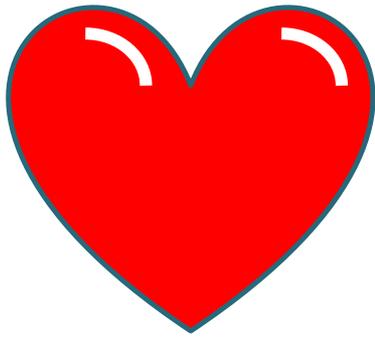


Mini Atlas On The Electrolytes and the Heart

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Life looks at electrolytes – answering how it rolls over with the grandiose gust of its orderly movement across its cellular boat and so it embraces celestial harmony to enjoy its existence to its full bloom. The heart twinkles with the same spirit.

- Sodium is mainly extracellular and the potassium mainly intracellular – the movement of these ions across the cardiac membrane is the key how the heart smiles.
- The sodium-potassium adenosine triphosphate pump is primarily responsible for resetting again the cardiac cellular homeostasis.

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Introduction



**Svante August Arrhenius
(1859-1927)
Swedish Physicist and
Physical chemist
(Father of Electrolytes)**

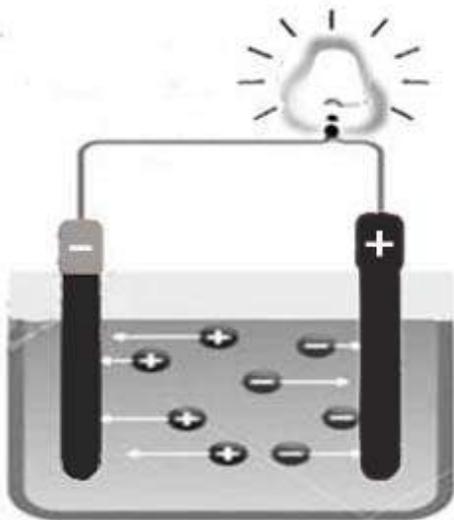
Arrhenius's main contribution is his theory of 'Electrolytic dissociation' (1887) – the Nobel prize in chemistry 1903

To conduct electricity one must have free-moving ions.

He noticed that the solution of acid conducts electricity by dissolving the substance in the solution, which dissociates into ions. This theory is known as "Electrolytic dissociation."

As per Arrhenius's theory of 'electrolyte dissociation' :

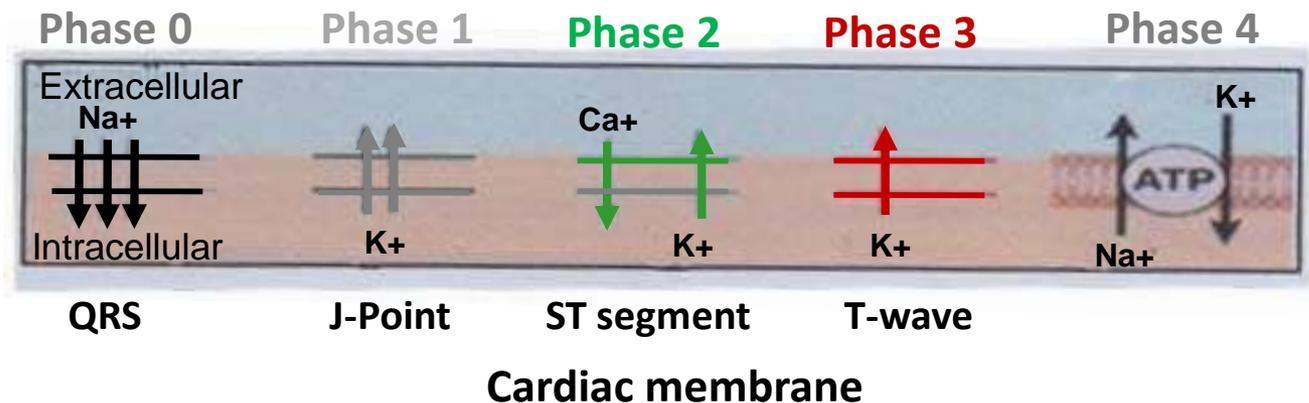
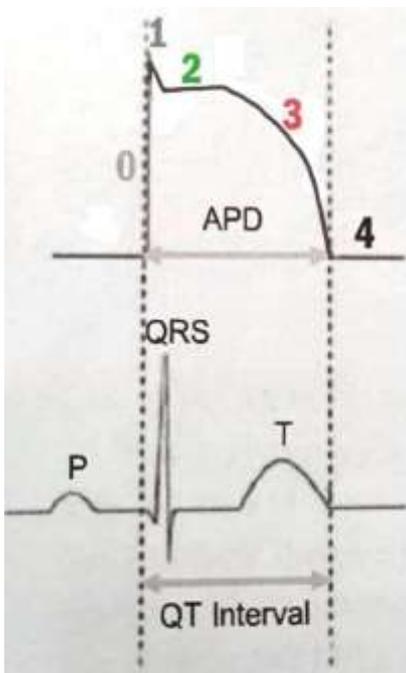
The word 'electro' denotes the state of electrolytes being in charged ionic state and the word 'lyte' (lytos) indicates the capability of electrolytes to undergo lysis when dissolved in a solvent – electrolyte dissociation.



An Electrolyte is a chemical substance when dissolved in aqueous solvent, it gets dissociated into cations (+Ve) and anions (-Ve) which are capable of producing electricity

The Cardiac Action Potential is a series of brief changes in voltage across the cardiac cell membrane, brought about by fluxes of ions through ion channels

- ❑ Sodium and calcium ions predominantly exist in the extracellular space and potassium ion exists mainly in the intracellular space
- ❑ Normal range (conventional units)
 - Normal serum potassium = 3.5-5.5 mEq/L
 - Normal serum calcium = 8.5-10.5 mg/dL
 - (Ionized serum calcium = 4.5-5.6 mg/dL)



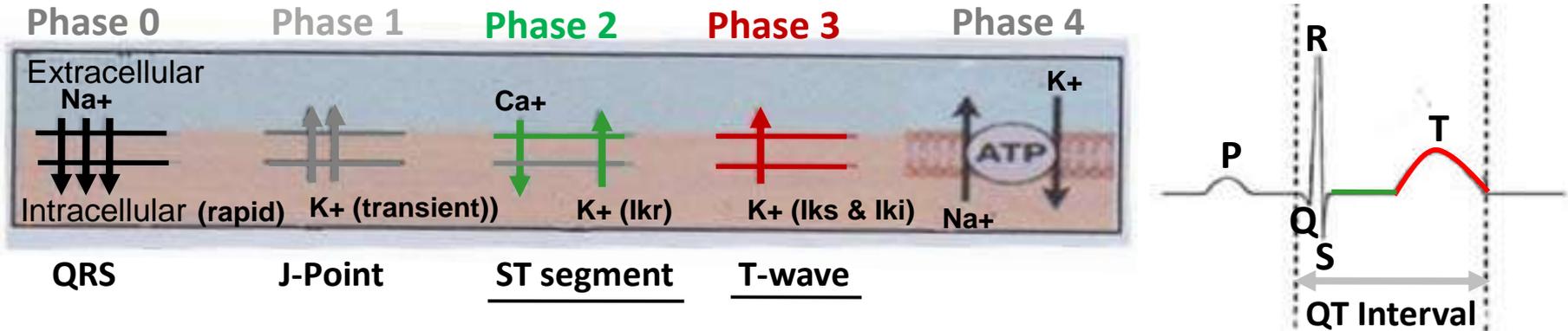


Electrolyte balance in a normal person

- **To maintain cellular homeostasis , the extracellular fluid must maintain specific concentrations of electrolytes so that the movement of ions across the cellular membrane may take place properly.**
- **One important function of electrolytes , particularly sodium , is to control fluid movement in between the fluid compartments.**
- **Sodium and potassium balance are maintained by the kidney through the action of hormone aldosterone**
- **Calcium balance is maintained by the integrated action of parathyroid hormone , calcitonin , and vitamin D.**



**A bird's eye view on
ECG changes
with electrolyte imbalance**



- ❑ In suspected cases of electrolyte disturbances one should carefully see the 'QTc' interval
 - Shortening of QTc interval , mainly due to shortening of Phase 2 (Ca⁺ - K⁺ exchange)
 - Hyperkalemia : QTc shortening with peaked tall T-wave.
 - Hypercalcaemia : QTc shortening , may be with complete absence of ST segment with compensatory wide based T-wave.
 - Prolongation of QTc interval
 - Hypokalemia : The merging of U-wave with T forming 'TU' complex
 - Hypocalcaemia : Due to the lengthening of ST segment (unlike , hypokalemia the ST segment is not displaced from the baseline)
- ❑ Other ECG changes associated with hyperkalemia should also be looked into : such as peaked tall T , ongoing atrial paralysis , SA nodal and AV nodal dysfunctioning with its appendages involvement, diffuse intraventricular conduction delay , sine wave .



