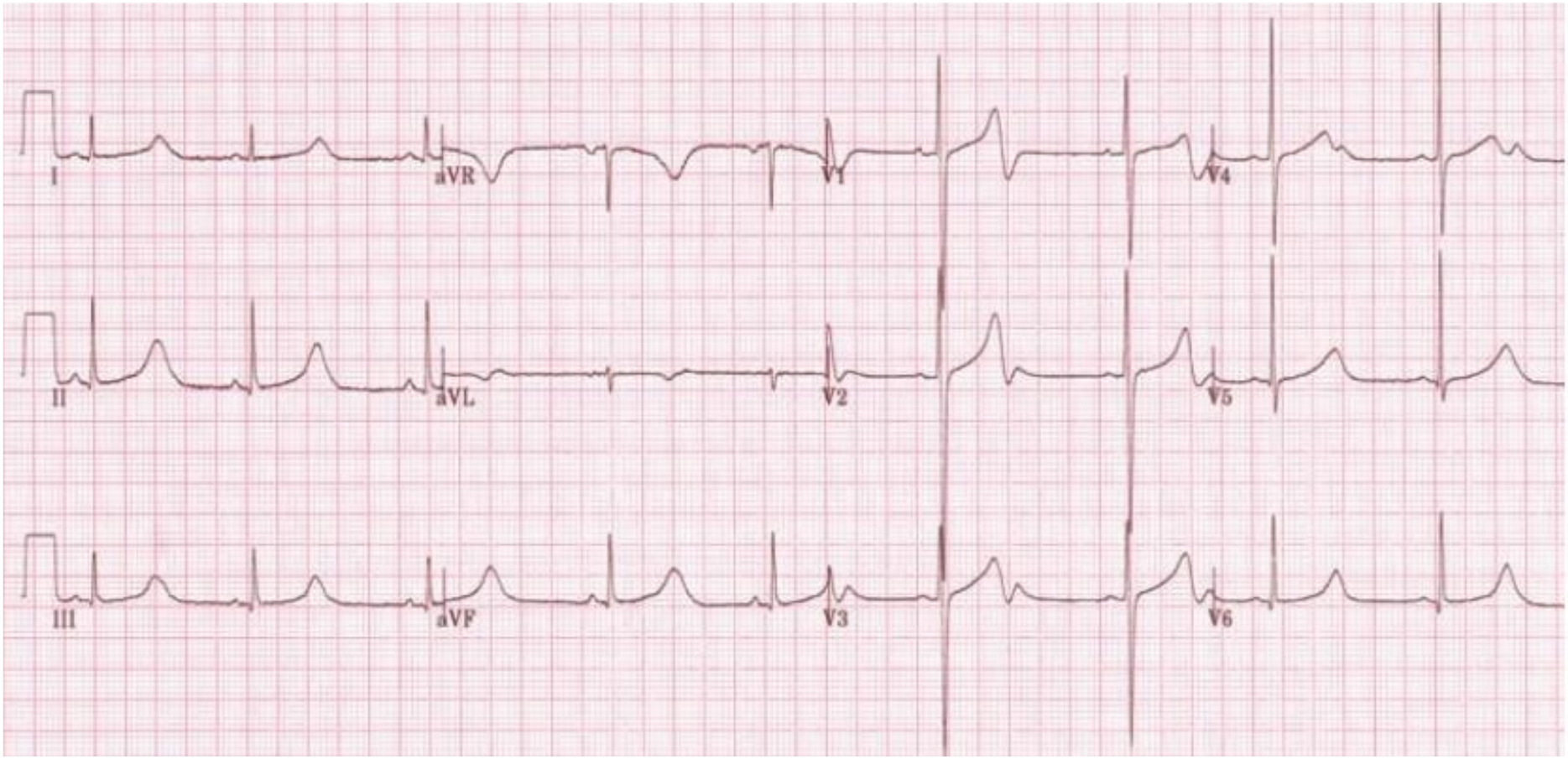
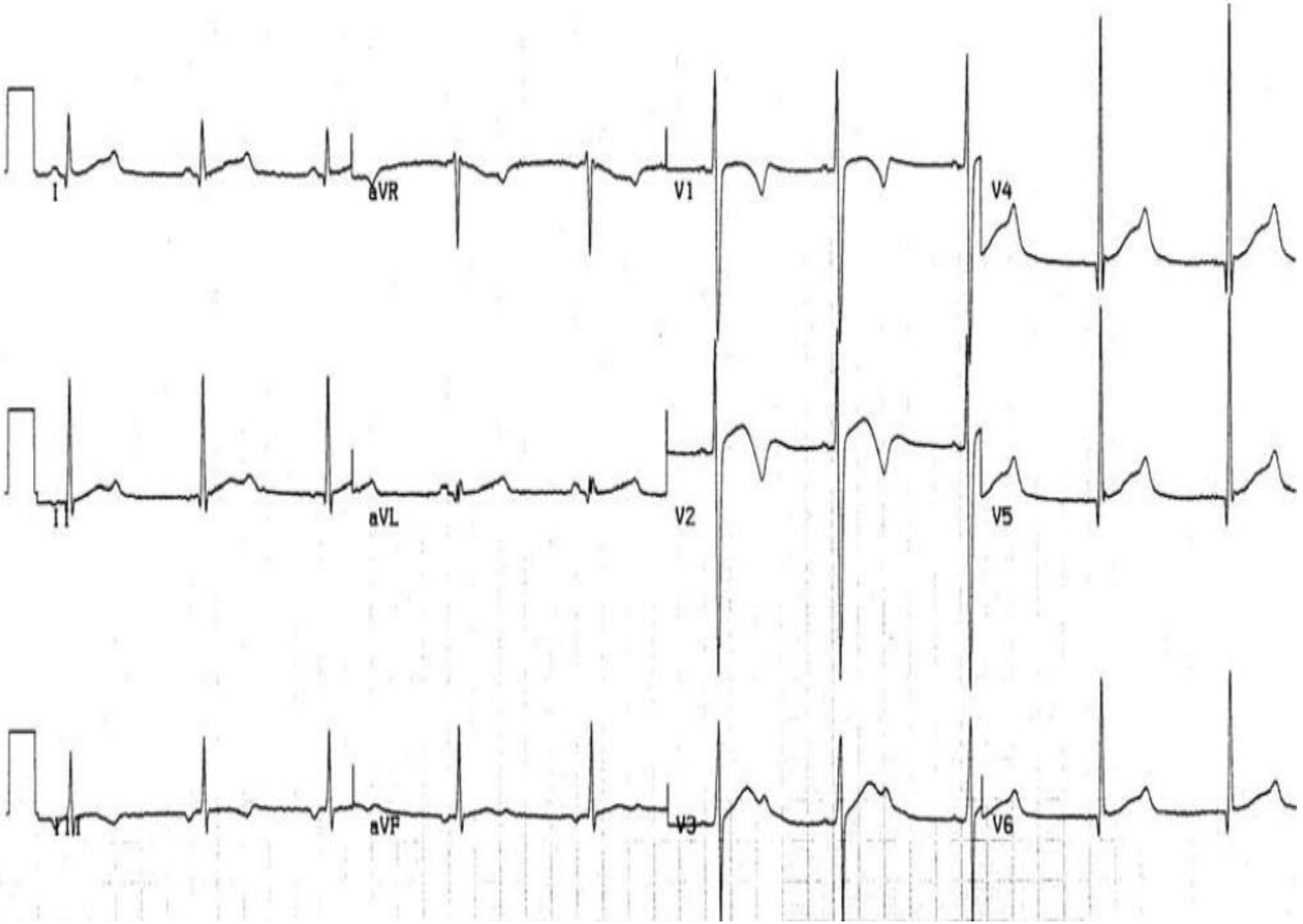


Figure 25.2

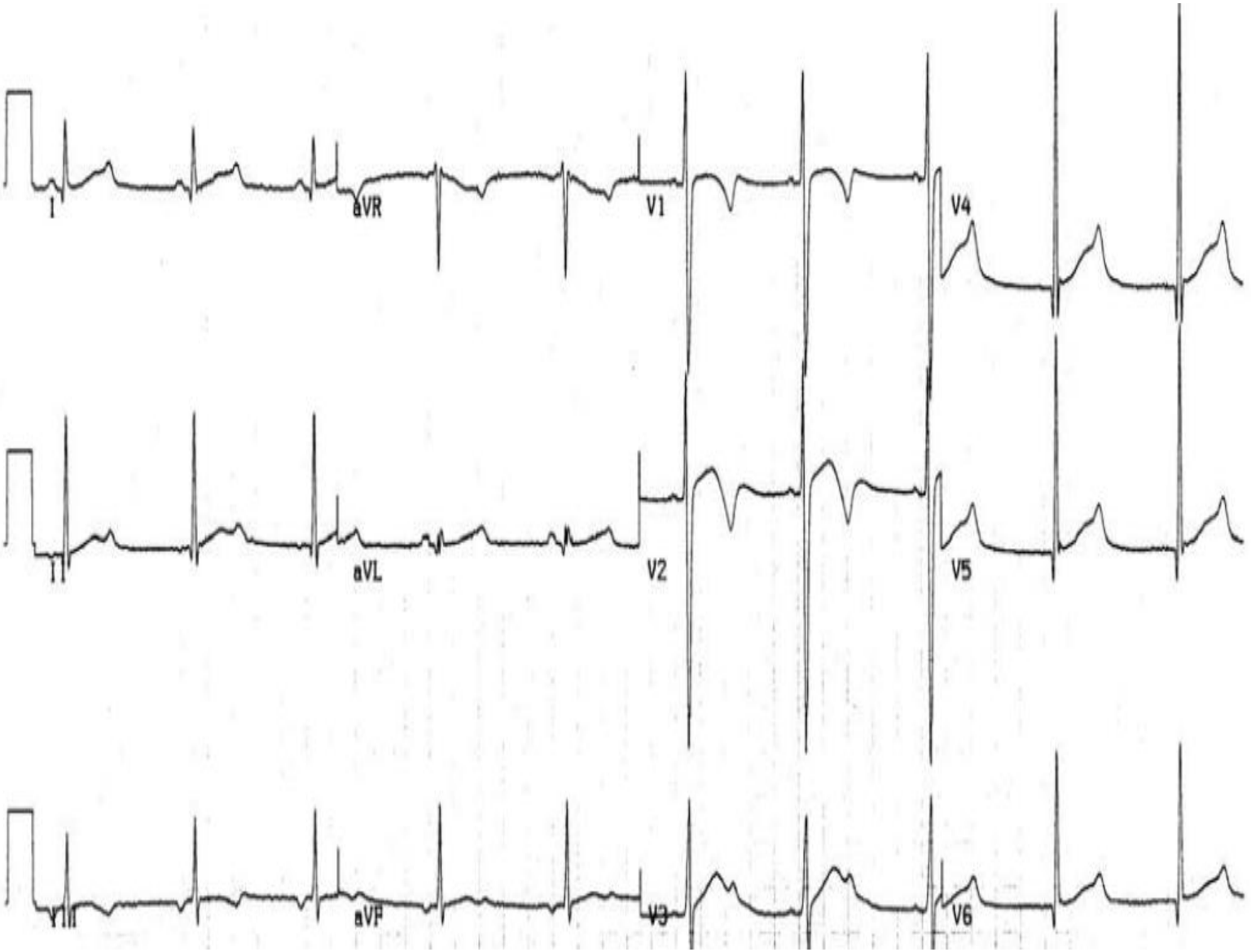
a 12-year-old boy with a typical presentation with syncope on exercise and a proven LQT1 mutation. His heart rate is around 55/min and his QT interval is 600 ms. Symptoms are mostly related to exertion in LQT1 and occur at a relatively young age, with 60% of patients experiencing their first symptoms before the age of 10


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

a 10-year-old boy
with syncope. Symptoms are less common in
LQT2 but the risk of death is probably
higher than LQT1. Syncope in LQT2 more often
occurs during emotional stress or
sleep or at rest

