

# GP Study Webinar

Acute Blood Results 12<sup>th</sup> May 2022



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Metabolic Medicine Registrar



Patient focused



Collaborative



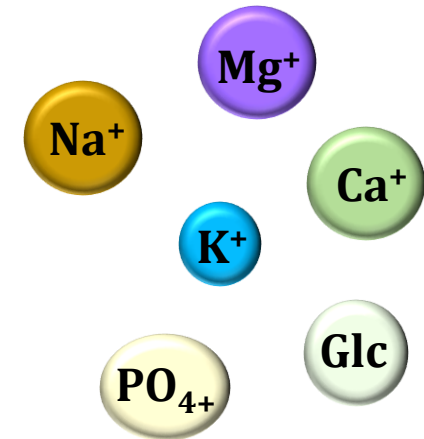
Expert



Caring

# Overview

- How the lab deals with critical results
- Action Limits
- Common electrolyte disturbances
- Interpretation & Investigation of abnormalities
- Erroneous Results
- Role of the Duty Biochemist



# Action Limits DAY

	Lower limit ( ≤ )	Higher Limit ( ≥ )	Units	Notes
Sodium	125 (>16y) 130 (<16 y)	150	mmol/L	120 -155 if in-patient (IP)
Potassium	2.5	6.5	mmol/L	Not haemolysed <2.0 on known renal patients.
Urea	None	30 (adult) 10 (<16 y)	mmol/L	Only phone GP/OP & only if first time call out limit exceeded
Creatinine	None	354 adult 200 (<16 y)	μmol/L	
Bicarbonate	10	None	mmol/L	
Adj. Calcium	1.8	3.2	mmol/L	Adj only (consider Tot Ca if no Alb)
Magnesium	0.4	None	mmol/L	
Phosphate	0.3	None	mmol/L	
Glucose	2.5 (>28d) 1.9 (0-28d)	25.0 (adult) 15.0 (<18 y)	mmol/L	2.0 - 30.0 if known diabetic
Antenatal, GAN	as above	7.8	mmol/L	Refer to DB to email
Total bilirubin	None	300	μmol/L	Only if patient is under 1 year of age
Conj Bilirubin	None	25	μmol/L	< 6 weeks old only
Ammonia	None	100	μmol/L	Sample not haemolysed or >4h old
Triglycerides	None	20	mmol/L	Above 30 if IP/OP
ALT/AST	None	600	IU/L	
CK	None	5000	IU/L	
Amylase	None	400	IU/L	
CRP	None	300	mg/L	Excluding hospital patients
Bile acids	None	30	μmol/L	
TSH	None	50	mIU/L	
Free T4	5.4	40	pmol/L	(<5.4 Alinity, < 5.2 Architect)
Free T3		20	pmol/L	
Cortisol	100	None	nmol/L	< 50 phone (not if part of ONDST), 50-100 nmol/L refer to DB.
30 min cortisol	250			COR30 refer to DB
Oestradiol	None	15000	pmol/L	
Carbamazepine	None	25	mg/L	
Digoxin	None	2.0	μg/L	If taken 6h post dose. Upper limit 2.5 μg/L if K >3.0 mmol/L
Lithium	None	1.5	mmol/L	If 12h post dose. Check tube type.
Paracetamol	None	1	mg/L	Cut off will remain 100 mg/L for A&E
Phenobarbitone	None	70	mg/L	
Phenytoin	None	25	mg/L	
Salicylate	None	300	mg/L	
Theophylline	None	25	mg/L	
Urate		340		Pregnant women only; not renal
Ethanol		4000	mg/L	
Troponin		>15F, >34M	ng/L	GP only
AKI		2,3 1		Call only if K > 6.0 mmol/L

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Ammonia	None	100	μmol/L	Sample not haemolysed or >4h old
Triglycerides	None	20	mmol/L	Call if IP > 30 mmol/L
ALT/AST	None	600	IU/L	* GP/OP Refer to DB/call in morning
CK	None	5000	IU/L	* GP/OP Refer to DB/call in morning
Amylase	None	400	IU/L	
CRP	None	300	mg/L	Excluding hospital patients
Bile acids	None	30	μmol/L	Refer pregnant only next working day
TSH	None	50	mIU/L	* GP/OP Refer to DB/call in morning
Free T4	5.4	40	pmol/L	* GP/OP Refer to DB/call in morning <5.4 Alinity, <5.2 Architect
Free T3		20	pmol/L	* GP/OP Refer to DB/call in morning
Cortisol	100	None	nmol/L	* GP/OP Refer to DB/call in morning
30 min cortisol	250			* GP/OP Refer to DB/call in morning
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Urate		340		Pregnant women only; not renal
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Troponin		>15F, >34M	ng/L	GP only
AKI		2,3 1		Call only if K >6.0mmol/L, *For GP call AKI1 next am

# Action Limits OOH

Is it Normal or Expected?