Hyperkalemia

serum potassium > 5.5 mEq/L

Causes :

- **Decreased excretion by the kidneys**
 - **Renal failure**
 - **Potassium sparing diuretics such as spironolactone , triameterene , amiloride**
- **Shift of potassium in the extracellular space due to metabolic acidosis , crushed muscular injury , internal bleeding**
- **Increased consumptions of potassium rich fruits specially in a patient with renal failure**
- **Miscellaneous conditions**
 - **Endocrinopathy : Hypoaldosteronism , Addison's disease**
 - **Insulin deficiency**
 - **Hyperkalemic periodic paralysis**

NB : The common cause of hyperkalemia is the laboratory error due to potassium released as blood cells from the sample breakdown.

Electrophysiology in hyperkalemia

Peaked and tented T-wave is the initial manifestation due to increased electrochemical gradient (surplus K⁺) during phase 3

SA node is less sensitive to hyperkalemia and so is the interconnective link in between SA node and AV node

Ongoing atrial paralysis • P wave broadening /flattening

• PR prolongation

• Eventually complete disappearance of P wave

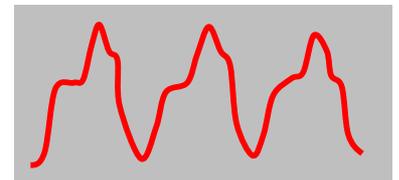
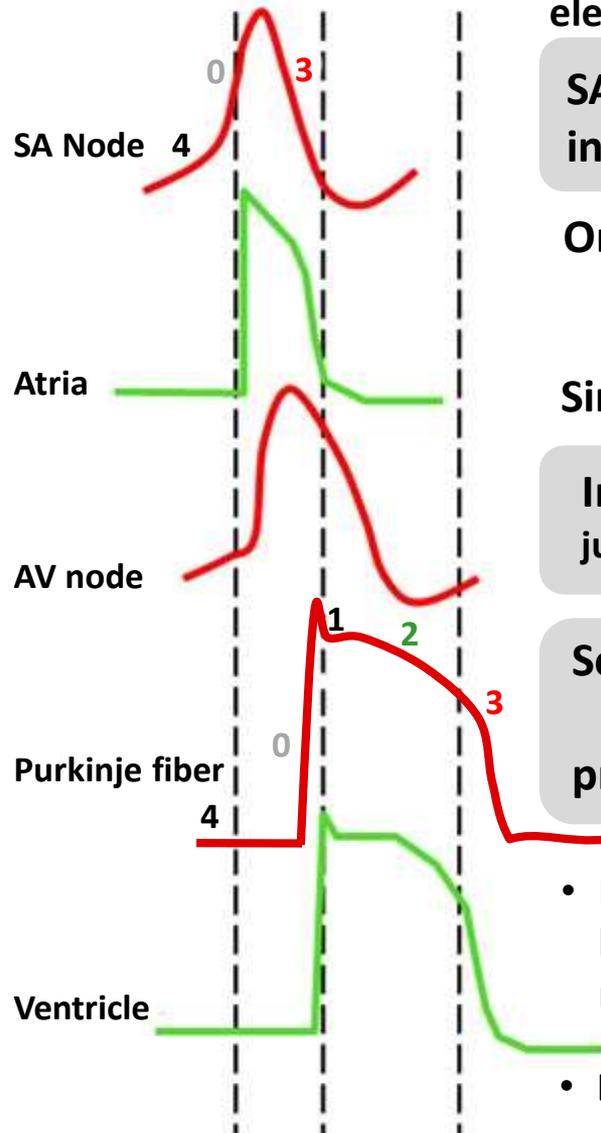
Sinus bradycardia / Sinus arrest (intact sinoventricular conduction)

Impaired AV conduction → different degree AV block (with slow junctional /ventricular escape rhythm), ± Bundle branch block, hemiblock

Secondary pacemaker capability of Purkinje fibers is suppressed → Infra nodal escape pacemaker is unreliable in the presence of heart block → frank asystole

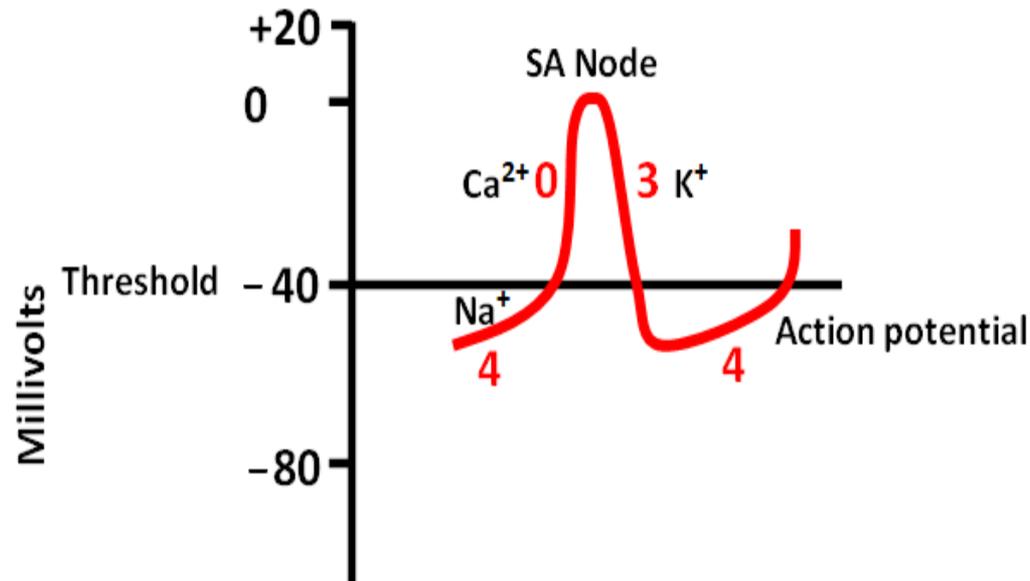
• Diffuse intraventricular conduction delay with broadening of QRS complex and T wave, both may merge together → Sine wave

• Even ventricular fibrillation



Red signal sign

Nodal system is less sensitive to hyperkalemia



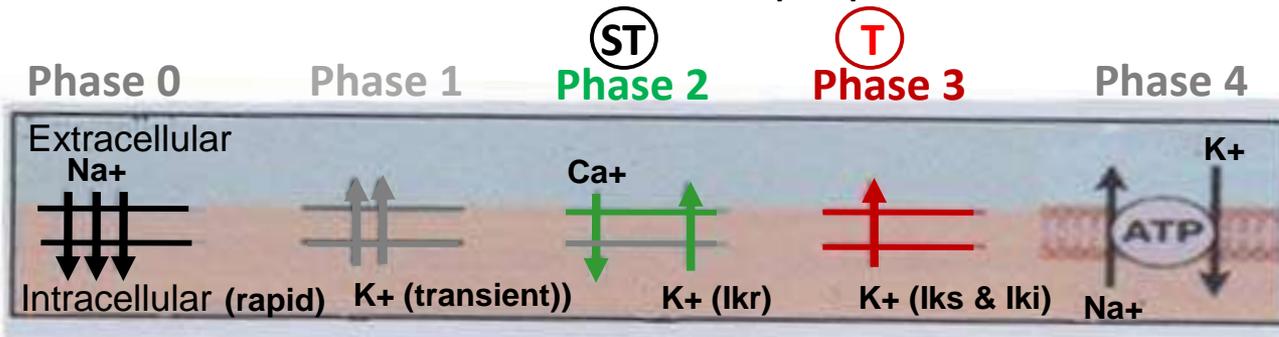
- Sinus node itself is less sensitive to hyperkalemia because of its low negative membrane potential with more steeping rise during Phase 4 (equipped with more stable automaticity)
- Sinoventricular conduction – hyperkalemic resistance to the internodal tracts connecting the SA node to the AV node.
- AV node is also relatively less sensitive to hyperkalemia (an important secondary pacemaker station , with lesser steep rise during Phase 4)

Mechanism of ECG changes in hyperkalemia

- ❑ Extracellular surplus K⁺ → Decrease phase 2 → shortening of QT (QTc) interval → T-wave near to QRS complex
- ❑ ↑ Electrochemical gradient (surplus K⁺) → Peaked Tall T



Surplus potassium



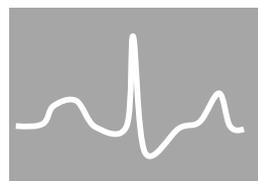
Normal



Tall peaked T with narrow base



ST depression



Broad low P with prolonged PR interval



Atrial arrest (No P wave)



Diffuse intraventricular conduction delay and merging of QRS with T

Sine wave

ECG changes as per potassium level (mEq/L)

