Department of Anesthesia Techniques
The lect. 4: ECG & Arrhythmia

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Learning objectives

After reading this lecture, you should be able to:

1-recognize two common variations of each of the normal P, QRS and T waves

2-distinguish the three atrioventricular blocks

3- describe three common tachyarrhythmias

Electrocardiogram and arrhythmias

Electrocardiography was **introduced by Willem Einthoven**, who first recorded the electrical activity of the heart using his string galvanometer. His work laid the foundation for modern cardiac diagnostics.

Electrocardiography is a **graphical representation of the electrical potential differences** generated by the heart as the impulse spreads through the myocardium, recorded from the **body surface** using electrodes.
Clinical Importance: The **electrocardiogram (ECG)** is one of the **most commonly used and accessible diagnostic tools** in both **primary and secondary healthcare settings**.

It is **indispensable** for:
- **Screening and monitoring** patients with cardiac conditions.
- **Evaluating arrhythmias** and **conduction abnormalities**.
- **Assessing myocardial ischemia or infarction.**

Specialized Applications:
- **Exercise (stress) ECG:** Used for diagnosing **coronary artery disease (CAD)** by recording the heart's electrical activity during physical exertion.
- **Ambulatory (Holter) ECG:** Used for **continuous monitoring** of cardiac rhythm to detect **intermittent arrhythmias** in daily life.

ECG Lead System

Basic Concept:

The **heart muscle generates electrical currents** during its depolarization and repolarization cycles. These currents can be **recorded from the surface of the body** using an **electrocardiogram (ECG)**.

The 12-Lead ECG:

The **standard ECG** consists of **12 conventional leads** that record the **difference in electrical potential** between electrodes placed on specific points of the body surface.

Classification of ECG Leads:

The 12 leads are divided into **two main groups**:

1. **Six Limb Leads**
2. **Six Chest (Precordial) Leads**

1. Limb Leads

The **six limb leads** are derived from electrodes placed on the **right arm (RA), left arm (LA),** and **left leg (LL)**.

They provide a view of the heart's electrical activity in the **frontal plane**.

a. Bipolar Limb Leads (Einthoven's Triangle)

Lead I: Left arm (+) – Right arm (−)

Lead II: Left leg (+) – Right arm (−)

Lead III: Left leg (+) – Left arm (−)

These three leads have been in clinical use for **over a century** and were first introduced by **Willem Einthoven**.

b. Unipolar Augmented Limb Leads

Introduced later, these are **unipolar leads**, designated by the letter **"a"** for **augmented**:

- **aVR:** Voltage at the **right arm**
- **aVL:** Voltage at the **left arm**
- **aVF:** Voltage at the **left leg (foot)**

