Bradyarrhythmias

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Normal Sinus Rhythm

Sinus rhythm is the name given to the normal rhythm of the heart where electrical stimuli are initiated in the SA node, and are then conducted through the AV node and bundle of His, bundle branches and Purkinje fibers.

Depolarization and repolarization of the atria and ventricles show up as 3 distinct waves on ECG.

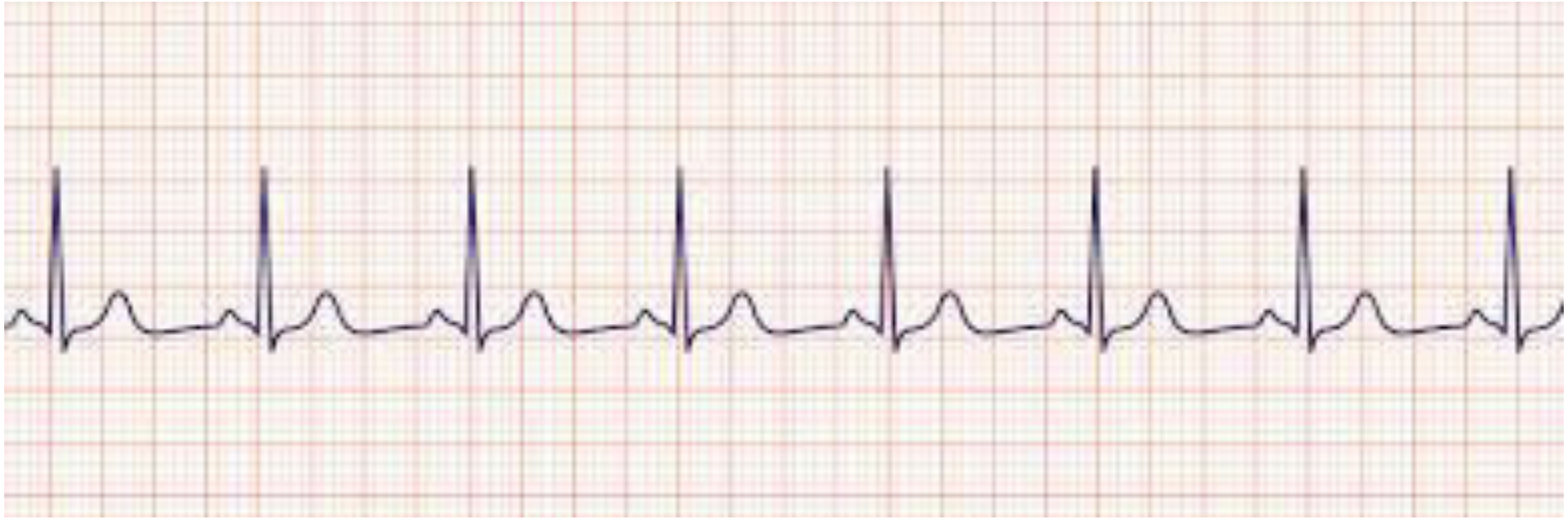
Heart rate between 60 and 100 beats per minute.

In general, sinus arrhythmias can be:

Sinus tachycardia: a faster heart rate, beating faster than 100 beats per minute (bpm)

Sinus bradycardia: a slower heart rate, beating slower than 60 bpm

Normal Sinus Rhythm



ECG features in a Normal Sinus Rhythm:

1) Regular
2) Each QRS comples is preceded by a P-wave in a 1:1 ratio
3) Normal P-wave axis: P-wave is positive in lead II and negative in aVR

How to measure heart rate :

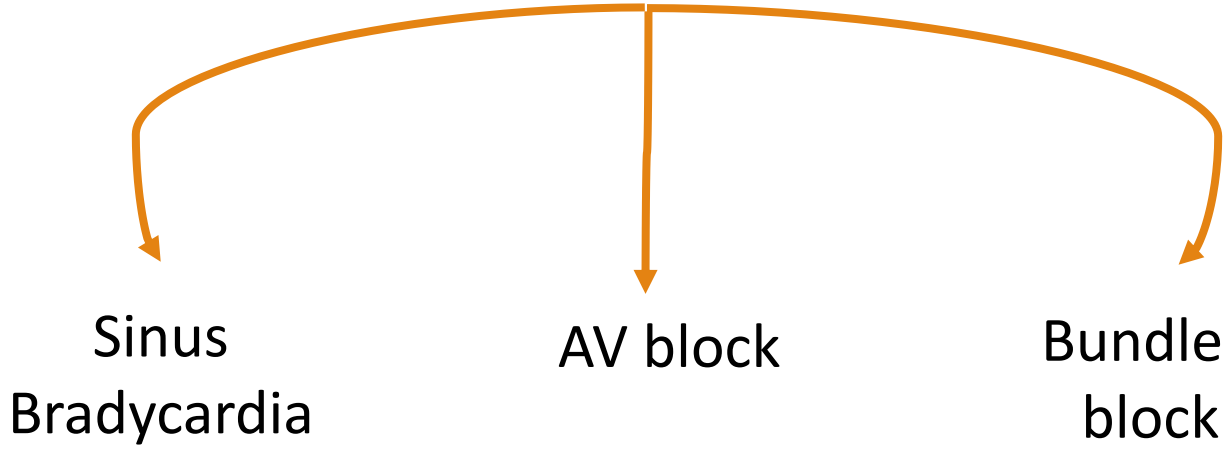
1) Regular: 300/big boxes
2) Irregular: #R in 10 big boxes (2s) X 30

Bradyarrhythmias

Bradyarrhythmia is defined as having a ventricular rate of less than 60 beats per minute (bpm) and has an arrhythmia (irregular beat).

Bradyarrhythmias are a common clinical finding and can be physiological, as in athletes, or caused by a dysfunction in the cardiac conduction system at the level of the sinus node, the atrioventricular node or the His/Purkinje system. The most important prognostic factor is the location of the block and the underlying cardiomyopathy.

Bradyarrhythmias



1) Sinus Bradycardia

Is a rhythm in which the rate of impulses arising from the sinoatrial (SA) node is lower than expected. (Rate less than 60beat/min), and becomes clinically significant when the heart rate is persistently below 45 bpm.

Relative bradycardia is used to explain a heart rate that, although not actually below 60 bpm is considered too low for the patient medical condition. For example 70 bpm for septic patient.

Etiology

1) Physiological:

a) Sleeping

b) Well conditioned athletes

Heart rates as low as 40bpm at rest and 30bpm in sleep can be accepted in asymptomatic trained athletes.

Etiology

2) Pathological:

a) Intrinsic factors:

Degenerative changes causing fibrosis of conduction pathways (risk in elderly patients, may have previous ECGs showing bundle branch block or 1st or 2nd degree heart block),

Post MI- Particularly after an inferior MI.(particularly those involving the right coronary artery as it supplies the SA node)

Sinus node dysfunction/ Sick Sinus Syndrome

Iatrogenic: Ablation, radiation, surgery

Aortic valve disease

Myocarditis, cardiomyopathy, SLE.

Etiology

2) Pathological:

b) Extrinsic factors:

1. Vasovagal- Very common

2. Endocrine-Hypothyroidism, adrenal insufficiency.

3. Metabolic- Hyperkalemia, hypoxia.

4. Other: Hypothermia, increased ICP.

5. Infectious causes :Infectious agents associated with relative sinus bradycardia include Lyme disease, Chagas disease, typhoid fever, typhus, malaria, viral hemorrhagic fevers, and Rocky Mountain Spotted fever

6. Drug induced:Parasympathomimetics(acetylcholine),B-blockers, amiodarone, CCBs(e.g: verapamil,diltiazem), digoxin, cimetidine, Chemotherapeutic agents, Opioids and sedatives.

7. Familial: May represent a manifestation of rare familial forms of sinoatrial node dysfunction, Brugada syndrome and progressive cardiac conduction disease.

Signs and Symptoms

Symptoms

1. Chest discomfort
2. SOB
3. Decreased LOC
4. Weakness and fatigue
5. Lightheadedness and dizziness
6. Presyncope and syncope

Signs

1. Hypotension-orthostatic hypotension
2. Pulmonary congestion on xray and auscultation
3. Features of CHF; Cyanosis, Peripheral edema
4. Features of pulmonary edema; Dyspnea, Rales and crackles

Diagnosis

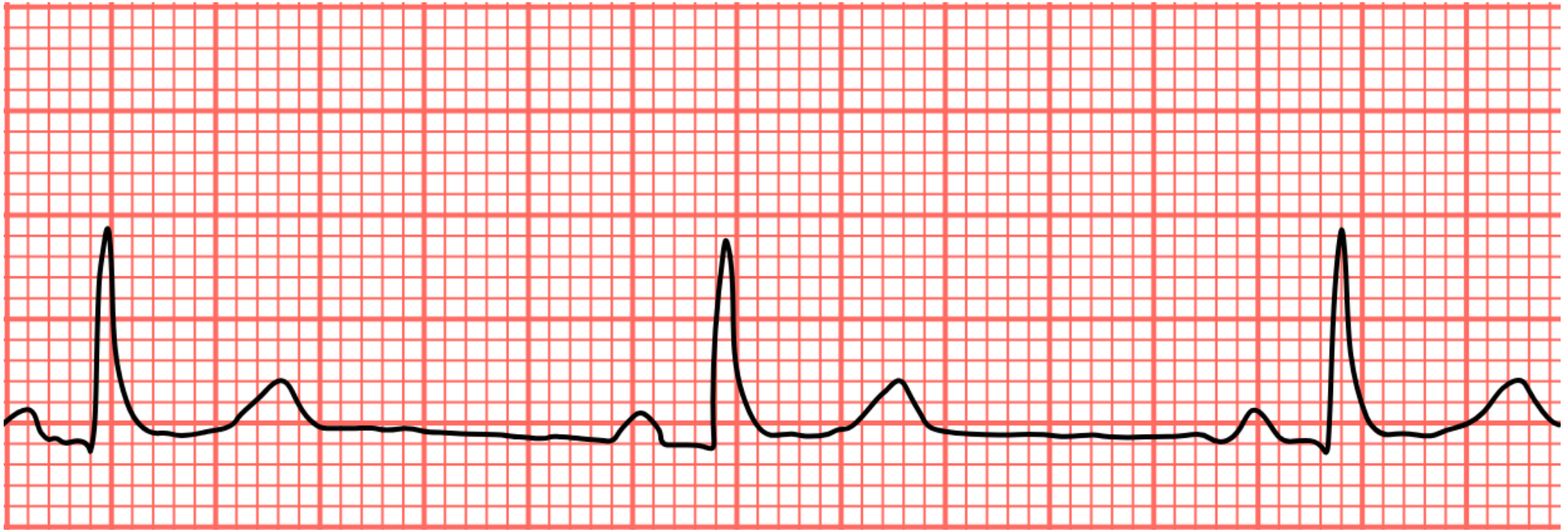
Sinus bradycardia is generally confirmed by ECG after a slow pulse is identified on physical examination, with the diagnosis usually being easy to establish from the surface ECG. An upright P wave in leads I, II and aVL, and a negative P wave in lead aVR, indicates a sinus origin of the bradycardia. It is vital to exclude other causes of bradyarrhythmias such as AV block.

Except for the rate, other features, including P-wave morphology, PR interval, and 1:1 AV conduction, are similar to the normal sinus rhythm.

ECG criteria follows:

- Regular rhythm with ventricular rate slower than 60 beats per minute.
- P-waves with constant morphology preceding every QRS complex.

ECG



Management

Sinus bradycardia usually doesn't need treatment unless you have symptoms.

Hemodynamically Stable vs Unstable?

The aim of the initial evaluation is to establish the presence or absence of symptoms, and any evidence of hemodynamic compromise as a result of the bradycardia. This may include hypotension, chest discomfort, altered mental status, or shortness of breath.

Management

1) Hemodynamically Unstable:

For patients with symptoms and hemodynamically unstable, administer IV atropine (1.0 mg IV push, which can be repeated every 3-5 minutes, if needed, to a total of 3 mg).

If symptoms do not improve, proceed with a temporary pacemaker until finding a reversible cause, if a reversible cause is not found, proceed with permanent pacemaker.