Potassium level (mEq/L)	Mechanism	ECG changes
5.5 -6.5	↑ K ⁺ electrochemical gradient during phase 3	Peaked and tall T waves
6.5-7.0	Ongoing atrial paralysis	P wave broadening /flattening , PR prolongation Eventually complete disappearance of P wave
7.0-9.0	Conducting pathways abnormalities	<p>✓ Conduction pathway abnormalities with Bradyarrhythmias in association with <u>±</u> other evidences of hyperkalemia , serum potassium level should be estimated for the purpose</p> <ul style="list-style-type: none"> • Suppression of SA Node – Sinus Bradycardia • Different degree of AV block of high grade nature with slow junctional /ventricular escape rhythm. • Bundle branch block , hemiblocks
>9.0	Fewer Na ⁺ channels in operation due to its marked inactivation + very early onset of repolarization	Sine-wave

NB : These ECG changes in hyperkalemia do usually run as per quantum of the serum potassium level

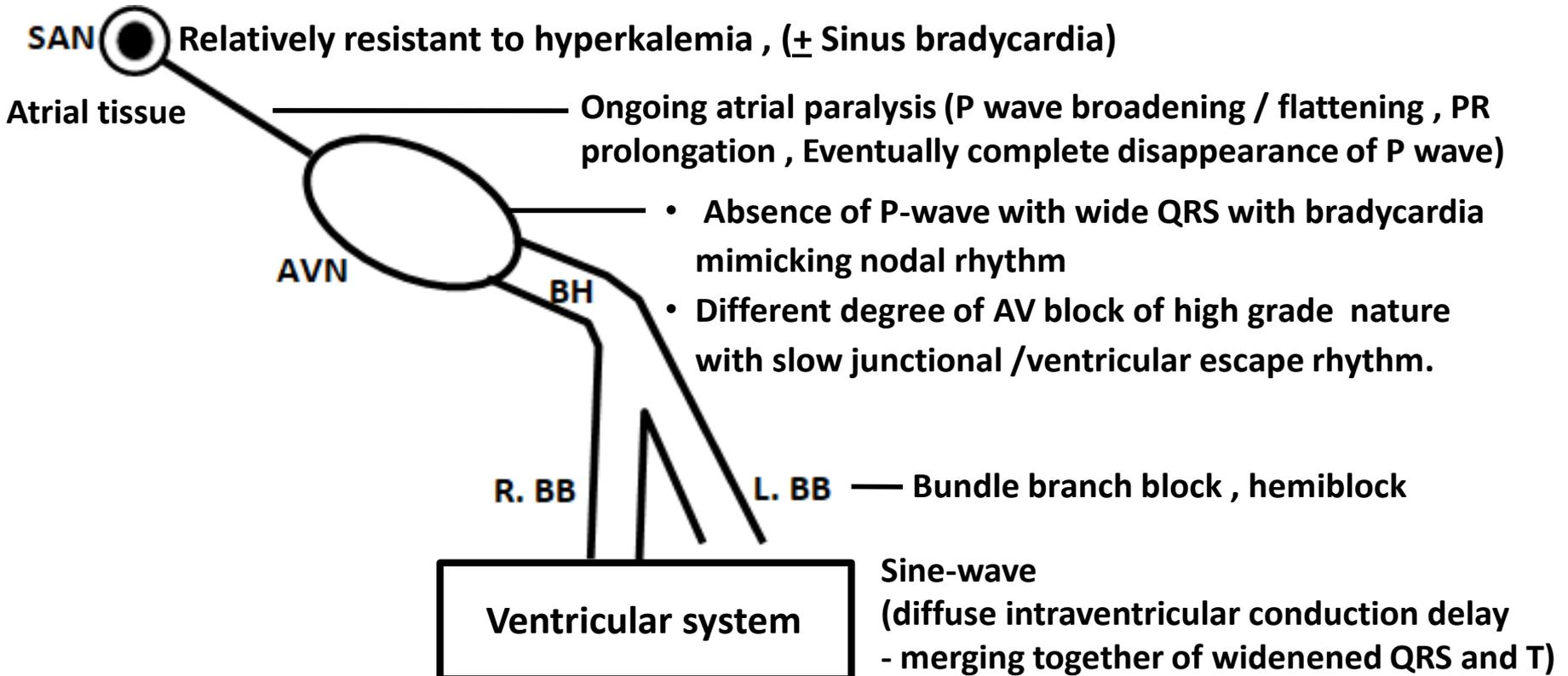
- ❑ The earliest ECG manifestation of hyperkalemia is seen with Serum potassium near about 6 mEq/L by the presence of high peaked tall tented T-waves , seen in all the leads especially over the precordial leads. With the further rise in serum K⁺ level , its amplitude and peaking character decreases but width increases. At much higher levels , the T-waves becomes broad with bizarre QRS running together to inscribe sine wave.
- ❑ 'Braun et al. found that the characteristic tall , steep , narrow and pointed T-wave were present in only 22% of patients with hyperkalemia , whereas in the remainder the tall T-wave could not be distinguished from similar T-waves of other etiology.'
(Chou's Electrocardiography in Clinican Practice – Sixth Edition – P 532)

This flow chart only aims at to organize the mind setup of the clinician to plot ECG changes in hyperkalemia

T-wave changes

- High-peaked tall tented T-waves
- With further rise in serum K level , its amplitude and peaking character decreases but its width increases

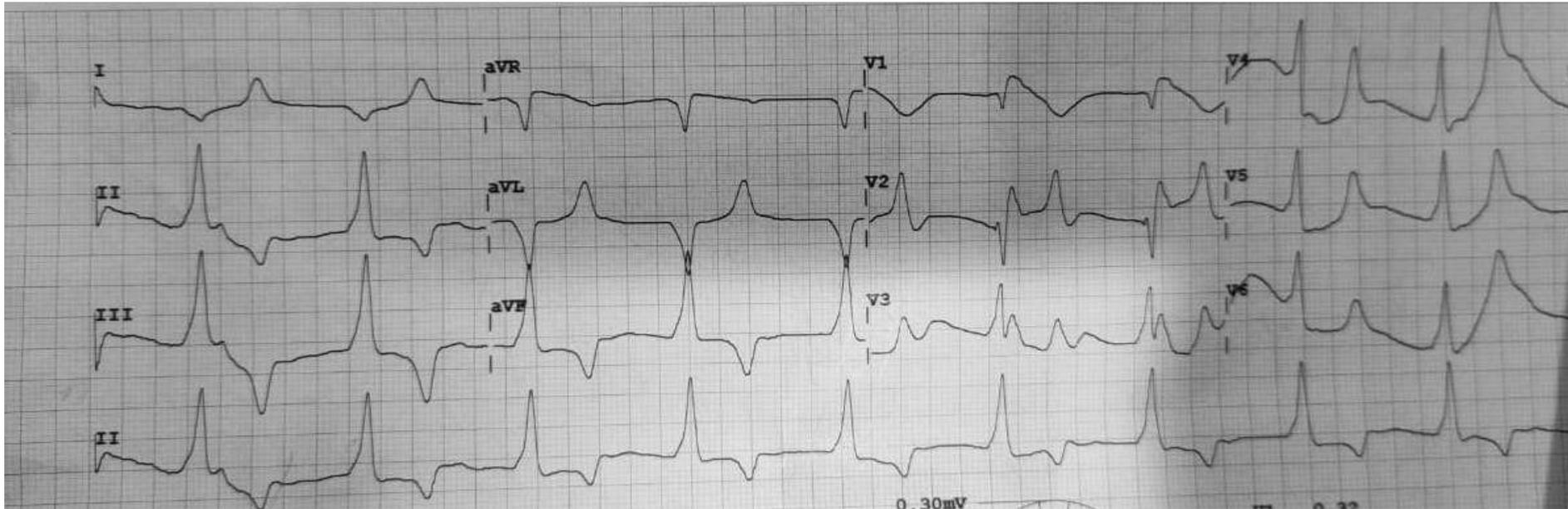
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ECG 2