Analytical  
Considerations



**RESULT**

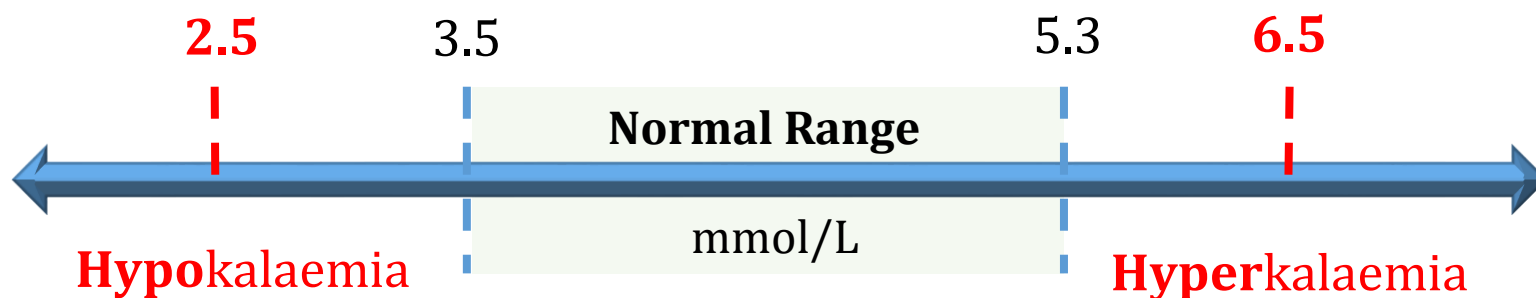


Clinical  
Context

Does it fit  
clinically?

Significant difference to  
previous result?

# POTASSIUM



**Lab calls out  $K^+ \leq 2.5$**   
 $K^+ < 2.0$  in known renal patients

**Lab calls out  $K \geq 6.5$**   
*Not haemolysed*

# PseudoHyperkalaemia

- **Delayed Separation**

- **Haemolysis**

Release of  $K^+$  from erythrocytes  $\longrightarrow$  falsely elevated results

- **EDTA Contamination**

e.g. blood is poured from one tube to another  
Usually accompanied by a markedly low  $Ca^{2+}$

- **↓ Temperature**

e.g. cold storage of whole blood samples

- **↑ Platelets & ↑ WBC (thrombocythaemia & leukocytosis)**

RA, Haem malignancies e.g. CLL

Measurement on a Lithium Heparin sample

- **Familial Pseudohyperkalaemia (rare)**



# HYPERKalaemia

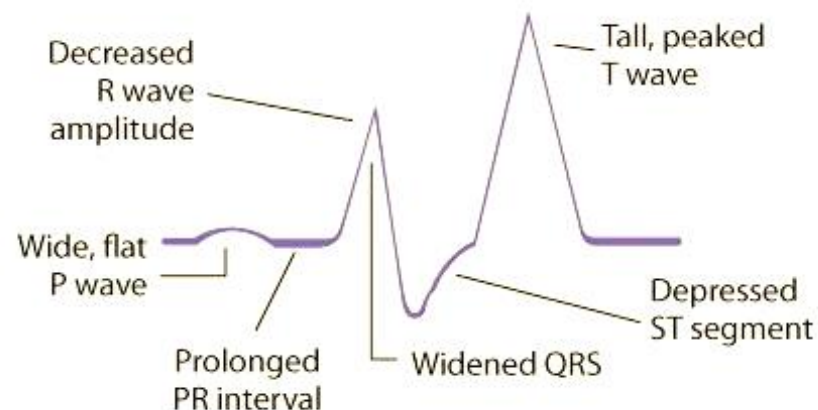
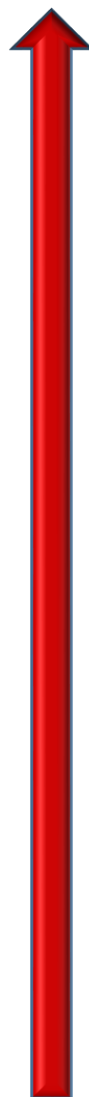
- **ECG CHANGES**

- **SYMPTOMS**

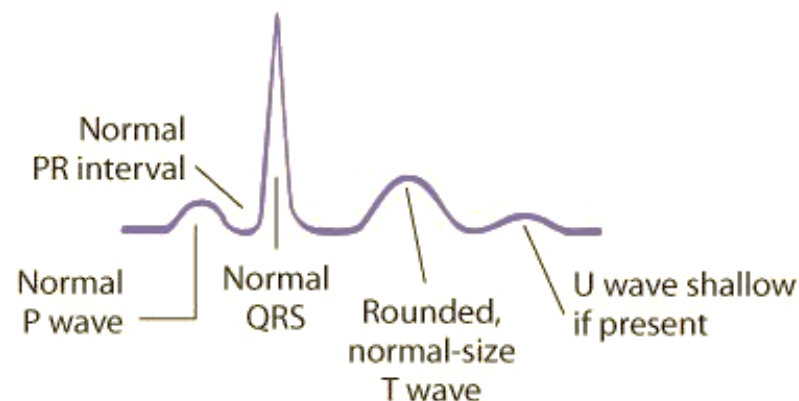
Palpitations  
Paraesthesia  
Muscle weakness  
Arrhythmias

- **CLINICAL CONTEXT**

Previous results  
Acute illness (AKI, DKA)  
Renal Disease (CKD)  
Obstructive Uropathy  
Medication (K<sup>+</sup> sparing diuretics, ACEi, ARBs, NSAIDs)  
Cellular Lysis (Tumour, Rhabdo)  
Renal Tubular Acidosis type 4