Management

2) Hemodynamically Stable:

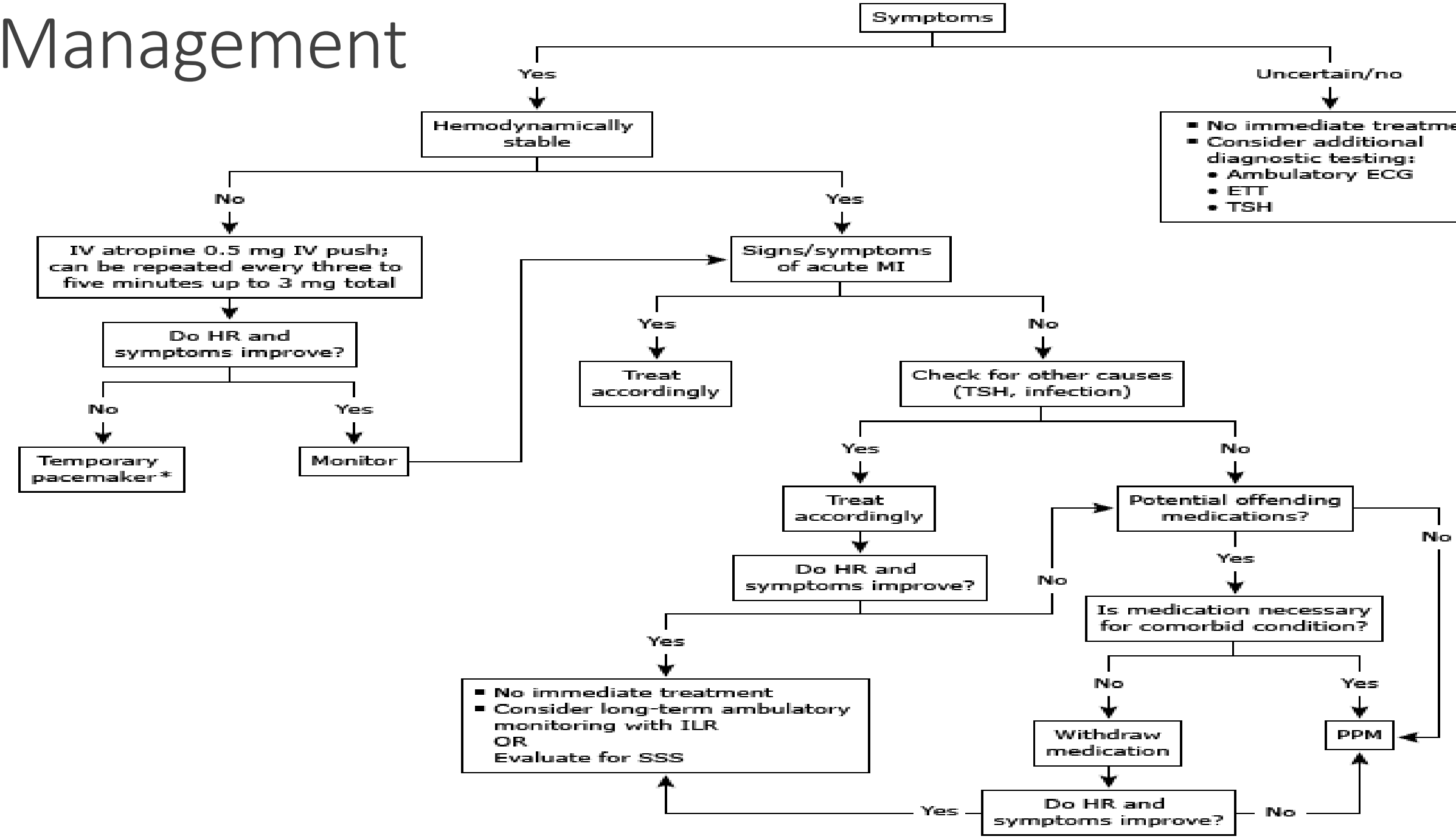
A. Patients with signs and symptoms of acute myocardial infarction should be treated accordingly.

B. Patients with evidence of another systemic condition associated with sinus bradycardia (eg, hypothyroidism, infection…..) should be treated accordingly.

C. Patients in whom a medication is suspected to be causing the symptomatic bradycardia should have the medication withheld. If the medication is mandatory for the treatment of comorbid condition as in beta blockers for a severe angina, a permanent pacemaker should be required.

If the symptoms resolve and the heart rate improves following the withdrawal of the suspected offending agent, no additional treatment is required.

Management


Symptoms
Yes
Uncertain/no
Hemodynamically stable
No immediate treatment
Consider additional diagnostic testing:
Ambulatory ECG
ETT
TSH
No
Yes
IV atropine 0.5 mg IV push; can be repeated every three to five minutes up to 3 mg total
Signs/symptoms of acute MI
Do HR and symptoms improve?
Yes
No
Treat accordingly
Check for other causes (TSH, infection)
No
Yes
Temporary pacemaker*
Monitor
Yes
No
Treat accordingly
Potential offending medications?
Do HR and symptoms improve?
No
Yes
No
Is medication necessary for comorbid condition?
Yes
No
Yes
No immediate treatment
Consider long-term ambulatory monitoring with ILR
OR
Evaluate for SSS
Withdraw medication
PPM
Yes
Do HR and symptoms improve?
No

Sick Sinus Syndrome

Sick sinus syndrome, also known as sinus node dysfunction (SND), is a disorder of the sinoatrial (SA) node caused by impaired pacemaker function and impulse transmission producing a constellation of abnormal rhythms. These include atrial bradyarrhythmias, atrial tachyarrhythmias and, sometimes, bradycardia alternating with tachycardia often referred to as “tachy-brady syndrome.” These arrhythmias may result in palpitations and tissue under-perfusion leading to fatigue, lightheadedness, pre-syncope, and syncope.

These patients usually need a permanent pacemaker inserted.

Atrioventricular block

Atrioventricular block

- Is defined as a delay or interruption in the transmission of an impulse from the atria to the ventricles due to an anatomical or functional impairment in the conduction system
- The conduction disturbance can be transient or permanent.

Atrioventricular block may be partial or complete:

- First-degree and second-degree blocks are partial.
- Third degree blocks are complete.

Etiology

Physiologic AV block:

AV block can result from physiologic slowing of cardiac conduction in response to increased parasympathetic nervous system output. Enhanced vagal tone due to athletic training, sleep, pain, carotid sinus massage, or carotid sinus hypersensitivity syndrome can result in slowing of the sinus rate and/or the development of AV conduction disturbances. In general, enhanced vagal tone leads to lower degrees of AV block (ie, first degree or Mobitz type I second degree); higher degree AV block that occurs in the setting of enhanced vagal tone could suggest other pathologic contributions to AV conduction disturbance.

Pathophysiologic AV block:

- Fibrosis and sclerosis of the conduction system, which appears idiopathic, accounts for about one-half of cases of AV block**,** they are the most common cause of AV Block.
- Idiopathic AV conduction abnormalities are characterized by progressive impairment of the conduction system which occurs gradually over decades:

1. Lenegre disease – The term Lenegre disease has been traditionally used to describe a progressive, fibrotic, sclerodegenerative affliction of the conduction system in younger(age <60) individuals. Lenegre disease is frequently associated with slow progression to complete heart block and may be hereditary.

2. Lev disease – The term Lev disease has been used to refer to "sclerosis of the left side of the cardiac skeleton" in older patients (age >70 years old), such as that associated with calcific involvement of the aortic and mitral rings. Lev disease is caused by fibrosis or calcification extending from any of the fibrous structures adjacent to the conduction system into the conduction system.

Depending upon the anatomic location of the areas of fibrosis and sclerosis, various conduction abnormalities can result:

- Fibrosis of the superior and basal aspect of the muscular septum is a common cause of right bundle branch block (RBBB) with left anterior fascicular block in the older adult.
- Involvement of the mitral ring or the central fibrous body, for example, may be the most common cause of complete heart block with a narrow QRS complex in the older adult.
- Aortic valve calcification, on the other hand, can invade the bundle of His, the right and/or left bundle branch as well as the left anterior fascicle. Thus, the QRS complex may be prolonged.

Associated with other cardiac disease

- **Ischemic heart disease:** Ischemic heart disease accounts for about 40 percent of cases of AV block . Conduction can be disturbed with either chronic ischemic heart disease or during an acute myocardial infarction (MI).
- **Cardiomyopathies and myocarditis:** – AV block can be seen in patients with cardiomyopathies, including hypertrophic obstructive cardiomyopathy and infiltrative processes such as amyloidosis and sarcoidosis, and in patients with myocarditis due to a variety of causes including rheumatic fever, SLE, bacterial endocarditis. Patients with COVID-19-associated myocarditis have been observed to have varying degrees of AV block.
- **Congenital heart disease:** AV block of varying degrees can be associated with a variety of congenital heart defects that result in structural abnormalities (congenitally corrected transposition of the great arteries, large AV septal defects). Additionally, complete heart block may be an isolated lesion, not associated with structural heart disease, this is most commonly associated with neonatal lupus.

Familial AV block

- Familial AV block, characterized by a progression in the degree of AV block in association with a variable apparent site of block, may be transmitted as an autosomal dominant trait.
- One form of AV conduction block has been mapped to a genetic locus at chromosome 19q13 and the other to chromosome 3p21, where the cardiac sodium channel, SCN5A, is encoded.

Miscellaneous causes

- Hyperkalemia, usually when the plasma potassium concentration is above 6.3 meq/L.
- Hyperthyroidism and hypothyroidism, myxedema, and thyrotoxic periodic paralysis.
- Hereditary neuromuscular degenerative disease such as myotonic dystrophy and Erb's dystrophy.
- Cardiac tumors, cysts, myocardial bridging, and trauma .
- Rheumatologic disorders including dermatomyositis.

IATROGENIC AV BLOCK

A. Medication

- Beta blockers
- Non-dihydropyridine calcium channel blockers (especially verapamil and to a lesser extent diltiazem)
- Digoxin
- Adenosine
- Antiarrhythmic medications, commonly amiodarone but also drugs that modulate the sodium channel (eg, quinidine, procainamide, etc)

B. Cardiac procedures

- Open heart surgery
- Transcatheter aortic valve implantation (TAVI)
- Catheter ablation for arrhythmias
- Alcohol (ethanol) septal ablation

AV Block Degrees

First-Degree AV Block

Delayed conduction from the atrium to the ventricle (defined as a prolonged PR interval of >200ms – 5 small sq).

Every QRS complex is preceded by a single P wave.

If >300ms: can progress rapidly to complete heart block.

May be a normal variant

It rarely causes symptoms and does not usually require treatment.

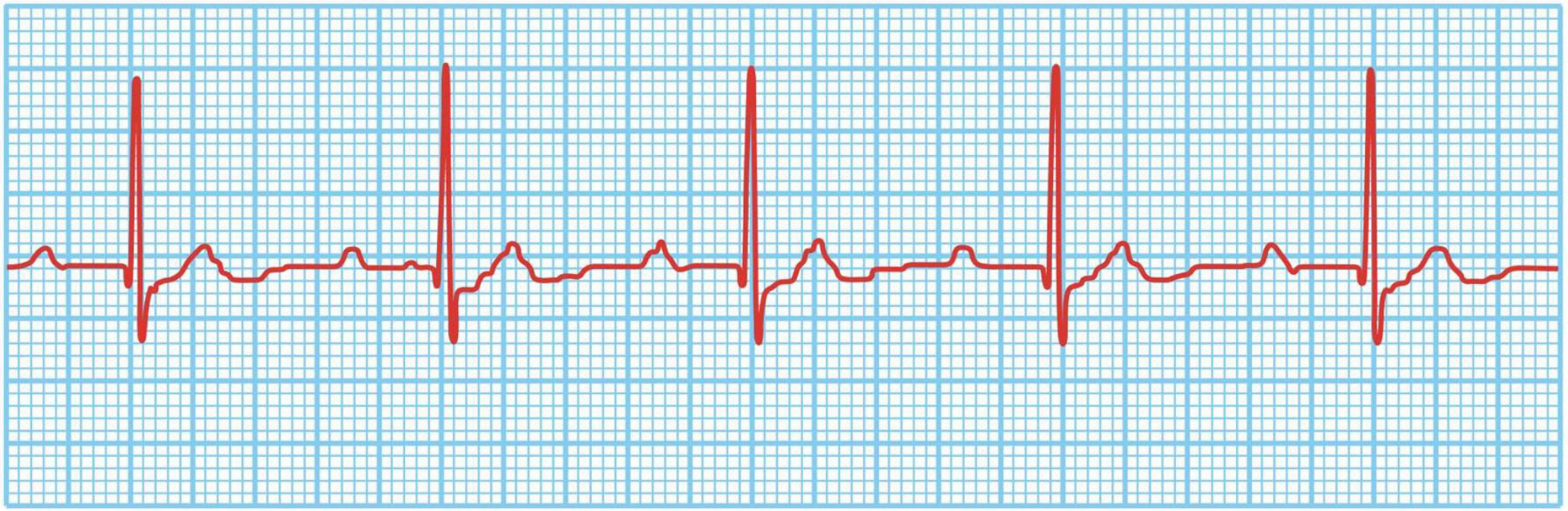


Fig. 16.44 First-degree atrioventricular block. The PR interval is prolonged and measures 0.26 sec.

Pacemaker syndrome and Pseudo-pacemaker syndrome

Pacemaker syndrome is a phenomenon in which a patient feels symptomatically worse after pacemaker placement and presents with progressively worsening symptoms of congestive heart failure (CHF). This is mainly due to the loss of atrioventricular synchrony whereby the pathway is reversed and now has a ventricular origin.

It describes the uncomfortable awareness of one's heart beat due to atrial contraction against a closed mitral valve or when atrial contraction occurs shortly after ventricular systole with incomplete atrial filling.

Pseudo-pacemaker syndrome refers to a similar constellation of symptoms that can occur with first degree AV block and other heart rhythms that have AV dissociation

Second-Degree AV Block

Intermittent atrial conduction to the ventricle.

Not every atrial impulse is able to pass through the AV node into the ventricles which results in dropped beats occurring.