Diabetic middle aged male

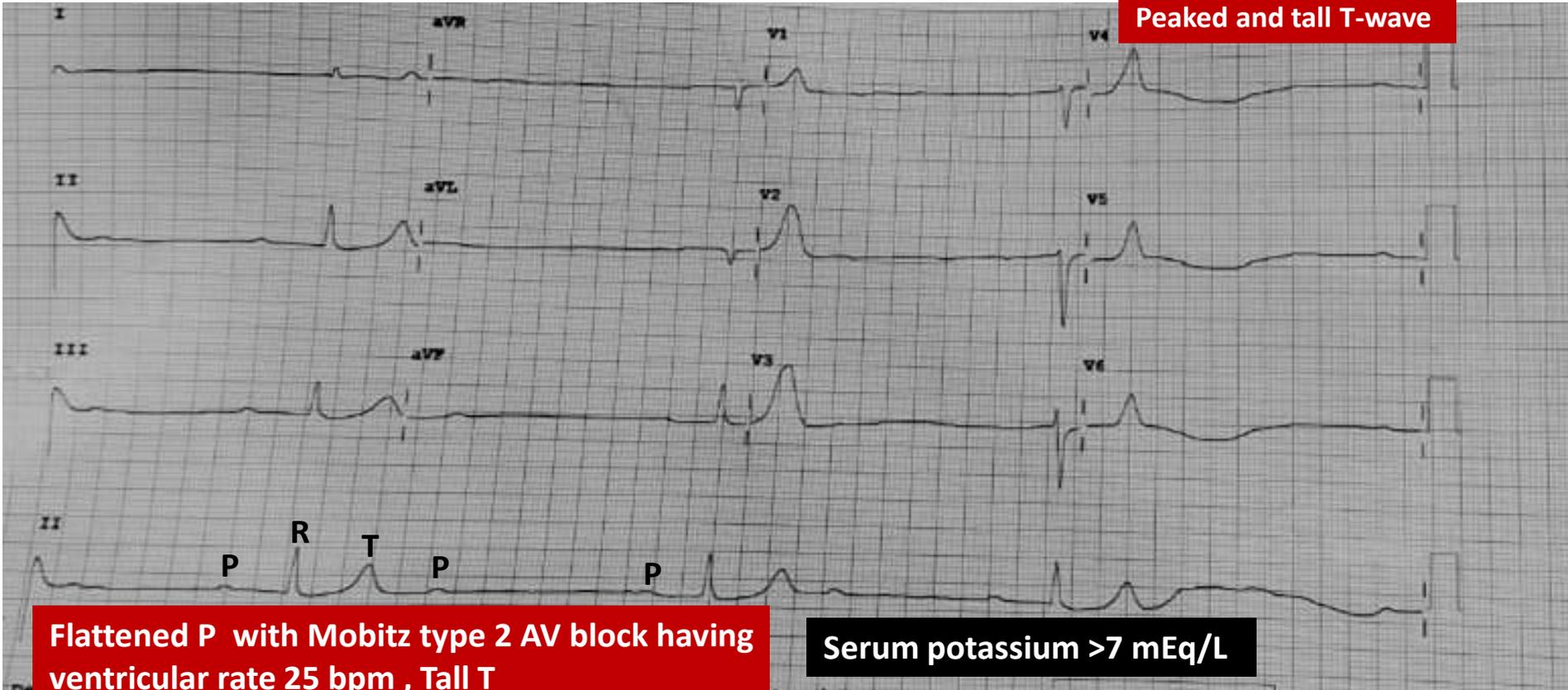
Serum K 6.5 mEq/L , Serum Creatinine 3.5 mg



- ❑ ECG with hyperkalemia (tall peaked T wave and absence of P wave with wide QRS and bradycardia mimicking nodal rhythm).

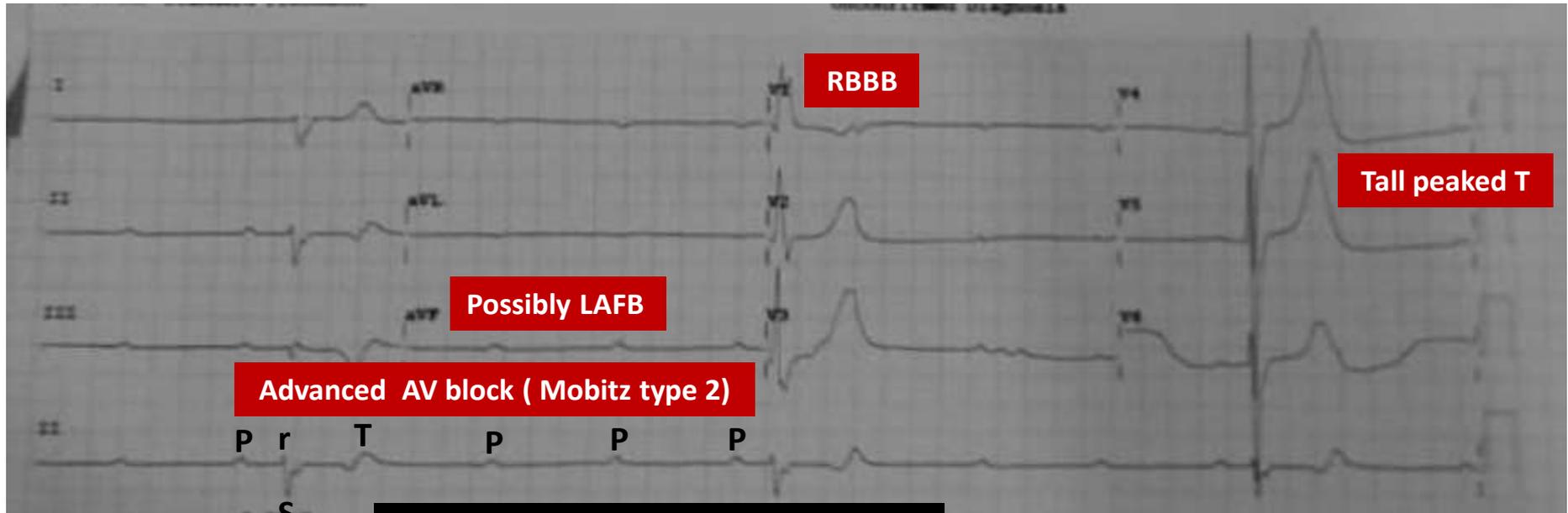
ECG 3

Male aged 41 years with h/o giddiness and fainting attack



ECG 4

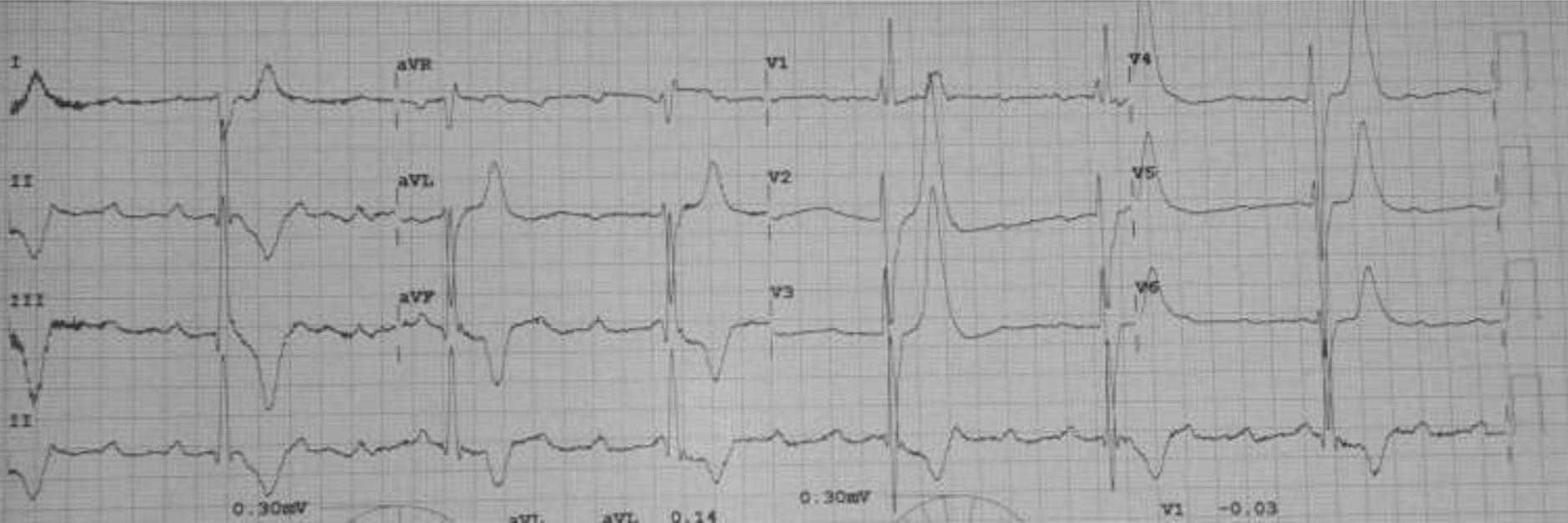
A male aged 45 years with h/o fainting attack
Serum K 7.4 mEq/L