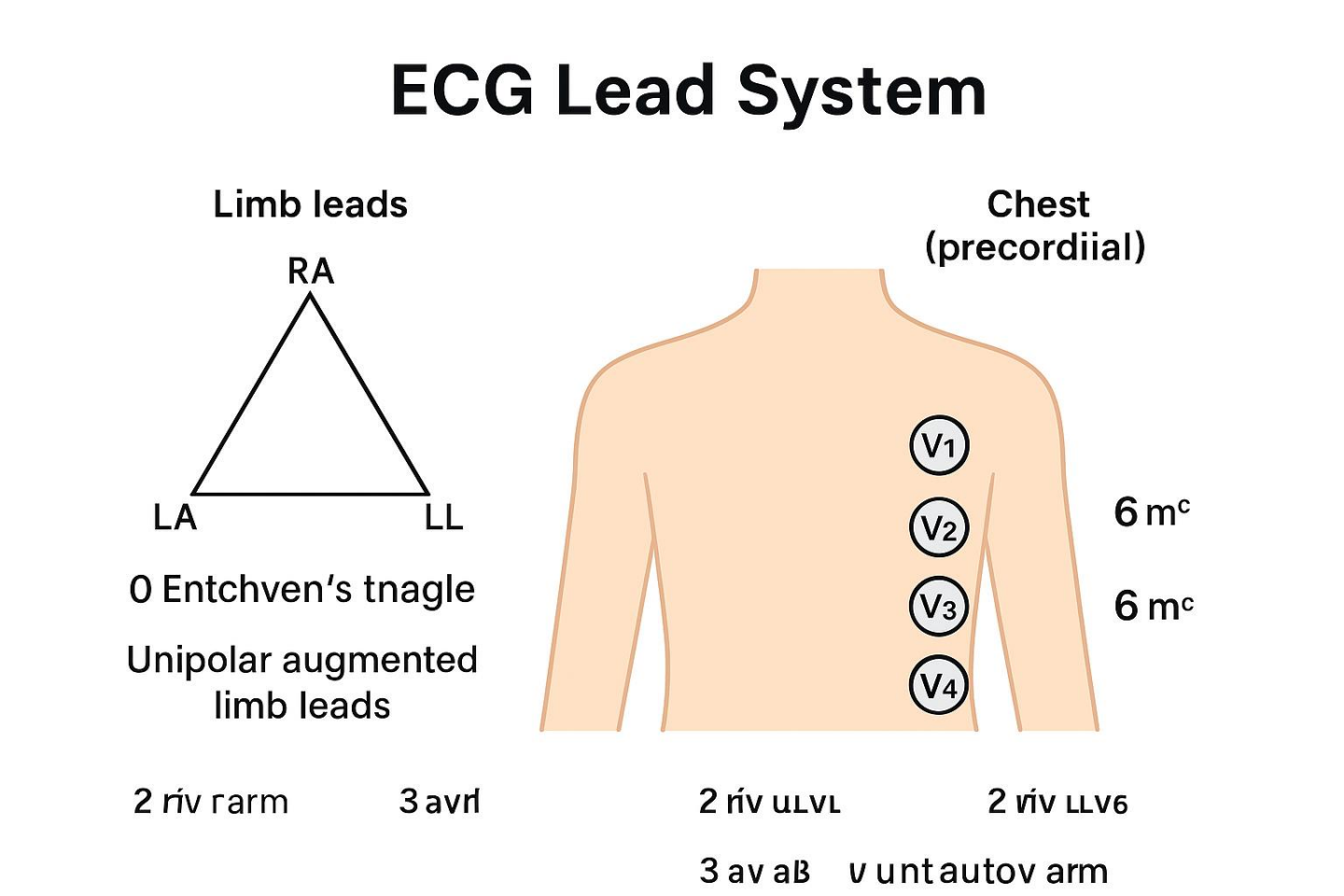
Each lead measures the voltage at one location **relative to a common central terminal** (an **indifferent electrode**) with approximately **zero potential**.

The ECG machine **augments** these small voltages by **50%**, which is why the prefix **"a"** (for *augmented*) is used.

2. Chest (Precordial) Leads

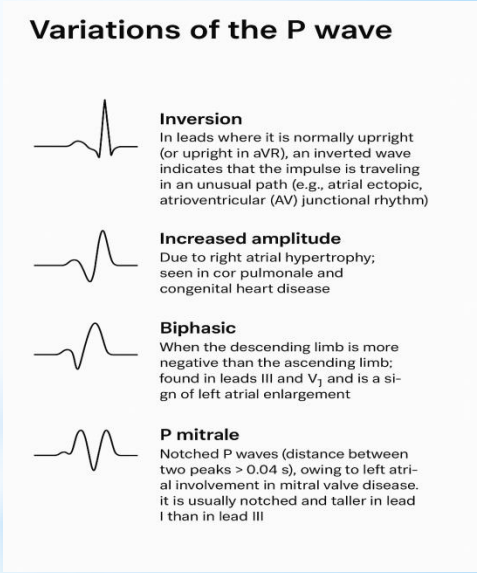
The **six unipolar chest leads**, labeled **V1–V6**, are placed directly on the **anterior chest wall**.

Each chest lead records the **electrical impulse immediately beneath the electrode**, providing a view of the heart's activity in the **horizontal plane**.



Variations of the P wave:

1. **Inversion**
    1. Seen in leads where the P wave is normally upright (or upright in aVR).
    2. Indicates that the **atrial impulse is traveling in an abnormal direction**, suggesting:
        1. **Atrial ectopic rhythm**, or
        2. **Atrioventricular (AV) junctional rhythm**.



2.Increased Amplitude

1. Caused by **right atrial hypertrophy**.
2. Commonly observed in conditions such as:
    1. **Cor pulmonale**, and
    2. **Congenital heart disease**.

3.Biphasic P Wave

1. The **descending limb is more negative** than the ascending limb.
2. Typically found in **leads III and V1**.
3. Indicates **left atrial enlargement**.

4.P Mitrale (Notched P Waves)

1. Characterized by **two peaks separated by more than 0.04 seconds**.
2. Due to **left atrial involvement** in **mitral valve disease**.
3. Usually **notched and taller in lead I** than in lead III.