There are 2 subtypes:

1. Mobitz I (Wenckebach block)

2. Mobitz II (Hay block)

Mobitz type I- Wenckebach phenomenon

Progressive lengthening of successive PR intervals, culminating in a dropped beat. The cycle then repeats itself. This is known as the Wenckebach phenomenon and is usually due to impaired conduction in the AV node itself.

The phenomenon may be physiological and is sometimes observed at rest or during sleep in athletic young adults with high vagal tone.

Rarely progresses to 3rd degree heart block.

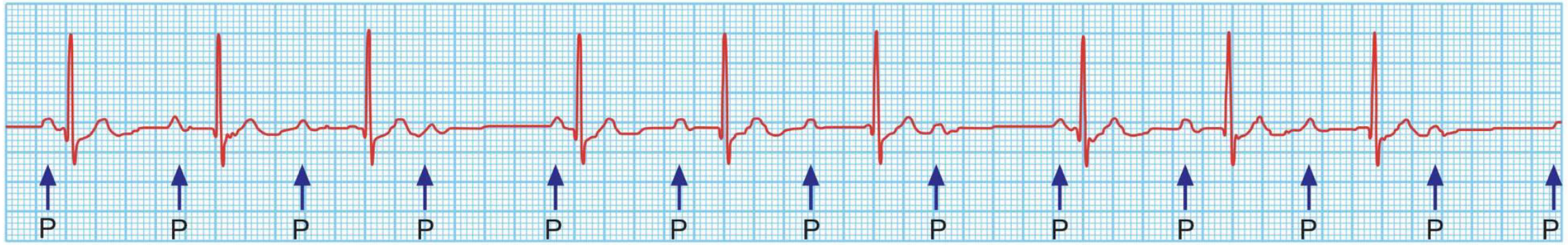


Fig. 16.45 Second-degree atrioventricular block (Mobitz type I – the Wenckebach phenomenon). The PR interval progressively increases until a P wave is not conducted. The cycle then repeats itself. In this example, conduction is at a ratio of 4:3, leading to groupings of three ventricular complexes in a row.

Mobitz type 1- Wenckebach phenomenon

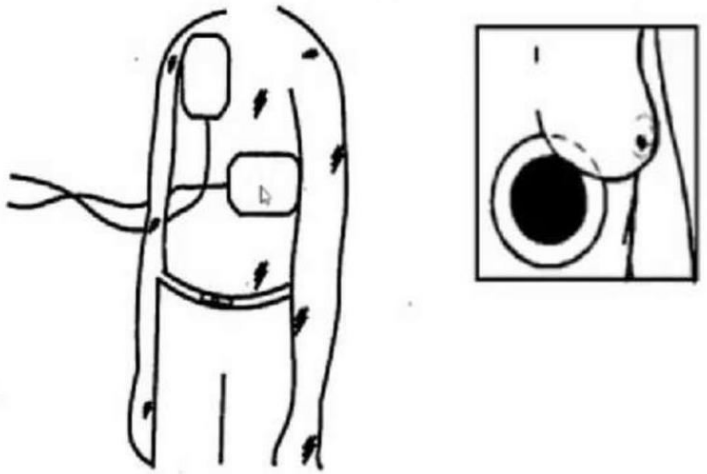
Management:

Depends on the presence or absence of symptoms*

Symptomatic and Unstable:

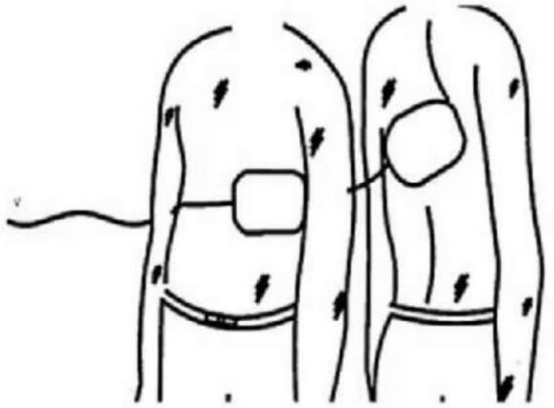
Urgently treated with Atropine (0.5 mg IV), which may be repeated every three to five minutes to a total dose of 3 mg.

Temporary cardiac pacing if not responsive to atropine (either with transcutaneous or, if immediately available, trans-venous pacing).



Antero-lateral Pacer
Pad Placement

Antero-posterior Pacer
Pad Placement



Mobitz type 1- Wenckebach phenomenon

Symptomatic and Stable:

Do not require urgent therapy with Atropine or temporary cardiac pacing.

However, patients should be continuously monitored with transcutaneous pacing pads in place in the event of clinical deterioration.

Asymptomatic:

Do not require any initial treatment.

Mobitz type 2- Hay AV Block

Occurs when the dropped QRS complex is not preceded by progressive PR prolongation.

The PR interval of the conducted impulses remains constant

RR interval surrounding the dropped beat is an exact multiple of the preceding RR interval.

It is more serious and often signifies a serious heart disease. (Can progress to 3rd block)

Management: needs immediate admission for cardiac monitoring, backup temporary pacing and ultimately insertion of a permanent pacemaker.

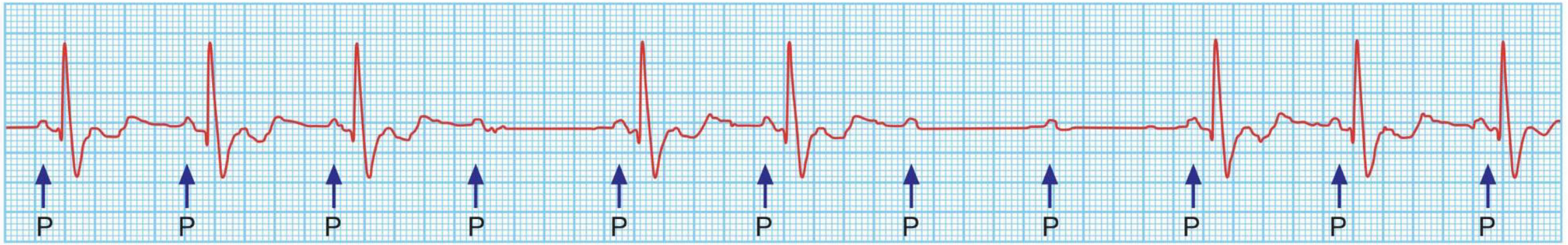


Fig. 16.46 Second-degree atrioventricular block (Mobitz type II). The PR interval of conducted beats is normal but some P waves are not conducted. The constant PR interval distinguishes this from the Wenckebach phenomenon.

Fixed Ratio AV Blocks

Is a 2nd degree heart block with a fixed ratio of P waves: QRS complexes.

E.g. 2:1, 3:1, 4:1

May result from either Mobitz I or Mobitz II.

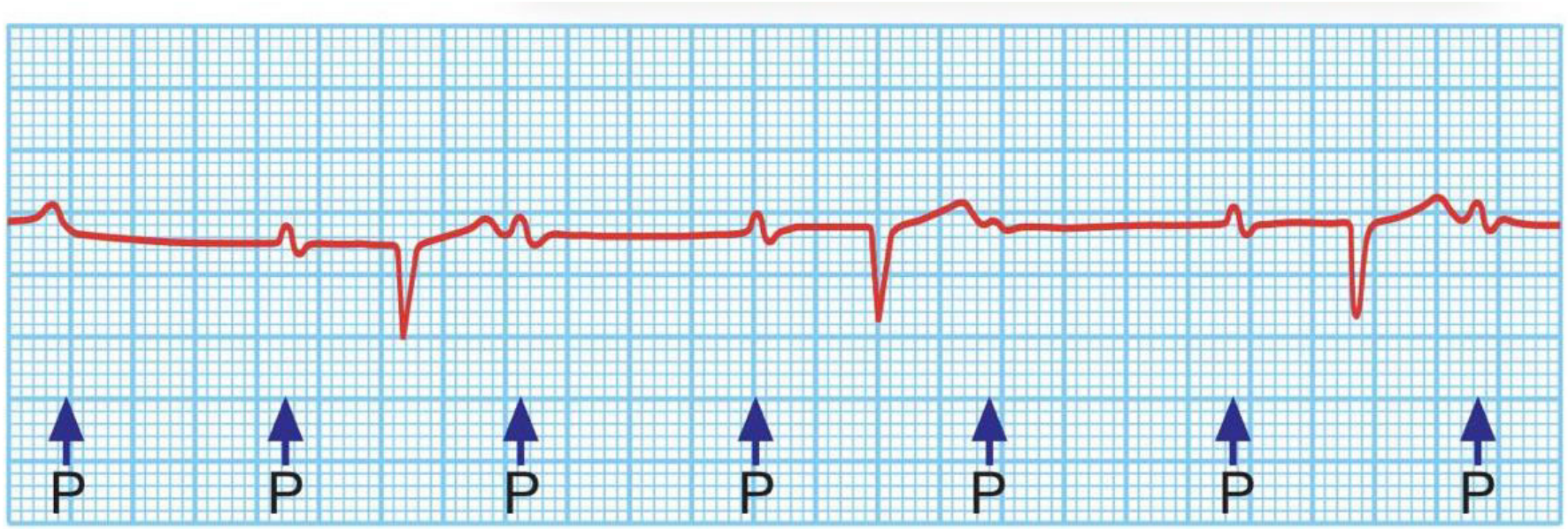


Fig. 16.47 Second-degree atrioventricular block with fixed 2:1 block. Alternate P waves are not conducted. This may be due to Mobitz type I or II block.

Third-Degree AV Block

Conduction fails completely and the atria and ventricles beat independently. This is known as AV dissociation.

Ventricular activity is maintained by an escape rhythm arising in the AV node or bundle of His (narrow QRS complexes) or the distal Purkinje tissues (broad QRS complexes). Distal escape rhythms tend to be slower and less reliable.

Third-Degree AV Block

Third Degree AV Block is electrocardiographically characterized by:

- Regular P-P interval (60-100)
- Regular R-R interval (30-45)
- Lack of relationship between P waves and QRS complexes
- More P waves are present than QRS

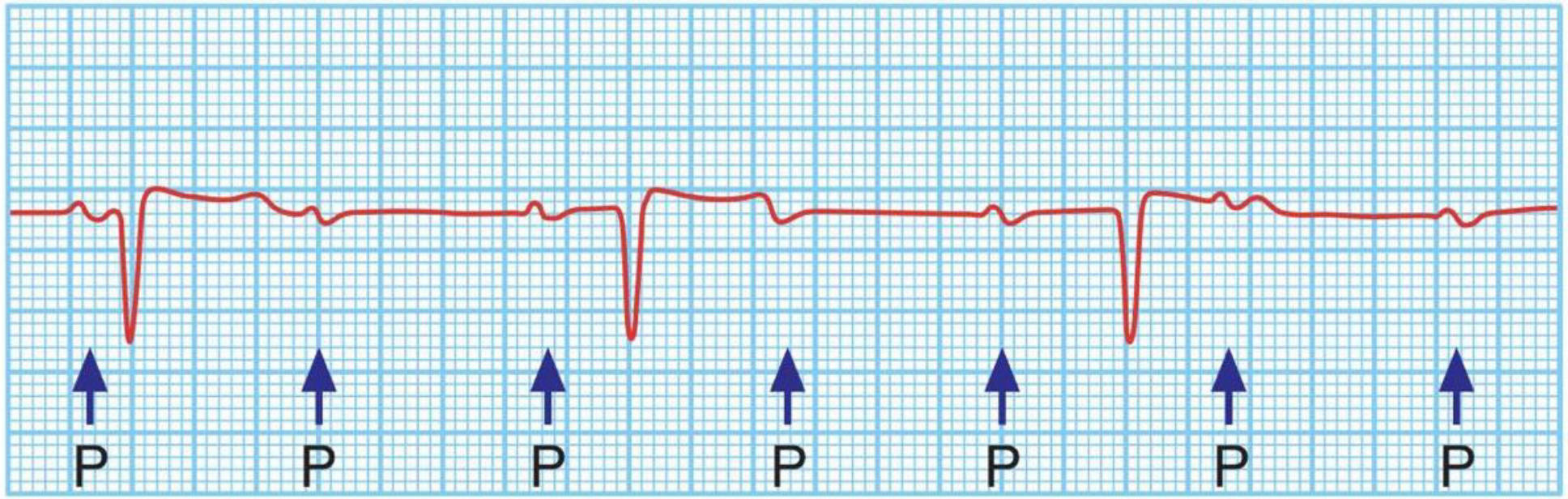


Fig. 16.48 Complete (third-degree) atrioventricular block. There is complete dissociation of atrial and ventricular complexes. The atrial rate is 80/min and the ventricular rate is 38/min.