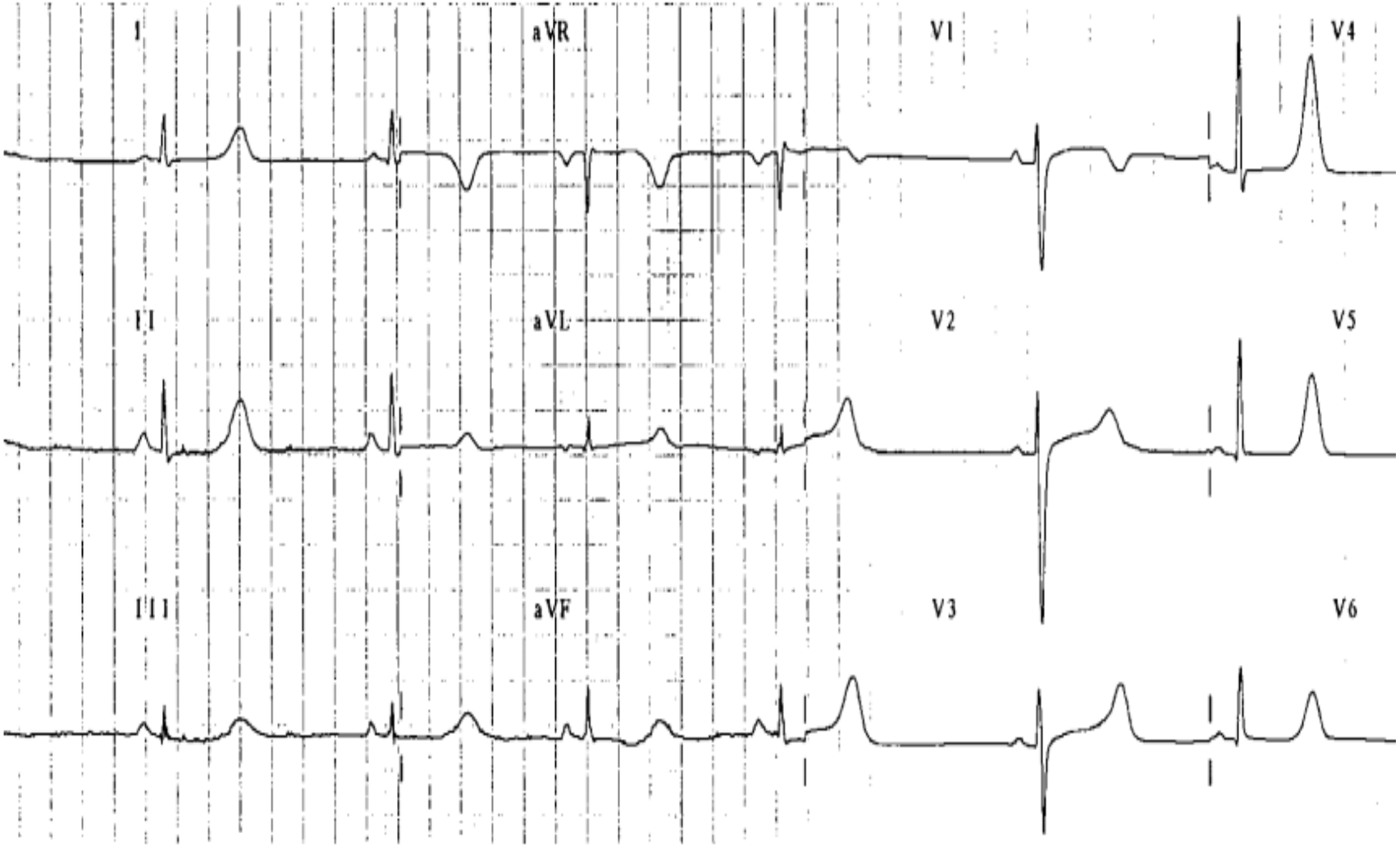

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

the typical appearance of LQT3 in an 8-year-old boy with mild aortic stenosis who was found by chance to have QT prolongation. The sharply peaked T wave in chest leads follows a long isoelectric ST segment. Symptoms during rest or sleep are also more common than during exercise in LQT3

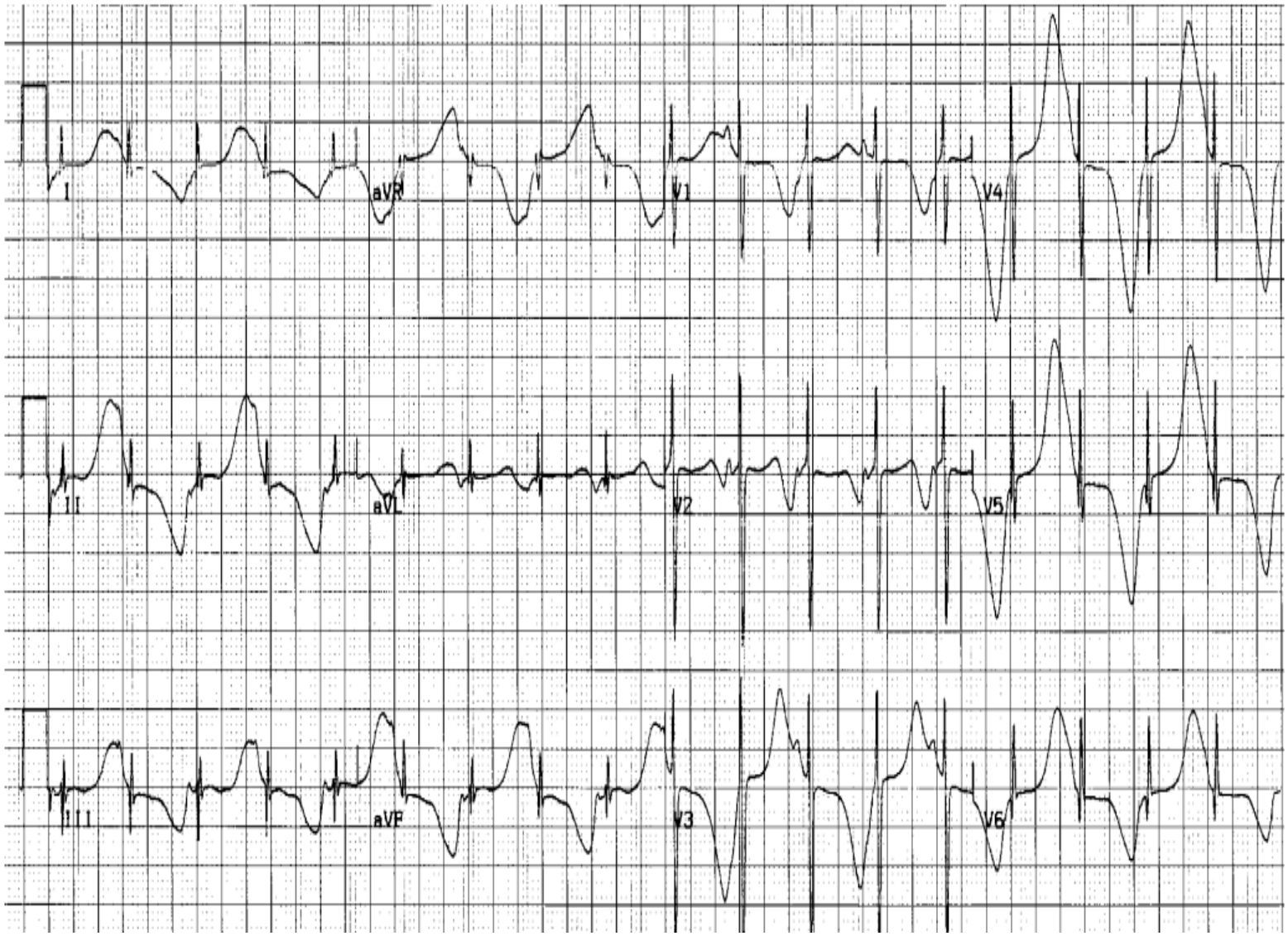

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

a boy with Jervell Lange-Nielsen syndrome

. In this rare condition most known patients are symptomatic, presenting
with syncope on exercise or during emotional stress at a young age, usually before
school age. The QT interval is usually markedly prolonged, as shown here. In this
example the T waves are flat in many leads and the QT interval is impossible to
measure in leads II and V5, so others leads have to be used


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

a 4-year-old boy undergoing transcatheter closure of a
patent ductus arteriosus under general anesthetic. His QT prolongation was an incidental finding and he had never had symptoms or arrhythmias. So far his genotype has not been identified


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Acute management of acquired LQTS involves withdrawal of the offending agent,
correction of hypokalemia to a potassium4 mmol/L and giving intravenous magnesium. Isoprenaline infusion or temporary pacing may also be useful to prevent
any triggering bradycardia or pauses. Further management avoids future avoidance
of any positive drug. Acquired LQTS is generally considered not to be familial but
similar advice may be offered to first-degree family mem

Brugada syndrome

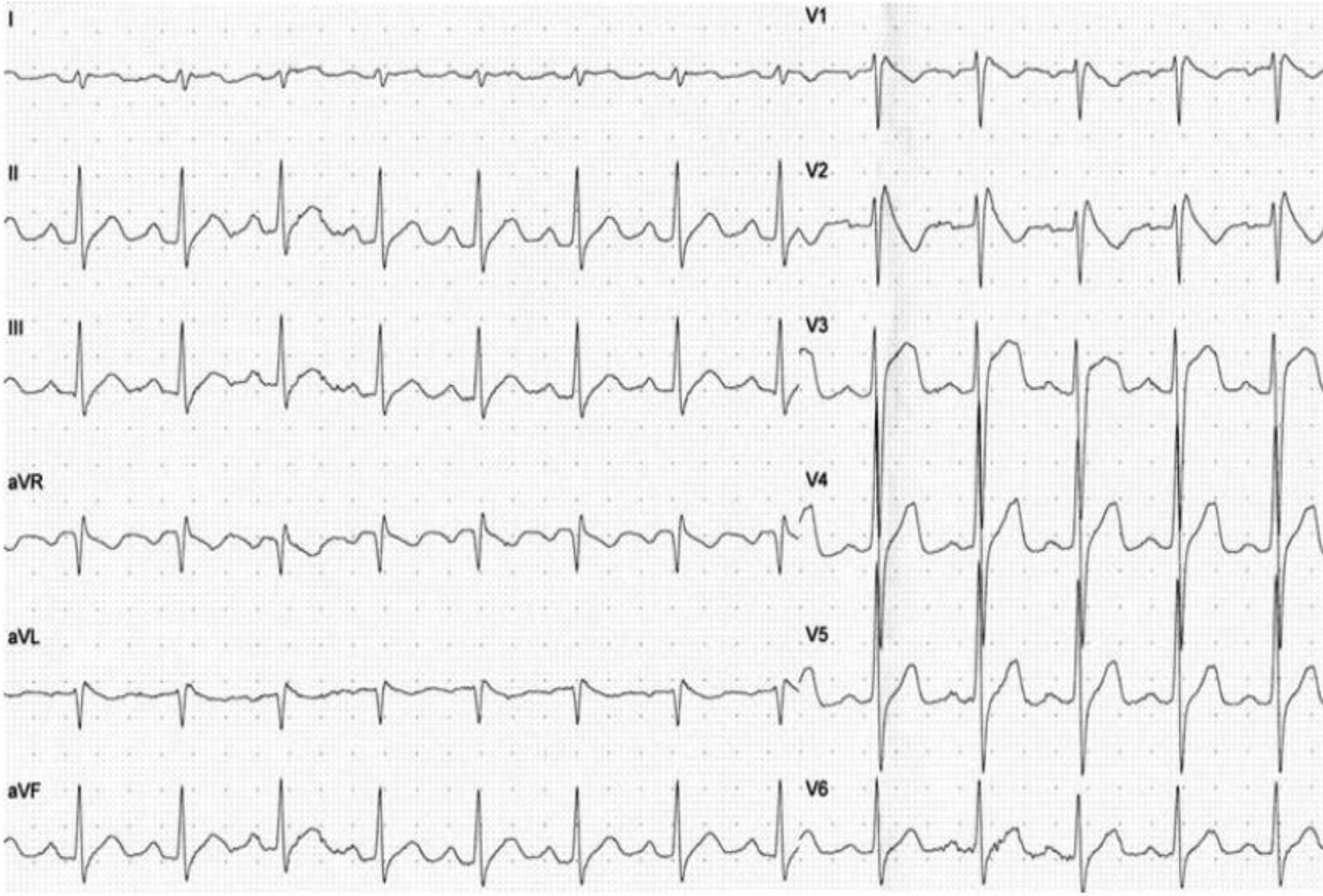


Figure 27.2

an 11-year-old girl whose father died in his sleep at the age of 39 years. The autopsy was normal and the cause of his death was not established

Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is one of the rarest but most dangerous ventricular arrhythmias encountered in childhood and is associated with a high risk of syncope and sudden death. It usually has autosomal dominant inheritance and 20–50% of cases show a mutation in the cardiac ryanodine receptor gene (RYR2).RYR2is the main calcium release channel on the sarcoplasmic reticulum within cardiac myocyte

