ECG

1. **action - regular**
   - irregular

2. **rythm: - sinus** / P wave at least in 1 lead present
   - non sinus – without P wave: (atrial fibrilation, atrial flutter, AV junction rythm)

3. **typ of curve (position of axis):**
   normal – highest R in the II[nd.] lead
   left – highest R in the I[st] lead
   right – highest R in the III[rd] lead

4. **intervals: normal**
   - **PQ interval**: 0,12-0,20 s (time of leading the fizz from atria to ventricules)
   Pathology:
     - **standard longer** than **0,20 s**: AV blok I. st. degree
     - **gradually longer PQ interval, at last 1 QRS is missing** (1 impuls is not leaded to ventricles)- AV blok II.nd degree Wenkelbach type
     - **shorter** PQ than **0,12 s** – syndrom of preexcitation (WPW syndrom, LGL syndrom)

   - **QRS interval** (time of leading of the fizz through the ventricles by Tawar arms) **0,10-0,12 s**
   Pathology: **longer as 0,12 s**: complete block (right RBB or left LBB)

   - **QT interval**: 0,32-0,42 s.
   Pathology: longer: in antiarythmic treatment (Sotalol) – risk of SCD sudden†

5. **ST segment – normal** / t.j. without depression, elevation/
   Pathology: **depression** of ST segment – sign of ischemia- which leads
   elevation of ST segment (Paarde´ wave in AMI)

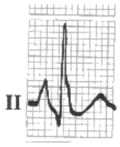
6. **T wave- positive**
   -high peak T- 1. st of AMI, hyperkalemy, neurocirculat. astenia
   -negative: negative coronary T (symetric in AMI)- which leads ???
   secondary neg., assymetric

7. **Transitional zone: V3-V4**
   (in chest leads – if size of positive and negative excursions are equal)

8. **Frequency**

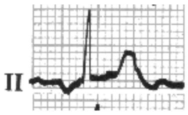
P wave (atrial excitation-depolarization in NSA)

– **till 0,10 s, amplitude till 2,5 mm (0,25 mV)**

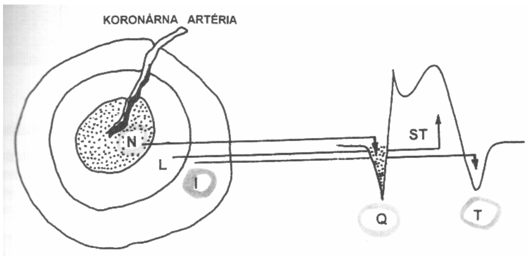


- **P pulmonale – amplitude higher as 2,5 mm** (high peak) – in cor pulmonale chr, chr. bronchitis (hypertrophy of RV)

- **P mitrale (sinistrocardiale)- longer as 0,10 s-** 2-peak wave - in mitral. stenosis (hypertrofy / dilatation of LA)



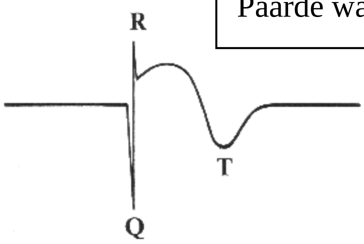
Negative P wave **(before or after QRS complex)**- AV junction rythm



ECG pathology according to the leads:

RV (right ventricle): II, III, aVF, V1, V2
LV(left ventricle): I, aVL, V5, V6
Anterior wall: V1-V6,I, aVL
Posterior (diaphragmatic) wall: II, III, aVF
Lateral wall: V5-V6
Septum: V3-V4

Paarde wave

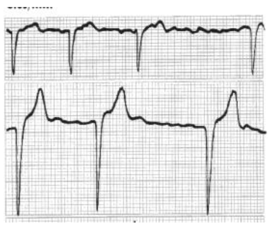


ECG according to the pathologic state:

AMI:

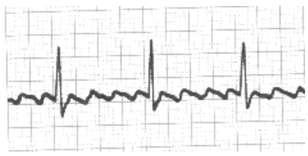
1. **ischemia : negative coronary T (symetric)**
2. **lesion : Paarde wave - elevation of ST segm.**
3. **necrosis : pathologic q** (deep- 1/3- ¼ of R and wide – longer as 0,04 s)
4. later: lack of elevation of ST segment, only patholog. Q + neg. coronary T
5. more later: lack of neg. T, only path. Q, or qS configuration (state after MI transmural)

localisation of AMI:
I, II, aVL, V1-V6: anterior wall, anteroextensive
II, III, aVF : posterior wall (diaphragmatic)
V3-V4: septal
V5,V6 (I, aVL): anterolateral
V5, V6, II, III, aVF: diaphragmatic
High R in V1: right posterior



Atrial Fibrilation (arytmia perpetua vera):
- **irregular** action, non sinus rythm (no **P wave**)
- irregular fibrilatory waves + frequency 100-160/min.