COMPLETE HEART BLOCK

» CAUSES-

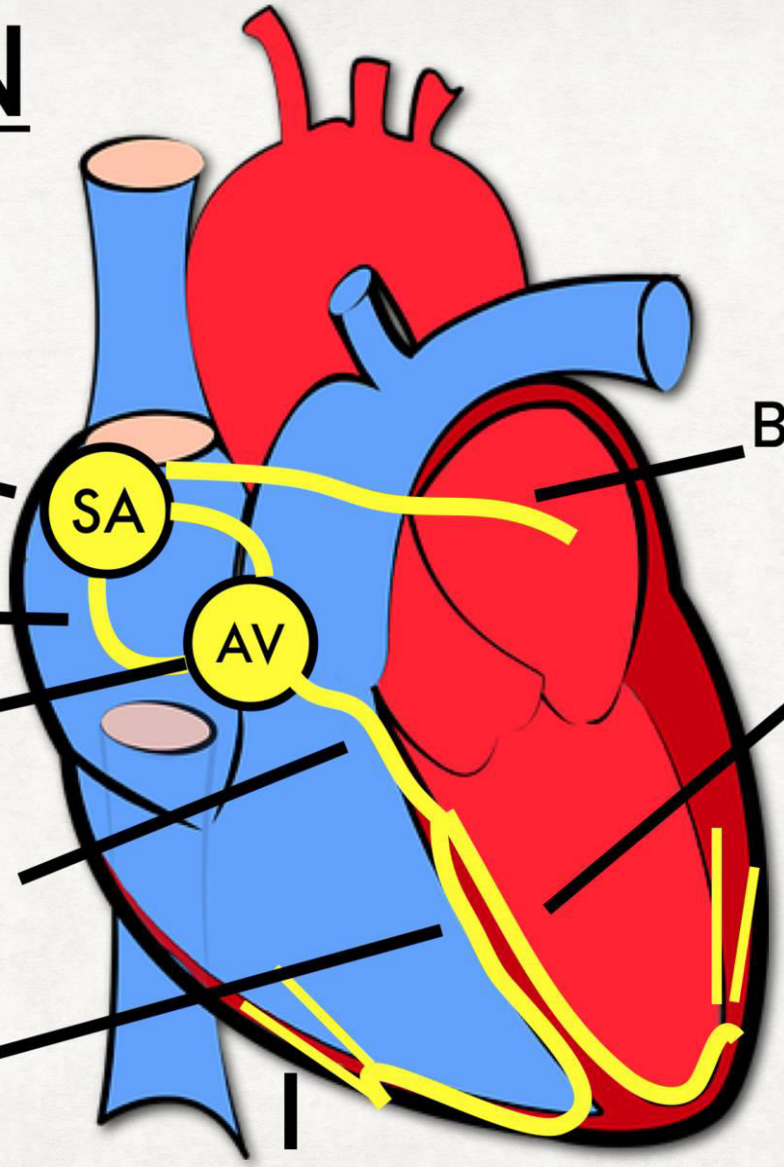
- Idiopathic / conduction tissue fibrosis.
- congenital.
- ischaemic heart disease.
- Associated with Aortic valve calcification.
- cardiac surgery & trauma.
- digoxin intoxication.
- bundle interruptions by tumors, parasites,granulomas, injury, abscess.

The level of block

In third degree (complete) AV block, the escape rhythm that controls the ventricles can occur at any level below that of the conduction block and the morphology of the QRS complex can help to determine the location where this is occurring

CONDUCTION

1. SA Node
2. AV Node
3. Bundle of His
4. R/L Bundle Branches
5. Purkinje Fibers



SA Node

Internodal Pathway

AV Node

Bundle of His

Right Bundle Branch

Purkinje Fibers

Bachmann's Bundle

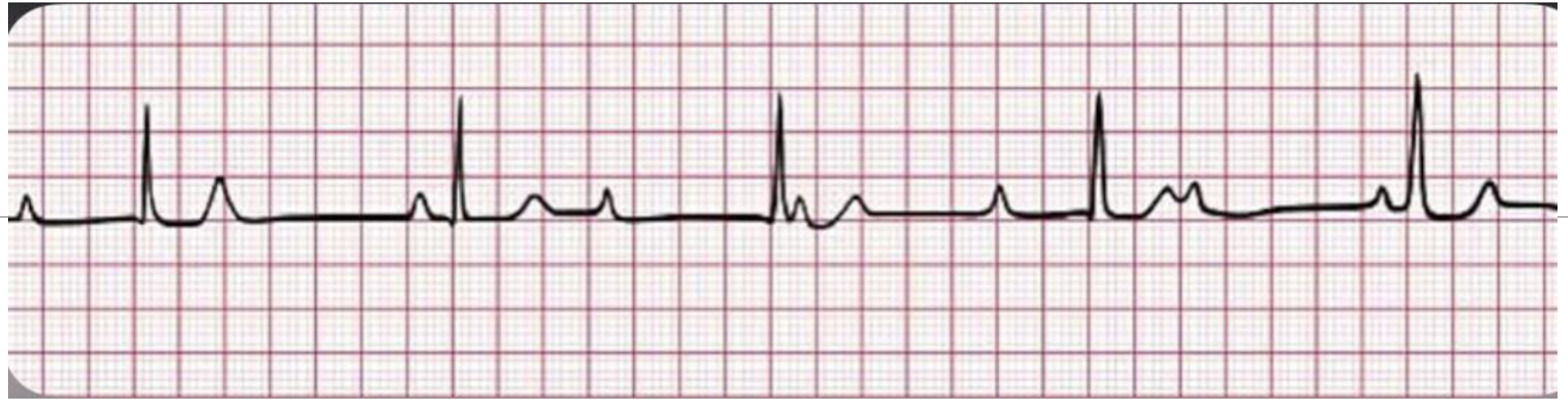
Left Bundle Branch

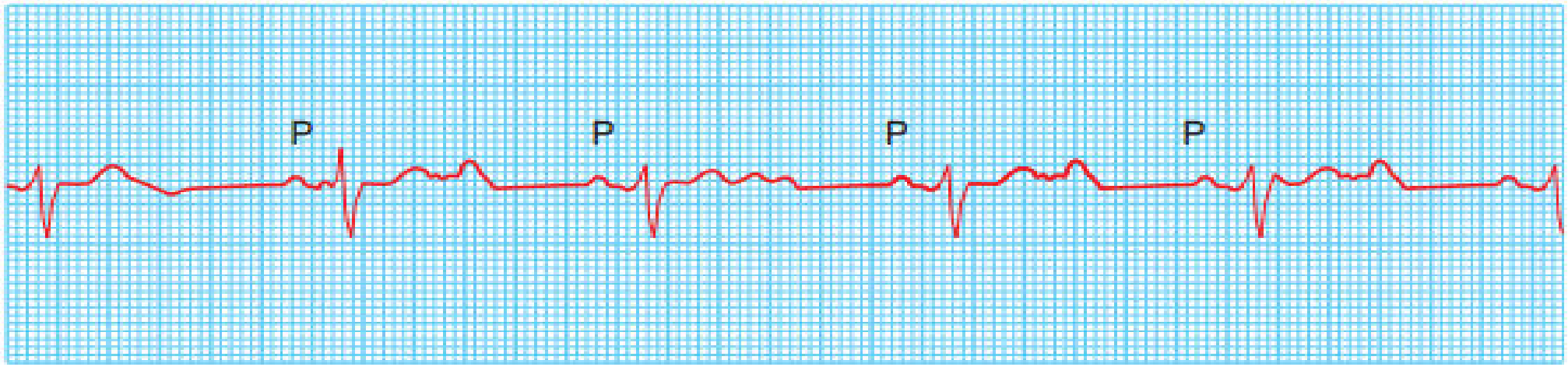
Purkinje Fibers

1) Narrow complex escape rhythm :

QRS complex < 0.12 s

If the block occurs above or at the crest of the AV node, a junctional rhythm will take over and drive the ventricles. The resulting QRS complexes will be narrow and occur at the intrinsic rate of the AV node (40 to 55 beats/minute).

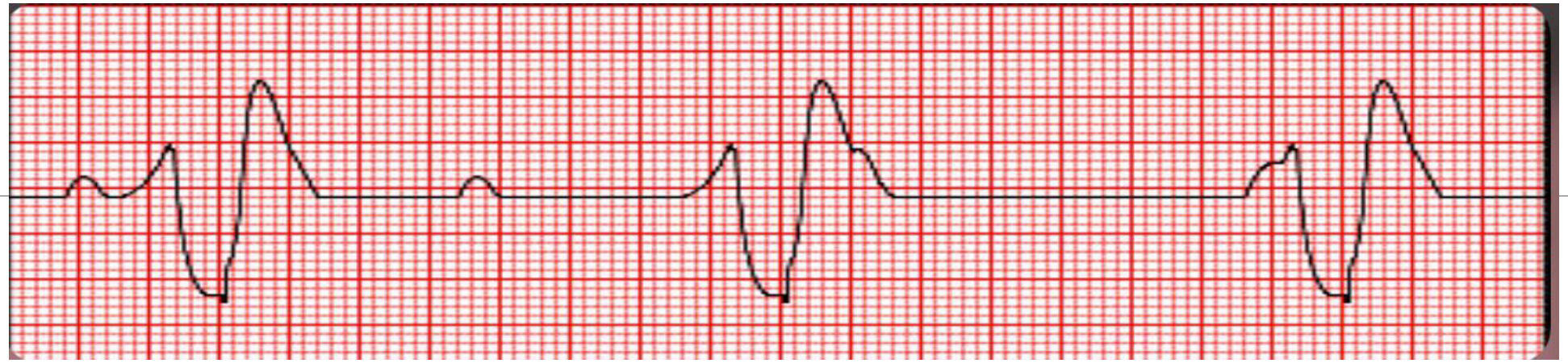


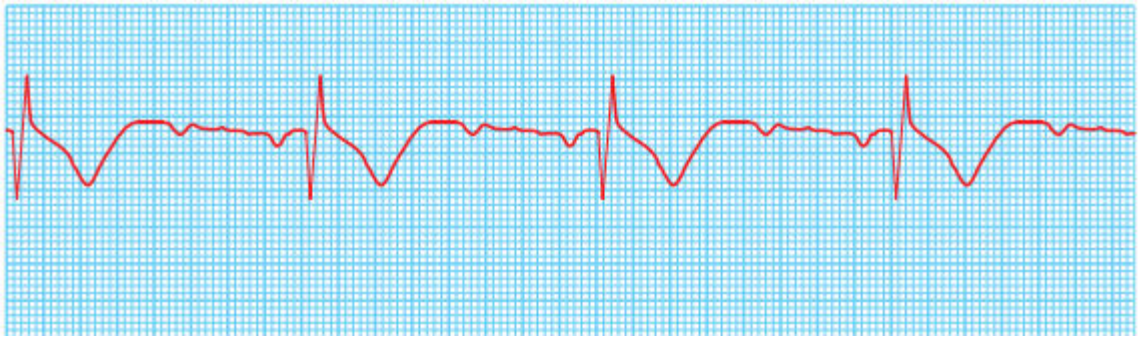

P
P
P
P

2) Broad complex escape rhythm

QRS complex > 0.12 s

- Whereas if the block occurs below the AV node, a ventricular pacemaker must take over. In such cases, the QRS complexes will be wide and at the intrinsic rate of the ventricular pacemaker (20 to 40 beats/minute).





Clinical presentation

The clinical presentation of third degree (complete) AV block is variable depending upon:

- the rate of the underlying escape rhythm

- and the presence of comorbid conditions.

Symptoms

Patients with **narrow complex escape rhythms** are more likely to have minimal symptoms.

Fatigue

Dizziness

exercise intolerance

Chest pain

Patients are profoundly symptomatic, especially if a **wide-complex escape rhythm** is present, In such cases, symptoms can include the following:

Syncope

Confusion

Dyspnea

Severe chest pain

Hypotension

Sudden death

Management

The initial management of the patient with third degree (complete) AV block depends on the presence and severity of the signs and symptoms related to the ventricular escape rhythm.

For an Unstable patient :

- **Urgently Atropine**
  - Initial dose 0.5 mg IV
  - This dose may be repeated every three to five minutes to a total dose of 3 mg
- **Temporary cardiac pacing**
  - Either with transcutaneous or, if immediately available, transvenous pacing
- **Dopamine** may be administrated in hypotensive patients
- **Dobutamine** an option for patients with heart failure symptoms.