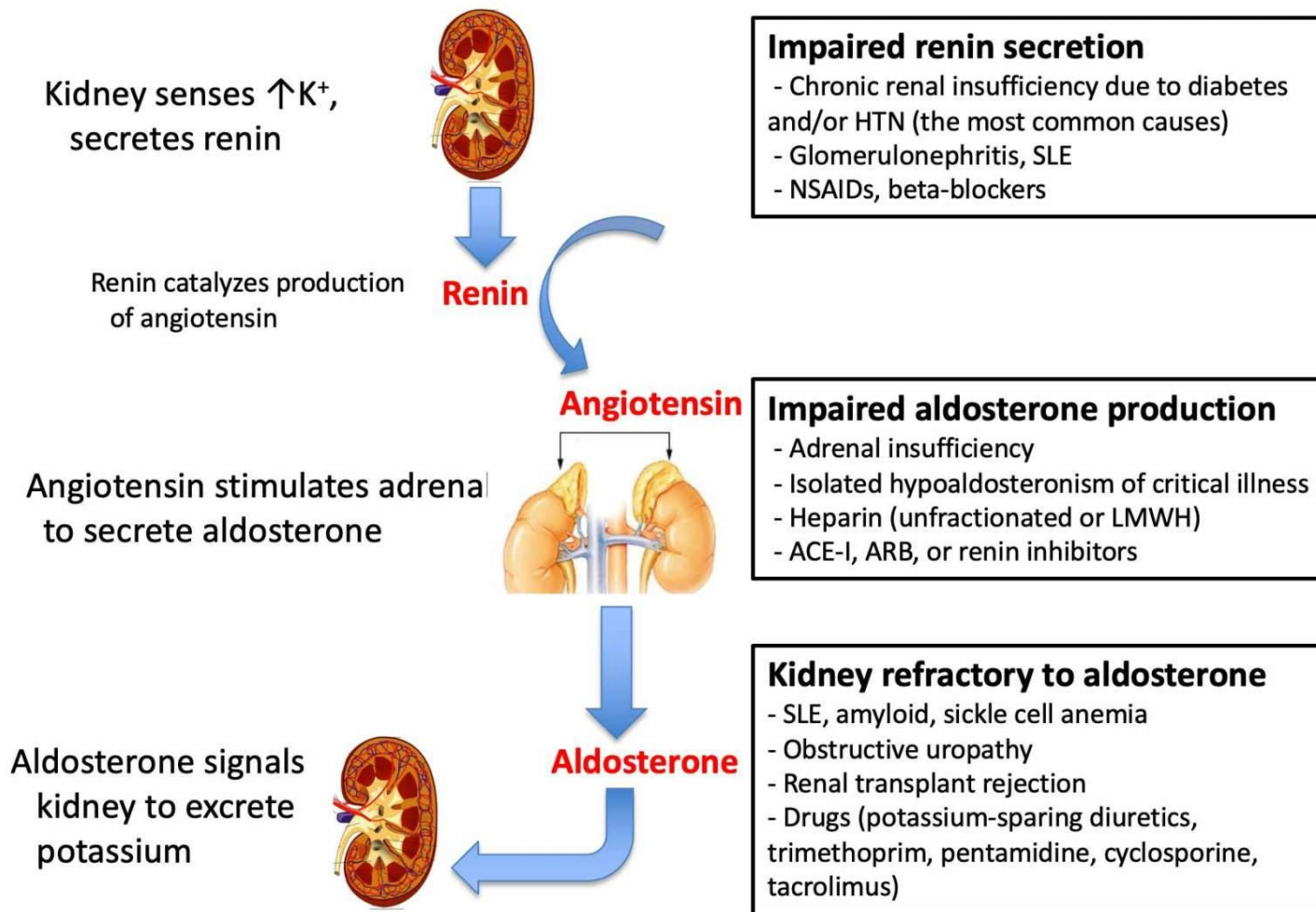
**K<sup>+</sup>**





# HYPERKalaemia

## Hyperkalemia due to dysfunction of the Renin-Angiotensin-Aldosterone system



# HYPOKalaemia

- **ECG CHANGES**

Potentialiation of digoxin toxicity

- **SYMPTOMS**

**Cardiac:** Palpitations, arrhythmias

**Neuromuscular:** Muscle weakness, cramps, hypotonia, paralytic ileus & constipation

- **CLINICAL CONTEXT**

Dialysis

Medication (diuretics)

GI disturbance (D&V)