Atrial Flutter

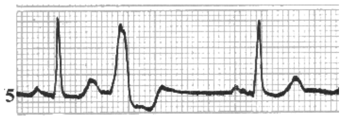


- regular action of atrium and ventricles
- flutter waves (2-4) before each QRS complex (block 2:1, 3:1, 4:1)

Extrasystols: (immature fizzes created except of NSA)
- **atrial**: premature QRS complex, normal QRS complex

- **ventricular**: **abnormal** QRS with compensatory pause

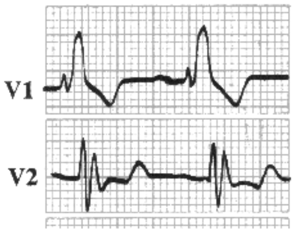
frequency: sporadic/ frequent
place of creation (1/ more): monotopic/ polytopic
bonding: bigeminy (each 2nd is ES, trigeminy (each 3rd ES),
couple – 2 ES, triplet: 3 ES together



! very dangerous: R to T phenomenon – very early ES, sitting to T wave

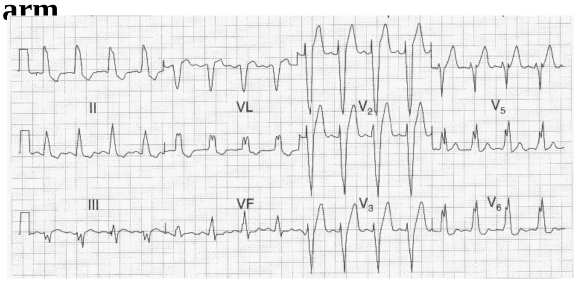
Right branch block: (blocked spread of fizz by right Tawar arm)

- **right position of axis**
- **rsR´, rsr´ configuration: V1-V2**
- complete RBB: QRS longer as 0,12 s
- incomplete RBB: QRS till 0,12 s.



Left branch block: blocked spread of fizz by left Tawar. arm

- left position of axis
- broad QRS complex-configuration of M in V5-V6
- ST elevation V1-V6
- complete LBB: QRS longer as 0,12 s
- incomplete LBB: QRS till 0,12 s.



Left branch hemiblock:

- extreme left side position (very high R in I.st lead , small R: II. III. lead)
- transitional zone in V5-V6

AV block of I.$^{st.}$ degree

- standard longer PQ interval as 0,20 s.

AV block II.nd degree Mobitz I.(Wenkelbach period)

- gradually longer PQ interval till 1 QRS is absent

AV block II. nd degree Mobitz II. – more severe emergency

- just only 2nd 3rd or 4th sinus impuls is conducted to ventricle
- 2 or 3 P waves + 1 QRS complex
- bradykardia- ventricules freq. is slower than atrial freq.

AV blok III.rd degree (complete absence of AV conduction)

- atrials and ventricules are independent
- regular action of A / V, but independent
- frequency of **A is faster** as freq. of V - bradykardia:

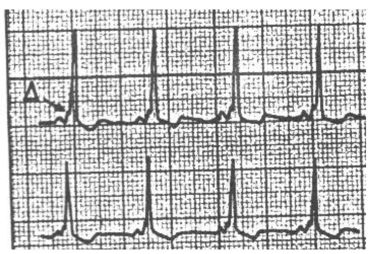
Morgan-Adams-Stokes attack + transient episode of syncope

Dif.dg.: AV- disociation: V freq. is **higher** as **A** frequency.

Syndrom of preexcitation:

LGL syndrom: PQ shorter as 0,12 s
QRS longer as 0,12 s
ST, T changes

WPW syndrom – as LGL **+ delta wave on** QRS



Hypertrophy of left Ventricle:

- **left** side position of axis
- V1, V2: small R, deep S
- **V5 (V6), I, aVL** : high **R** more than 25 mm
- **Index acc. Sokolow: S (V1,V2) + R (V5,V6) ≥ 35 mm**
- **Index Mc Phie: the highest R $^{\text{in precordial leads}}$ + deepest S $^{\text{in precordial leads}}$ ≥ 40 mm**
- **LV strain** pattern: neg T, ST depression

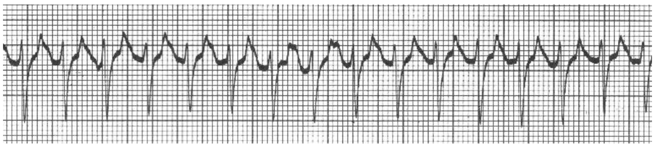
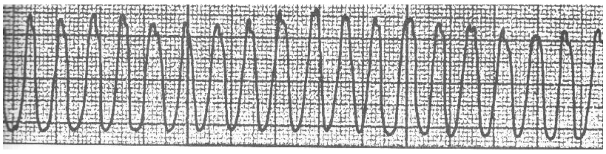
Hypertrophy of right ventricle (RV):