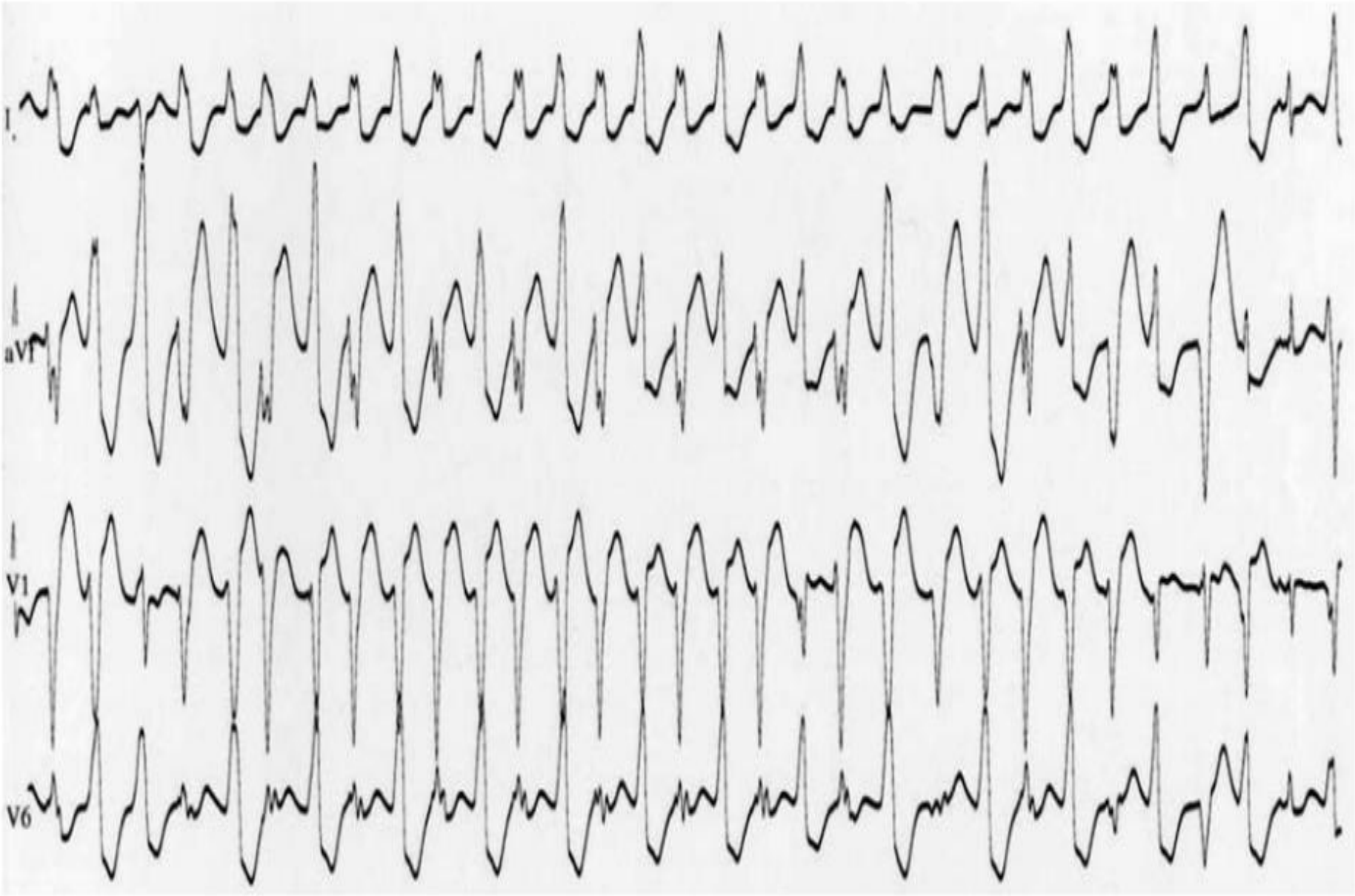


Figure 26.3

Antiarrhythmic drug treatment

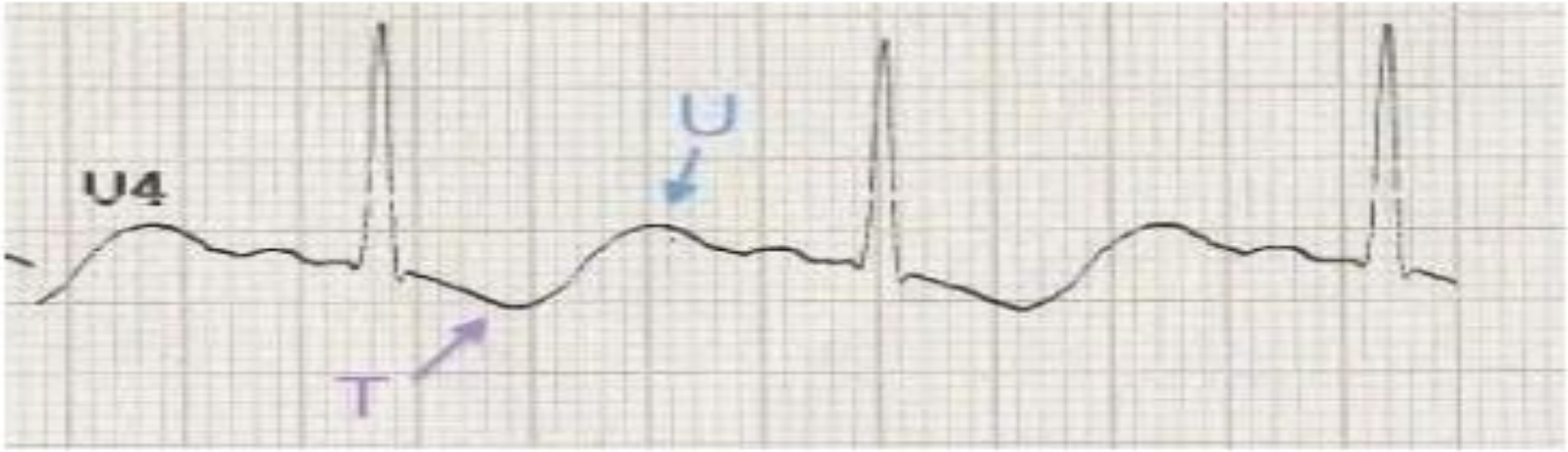
Classification of Antiarrhythmic Agents

Class	Subclass	Drug	Pharmacologic Effect
I		Moricizine	Depression of rate of increase of action potential
	IA	Quinidine	Increased AP duration, and increased atrial and ventricular ERP; increased JT interval; vagolytic action
		Procainamide	
		Disopyramide	
	IB	Lidocaine, Mexiletine, Tocainide	Decreased AP duration but increased ventricular ERP; unchanged QRS complex; unchanged JT interval
	IC	Propafenone	Depressed rate of increase of action potential, causing widening of QRS complex; unchanged AP duration, but increased atrial and ventricular ERP; unchanged JT interval
		Flecainide (Encainide)	
II		Beta-blockers	Inhibition of beta-adrenergic receptors
III		Amiodarone	Increased AP duration
		Sotalol	Increased JT interval
		Bretylium	
IV		Verapamil, Diltiazem	Blockade of Ca_{++} channels

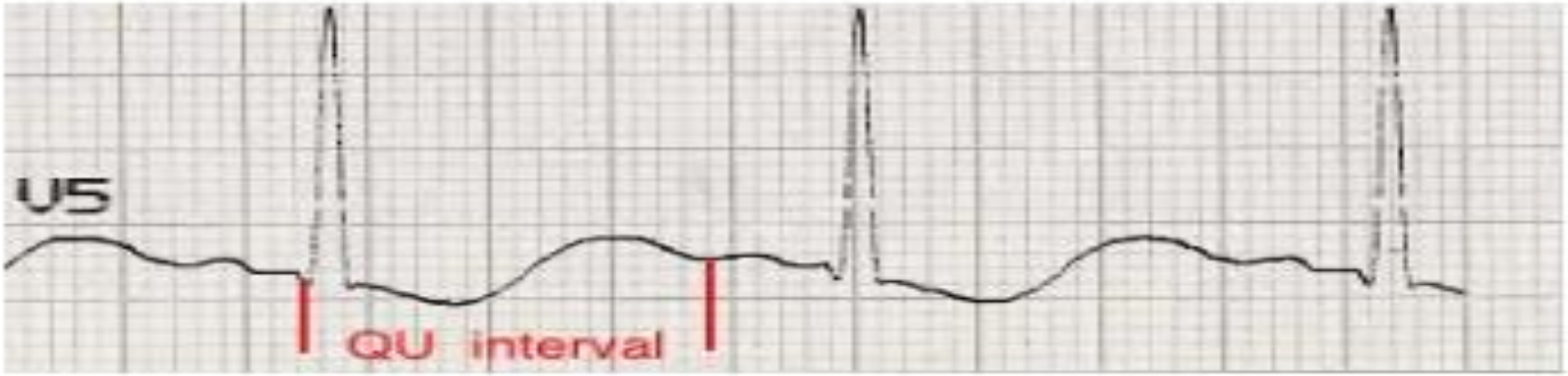
Electrolyte Imbalance

Potassium Imbalance

Calcium and Magnesium Imbalance



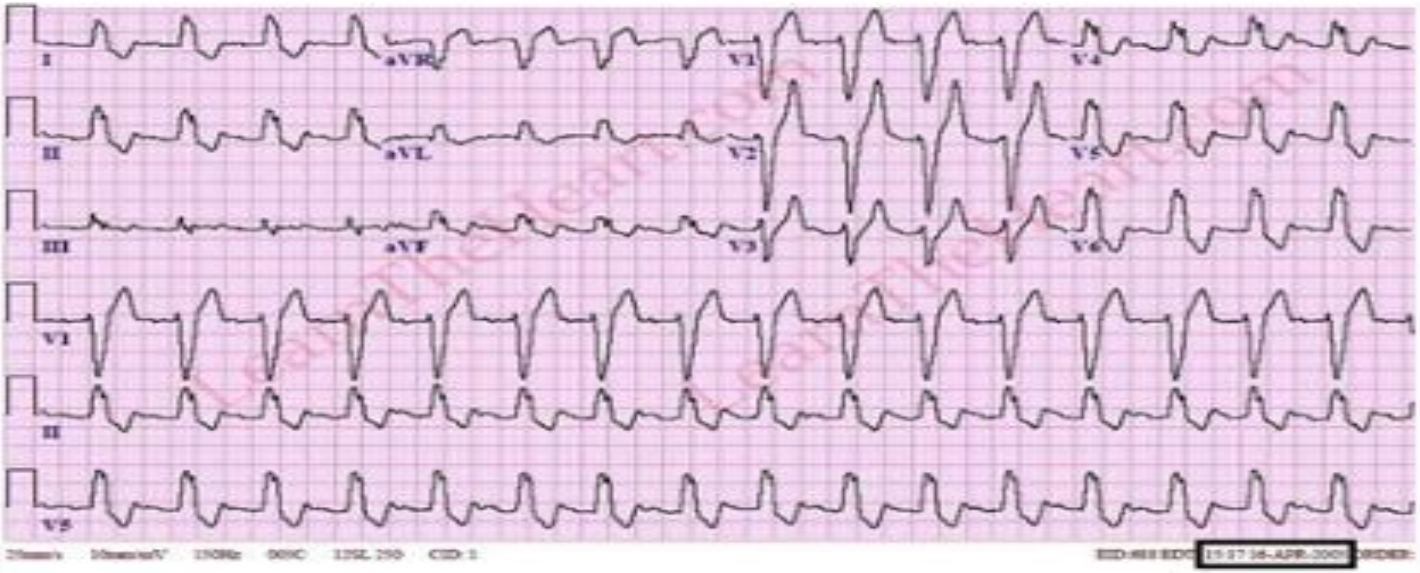
Hypokalaemia: T wave inversion and prominent U waves



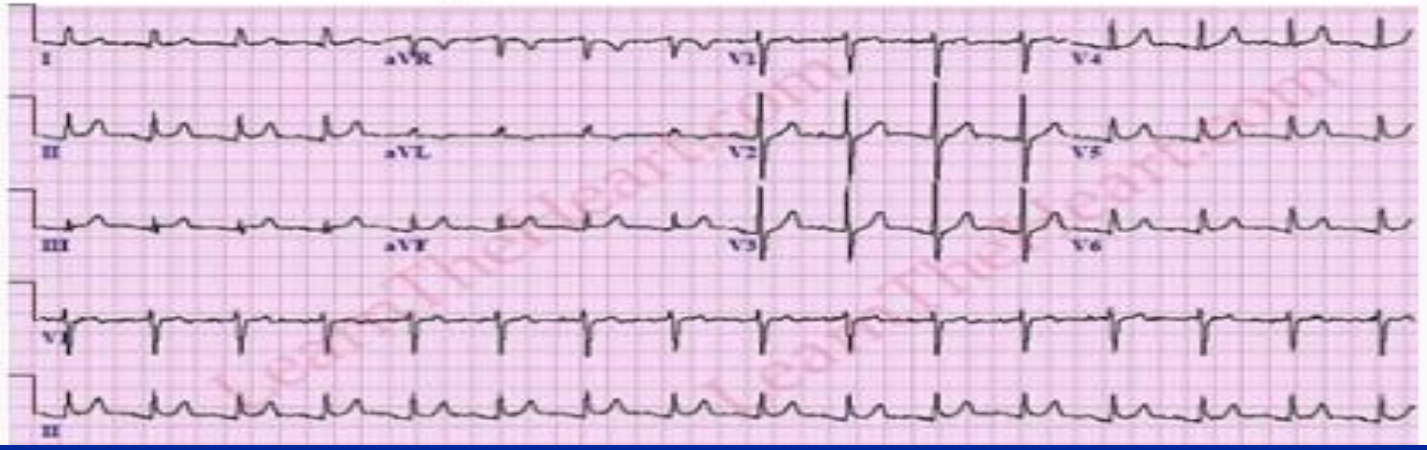
QU interval: The apparent pseudo-prolonged QT interval is