Nutritional status, dietary K<sup>+</sup> intake

Cushingoid features

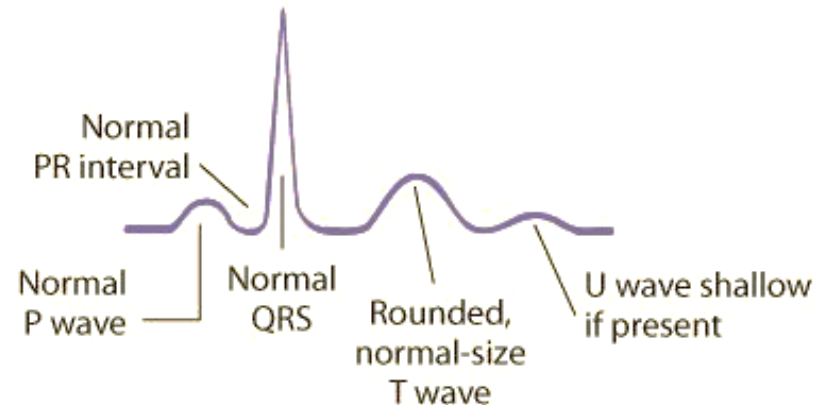
Hypertensive

- **OTHER Ix**

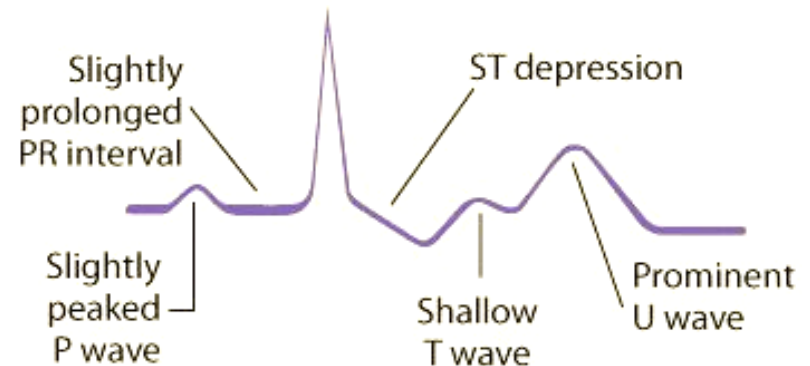
Magnesium Level

Renin Aldosterone studies

pH / HCO<sub>3</sub> – alkalosis



**K<sup>+</sup>**



# HYPOKalaemia Causes



## Decreased K<sup>+</sup> intake

- Oral, Parenteral



## Transcellular K<sup>+</sup> movement

- Metabolic Alkalosis
- Iatrogenic – Insulin,  $\beta$ -adrenergic agonists, steroids
- Refeeding syndrome



## Increased K<sup>+</sup> excretion

### • Renal

Diuretics,

Diuretic phase of ARF, Dialysis

1° & 2° Hyperaldosteronism (Conn's)

Cushing's Syndrome

Bartter's, Gitelman's & Liddle's Syndromes

Renal Tubular Acidosis Type 1 and 2

### • Extrarenal

Diarrhoea, vomiting, laxatives

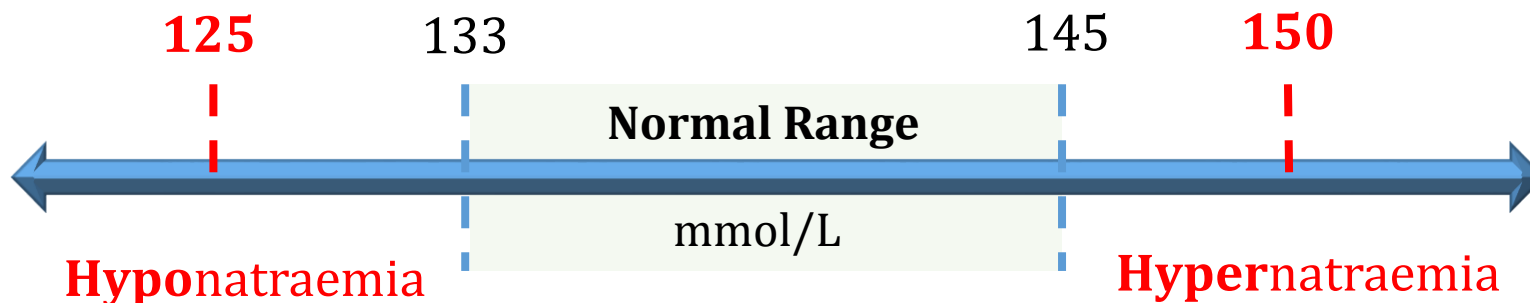
Pyloric Stenosis, Ileostomy

Excessive sweating

**Mineralocorticoid excess  
Endocrine causes**

**Gastrointestinal Loss**

# SODIUM



**Lab calls out**  
**Na  $\leq$  125 mmol/L**

**Lab calls out\***  
**Na  $\geq$  150 mmol/L**

Na  $\leq$  130 mmol/L in  $<16$ yo

**\*155  $\leq$  Na  $\leq$  125 mmol/L for inpatients**

# HYPONatraemia

- **SYMPTOMS**

Headache, confusion, weakness, convulsions, drowsiness – GCS level

- **CLINICAL CONTEXT**

Previous results - acute vs chronic

Rate of change - rapid vs stable

Volume status - hypovolaemic vs euvolaemic vs fluid overload

Organ Failure - renal (nephrotic syndrome), liver (cirrhosis), cardiac (CCF), adrenal

Medications (diuretics)