QRS complex

The QRS complex reflects rapid ventricular depolarization. An initial downward deflection is termed the Q wave and ensuing deflections are labelled in alphabetical order. The first positive deflection is designated R, whereas S is the first negative deflection that follows the R wave. This represents the terminal part of the ventricular activation.

The complex ventricular depolarization can be divided into two sequential phases.

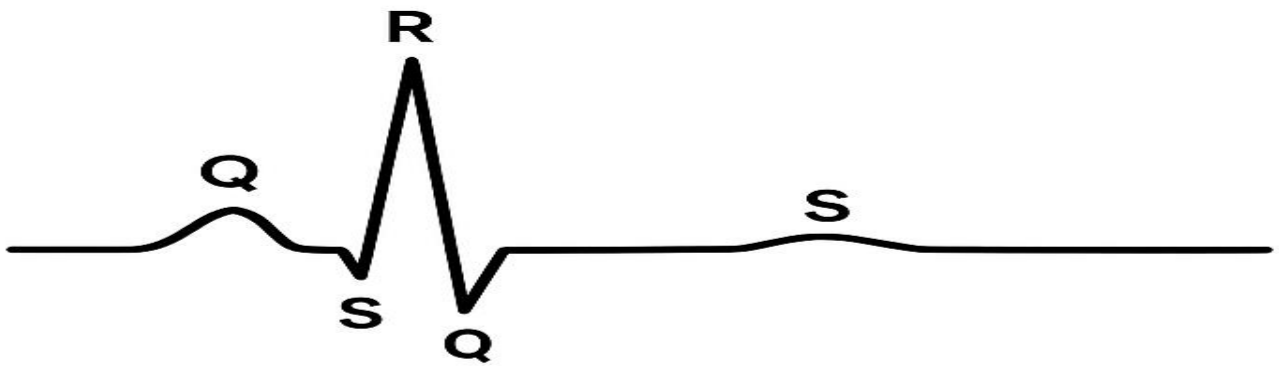
- The first phase is the activation of the ventricular septum from left to right.
- The second phase is the simultaneous activation of the right and left ventricles, usually

dominated by the bulky left ventricle.

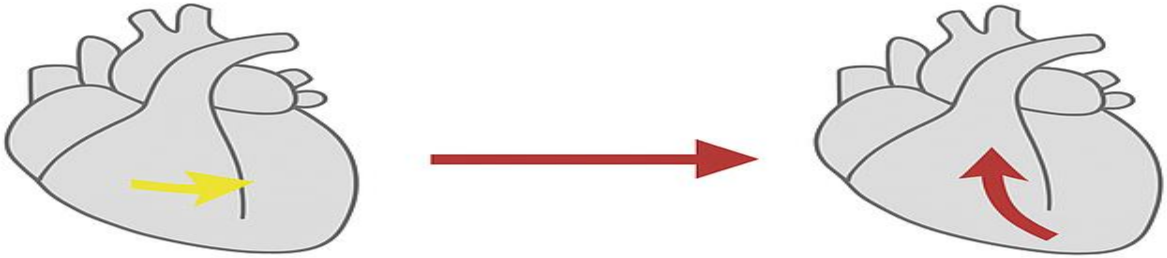
In the chest leads, as a consequence of this normal depolarization process, the right-oriented leads (V1 and V2) show a small upward deflection (septal R wave), followed by a deep S wave.

The QRS pattern in the limb leads varies and depends on the mean QRS axis. Lead aVR, which records from the right shoulder, effectively ‘looks’ from the cavity of the heart with all the vectors directed away and thus has all negative deflections. The normal duration of the QRS complex is 0.05e0.10 seconds

QRS Complex



Normal QRS Duration: 0.05–0.10 s



Phase 1 Phase 2

V1

Small upward septal R wave followed by deep S wave

V6

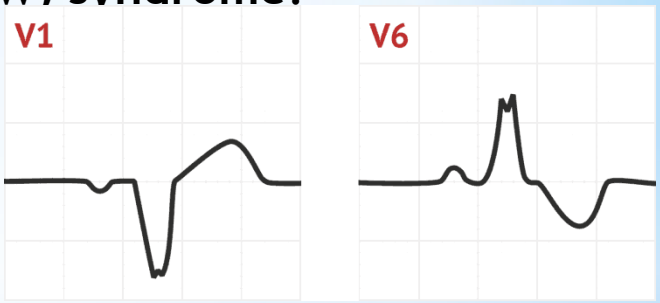
Tall R wave with small S wave

Variations of the QRS complex:

Variations of the QRS Complex

1.Prolonged QRS duration

1. A QRS duration of **0.12 seconds or more** indicates **conduction delay**, such as in **bundle branch block (BBB).**
2. It can also occur due to **pre-excitation of the ventricles** via an accessory pathway, as seen in **Wolff-Parkinson-White (WPW) syndrome.**



2.Right Bundle Branch Block (RBBB)

1. Occurs **more commonly** than left bundle branch block, even in people with structurally normal hearts.
2. May also occur in **acquired (valvular, ischemic)** and **congenital heart diseases**, especially **atrial septal defects.**

3-Left Bundle Branch Block (LBBB)

Usually associated with ischemic heart disease, hypertension, severe aortic stenosis, or cardiomyopathy.

Bundle branch blocks (QRS ≥ 0.12 seconds) can be chronic, intermittent, or rate-related.

Some patients with supraventricular tachycardia (SVT) may exhibit broad QRS complexes due to aberrant conduction.

T wave

The **T wave** represents **ventricular repolarization**, i.e., the recovery phase of the ventricles after depolarization.

Normal Characteristics

Direction: Usually **in the same direction as the QRS complex** (concordant).

Lead-specific appearance:

- **Upright (positive)** in:
  - Leads **I and II**
  - **Left-sided chest leads** (V4–V6)
- **Inverted (negative)** in:
  - Lead **aVR**
- **Variable** in other leads.

Clinical Significance

Changes in T wave **shape, height, or inversion** may indicate:

- **Ischemia** or **myocardial infarction**
- **Electrolyte disturbances** (e.g., hyperkalemia → tall peaked T waves)
- **Ventricular strain** or **pericarditis**

Variations of the T Wave

1. Tall Positive T Waves

Description: Exaggerated upward deflection of the T wave.

Possible Causes:

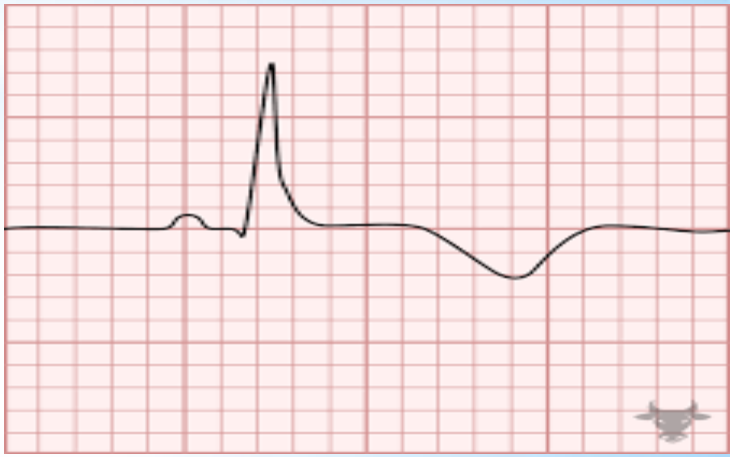
Can be a **normal variant**
Hyperkalemia
Hyperacute myocardial ischemia
Cerebrovascular injury (e.g., stroke, intracranial hemorrhage)
Left ventricular volume overload

2. T Wave Inversion

•**Description:** T wave is deflected **opposite to the QRS**.

•**Possible Causes:**

- **Cardiomyopathy**
- **Myocardial ischemia**
- **Ventricular hypertrophy**
- **Myocarditis**
- **Intracranial bleeds**



Arrhythmias

Arrhythmias are abnormalities in the rate, rhythm, or conduction of the heart.
Normal heart rate: ~70 beats per minute (bpm) at rest.
Bradycardia: Heart rate **<60 bpm**
Tachycardia: Heart rate **>100 bpm**

Bradycardia

1. Physiological Bradycardia

Normal during **sleep** or in **fit athletes** (“athletes’ heart syndrome”).
Heart rate **<60 bpm** without symptoms can be normal.
Mediated by **baroreceptors** (pressure sensors) in the **aorta and carotid arteries**, which modulate **vagal tone** via **acetylcholine release**.

2. Pathological or Exaggerated Bradycardia

Carotid sinus syndrome: Increased sensitivity of baroreceptors in the **carotid sinus**.
Pressure on the neck can cause: Extreme bradycardia,Dizziness, Syncope (fainting).