ENLARGE

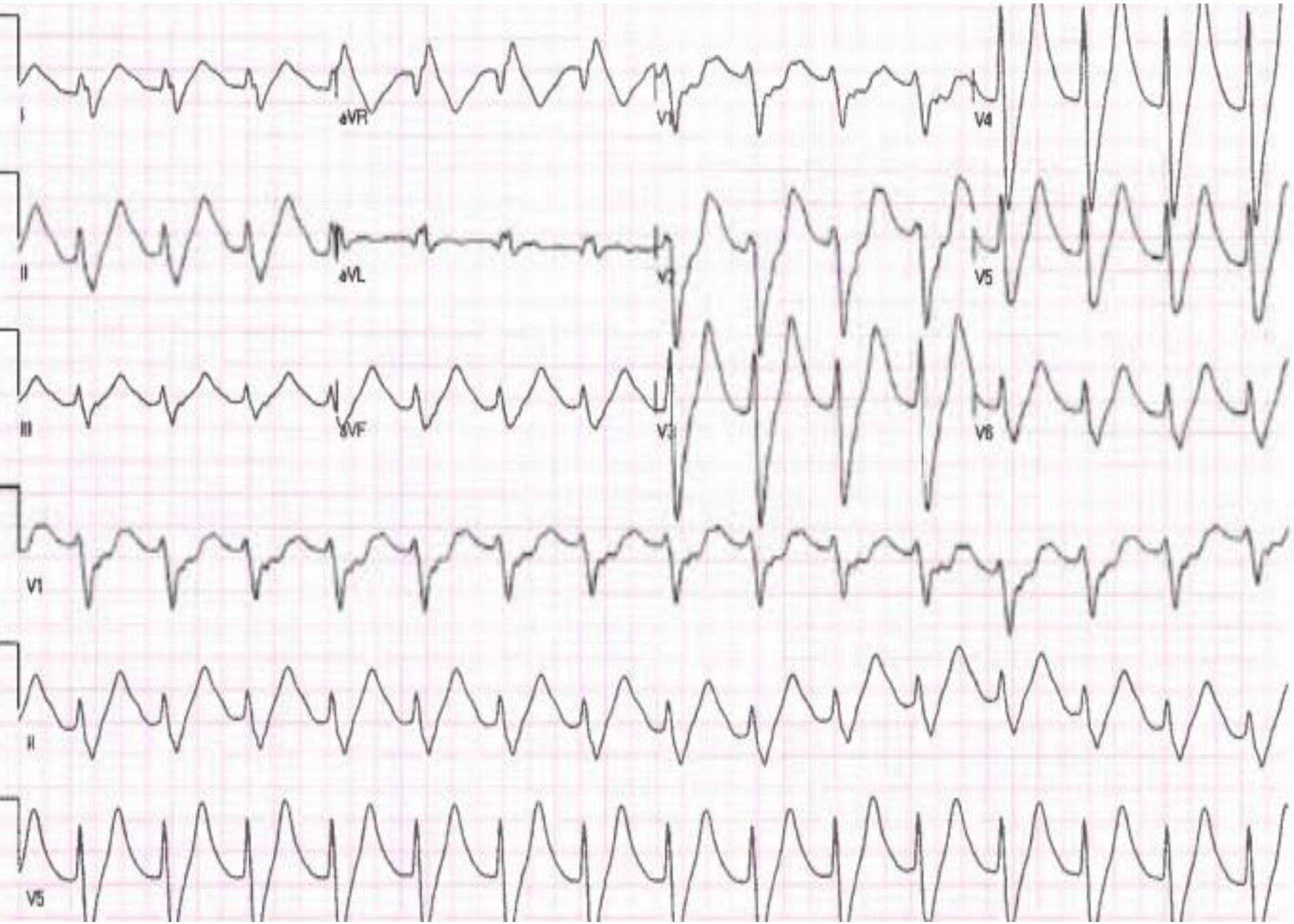
Hyperkalemia Before IV Calcium



Hyperkalemia After IV Calcium



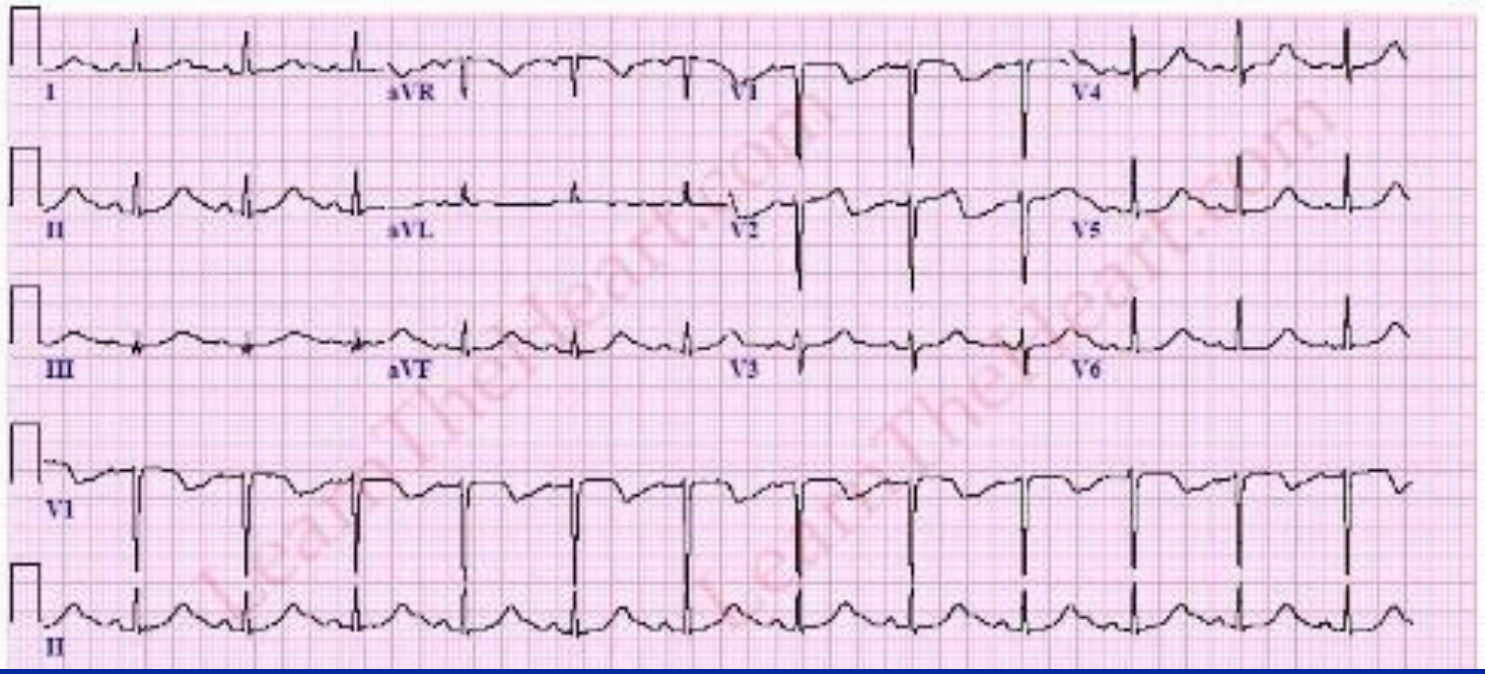
A 14-year-old
patient with renal failure and a
potassium level of 7.7 mmollL.
The ECG shows the characteristic findings of
hyperkalemia


I
aVR
V4
II
aVL
V5
III
aVF
V6
V1
II
V5

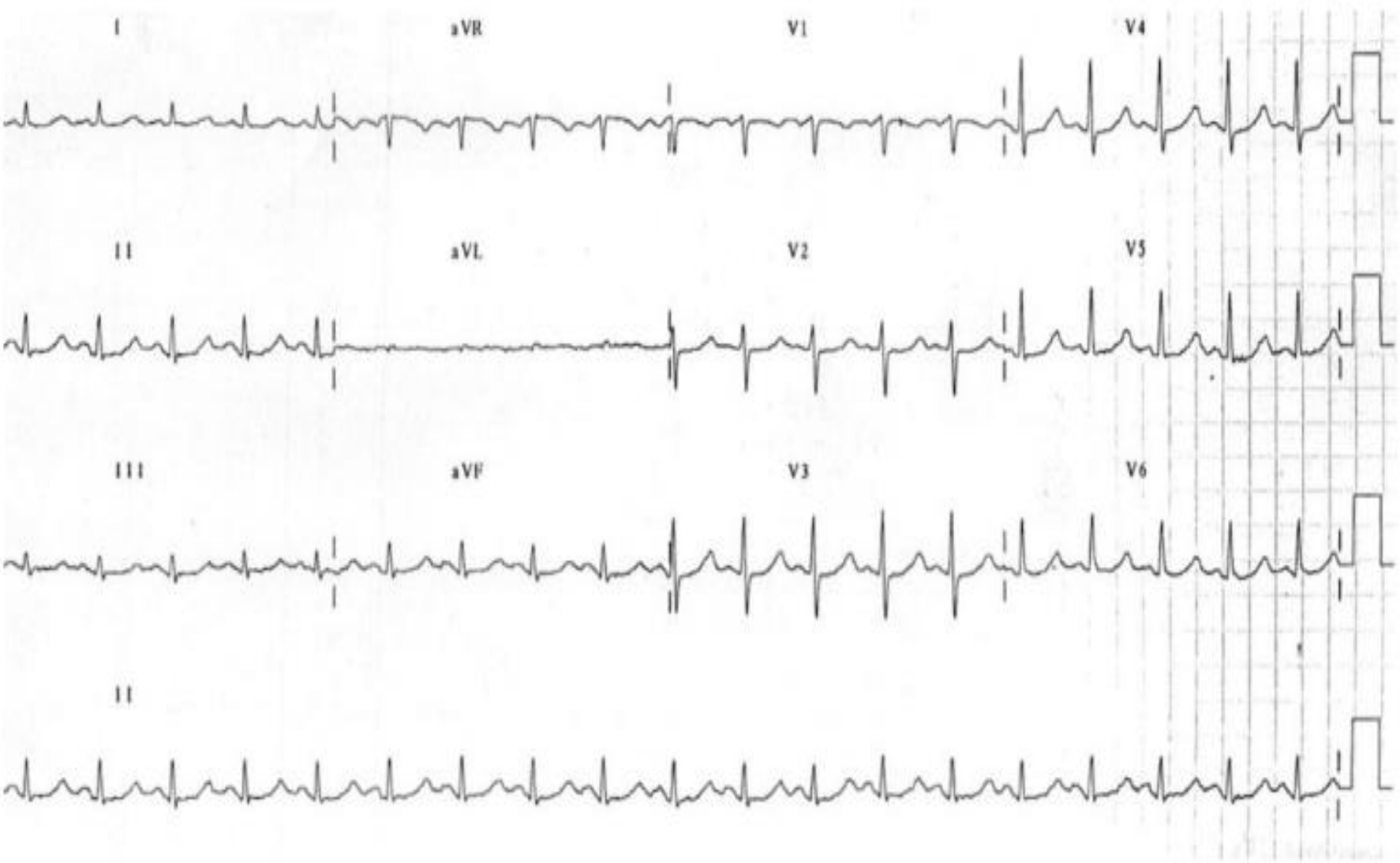
The ECG findings of hypocalcemia include:

1. A prolonged QT interval
2. A lengthened ST segment

ENLARGE



Example 1



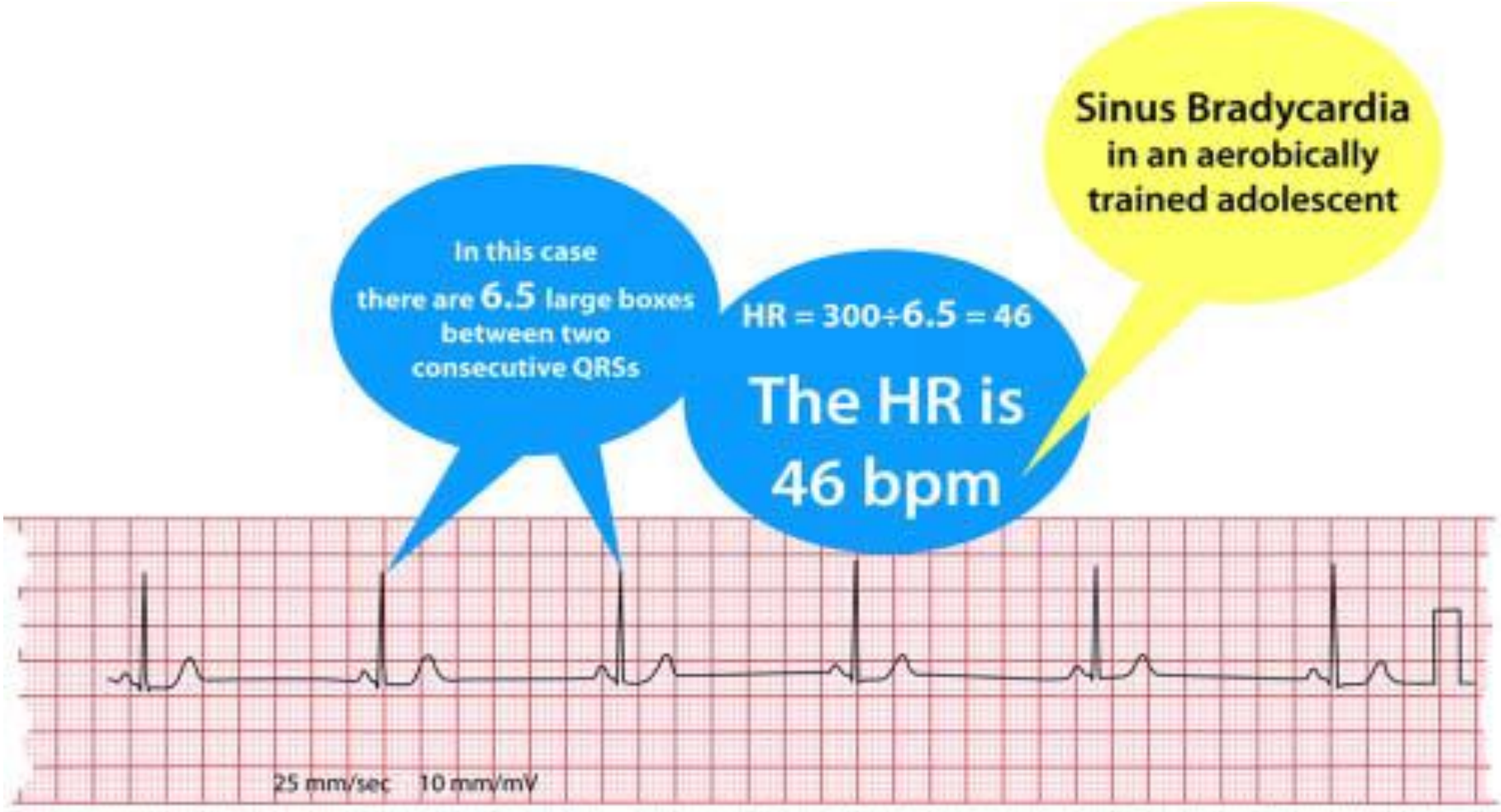
Hypomagnesaemia with QTC prolonged at 510ms

Bradyarrhythmias

Sinus Bradycardia

Sinus bradycardia may occur in the intensive care unit (ICU) setting during airway suctioning, elevated intracranial pressure, hypoxia, hypoglycemia, hypercalcemia, and acidosis. Drugs in therapeutic or toxic doses (e.g., digoxin, beta-blockers, amiodarone) may also cause sinus bradycardia. Surgical injury or trauma to the sinus node may also result in persistent sinus bradycardia

Sinus Bradycardia



Sinus Bradycardia in Children

Age	Rate (bpm)
Infants and children to 2 yr of age	<90
Children age 2–6 yr	<80
Children age 6–11 yr	<70
Children older than 11 yr	<60

bpm, beats per minute.

Atrioventricular Block

Atrioventricular block may be congenital, related to changes in autonomic tone, or develop in association with a number of disease states

First-degree AV block is present when the PR interval is longer than the age-specific norm.. As an isolated finding, first-degree AV block is benign and warrants no specific treatment or follow-up. When part of an evolving disease state or if there is associated bundle branch block, attention should be paid to the possible development of more advanced AV block

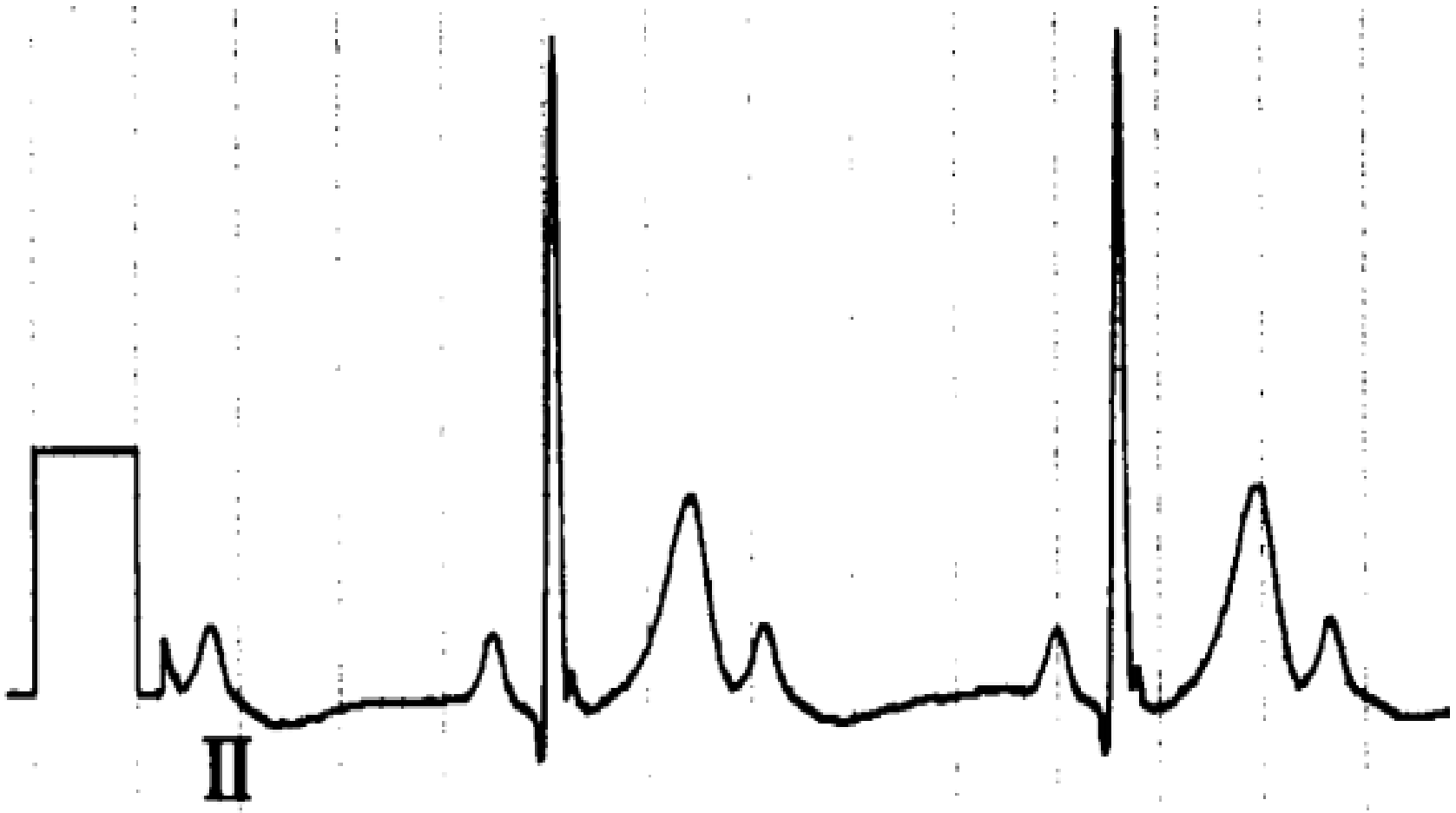
Increased parasympathetic (vagal) tone	Airway suctioning
	Intubation
	Pain
	Bowel distention
	Vasovagal syncope
Metabolic disorders	Hypokalemia
	Hyperkalemia
	Hypomagnesemia
	Hypocalcemia
	Hypercalcemia
	Hypoglycemia
Associated with congenital heart disease	Heterotaxies, especially “polysplenia”
	Congenitally corrected transposition of the great arteries
	Persistent left SVC with absent right SVC
Postcardiac surgery	VSD, especially in congenitally corrected transposition
	AV canal
	Others, much more rarely
Congenital complete atrioventricular block	
Hypothermia	
Infectious diseases	Rubella
	Mumps
	Lyme disease
	Rocky Mountain spotted fever
	Chagas disease (Trypanosomiasis)
	Rheumatic fever
	Diphtheria
	Enteroviral myocarditis
	Perivalvular abscess
Myopathies	Myotonic dystrophy
	Duchenne's muscular dystrophy
	Polymyositis

Second-degree atrioventricular block

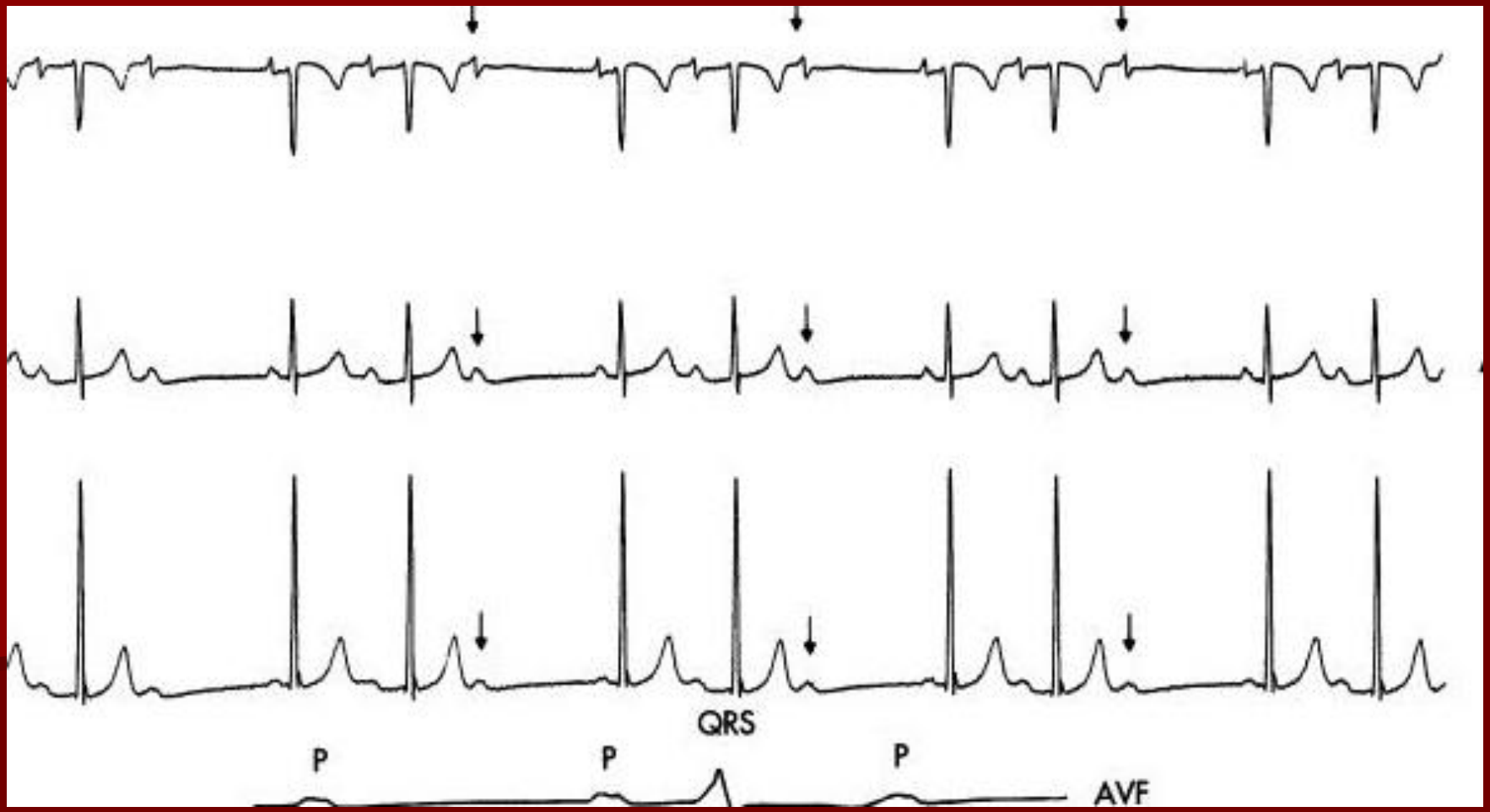
In second-degree AV block some P waves are conducted and others are not.

The type most commonly encountered isWenckebach block(also known as Mobitz

type I block) which is characterized by progressive lengthening of the PR interval


II

Second-degree AV block is present when an atrial depolarization intermittently is not conducted to the ventricles resulting in the appearance of a "dropped beat." Mobitz type I second-degree AV block (Wenckebach AV conduction) exhibits a gradual prolongation of the PR interval before the nonconducted P wave



the PR interval
lengthens over two or three beats before the third or fourth P wave is not conducted, so we have varying 3:2 and 4:3 conduction. P waves sometimes merge with the previous T waves and are less easy to see (arrows).