Addisonian features – BP, K<sup>+</sup> level

- **OTHER Ix**

Lipid profile

Albumin level


Glucose level

TFTs

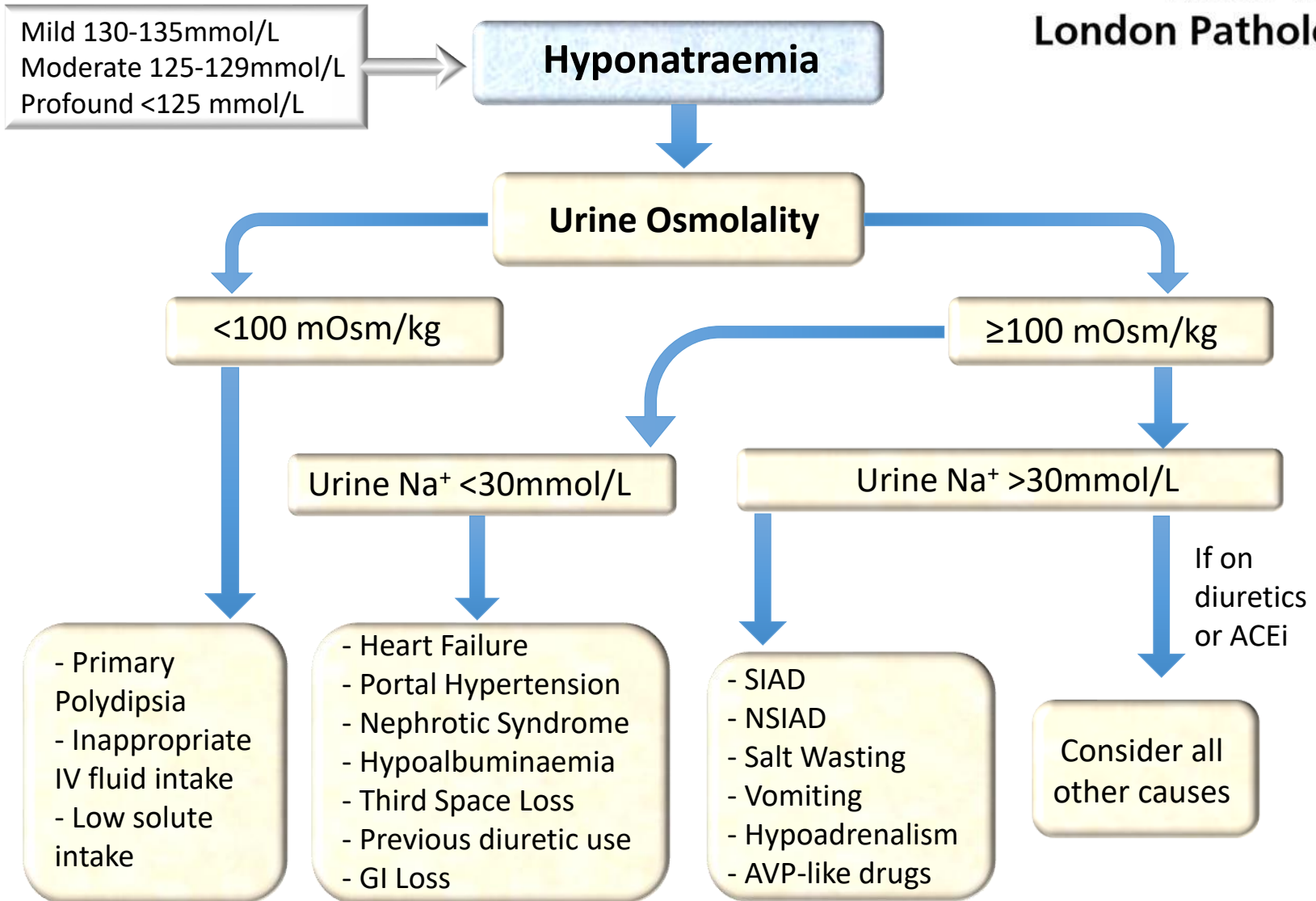
SST

Paired urine + serum osmolality & urinary Na level

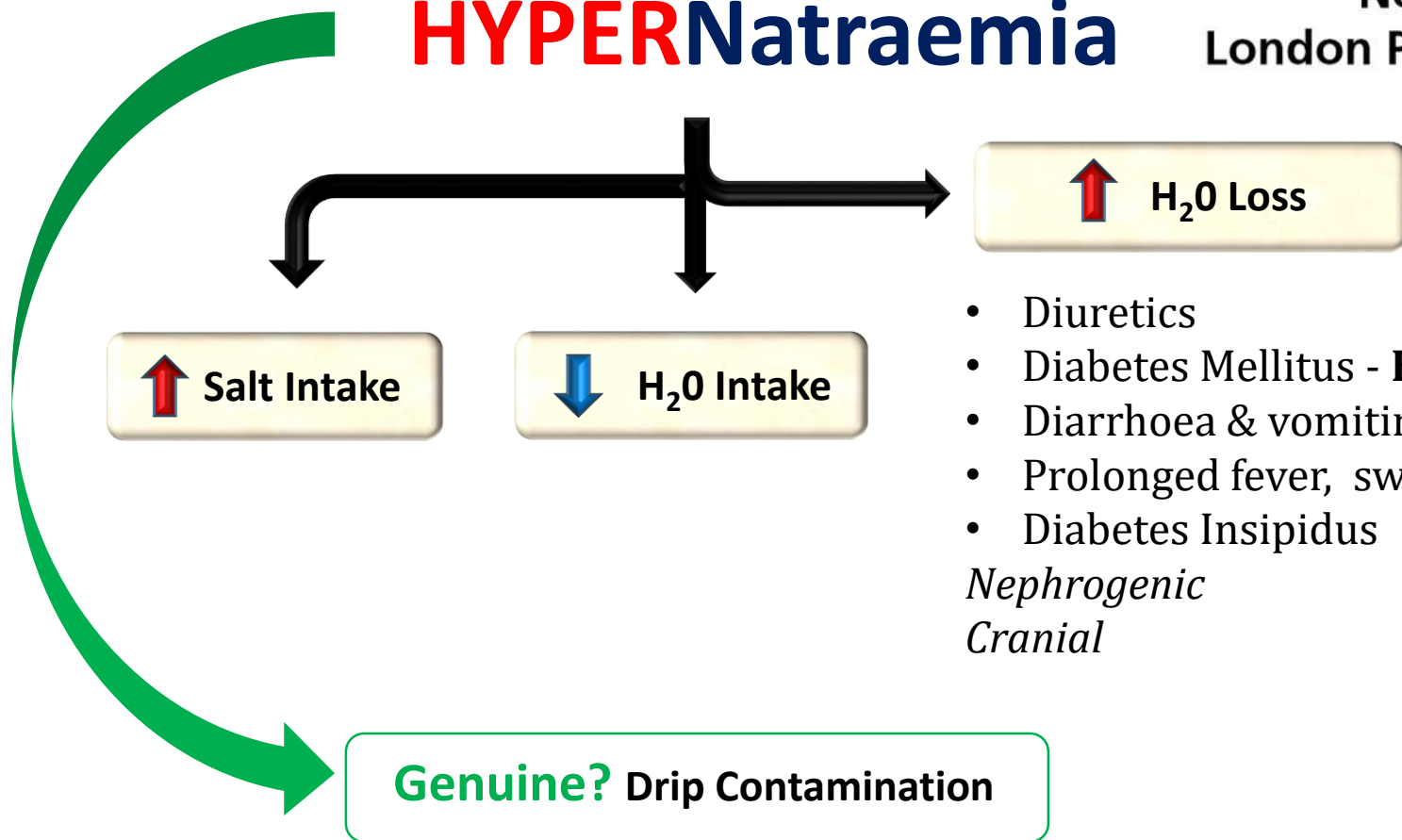
## Genuine?