3. Sinus Arrhythmia

Heart rate naturally varies with **respiration**: Increases during **inspiration**, Decreases during **expiration**. Considered a **normal physiological variation**.

Atrioventricular (AV) Block

AV block is a delay or interruption in the conduction of electrical impulses from the atria to the ventricles.

1. First-Degree AV Block

ECG Feature: PR interval > 0.20 seconds (prolonged), **constant delay**, no missed QRS.
Causes: Physiological (athletes), Ischemic heart disease, Acute rheumatic carditis, Drugs: Digitalis, beta-blockers
Symptoms: Usually **asymptomatic**
Management: Observation only

2. Second-Degree AV Block

Type 1 (Mobitz I / Wenckebach)

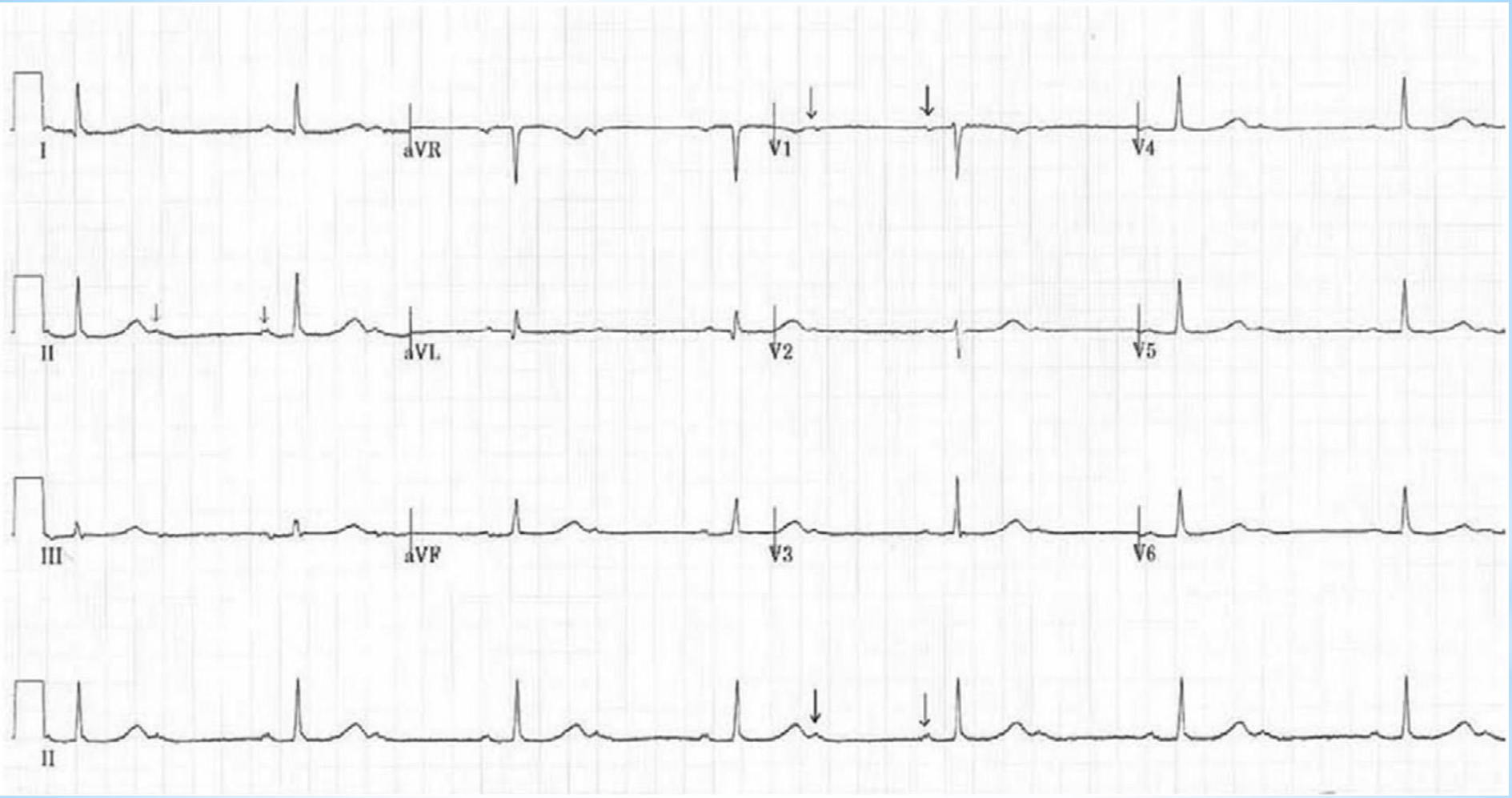
ECG Feature: Progressive **PR prolongation** until a **QRS is dropped**
Site of Block: Usually in **AV node**
QRS: Normal duration
Causes: Inferior wall MI, Drugs: Beta-blockers, digoxin, calcium channel blockers, Physiological (increased vagal tone, often at night)
Symptoms: Usually **asymptomatic if ventricular rate adequate**
Management: Observation if asymptomatic