- **Right** position of axis
- Tall **R: III, aVF, V1, V2**
- Deep **S till V6** (transition zone is down)
- RV strain pattern: neg T, ST depression
- cor pulmonale chronicum: P pulmonale

Paroxysmal tachycardia

Supraventricular (ectopic center in SV, or reentry)
narrow QRS
Fr. 180-220

Ventricular (ectopic center in V)
- more than 3 VES -
- broad deformed QRS complexes
- fr. 150-180

Reentry mechanism: is created on the borderline of ischemic/nonischemic place

- if 2 structure have different conductory features (1 is depolarised and second is repolarised) – stop of conduction of the fizz - circulatory conduit

http://lifeinthefastlane.com/ecg-library/basics/

ECG Rhythm

A useful 7 step approach to rhythm analysis is described.

1. Rate —

- Tachycardia or bradycardia?
- Normal rate is 60-100/min.

2. Pattern of QRS complexes —

- Regular or irregular?
- If irregular is it regularly irregular or irregularly irregular?

3. QRS morphology —

- Narrow complex — sinus, atrial or junctional origin.
- Wide complex — ventricular origin, or supraventricular with aberrant conduction.

4. P waves —

- Absent — sinus arrest, atrial fibrillation
- Present — morphology and PR interval may suggest sinus, atrial, junctional or even retrograde from the ventricles.

5. Relationship between P waves and QRS complexes —

- AV association (may be difficult to distinguish from isorhythmic dissociation)
- AV dissociation
  - complete — atrial and ventricular activity is always independent.
  - incomplete — intermittent capture.

6. Onset and termination —

- Abrupt — suggests re-entrant process.
- Gradual — suggests increased automaticity.

7. Response to vagal manoeuvres —

- Sinus tachycardia, ectopic atrial tachydysrhythmia — gradual slowing during the vagal manoeuvre, but resumes on cessation.
- AVNRT or AVRT — abrupt termination or no response.
- Atrial fibrillation and atrial flutter — gradual slowing during the manoeuvre.
- VT — no response.

Narrow Complex (Supraventricular) Tachycardias

	Regular	**Irregular**
Atrial	Sinus tachycardia Atrial tachycardia Atrial flutter Inappropriate sinus tachycardia Sinus node re-entrant tachycardia	Atrial fibrillation Atrial flutter with variable block Multifocal atrial tachycardia
Atrioventricular	Atrioventricular re-entry tachycardia (AVRT) AV nodal re-entry tachycardia (AVNRT) Automatic junctional tachycardia	

Broad Complex Tachycardias

Regular

- Ventricular tachycardia
- *Antidromic* atrioventricular re-entry tachycardia (AVRT).
- Any *regular* supraventricular tachycardia with aberrant conduction — e.g. due to bundle branch block, rate-related aberrancy.

All regular BCTs should be considered to be VT until proven otherwise.

Irregular

- Ventricular fibrillation
- Polymorphic VT
- Torsades de Pointes
- AF with Wolff-Parkinson-White syndrome
- Any *irregular* supraventricular tachycardia with aberrant conduction – e.g. due to bundle branch block, rate-related aberrancy.

Bradycardias

P waves present

Each P wave is followed by a QRS complex (= sinus node dysfunction)

- Sinus bradycardia
- Sinus node exit block
- Sinus pause / arrest

Not every P wave is followed by a QRS complex (= AV node dysfunction)

- AV block: 2nd degree, Mobitz I (Wenckebach)
- AV block: 2nd degree, Mobitz II
- AV block: 2nd degree, "fixed ratio blocks" (2:1, 3:1)
- AV block: 2nd degree, "high grade AV block"
- AV block: 3rd degree (complete heart block)