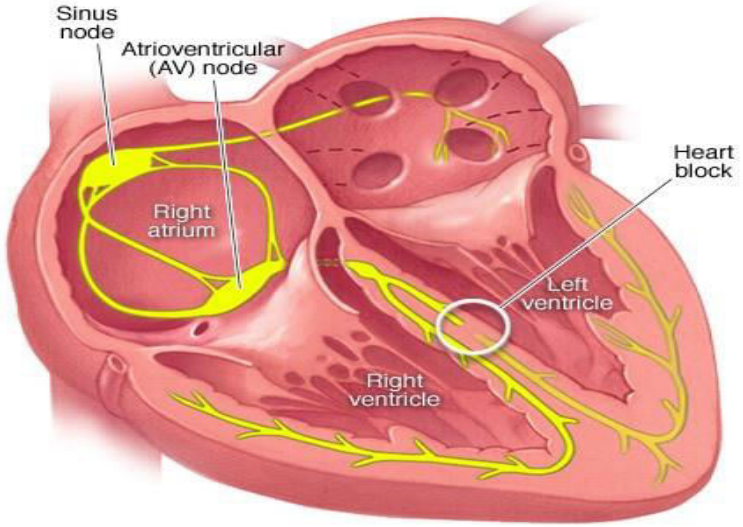
For a Stable patients :

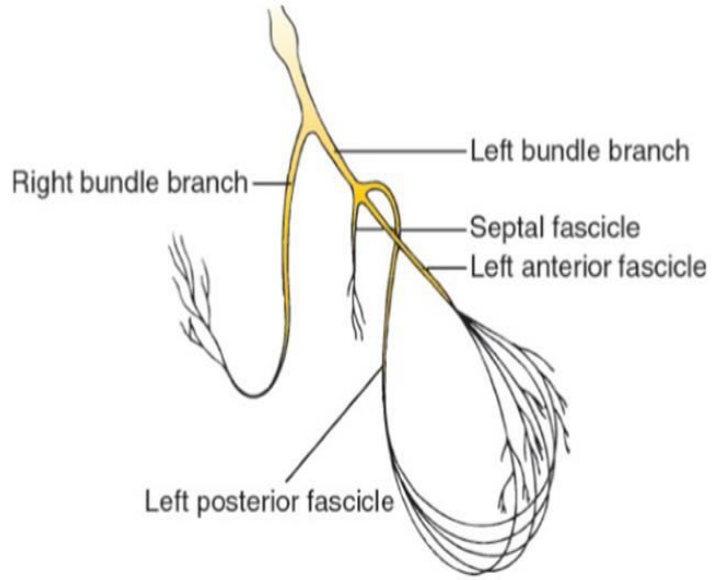
Identification and treatment of any reversible causes like:

- Acute myocardial infarction should be treated with temporary pacing and revascularization.
- Medication-induced should be observed while the offending agents are withdrawn
- Hyperkalemia
- Hypothyroidism

If no reversible causes are present, **definitive treatment** of third degree (complete) AV block involves **permanent pacemaker placement** in most patients.

Bundle Branch Block





The anatomy of the ventricular bundle branches.

Bundle Branch Block

Impulses, or electrical signals, travel through both the left and right chambers of your heart to make it pump. But if the pathway is blocked, the impulses may move slower than normal or irregularly. This is called a bundle branch block.

There are two types:

Right bundle branch block (RBBB)

Left bundle branch block (LBBB)

Types of bundle branch block :

1- Partial bundle branch block:

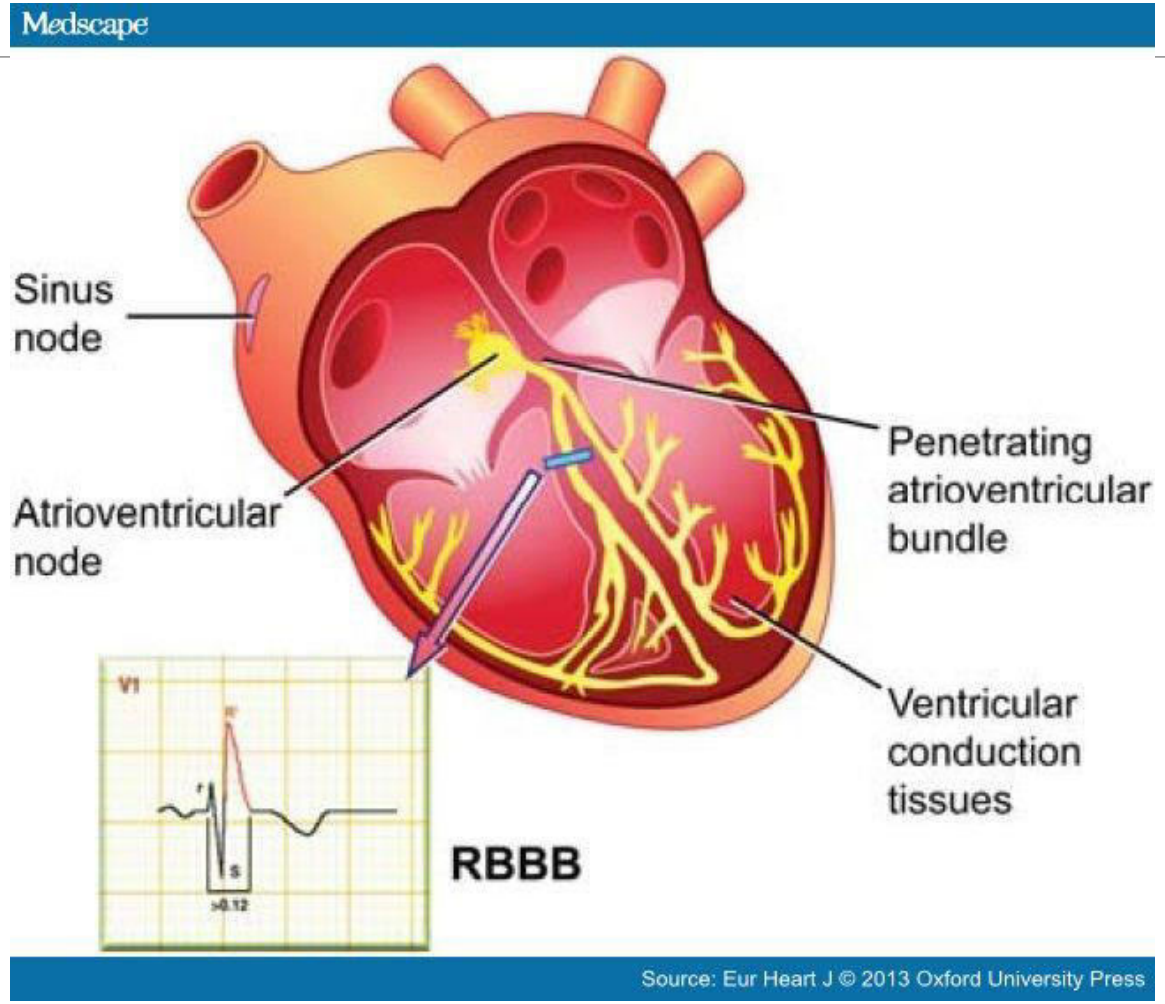
Slight widening of the QRS complex (0.1 -0.12 s) also called incomplete BBB

2- Complete bundle branch block:

This is associated with a wider QRS complex (more than 0.12 s) this can be on the right or left

Right Bundle Branch Block

This is the most common type.

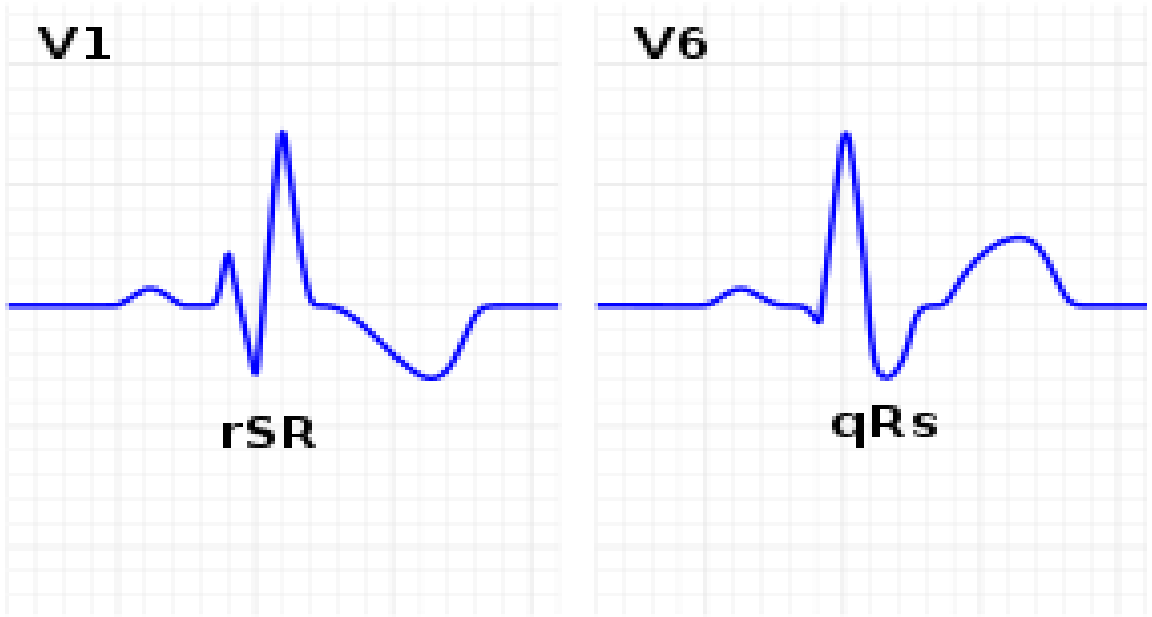


Causes of Right Bundle Branch Block :

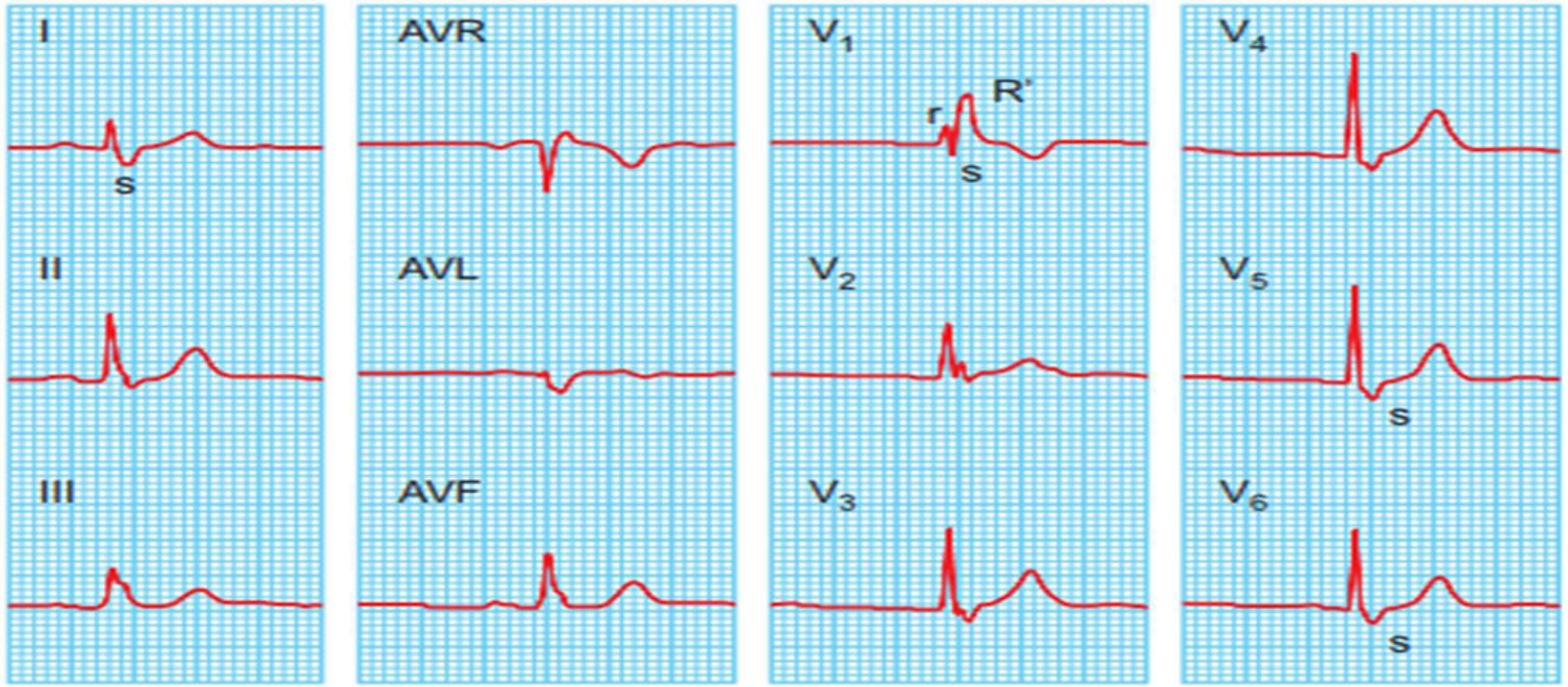
- Pulmonary embolism (a blood clot in the lungs)
- Heart attack
- Congenital heart defect (problems you were born with, like a hole in the wall between the upper chambers of the heart)
- High blood pressure in the arteries
- Infection of the heart muscle (myocarditis)
- Idiopathic

Right Bundle Branch Block

There is late activation of the right ventricle , seen in deep S wave in lead I & V6 and as a tall R wave in V1