ECG suggestive of hyperkalemia

ECG 5

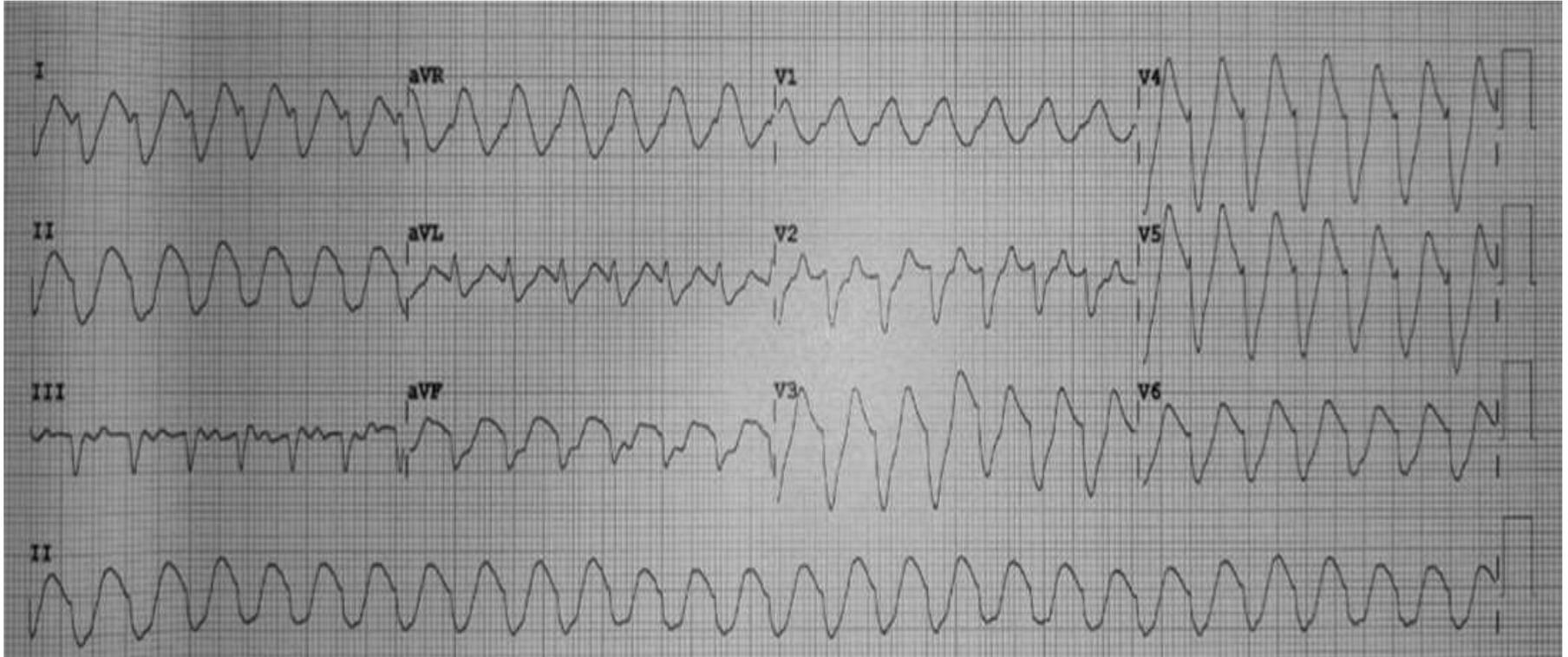
Middle aged Diabetic male with severe chest pain since 3 hours
(Random blood sugar 200mg, Serum K 5.7, Serum Creatinine 1.5 Trop I positive)



- One should suspect hyperkalemia in the presence of conduction pathway abnormality e.g. **CHB associated with tall and peaked T-wave**
- Inferior MI (Non-STEMI)

ECG 6

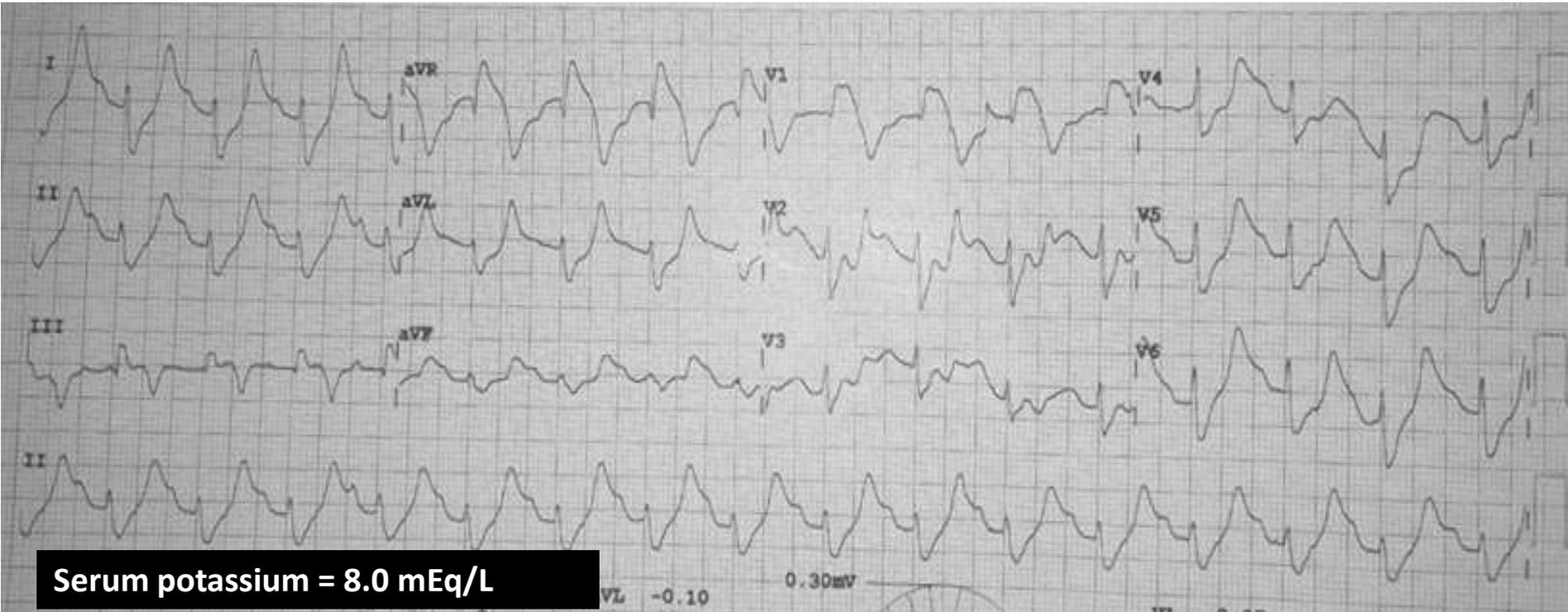
Middle aged diabetic female presenting with weakness , nausea and vomiting (serum potassium = 7.6 mEq/L and Serum creatinine = 4.80 mg/dL)



- Ventricular rate 186 bpm
- Rhythm lead II shows a run of up and down oscillations , almost with equal pacing and amplitude --- Sine wave
- Lead V1 is showing much widening of the QRS complex almost equivalent to 0.28 sec simulating with right bundle branch block pattern.

ECG 7 (A)

Patient aged 50 years in unconscious state
BP = 180/110 mmHg with deep rapid breathing



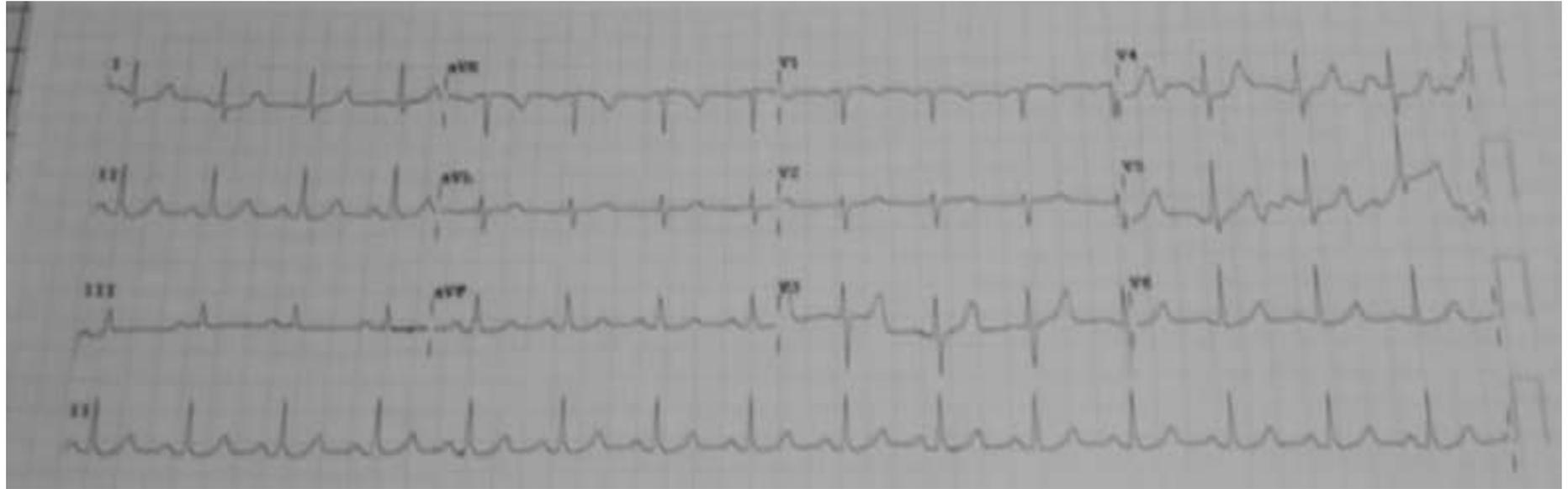
Sine wave (diffuse intraventricular conduction delay) with RBBB pattern in V1 with extreme width 220 ms.