P waves absent

Narrow complexes

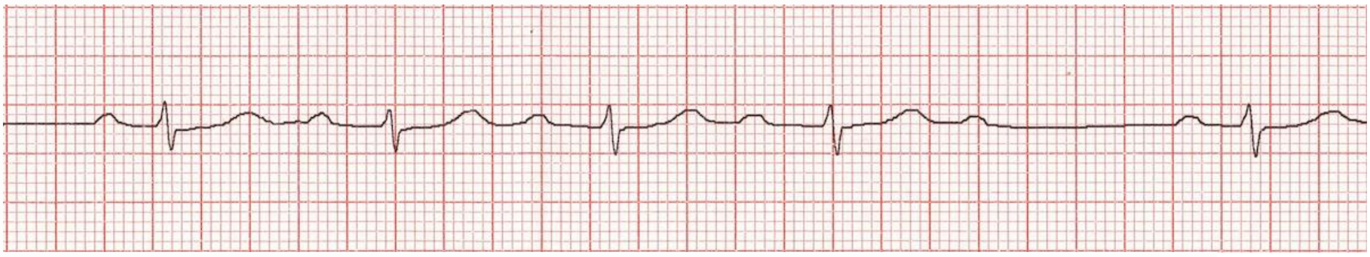
- Junctional escape rhythm

Broad complexes

- Ventricular escape rhythm

For escape rhythms to occur there must be a failure of sinus node impulse generation or transmission by the AV node.

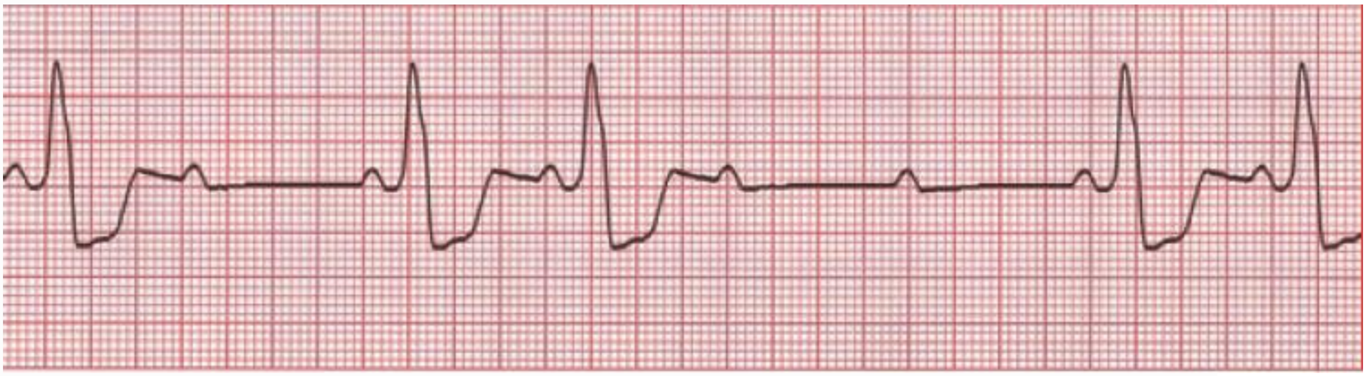
AV Block: 2nd Degree, Mobitz I (Wenckebach Phenomenon)



Definition

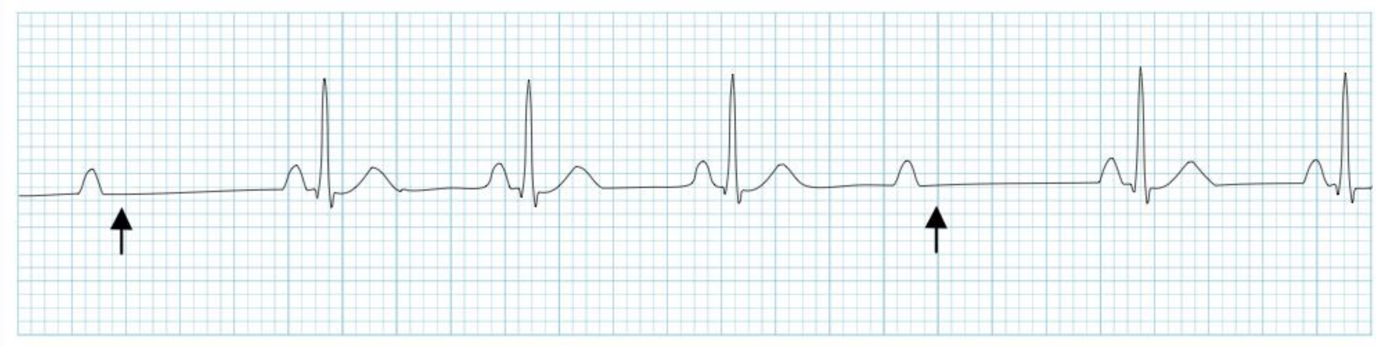
- Progressive prolongation of the PR interval culminating in a non-conducted P wave
- The PR interval is longest immediately before the dropped beat
- The PR interval is shortest immediately after the dropped beat

AV Block: 2nd Degree, Mobitz II



Definition

- Intermittent non-conducted P waves *without* progressive prolongation of the PR interval (compare this to [Mobitz I](#)).
- The PR interval in the conducted beats remains constant.
- The P waves 'march through' at a constant rate.
- The RR interval surrounding the dropped beat(s) is an exact multiple of the preceding RR interval (e.g. double the preceding RR interval for a single dropped beat, treble for two dropped beats, etc).