Type 2 (Mobitz II)

ECG Feature: PR interval constant, then sudden **failure of P wave to conduct**; can show 2:1 block
Site of Block: His-Purkinje system
QRS: Often abnormal
Causes: Anteroseptal MI, Degenerative disease of cardiac conduction system
High-Grade Block: Two or more successive P waves blocked
Risk: Can progress to **third-degree AV block or ventricular standstill**
Management: Permanent pacing indicated

3. Third-Degree AV Block (Complete Heart Block)

ECG Feature: Complete **dissociation** between P waves and QRS complexes
Pacemakers: Atria and ventricles controlled by **independent pacemakers**
Causes: Chronic degenerative bundle branch disease (Lev's or Lenegre's), CardiomyopathyInferior MI, Congenital (maternal autoantibodies: Ro/SSA or La/SSB)
Symptoms: Can be severe; high **mortality risk**
Management: Permanent pacing



Bradycardia: type II second-degree (Mobitz type II) atrioventricular block. There are two P waves for one QRS complex (2:1 block).

Tachycardia

Rapid heart rate due to abnormal electrical activity.

Normal Physiology: Myocardial cells generally do **not** discharge spontaneously; impulses are normally generated by the sinoatrial (SA) node.

Mechanisms of Tachyarrhythmias

Disorders of Impulse Formation (Enhanced Automaticity):

Causes:Exogenous catecholamines (e.g., adrenaline),Hyperkalemia,Hypoxia,Digitalis toxicity

Disorders of Impulse Spread (Re-entry):

Mechanism: A circuit allows the impulse to re-excite the myocardium repeatedly.

Example: Sustained supraventricular tachycardias (SVTs)

Classification of Tachyarrhythmias

1-By Rhythm Regularity:

Regular: Each beat follows a predictable interval

Irregular: Beat intervals vary

Usually **atrial fibrillation** (AF) or **atrial flutter with variable block**

2-By QRS Complex Morphology:

Narrow Complex (<120 ms): Usually originates above the ventricles (supraventricular), Example: SVT

Broad Complex (≥120 ms): Usually originates from the ventricles

Example: Ventricular tachycardia (VT)

Atrial Fibrillation (AF)

Atrial fibrillation is a common arrhythmia characterized by **disorganized atrial electrical activity**, resulting in the absence of discrete P waves on the ECG.

Types

Paroxysmal AF: Episodes terminate spontaneously, usually within 7 days.
Persistent AF: Episodes last longer than 7 days or require intervention to terminate.

Mechanism

Exact mechanism is unclear, but likely involves:
Multiple micro-reentry circuits in the atria
Triggers commonly from the **pulmonary veins entering the left atrium**
Atrial rate: 350–600 bpm
Leads to an **undulating baseline** on ECG

AV Node and Ventricular Response

Impulses from atria reach the AV node **via multiple paths at irregular intervals**
AV node is **partially refractory**, so:

- **Ventricular rate** is slower than atrial rate
- Rhythm is **irregularly irregular**

Acute Precipitating Factors

Infection,Alcohol intake, Dehydration, Congestive cardiac failure, PE

Atrial Flutter (AFL)

Atrial flutter is an arrhythmia characterized by **rapid, regular atrial activity**, producing a **saw-tooth or picket-fence pattern** on the ECG, especially in the **inferior leads** (II, III, aVF).

Mechanism

Usually caused by a **macro-reentry circuit in the right atrium**.

Atrial rate: 250–350 bpm

Ventricular rate: Typically about half the atrial rate (~150 bpm) due to AV nodal conduction block

Clinical Course

Episodes lasting **more than one week** often **convert to AF**.