- Hyperlipidaemia
  - Contamination
  - Hyperglycaemia (*causes redistribution*)
  - Increased proteins
- 

# Society of Endocrinology Clinical Guidelines



# HYPERNatraemia



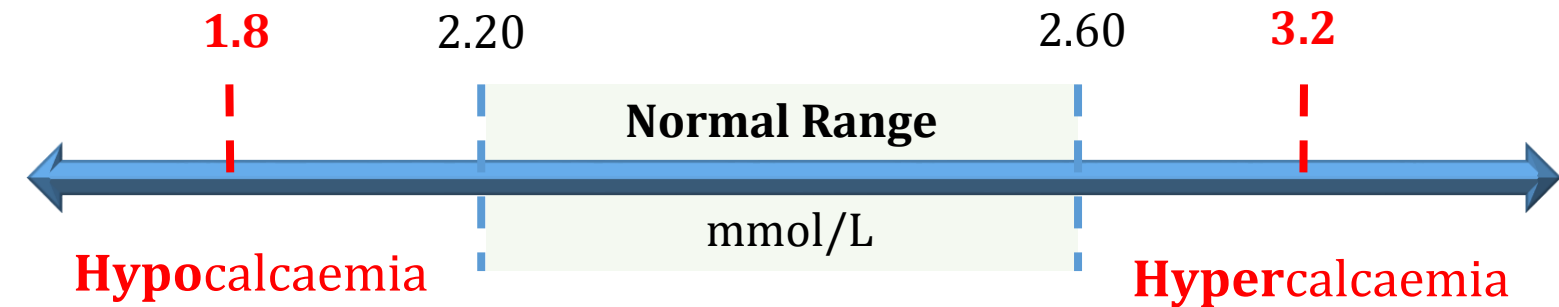
↑ H<sub>2</sub>O Loss

- Diuretics
- Diabetes Mellitus - HHS
- Diarrhoea & vomiting
- Prolonged fever, sweating
- Diabetes Insipidus

*Nephrogenic  
Cranial*

Genuine? Drip Contamination

# CALCIUM

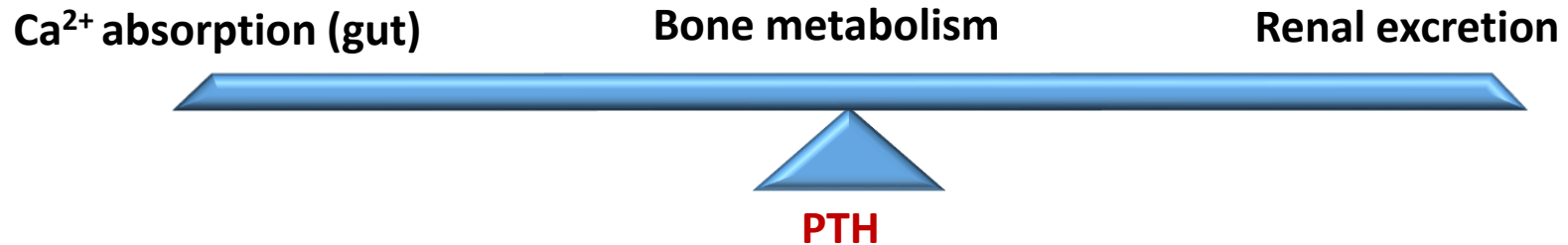


Lab calls out  
**Adj Ca<sup>2+</sup> ≤ 1.8 mmol/L**

Lab calls out  
**Adj Ca<sup>2+</sup> ≥ 3.2 mmol/L**



# Serum Calcium



## *adjusted* Calcium

- Serum Ca<sup>2+</sup> is expressed as adjusted values as it is ~ 50% bound to albumin

$$\text{Adjusted Ca}^{2+} = \text{measured [Ca}^{2+}] + \{40 - [\text{Alb}] \times 0.013\}$$