Right Bundle Branch Block



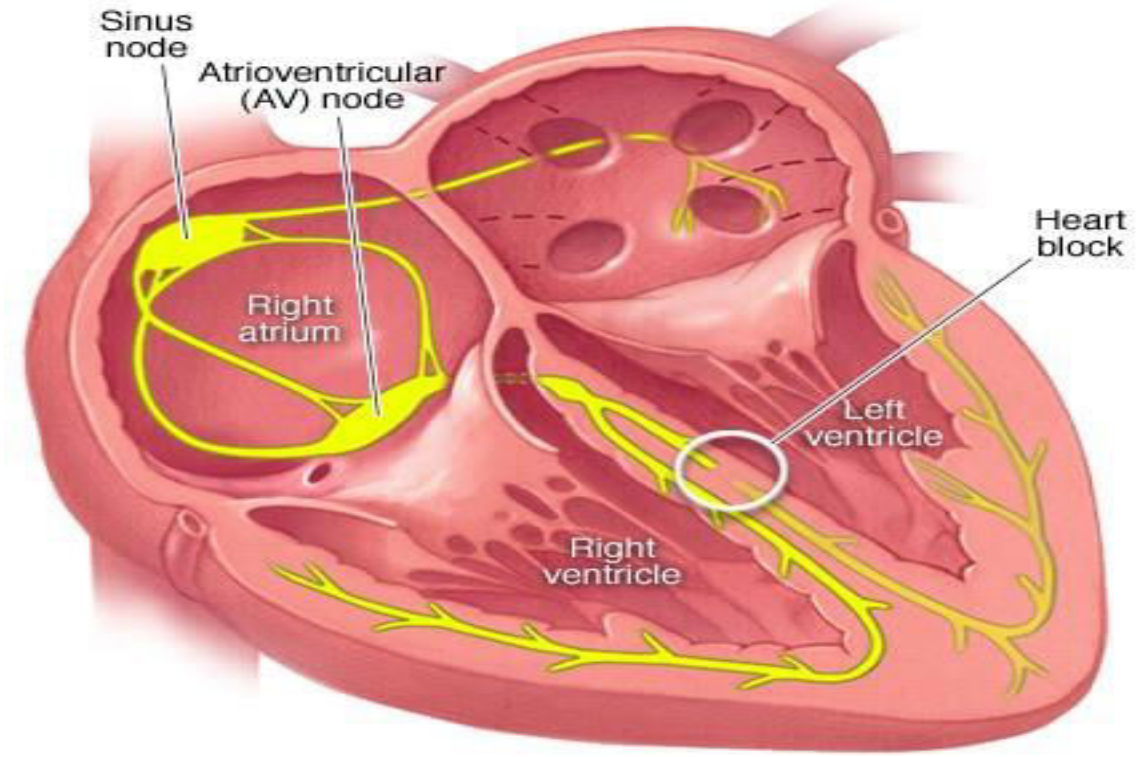
Criteria for RBBB

- ✓ QRS duration ≥ 120 milliseconds
- ✓ RSR' pattern in leads V1 and V2
- ✓ Greater duration of S wave than R wave in leads I and V6 or the S wave greater than 40 ms
- ✓ R wave peak time > 50 ms in V1
- ✓ Normal R wave peak time in leads V5 and V6

Right Bundle Branch Block

Right bundle branch block is usually asymptomatic and is typically found incidentally on ECG. The ECG finding itself does not cause any signs or symptoms. On physical examination, the patient may have a split second heart sound.

Left bundle branch block



Causes of Left bundle branch block :

Many of them are the same as RBBB and can include:

Left ventricular outflow obstruction

Aortic stenosis

Hypertension

Heart attack

High blood pressure

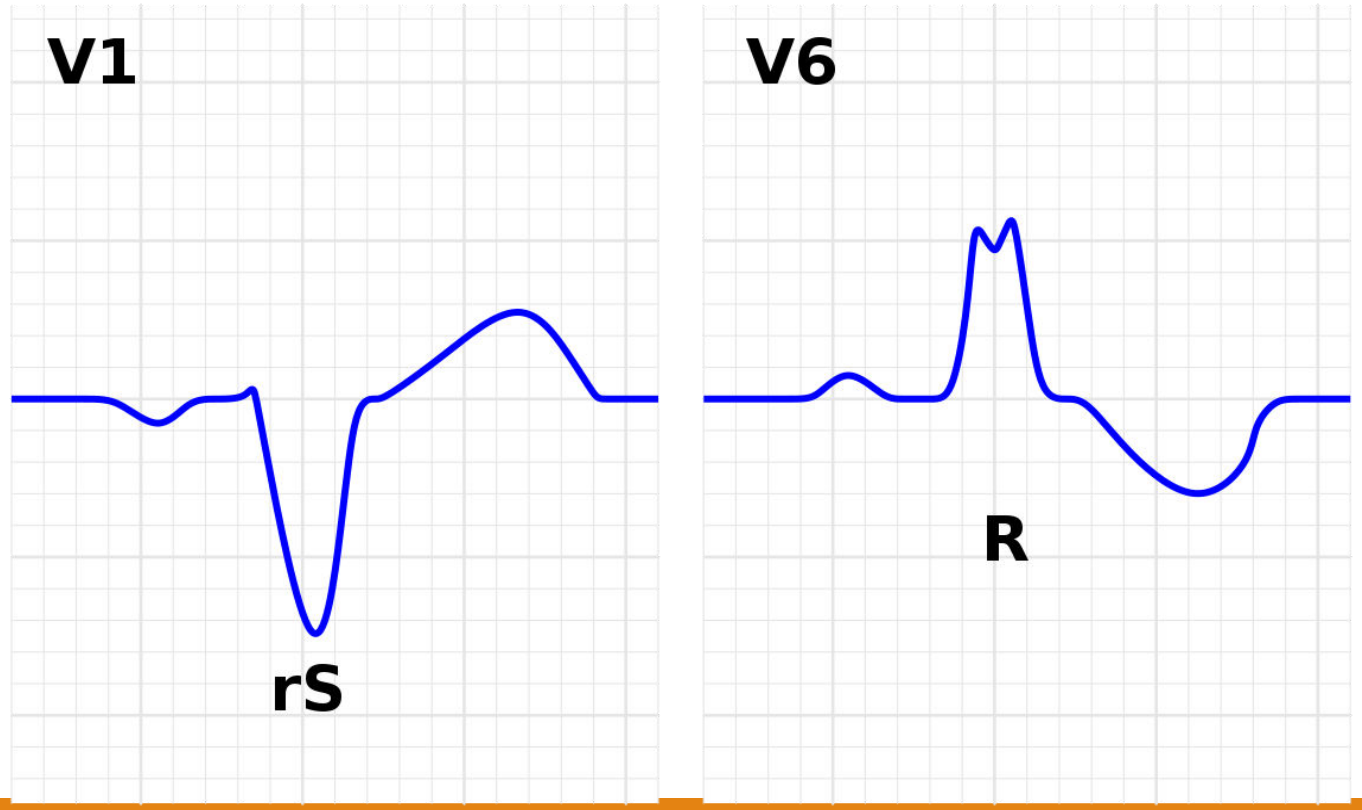
Infection of the heart muscle (myocarditis)

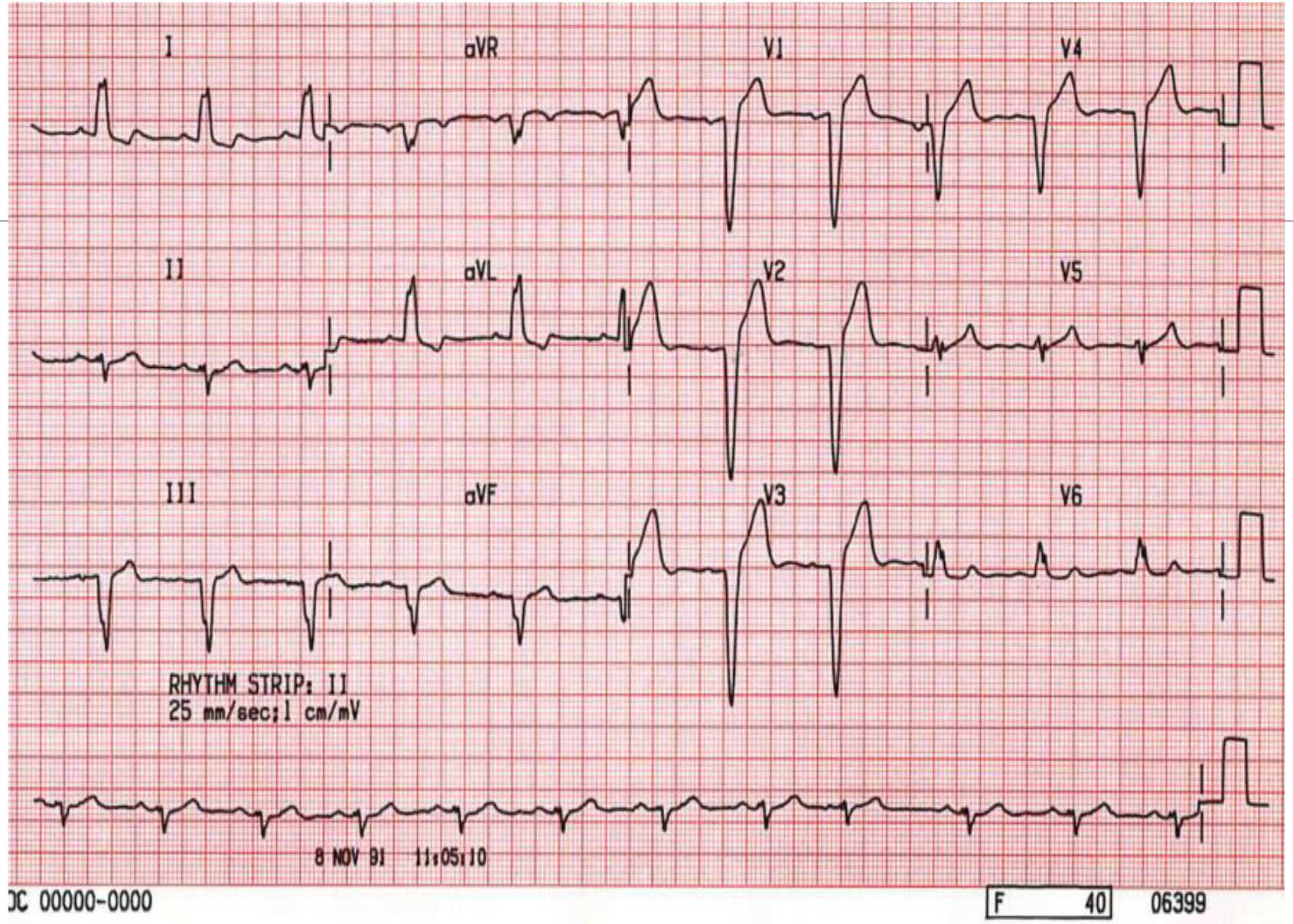
Thick, stiff, or weak heart muscle (cardiomyopathy)

Coronary artery disease

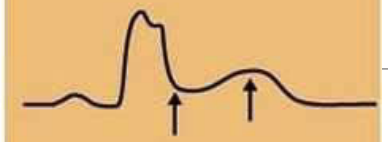
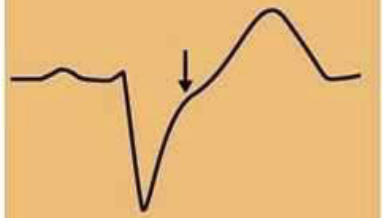
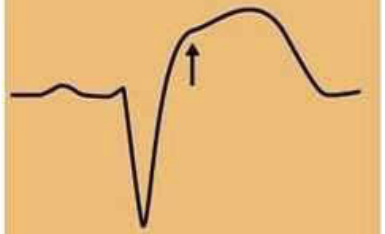
In LBBB there may be reversed splitting of the second heart sound