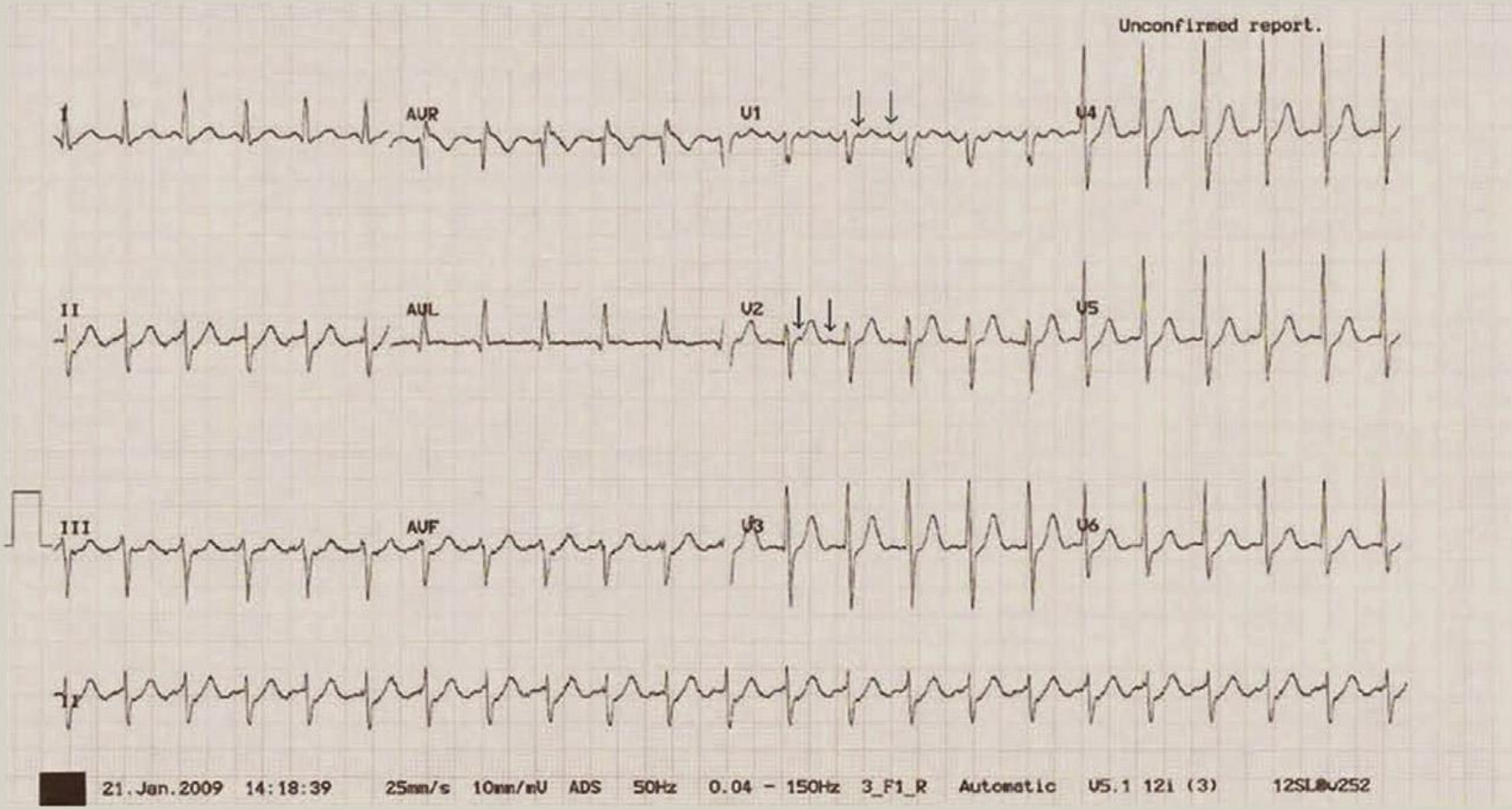
Risk of systemic embolization: Less than AF, but **management is similar** (rate/rhythm control, anticoagulation

ECG Features

Saw-tooth flutter waves (best seen in inferior leads)

Regular atrial rhythm

Ventricular rate usually ~150 bpm (2:1 block common)



Atrial flutter: regular narrow complex tachycardia showing 2:1 physiological block. P waves (arrows) are best seen in leads V1 and V2.

Ventricular Tachycardia (VT)

VT is defined as **three or more consecutive ectopic ventricular QRS complexes** at a rate **>100 bpm**.

Sustained VT: Lasts **>30 seconds** or requires intervention.