A fewer Na⁺ channels availability to be activated during depolarization contributes to widened QRS plus early initiation of repolarization phase having more widened T → fusion of T-wave with the preceding QRS complex.

D/D : when the heart rate is rapid the ECG simulates ventricular tachycardia

ECG 7 (B)

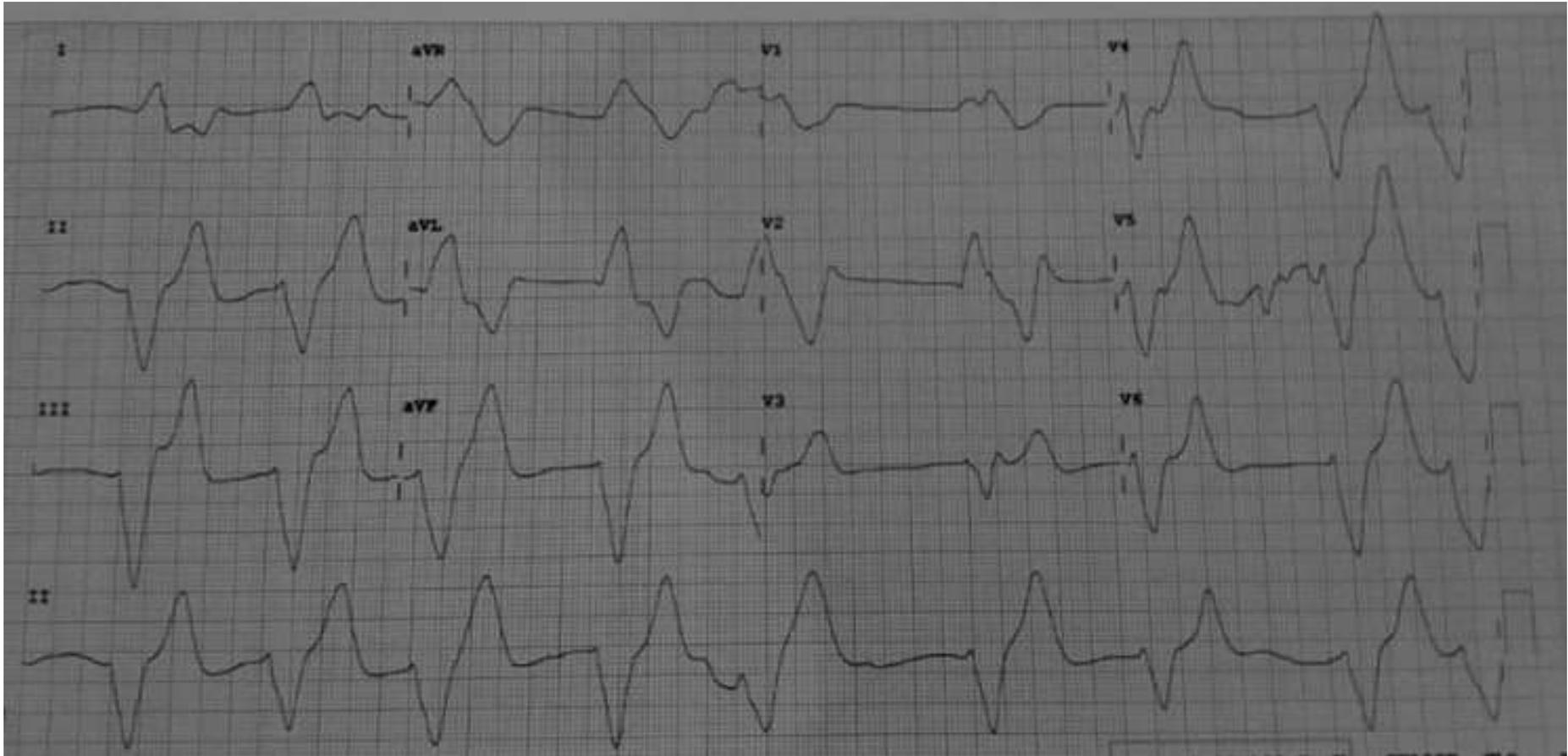
The same patient's ECG after recovery



Serum K corrected to normal = 4.5 mEq/L

ECG 8

A 24 years lady presented with breathlessness (ESRD with Serum K 7.88 mEq/L)



The ventricular rhythm has become irregular , most likely (? A possibility) due to simultaneous activity with several escape pacemakers in the depressed myocardium , attributed to hyperkalemia (serum K = 7.88 mEq/L). Usually this phenomenon sets in when the plasma K concentration exceeds 10 mEq/L.

– Chou's Electrocardiography in Clinical practice , 6th edition , P- 517

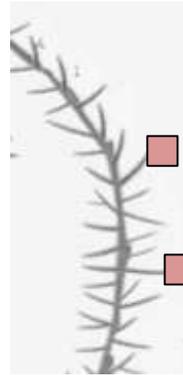


Hypokalemia

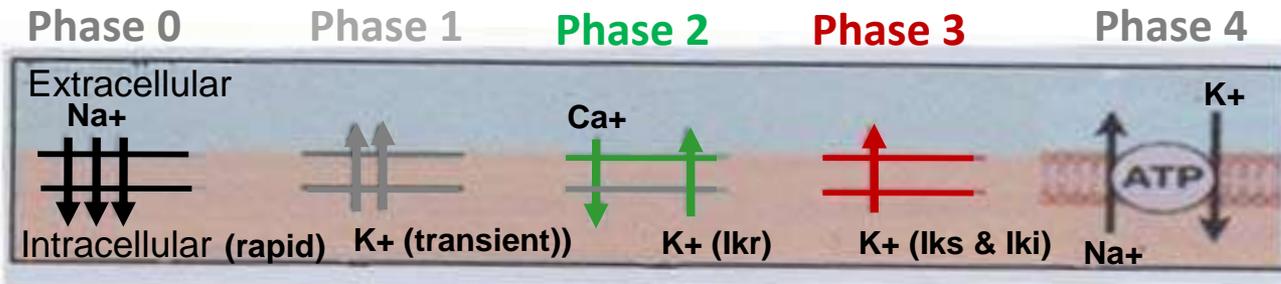
Normal serum potassium level is 3.5 to 5.5 mEq/L. The ECG changes in hypokalemia appear when K⁺ level falls below 3.0 mEq/L.

Causes :

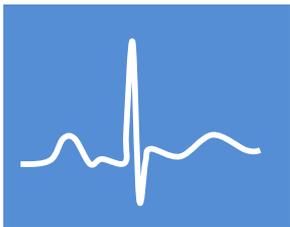
- **Potassium loss through vomiting and diarrhea**
- **Low dietary intake**
- **Increased excretion of potassium with diuretic use (especially loop diuretics) , hyperaldosteronism, renal tubular acidosis , diabetic ketoacidosis , glucocorticoids-use**
- **Conditions causing intracellular shift of potassium such as hypokalemia periodic paralysis , insulin effect , alkalosis**



- Normally Synchronized repolarization of cardiac myocytes and Purkinje fibres together
- In hypokalemia dichotomized repolarization : delayed and prolonged repolarization through purkinje fibres → T-U complex → fusion of T with U → QT prolongation



In hypokalemia the repolarization pattern is gradually shifted towards delayed and prolonged repolarization through purkinje fibres



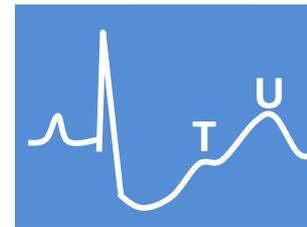
Normal



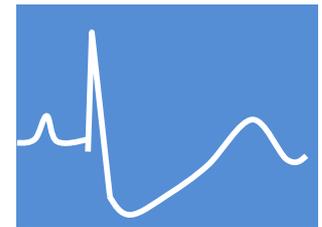
Low U wave



Prominent U wave

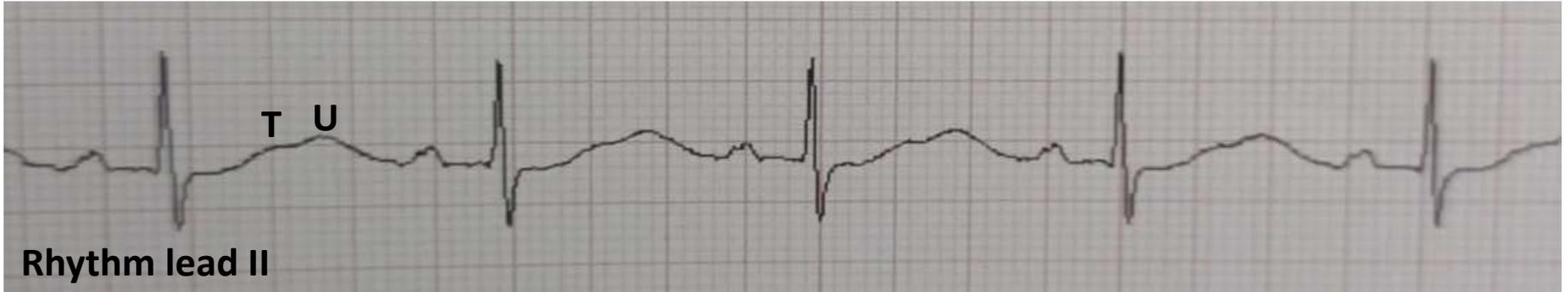


• T-U complex with ST depression



• Fusion of T with U → QT prolongation

40 years male with h/o loose motions since last 3 days (Serum Potassium = 2.5 mEq/L)



Findings :

- PR prolongation (0.22 sec)
- T-U complex (large U wave > 1 mm that is fused with the T-wave should be included in the measurement of QT interval) to be mentioned that QT interval is considered to be prolonged if it is more than half of the corresponding RR interval.
- Associated ST depression > 0.5 mm

✓ Surawicz et al. considered the following three ECG features : (1) depression of the ST segment of ≥ 0.5 mm; (2) U wave amplitude > 1 mm ; and (3) U wave amplitude greater than the T wave amplitude in the same lead .