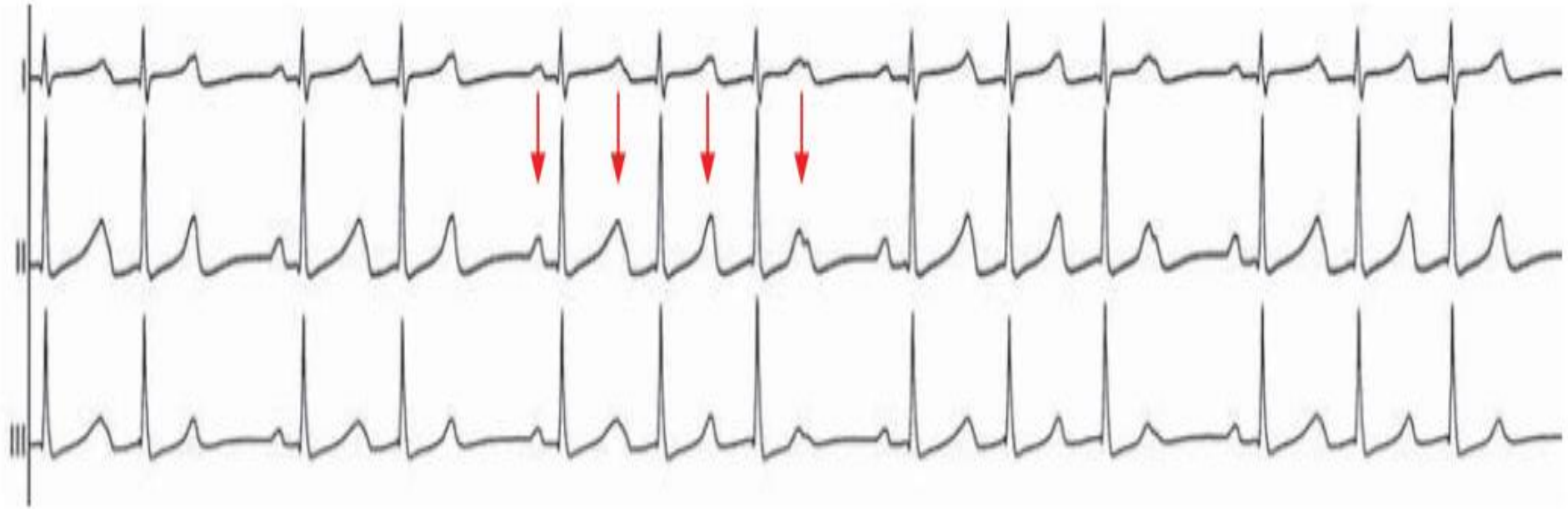
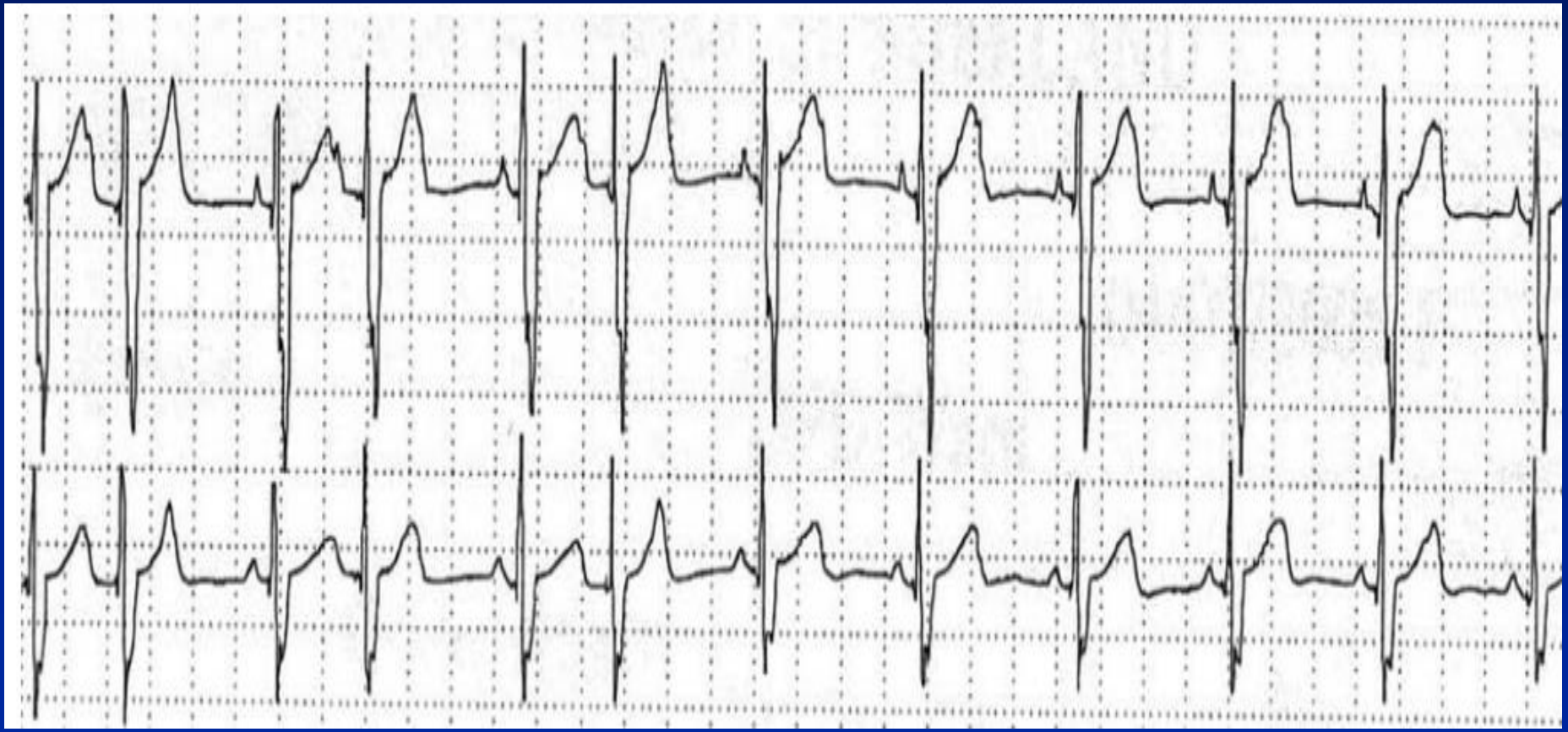
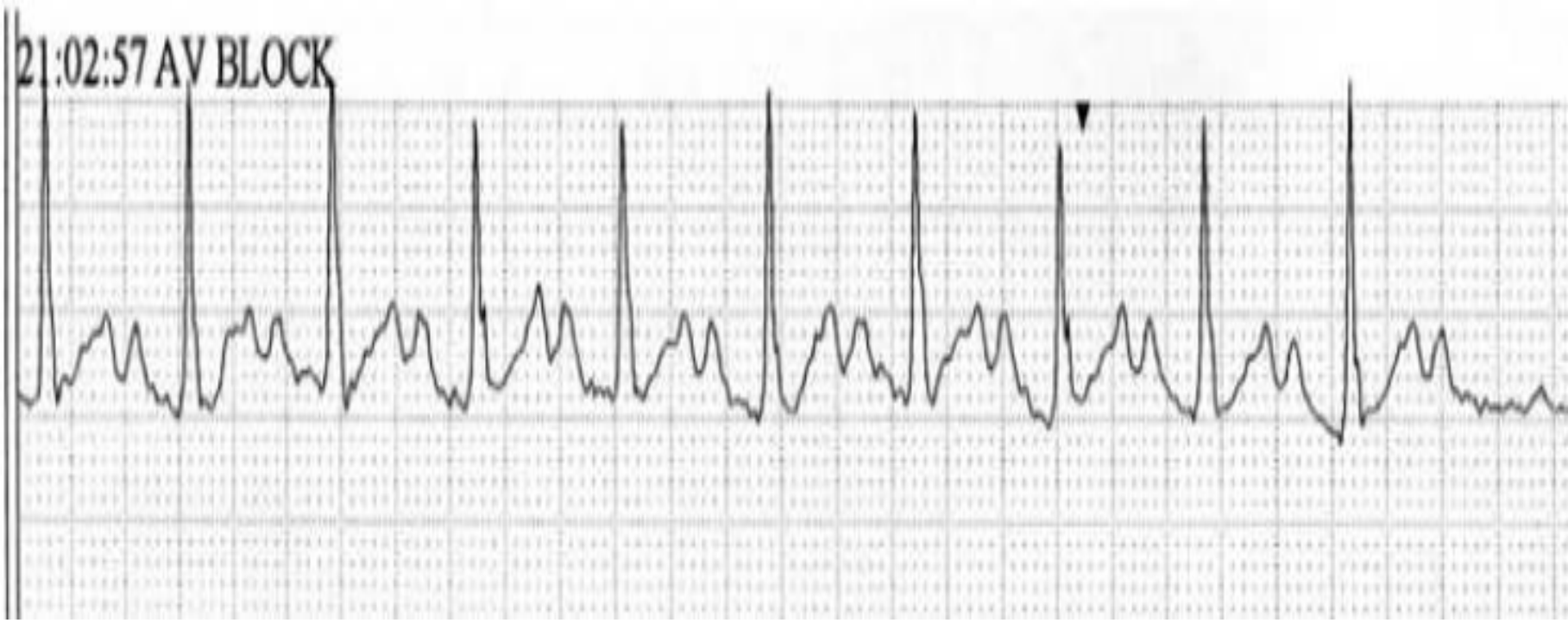


Figure 28.2

3:2 Wenckebach conduction followed by
2:1 conduction. 2:1 AV conduction may result from Mobitz I or Mobitz II conduction
but here it is clearly Mobitz I because of the 3:2 conduction

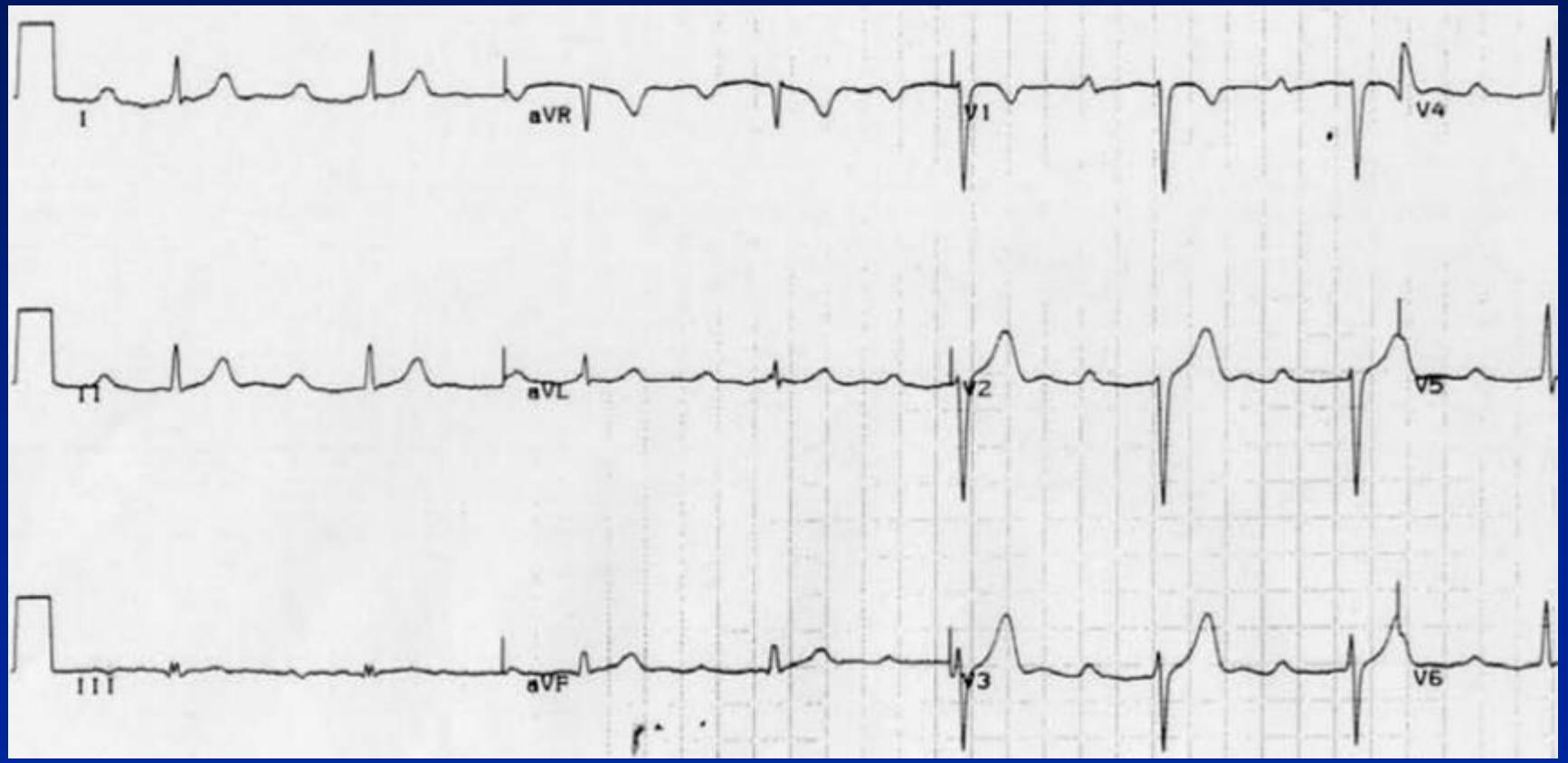


a 21-year-old man with congenitally corrected transposition of the great arteries. On ambulatory ECG recording he was noted
to have non-conducted beats. This may not be classic Mobitz II block because he has
a slightly prolonged PR interval all the time and his QRS is only slightly wider than
normal