Arrows indicate “dropped” QRS complexes (i.e. non-conducted P waves)

Mechanism

- Mobitz II is usually due to failure of conduction at the level of the His-Purkinje system (i.e. below the AV node) (*structural* damage to the conducting system (e.g. infarction, fibrosis, necrosis).

Causes Of Mobitz II

- Anterior MI, fibrosis of the conducting system, cardiac surgery
- Inflammatory conditions (rheumatic fever, myocarditis, Lyme disease).
- Autoimmune (SLE, systemic sclerosis).
- Infiltrative myocardial disease (amyloidosis, haemochromatosis, sarcoidosis).
- Hyperkalaemia.
- Drugs: beta-blockers, calcium channel blockers, digoxin, amiodarone.

Clinical Significance

- Mobitz II is much more likely than Mobitz I to be associated with haemodynamic compromise, severe bradycardia and progression to 3rd degree heart block.
- Onset of haemodynamic instability may be sudden and unexpected, causing syncope **(Stokes-Adams attacks**) or sudden cardiac death.
- The risk of asystole is around 35% per year.
- **immediate admission for cardiac monitoring**, backup temporary pacing and ultimately insertion of a permanent pacemaker.

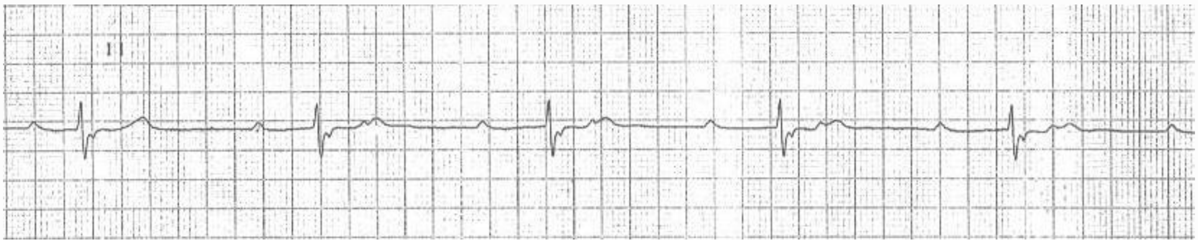
AV Block: 2nd Degree, “Fixed Ratio” Blocks

Definition

- Second degree heart block with a fixed ratio of P waves: QRS complexes (e.g. 2:1, 3:1, 4:1).

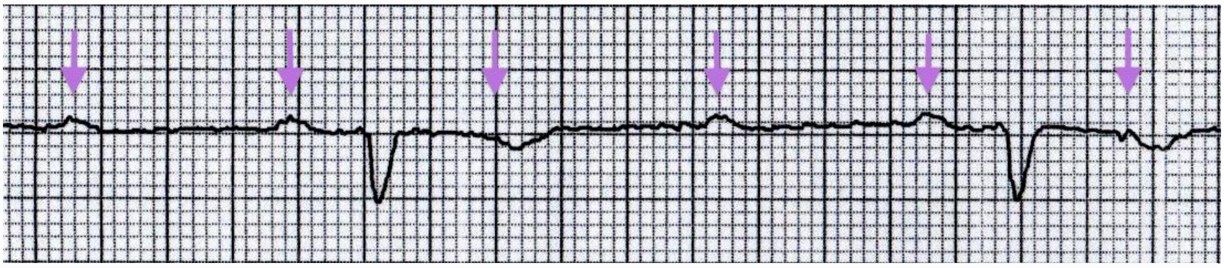
- Fixed ratio blocks can be the result of either Mobitz I or Mobitz II conduction.

2:1 block



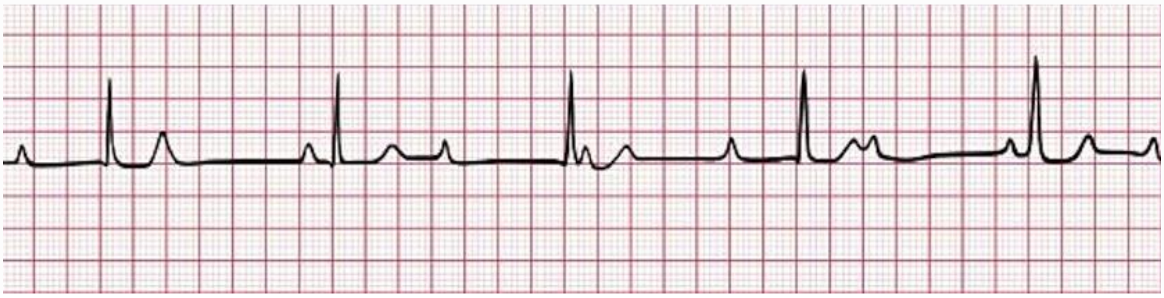
- The atrial rate is approximately 75 bpm.
- The ventricular rate is approximately 38 bpm.
- Non-conducted P waves are superimposed on the end of each T wave.