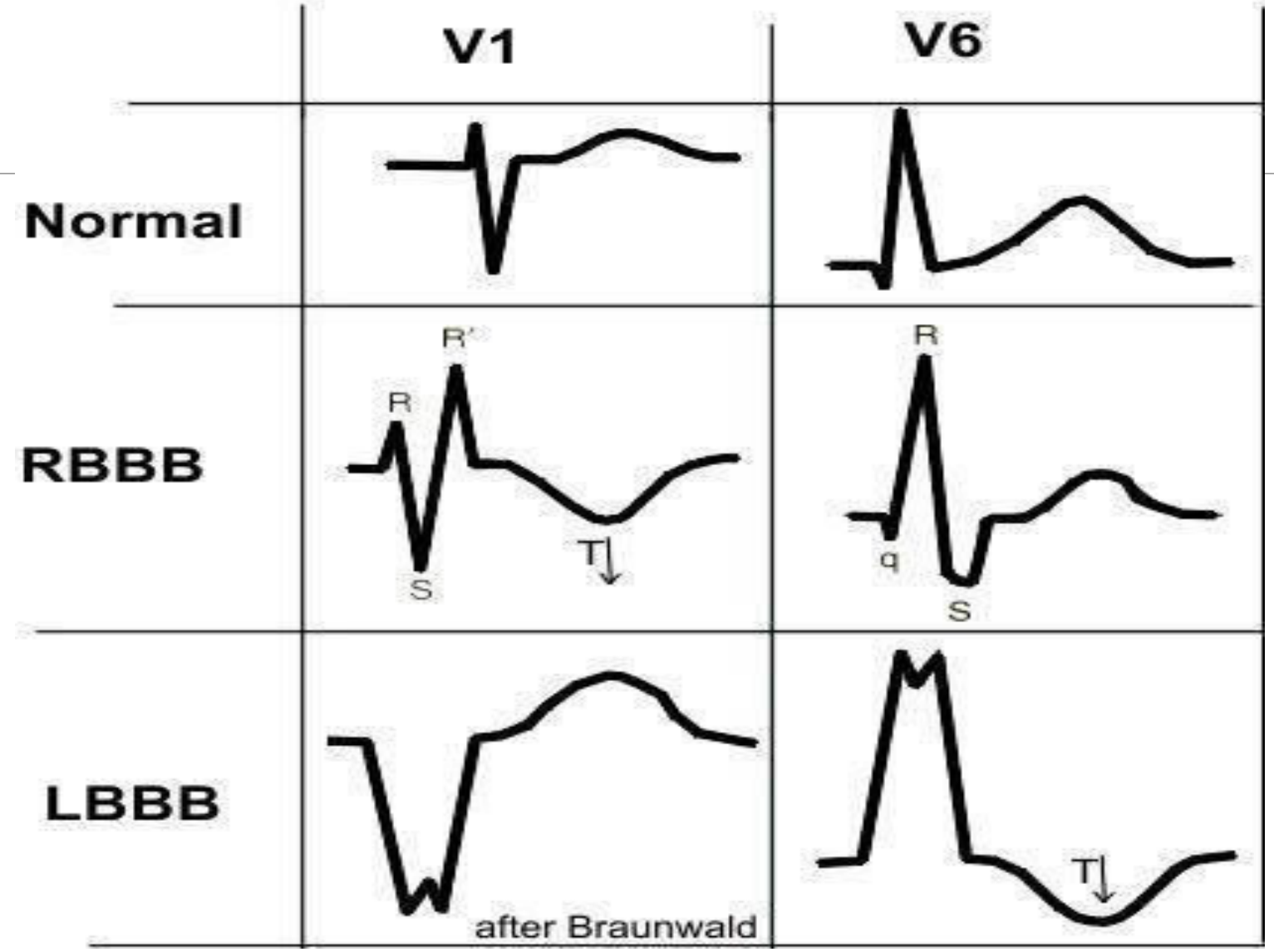
Produce a deep S in V1 and tall late R in lead I and V6




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec;1 cm/mV
8 NOV 91 11:05:10
DC 00000-0000
F 40
06399

Sgarbossa criteria for diagnosis of STEMI in the setting of LBBB

Criteria	Points		Normal LBBB	Ischemic LBBB
ST segment elevation ≥1 mm in any lead with positive QRS (V4, V5, V6, aVL, I) .	5	V4-V6, aVL, I		
ST segment depression ≥1 mm in V1, V2 and/or V3.	3	V1-V3		
ST segment elevation ≥5 mm in any lead with discordant QRS (V1, V2, V3)	2	V1-V3		
			These complexes show the normal (expected) appearance of LBBB.	These complexes show ischemic manifestations in the setting of LBBB.


V1
V6
Normal
RBBB
R'
R
S
T
R
q
S
LBBB
T
after Braunwald

Management :

If the patient doesn't already have heart disease, heart disease symptoms, or other electrical blocks, he may not need treatment. But we will look into treatment options if he had:

Heart attack of heart failure

Fainting or dizzying spells

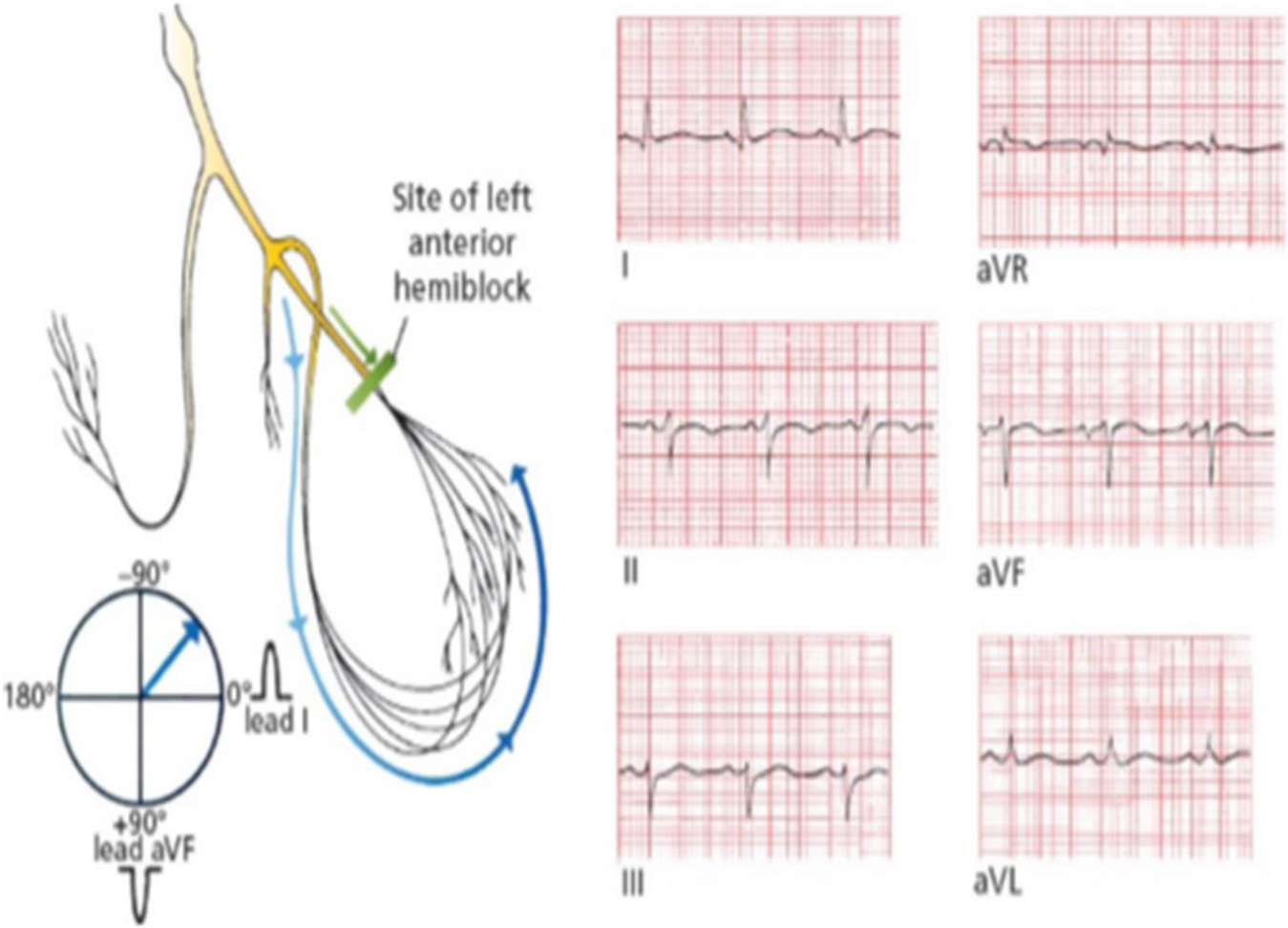
Chest pain

Hemiblock

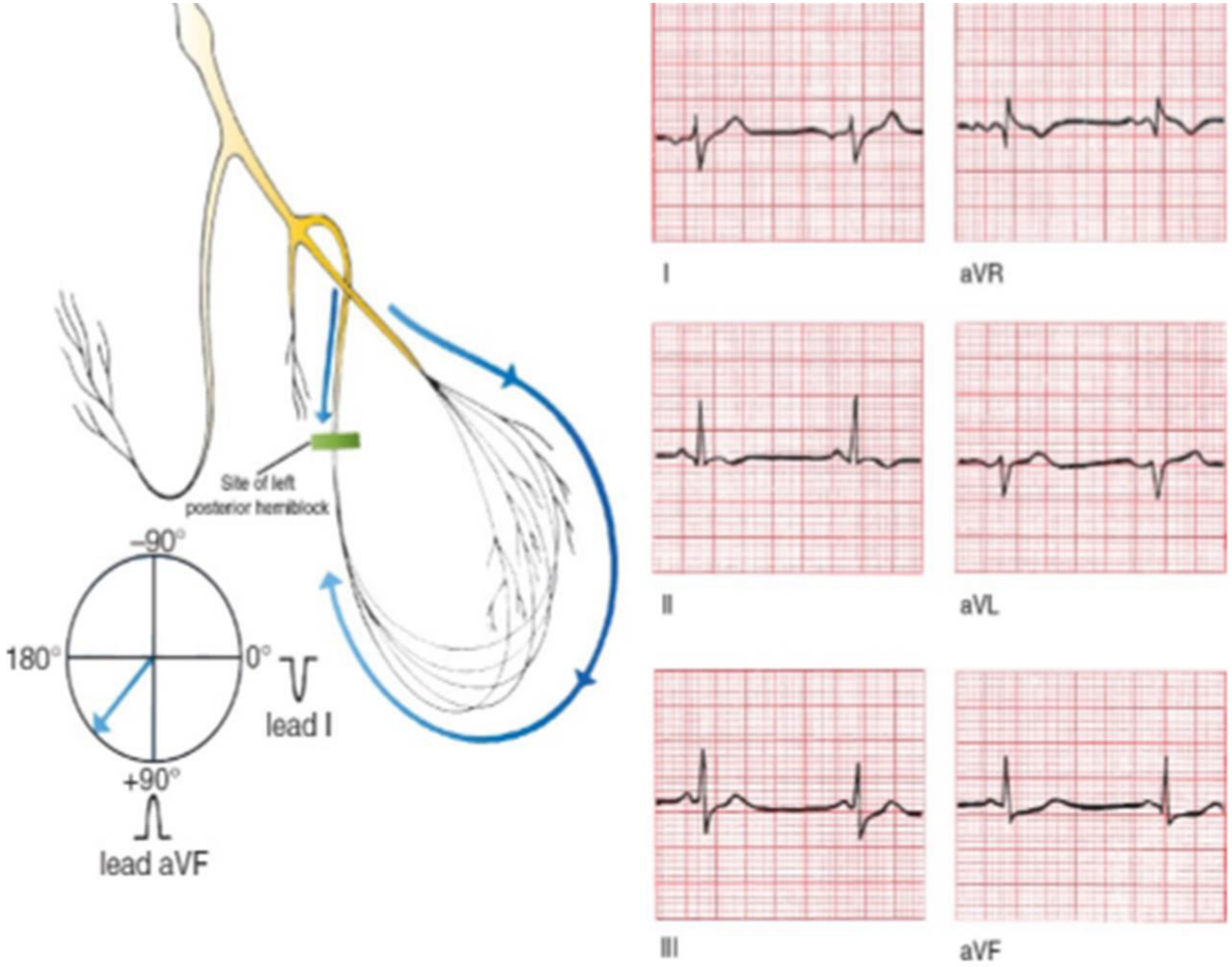
When there is block in one of the divisions of the left bundle, either anterior division or posterior division, this will produce a swing in the electrical axis of the heart.

Anterior hemiblock : cause left axis deviation

Posterior hemiblock : cause right axis deviation



Left anterior hemiblock. Current flow down the left anterior fascicle is blocked; hence, all the current must pass down the posterior fascicle. The resultant axis is redirected upward and leftward (left axis deviation).



Left posterior hemiblock. Current flow down the left posterior fascicle is blocked; hence, all the current must pass down the right anterior fascicle. The resultant axis is redirected downward and rightward (right axis deviation).

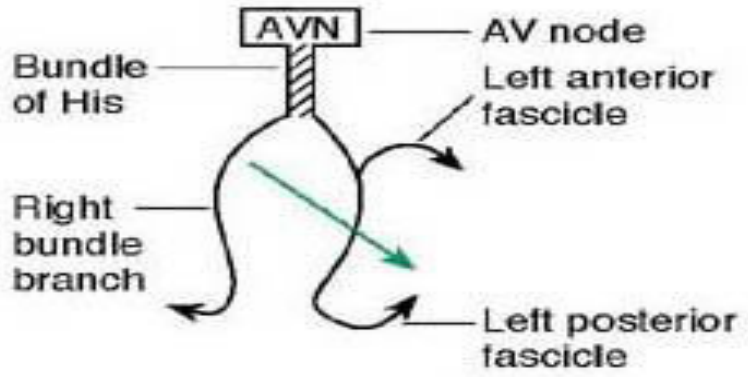
Bifascicular block

Right Bundle Branch Block + Left Anterior Fascicular Block or Left Posterior Fascicular Block

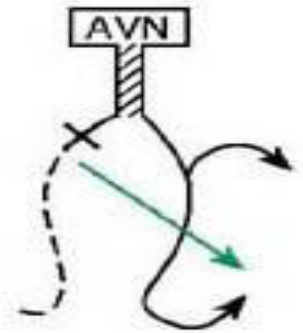
A block in all the above three will cause complete heart block.

Bifascicular block is often associated with structural heart disease (50-80%) and extensive fibrosis of the conducting system. There is a risk of progression to complete heart block with additional damage to the third remaining fascicle.