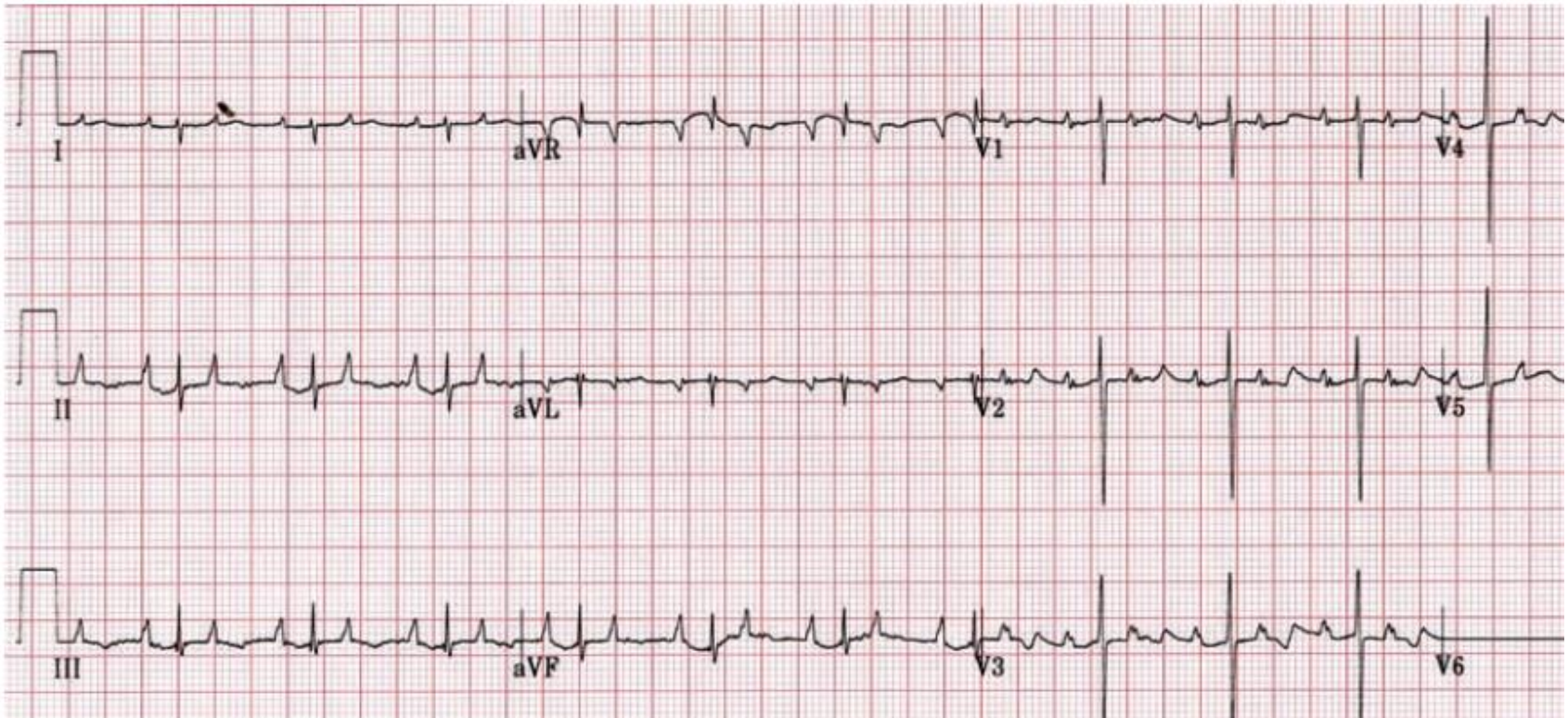


In the presence of stable 2:1, 3:1, or 4:1 conduction there is no opportunity to observe prolongation of the PR interval before the block, so characterization as Mobitz type I or type II AV block is not appropriate and this is best described as high-grade second-degree AV block

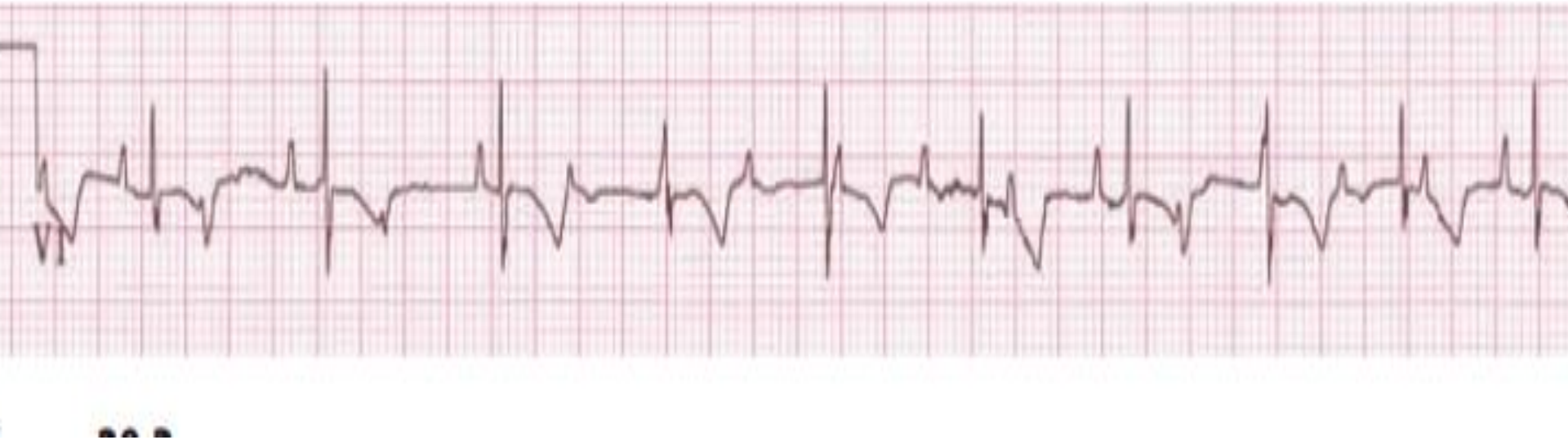
a PR interval of around 400 ms in a child with a
small ventricular septal defect. AV conduction remained 1:1 during exercise testing
and on ambulatory monitoring and did not change during 15 years of follow-up



another example of constant 2:1 AV conduction from an asymptomatic teenager with a normal heart. This needs to be distinguished from non-conducted atrial bigeminy (see Chapter 11) but note that here the PP interval is regular and the P wave morphology is constant (this is best seen in more than one lead)

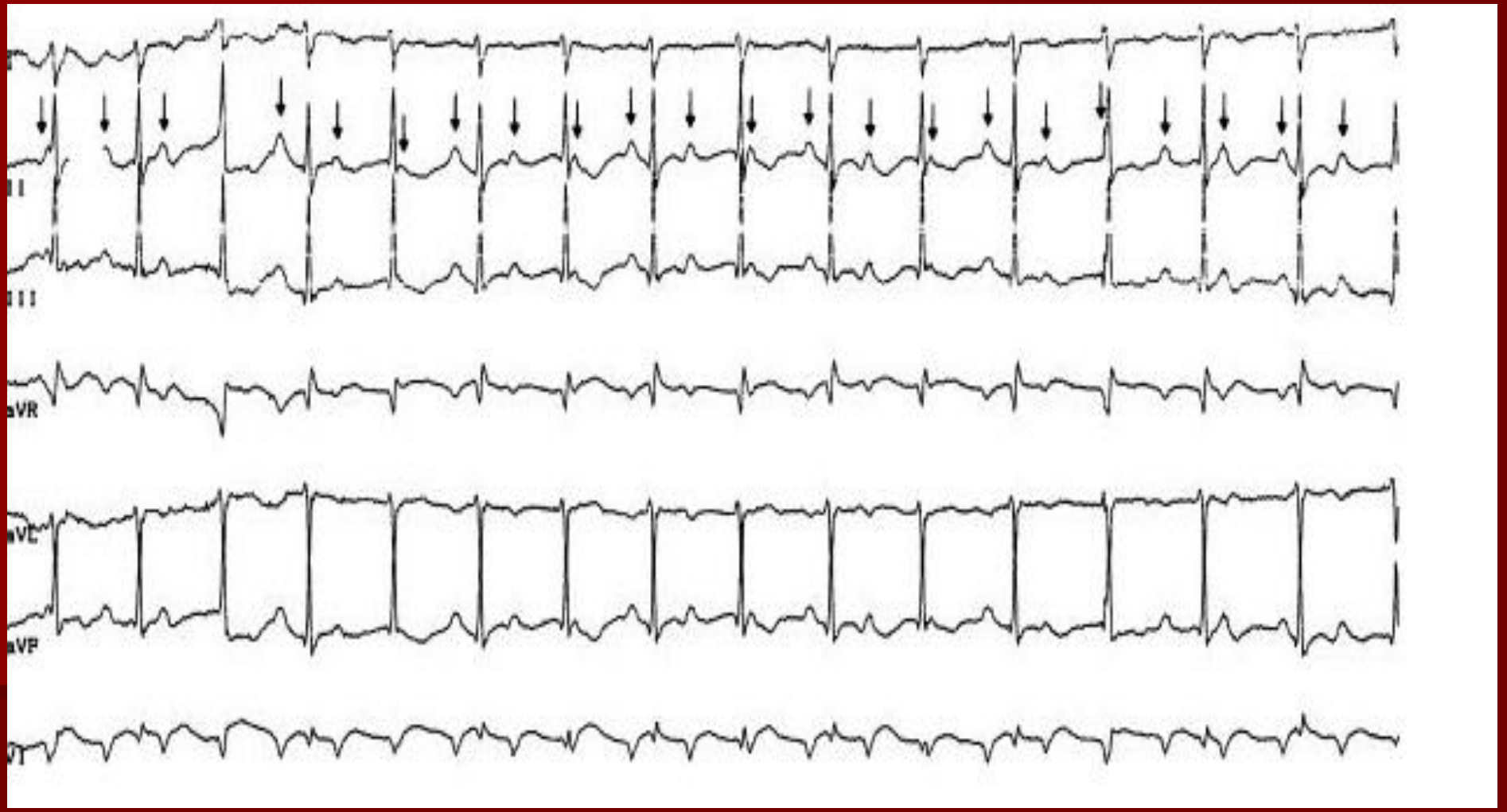


Complete atrioventricular block



Third-degree or complete heart block (CHB) may be congenital or acquired and indicates that no atrial impulses are conducted to the ventricles (Fig. 8-4). The ventricular rate is maintained

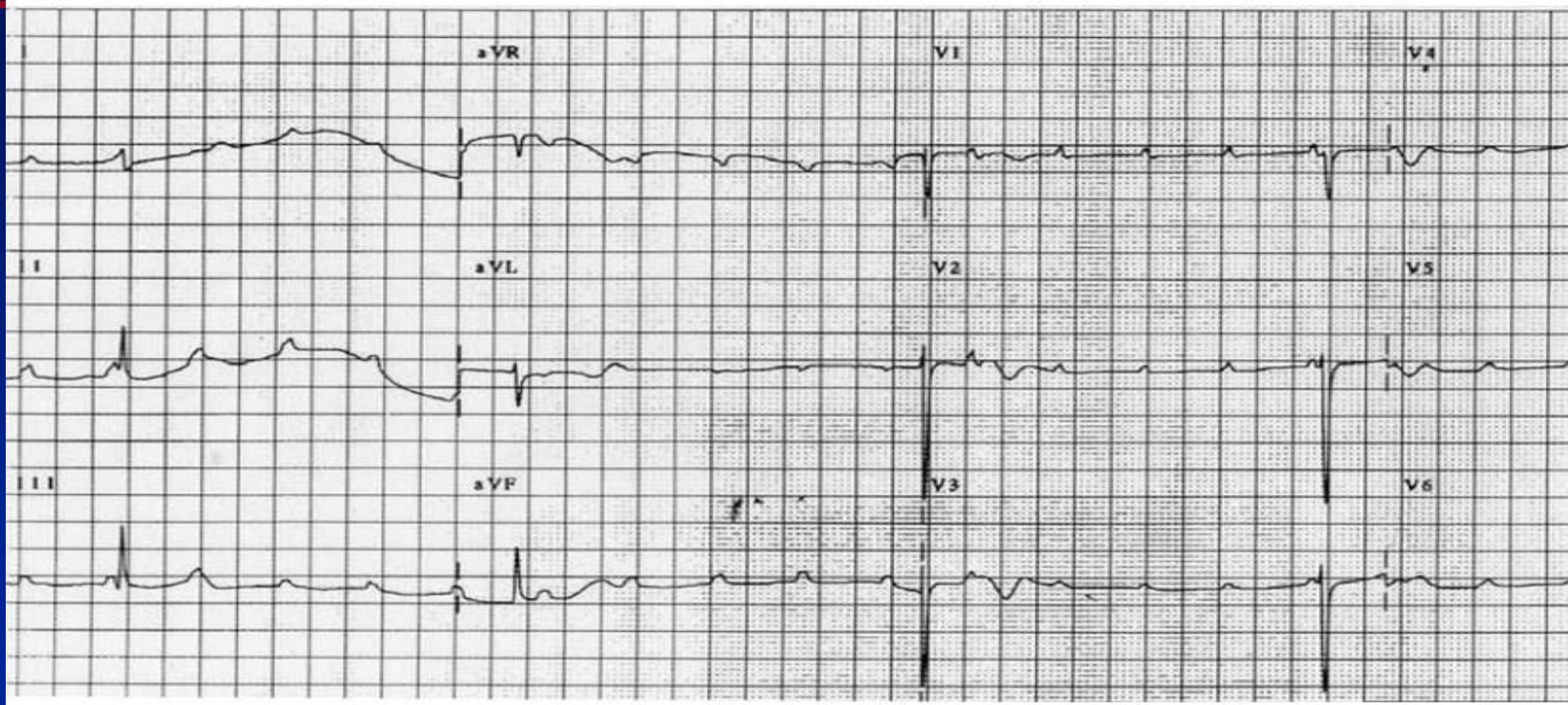
solely by escape pacemakers below the site of block in the AV node ,