Mechanism

Abnormal automaticity of ventricular cells **or**

More commonly, **re-entry distal to the His bundle**

ECG Characteristics

Broad complex tachycardia (QRS ≥ 120 ms)

Usually **regular rhythm**

Most common cause of broad complex tachycardia, especially in **structural heart disease**

ECG Features Favoring VT (vs. SVT with Aberrant Conduction)

1.Atrioventricular dissociation

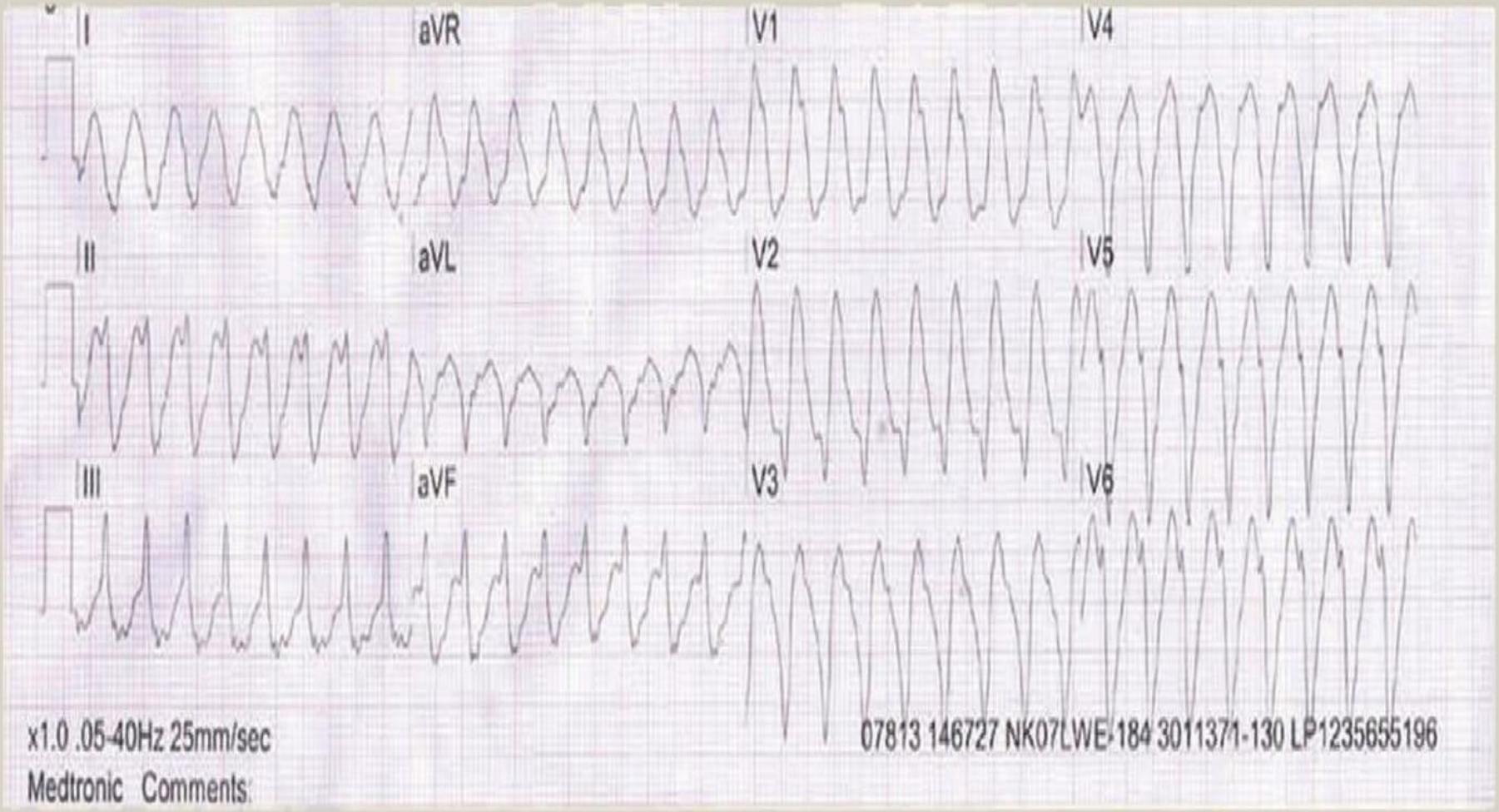
2.QRS duration:

- 0.14 sec with **RBBB morphology**
- 0.16 sec with **LBBB morphology**

3-QRS axis:

Left axis with RBBB morphology

Extreme left axis with LBBB morphology



Regular broad complex ventricular tachycardia