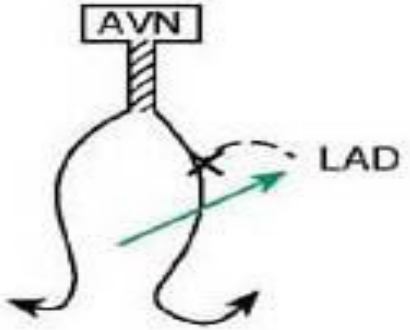
Syncope or presyncope in the context of a bifascicular block is an indication for admission and monitoring. If other causes of syncope are not identified on work-up, pacemaker insertion is recommended.



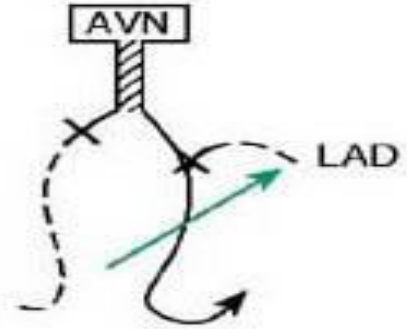
1. Normal conduction → normal axis



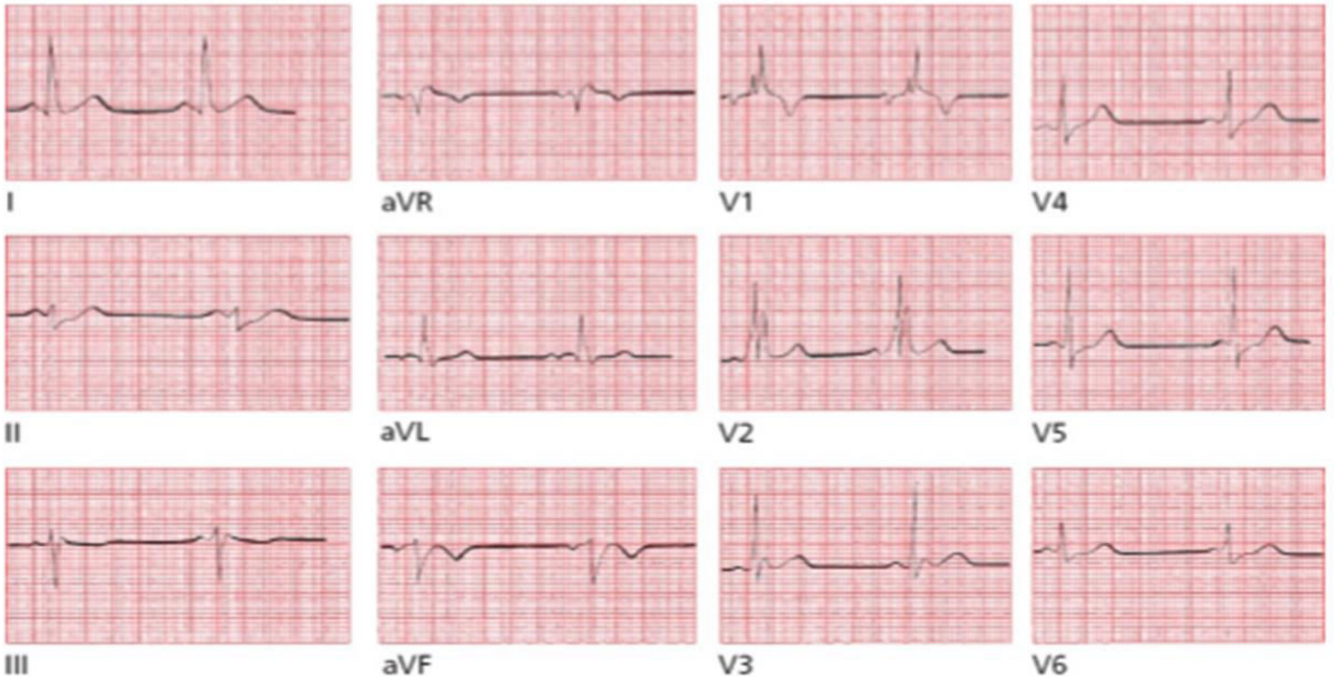
2. RBBB → normal axis (as left ventricle depolarizes normally)



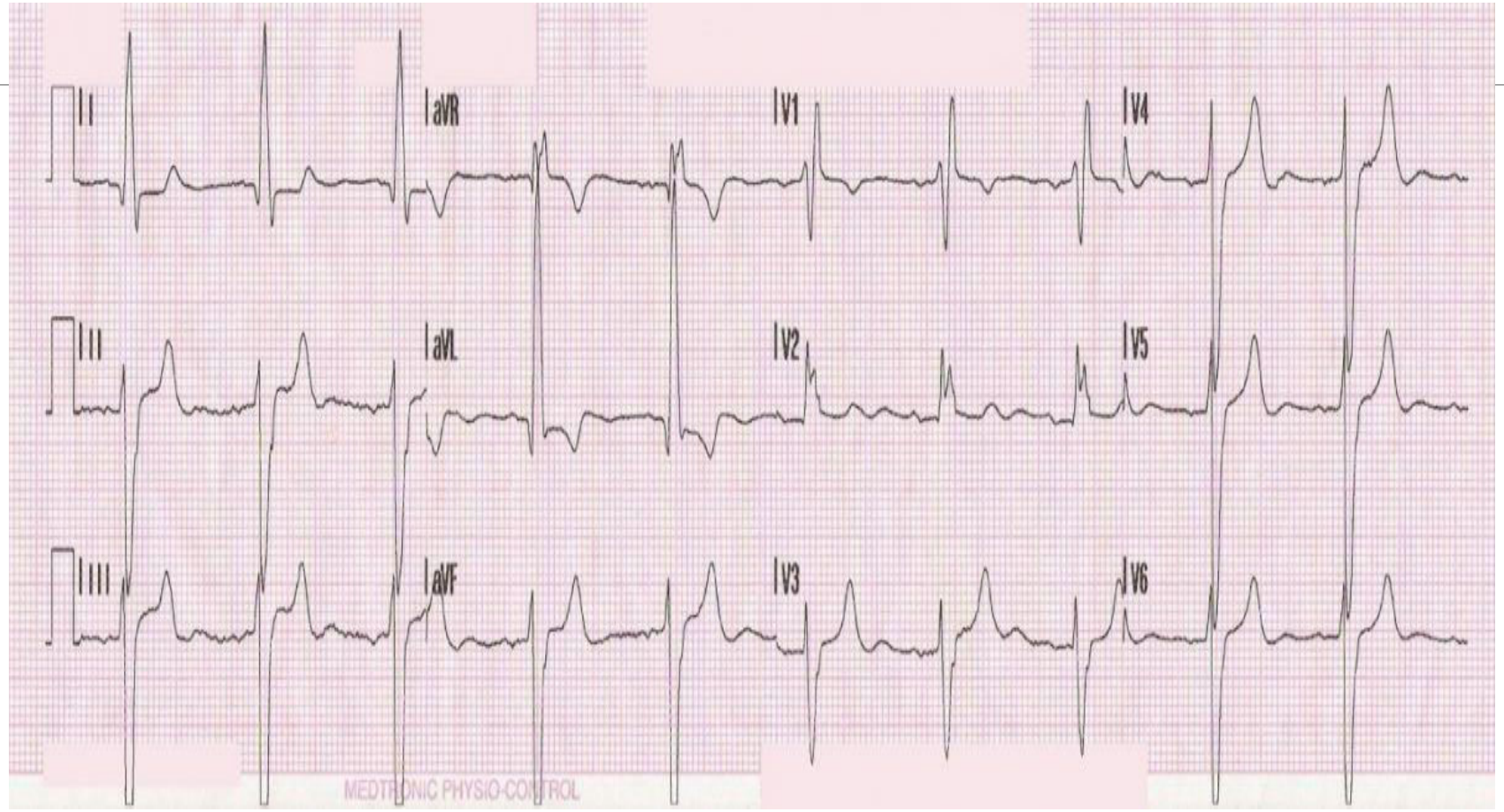
3. Left anterior hemiblock → left axis deviation



4. RBBB and left anterior hemiblock → left axis deviation



This is an example of right bundle branch block combined with left anterior hemiblock. Note the widened QRS complex and rabbit ears in leads V1 and V2, characteristic of right bundle branch block, and the left axis deviation in the limb leads (the QRS complex is predominantly positive in lead I and negative in leads aVF and II) that suggests left anterior hemiblock.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
MEDTRONIC PHYSIO-CONTROL

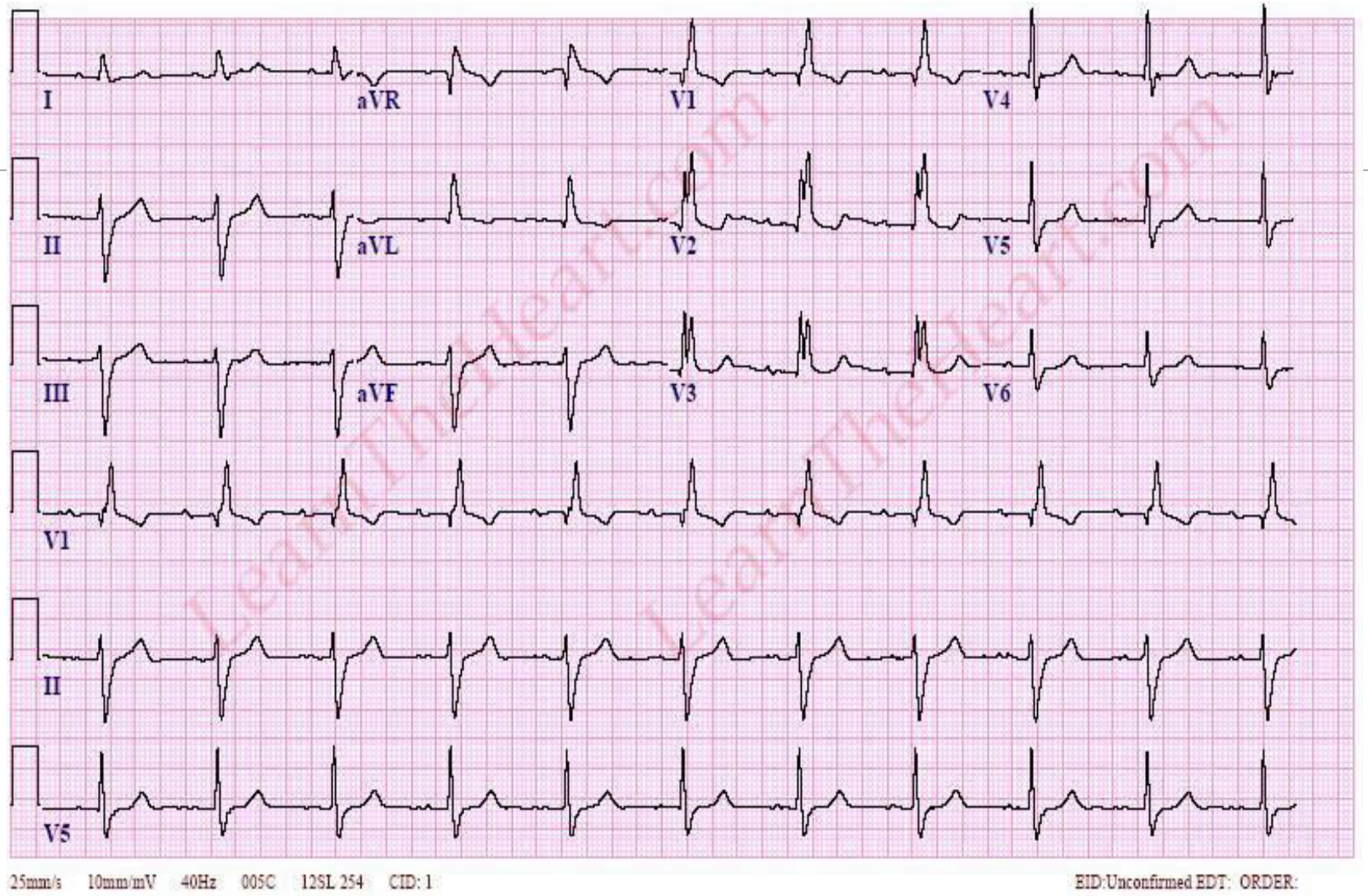
Trifascicular Block

A trifascicular block means there are signal problems with the right bundle branch and both of the left fascicles that make up the left bundle branch. This is also known as a complete heart block.

While a trifascicular block itself does not require any treatment, high doses of AV blocking agents likely should be avoided. Some series report a 50% lifetime need for a permanent pacemaker in the setting of a trifascicular block.

Trifascicular Block

- Conduction blocks in all 3 fascicles
  - Can be permanent or transient
- Criteria:
  - 1) RBB and LASF w/1st degree AV block
  - 2) RBB and LPIF w/1st degree AV block
  - 3) LBB w/1st degree AV block or
  - 4) Alternating RBBB and LBBB


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 254 CID: 1
EID:Unconfirmed EDT: ORDER:

Management

Generally , if patients with isolated BBB asymptomatic with no evidence of cardiac disease, they don’t require specific therapy.

Permanent pacemaker insertion indicated if patient become symptomatic (syncope) , or in the presence of other conduction abnormalities.

Cardiac resynchronization therapy indicated in patient with BBB (especially LBBB) and heart failure with reduced ejection fraction.

Thank you