Considered “compatible” with hypokalemia with two of these features or one related to the U wave are present.

(Ref : CHOU’S Electrocardiography in Clinical Practice – Sixth Edition , P 539)



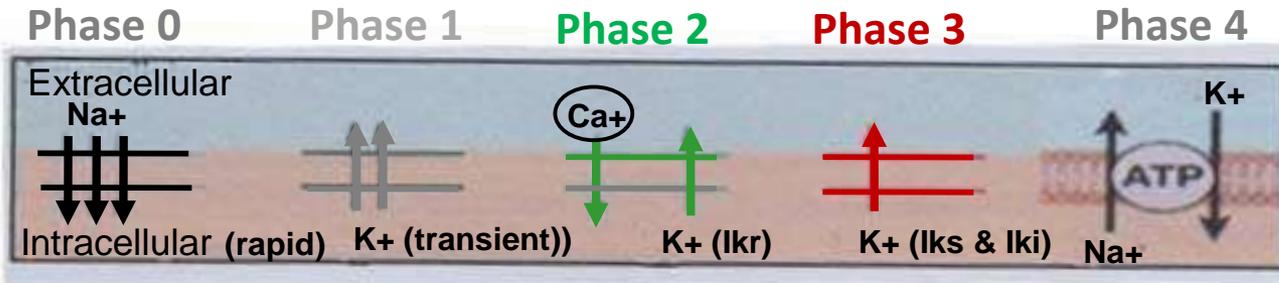
Hypercalcaemia

- Three overlapping regularity systems for calcium homeostasis are highly integrated by three hormones : PTH (parathyroid hormone) , Calcitonin and Vitamin D.
 - Each system operates through bone , intestine and kidney
-

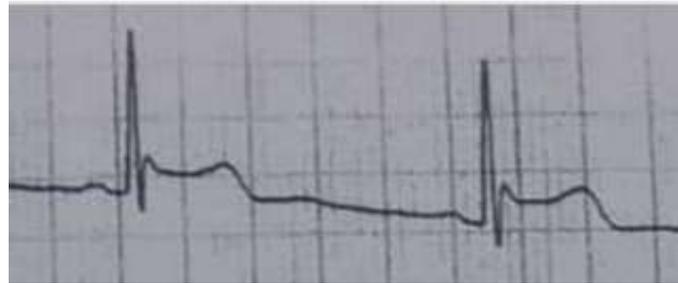
Causes :

In brief Hypercalcaemia usually occurs with hyperparathyroidism , renal failure and with the administration of excessive dose of vitamin D .

- Primary Hyperparathyroidism
- Bones related
 - Solid tumors with bone metastasis
 - High bone turnover as with immobilization
- Vitamin D related
 - Vitamin D intoxication
 - Sarcoidosis and other granulomatous diseases
- Associated with renal failure
 - Secondary hyperparathyroidism



- ❑ Shortening of Phase 2 \rightarrow Shortening of ST segment \rightarrow Shortening of QT interval (ST segment may be completely absent and is replaced by T wave having widened base to counteract the shorten QT interval – a compensatory effect of hypercalcaemia).
- ❑ Since the QT interval is shortened in hypercalcaemia , initial upstroke of the T wave may immediately start after the QRS complex , mimicking the hyperacute phase of myocardial infarction , especially when the T waves are rapid and taller than usual
- ❑ When the upstroke of T-wave starts immediately after the QRS complex , it indicates more rapid entry of calcium ions inside the myocardial cells. To equalize the electrochemical gradient there is somewhat exaggerated K^+ exit in phase 1 resulting in the augmentation of the J-wave – termed as Osborn wave.

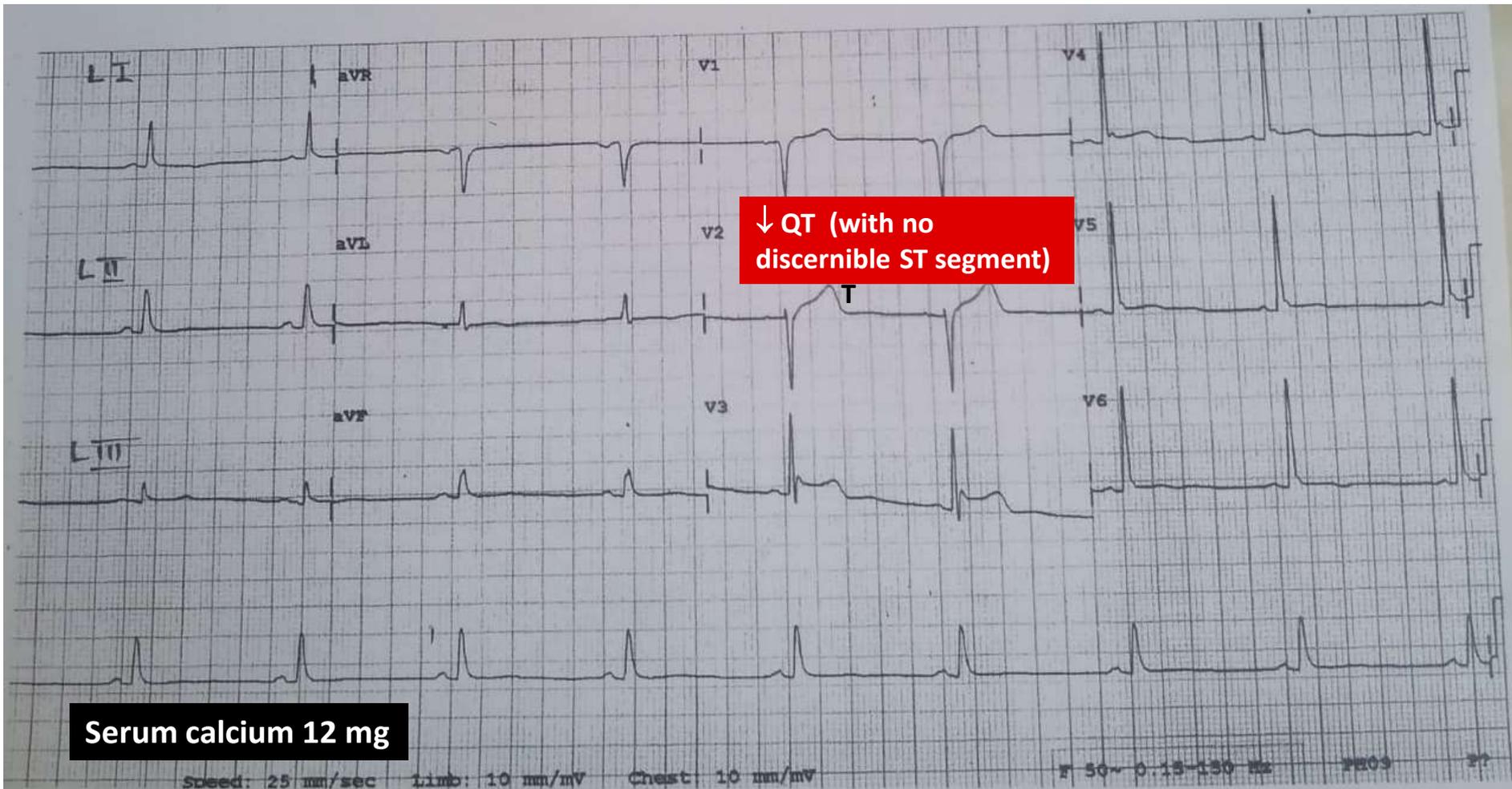


Elevation of the J point with shortened QT interval , followed by T wave having widened base (so counteracting the shortened QT interval - a compensatory effect).