# HYPOCalcaemia

- **ECG CHANGES**

Prolonged QT interval, arrhythmia

- **SYMPTOMS & SIGNS**

Peri-oral and digital paraesthesiae

Muscle cramps, carpopedal spasm & tetany

Laryngospasm / stridor

Convulsions

**Chronic:** BG calcification, cataracts, papilloedema, abnormal dentition

- **CLINICAL CONTEXT**

Thyroid / Parathyroid surgery

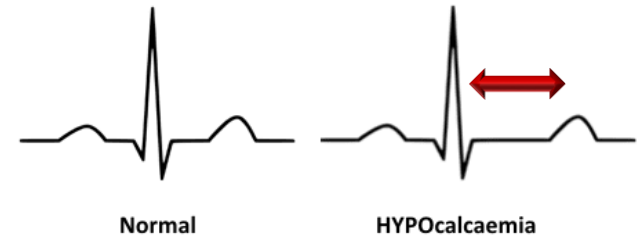
Malabsorption

Renal insufficiency

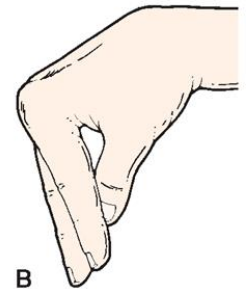
Medication (bisphosphonates, cytotoxics, furosemide, phenytoin)