3:1 block



- The atrial rate (purple arrows) is approximately 90 bpm.
- The ventricular rate rate is approximately 30 bpm.
- every third P wave is almost entirely concealed within the T wave.
- Broad QRS complexes suggest that this may be due to Mobitz II block

AV Block: 3rd Degree (Complete Heart Block)

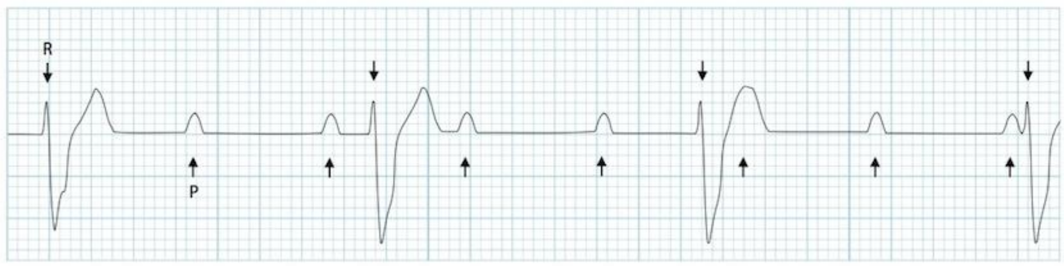


Definition

- complete absence of AV conduction – *none* of the supraventricular impulses are conducted to the ventricles.
- Perfusing rhythm is maintained by a junctional or ventricular escape rhythm. Alternatively, the patient may suffer ventricular standstill leading to **syncope** (if self-terminating) or **sudden cardiac death** (if prolonged).

- Typically the patient will have **severe bradycardia** with **independent atrial and ventricular rates**, i.e. AV dissociation.

Example of Complete Heart Block



- The atrial rate is approximately 100 bpm.
- The ventricular rate is approximately 40 bpm.
- **The two rates are independent**; there is no evidence that any of the atrial impulses are conducted to the ventricles.

Mechanism

- the end point of either Mobitz I or Mobitz II AV block
- progressive fatigue of AV nodal cells as per Mobitz I (e.g. secondary to increased vagal tone in the acute phase of an inferior MI).
- sudden onset of complete conduction failure throughout the His-Purkinje system, as perMobitz II (e.g. secondary to septal infarction in acute anterior MI).
- The former is more likely to respond to atropine and has a better overall prognosis.

Causes Of Complete Heart Block

- Inferior myocardial infarction
- AV-nodal blocking drugs (e.g. calcium-channel blockers, beta-blockers, digoxin)
- Idiopathic degeneration of the conducting system (Lenegre's or Lev's disease)

Clinical Significance

- high risk of ventricular standstill and sudden cardiac death
- urgent admission for **cardiac monitoring** is required, backup **temporary pacing** and insertion of a **permanent pacemaker**