The management of CHB is guided by the age of the patient, the degree of symptoms and whether it is acquired or congenital. The acute treatment of symptomatic CHB involves the use of chronotropic agents such as isoproterenol or atropine. If medical therapy is not effective or insufficient, some form of temporary pacing should be initiated until permanent pacemaker implantation can be performed. Patients with congenital CHB and a structurally normal heart may be completely asymptomatic, especially if their escape pacemaker has a relatively normal rate and increases with exercise

a 12-year-old girl who presented with syncope. The ventricular bradycardia is profound, with a rate of 28/min. Her heart was structurally normal and the poor R wave progression in the chest leads probably reflects ventricular dilation. The age of onset of her heart block, and its cause, remain unknown.

However, an ECG in the referring hospital records, when she was seen with a murmur at 4 years of age, also showed complete AV block



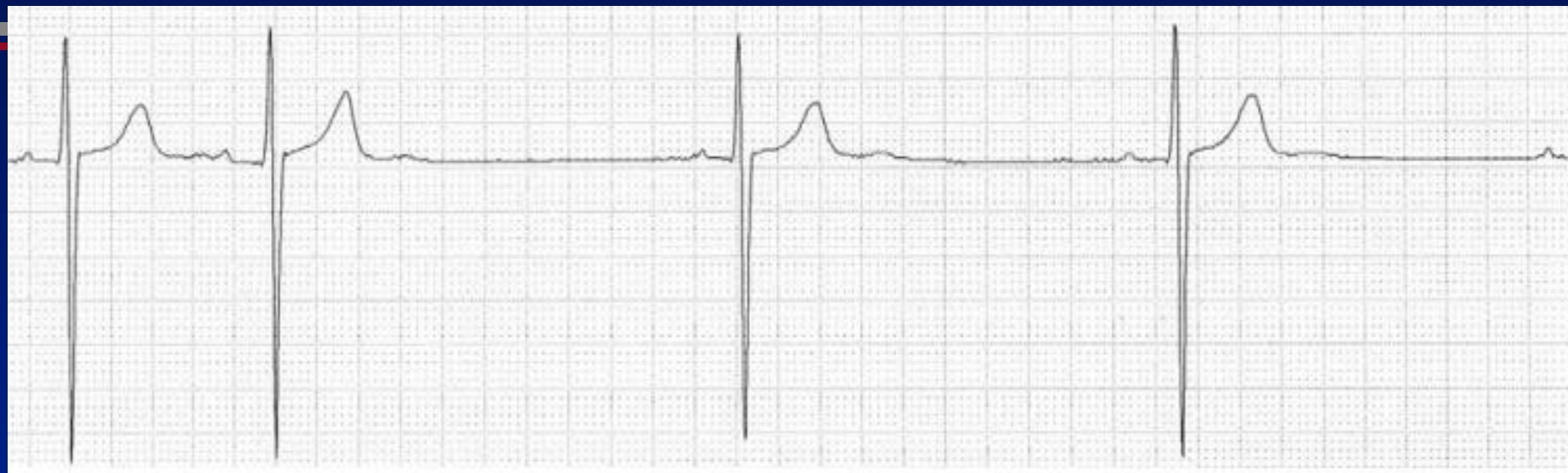
ure 29.5

Sinus node dysfunction and sinoatrial disease

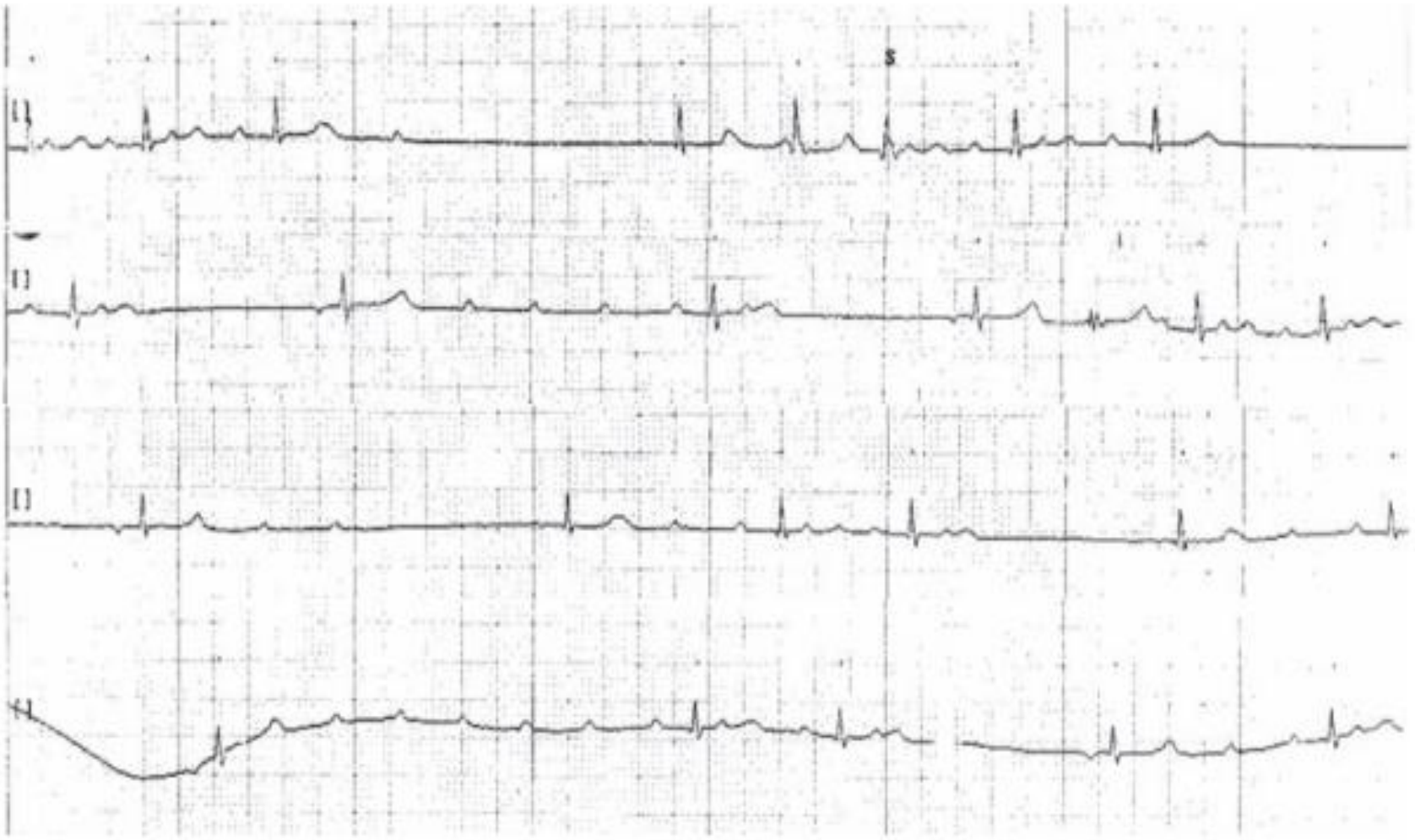
Sinus node dysfunction describes abnormalities of sinus node automaticity and

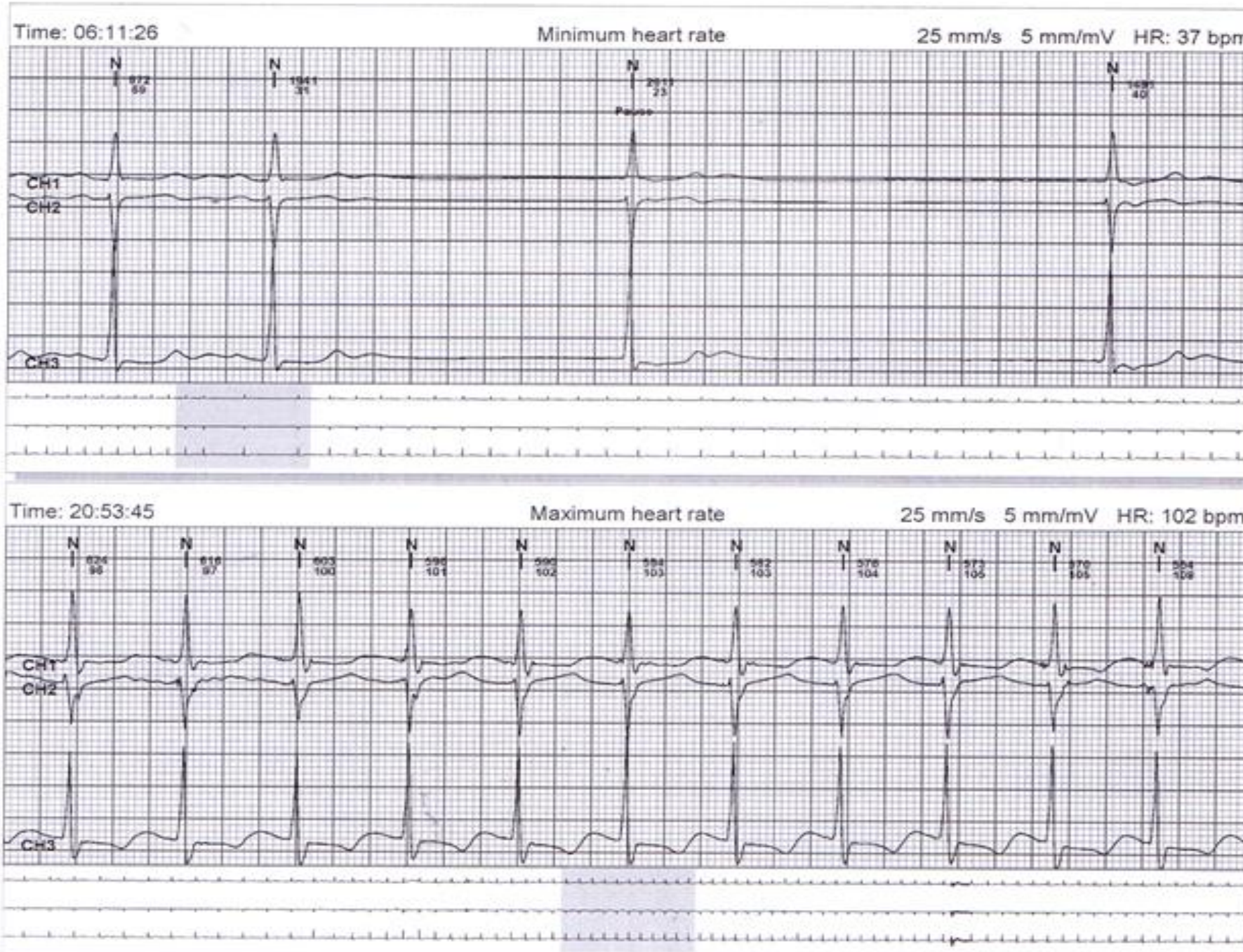
sinoatrial conduction that produce a variety of ECG abnormalities, including sinus

bradycardia, sinus pauses, sinus arrest, sinoatrial block, and atrial tachycardias



igure 30.4




Time: 06:11:26
Minimum heart rate
25 mm/s 5 mm/mV HR: 37 bpm
N
Pause
CH1
CH2
CH3
Time: 20:53:45
Maximum heart rate
25 mm/s 5 mm/mV HR: 102 bpm
CH1
CH2
CH3