20

74 years old man , a chronic smoker with h/o Polyuria , Polydysia , Fatigue and Pain abdomen. Physical examination was unremarkable , Chest X-ray normal , Blood sugar , urea , serum creatinine , Na^+ , Cl^- and HCO_3^- , Parathyroid hormone , Vitamin D – all were normal (no evidence of nephrocalcinosis or renal stone)

Illustration
by ECG



ECG features are suggestive of Hypercalcaemia

Familial Hypercalcaemia



Hypocalcaemia

Normal serum calcium = 8.5-10.5 mg/dL
(Ionized serum calcium = 4.5-5.6 mg/dL)

NB :

- The blood for calcium estimation should be taken without the help of the tourniquet
 - Hypocalcaemia diagnosis requires checking the serum albumin level to correct for total serum calcium
-

Causes :

- In a hypocalcaemic state a decrease in PTH quantum from the parathyroid glands and a decrease in the release of CT (calcitonin) from the parafollicular cells of the thyroid gland occur.

Hypoparathyroidism

- Vitamin D - decrease mobilization of calcium from bone , decreased absorption from intestine and kidney

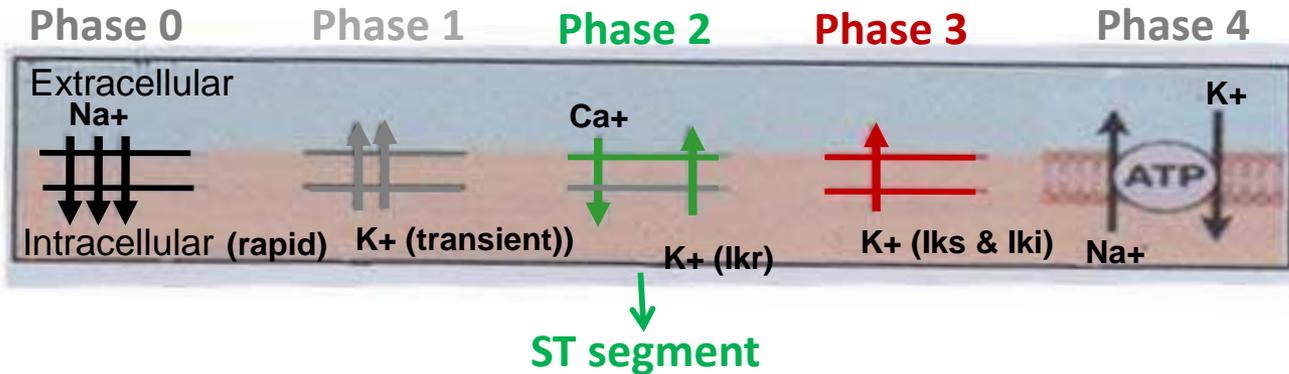
Vitamin D deficiency

- Miscellaneous causes –

Severe Diarrhoea

*** Acute pancreatitis , etc**

- * Usually , acute pancreatitis is associated with a decrease in serum calcium and thus it constitutes a prognosis factor during the first 48 hours of illness

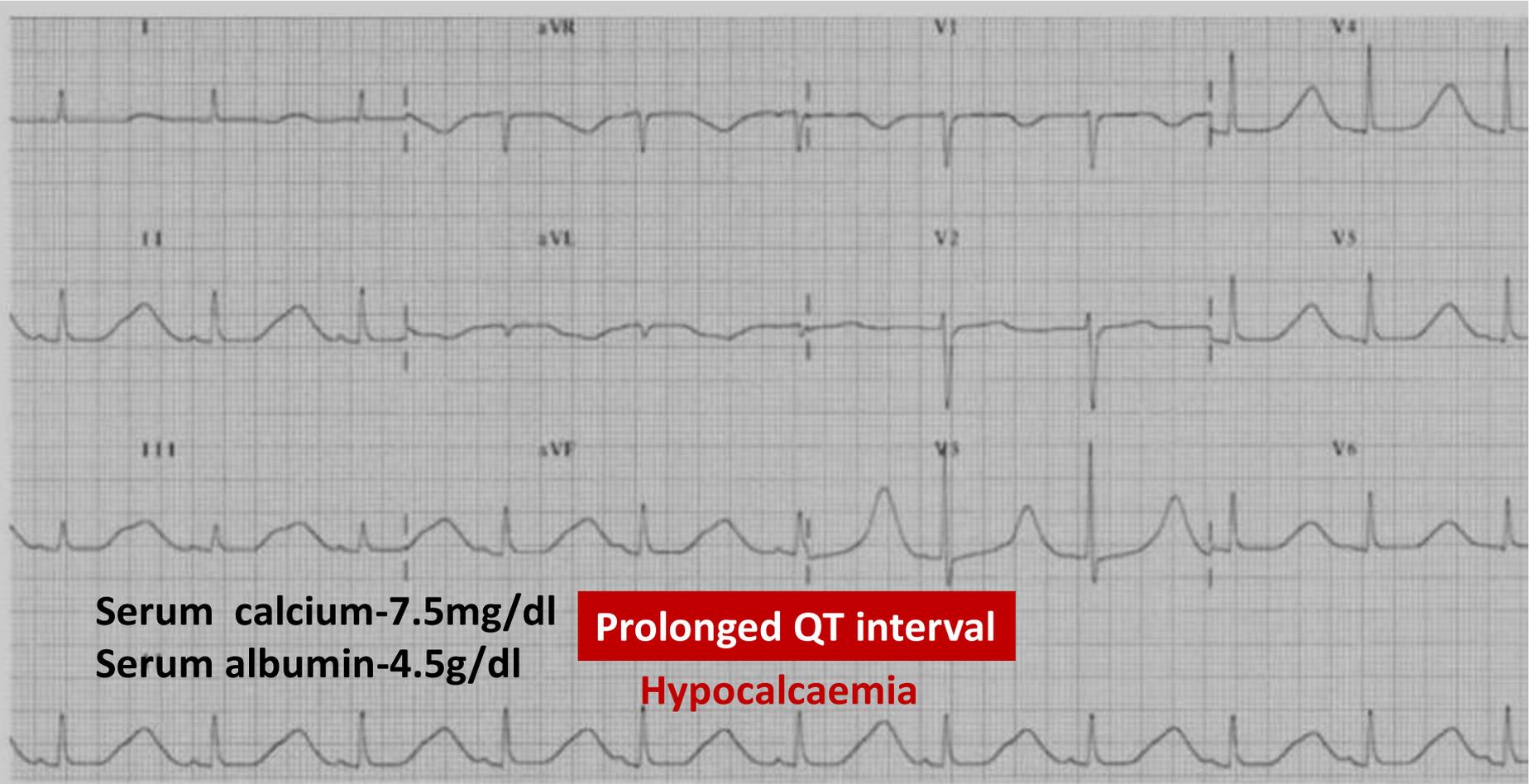


- ❑ **ST segment** : During this phase outward and inward currents (Ca^{2+} - K^+) are approximately equal but with reverse polarity on either side → practically no voltage gradient across the cardiac membrane is found during this period → Isoelectric ST segment
- ❑ Prolongation of Phase 2 → Prolongation of ST segment that tends to be isoelectric → QT prolongation
The prolongation of QT interval is inversely proportional to the serum calcium level



Hypocalcaemia : QT prolongation

45 years Female presented to the emergency department with recurrent seizures since 1 day. Her ECG shows prolonged QT interval (✓ QT interval exceeding half of the corresponding RR interval).





**Pertinent points to be
considered**

❑ Hypomagnesemia

The ECG changes are similar to hypokalemia. This should be stated here that if potassium supplementation tends not to normalize the QTc interval , hypomagnesemia must be suspected.

❑ Hypermagnesemia

The ECG findings are similar to hyperkalemia but no definite criteria has been laid down.

❑ Uremia

Uremia on ECG is recognized by hyperkalemia \pm hypocalcaemia with prolongation of QTc interval



Arrhythmogenicity

- ❑ **Severe hyperkalemia** can lead to heart block , asystole , pulseless electrical activity and ventricular arrhythmias including ventricular fibrillation.
- ❑ **With hypokalemia**
 - Supraventricular tachyarrhythmias : AF , atrial flutter, atrial tachycardia
 - Even moderate hypokalemia may inhibit the sodium potassium pump in myocardial cells promoting spontaneous early afterdepolarizations that lead to ventricular tachycardia/fibrillation. At times a few isolated ventricular extrasystoles.

- ❑ **Cardiac arrhythmias are uncommon in patients with hypercalcemia.** Hypercalcaemia decreases ventricular conduction velocity and shortens the effective refractory period. Ventricular arrhythmias ranging from VPCs to Frank Ventricular fibrillation can be encountered in severe hypercalcaemia
 - ❑ **In hypocalcaemia** arrhythmias are uncommon , although atrial fibrillation has been reported
Torsades de pointes may occur , but is much less common than hypokalemia and hypomagnesaemia.
-

Thanks