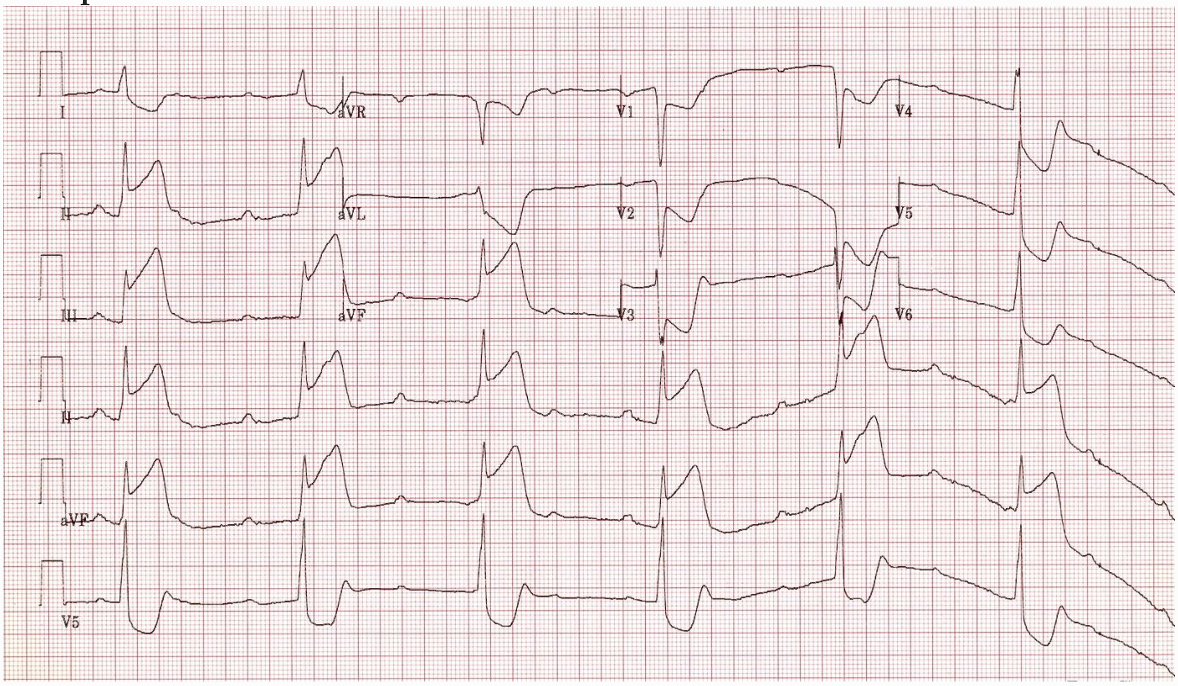
Differential Diagnosis

- **AV dissociation**:

occurrence of *independent atrial and ventricular contractions*

ECG Examples

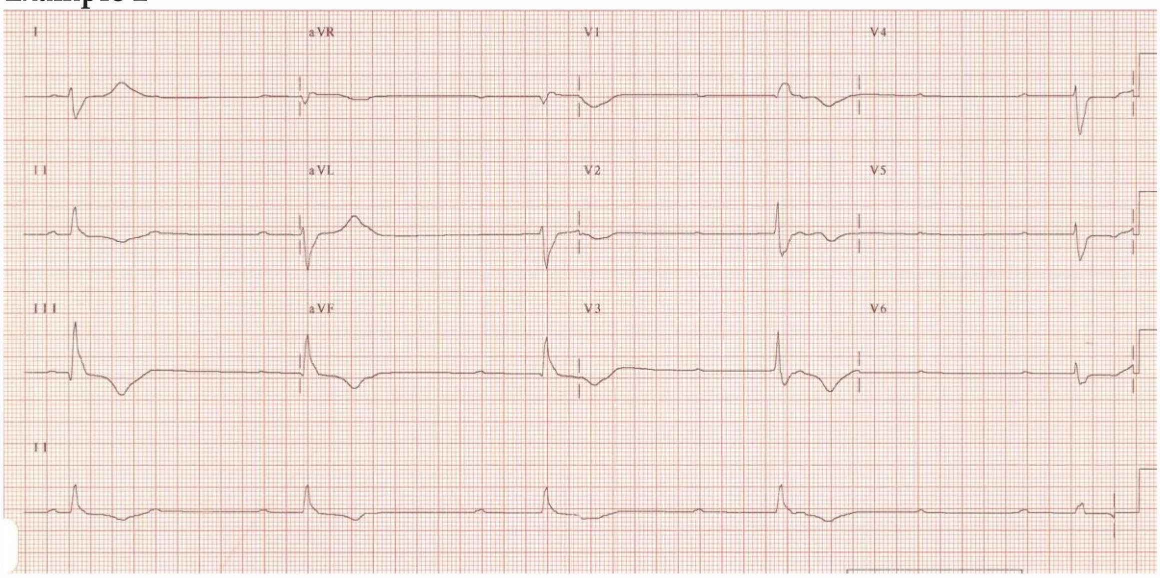
Example 1



Complete Heart Block:

- Atrial rate is ~ 85 bpm.
- Ventricular rate is ~ 38 bpm.
- None of the atrial impulses appear to be conducted to the ventricles.
- Rhythm is maintained by a junctional escape rhythm.
- Marked inferior ST elevation indicates that the cause is an inferior STEMI.

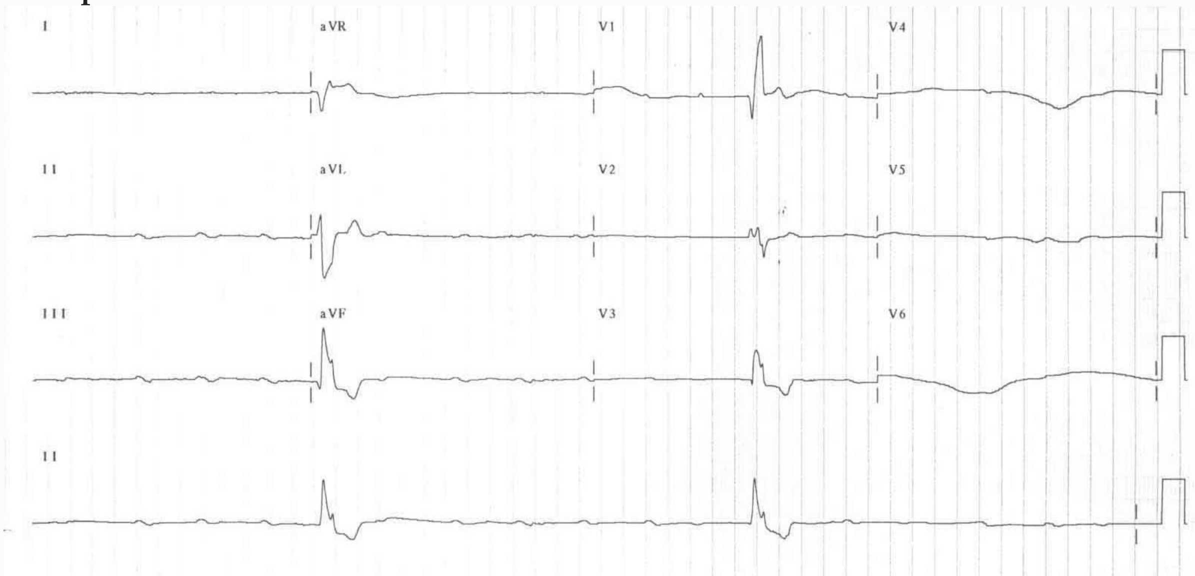
Example 2



Complete Heart Block:

- Atrial rate is ~ 60 bpm.
- Ventricular rate is ~ 27 bpm.
- None of the atrial impulses appear to be conducted to the ventricles.
- There is a slow ventricular escape rhythm.

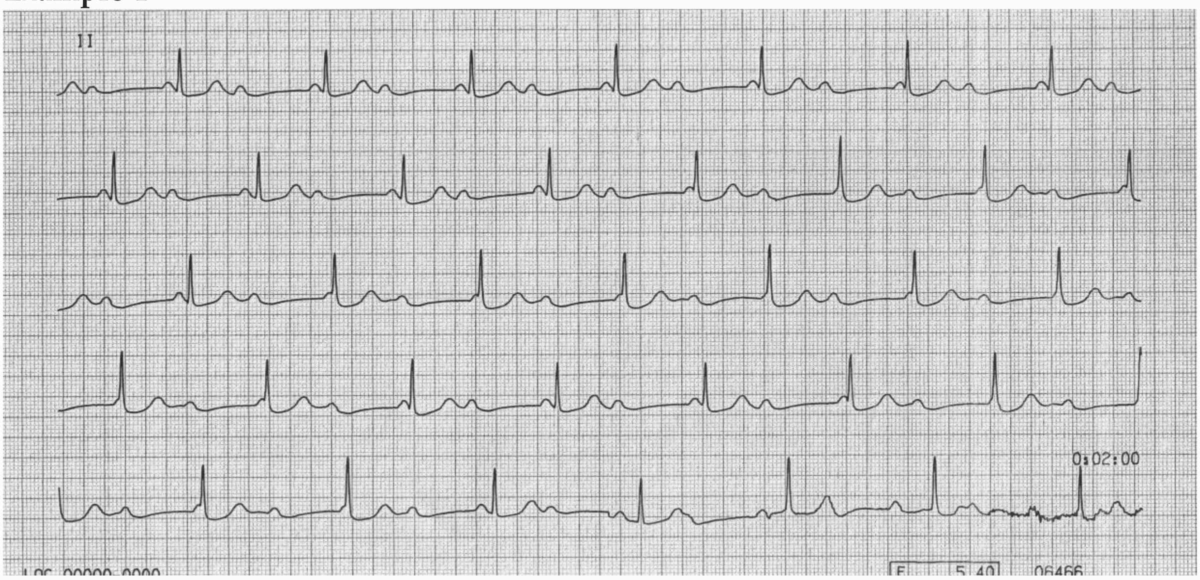
Example 3



Complete Heart Block:

- Atrial rate 100 bpm
- Ventricular rate only 15 bpm!
- This patient needs urgent treatment with atropine / isoprenaline and pacing!

Example 4



Complete Heart Block with Isorhythmic AV Dissociation (long rhythm strip):

- Atrial rate ~ 85 bpm
- Ventricular rate ~ 42bpm

- There is a junctional escape rhythm.
- As the ventricular rate is approximately half the atrial rate, this rhythm at first glance appears to be second-degree AV block with 2:1 conduction.
- However on closer inspection the PR interval varies, with some of the P waves superimposed on the QRS complexes. The ventricular rate remains regular.
- This confirms that **the atrial impulses are not being conducted to the ventricles**.
- The apparent relationship between the P waves and QRS complexes occurs merely by chance (= *isorhythmic* AV dissociation).