**OTHER Ix**

Bone profile, PTH , Vitamin D, Mg level



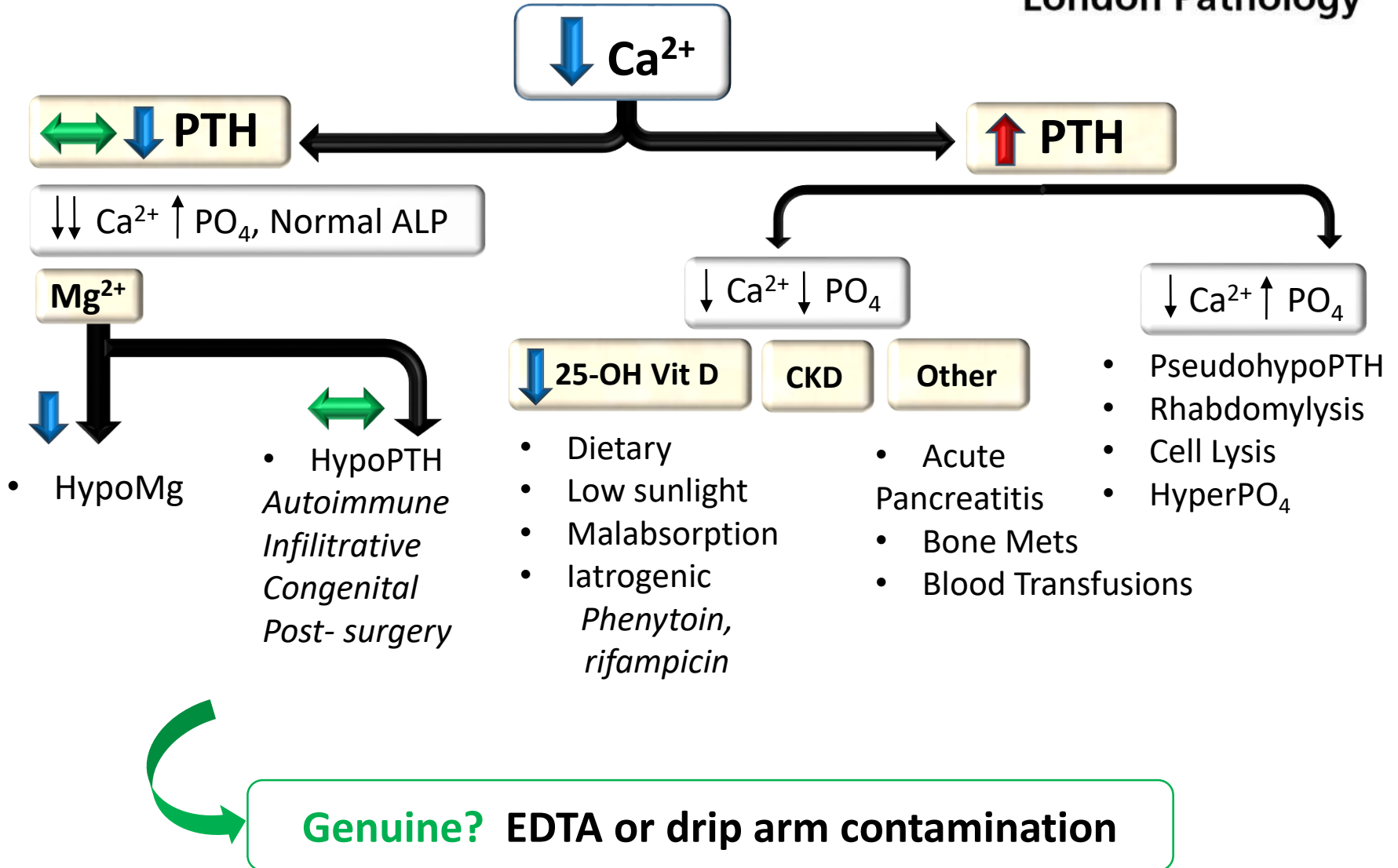
**Chovsteks's**



**Trousseau's**

**Ca<sup>2+</sup>**

# HYPOCalcaemia



# HYPERCalcaemia

- **ECG CHANGES**

Short QT interval, arrhythmia

- **SYMPTOMS & FEATURES**

Polydipsia & polyuria

Constipation

Abdominal pains, indigestion

Tiredness

Renal colic

- **CLINICAL CONTEXT**

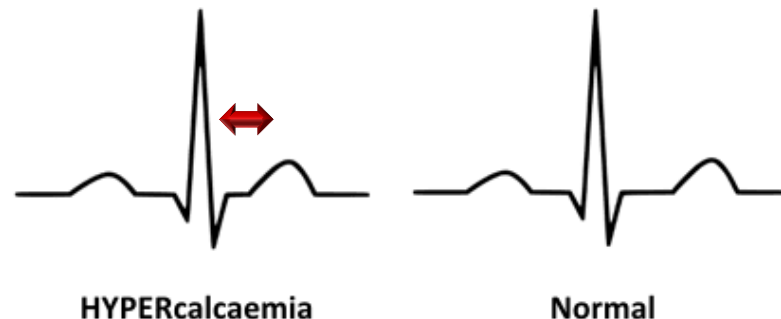
Symptomatic vs asymptomatic

Medication (thiazide diuretics)

Malignancy – known diagnosis vs red flag symptoms

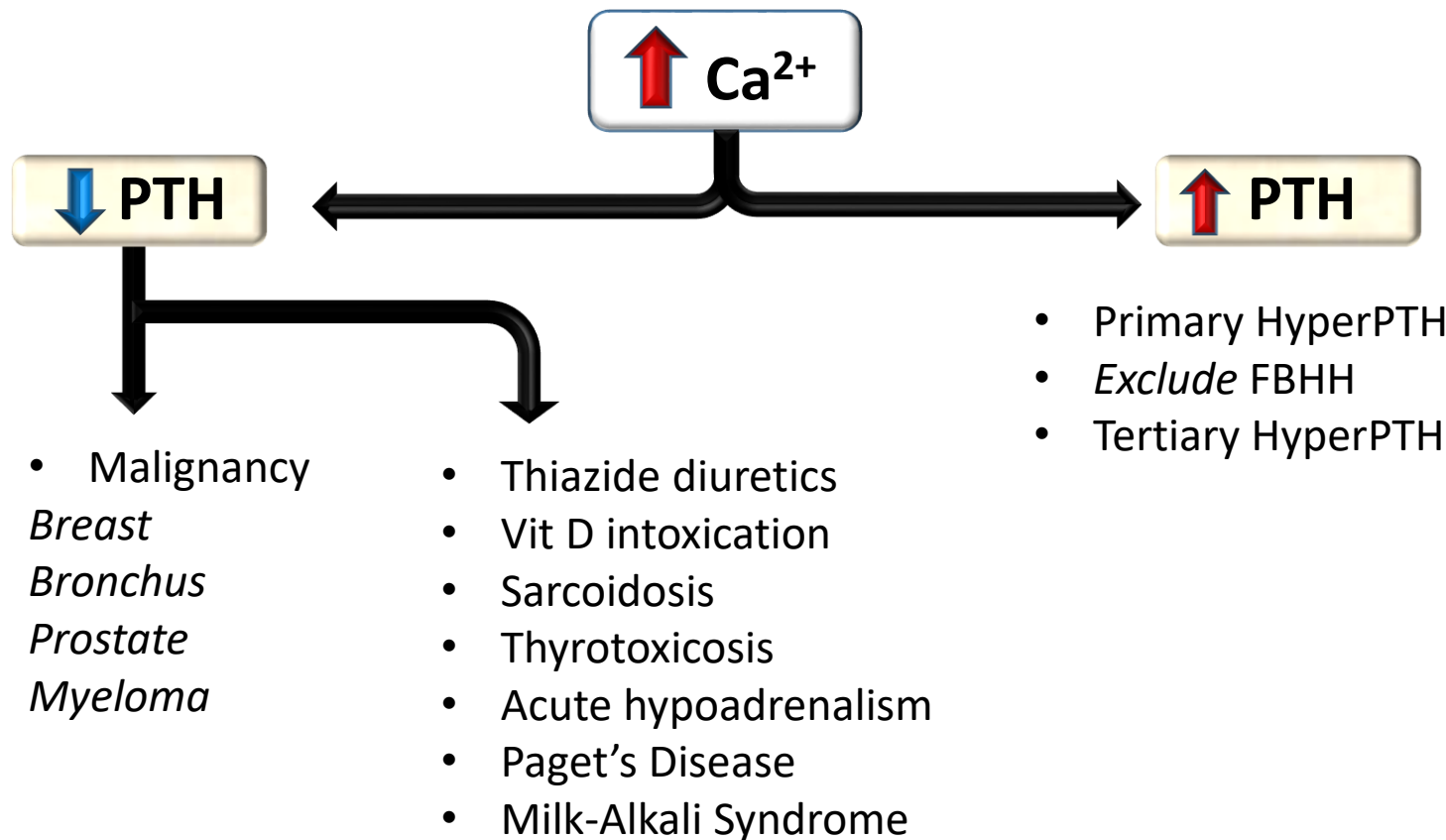
Renal failure

Known hyperPTH

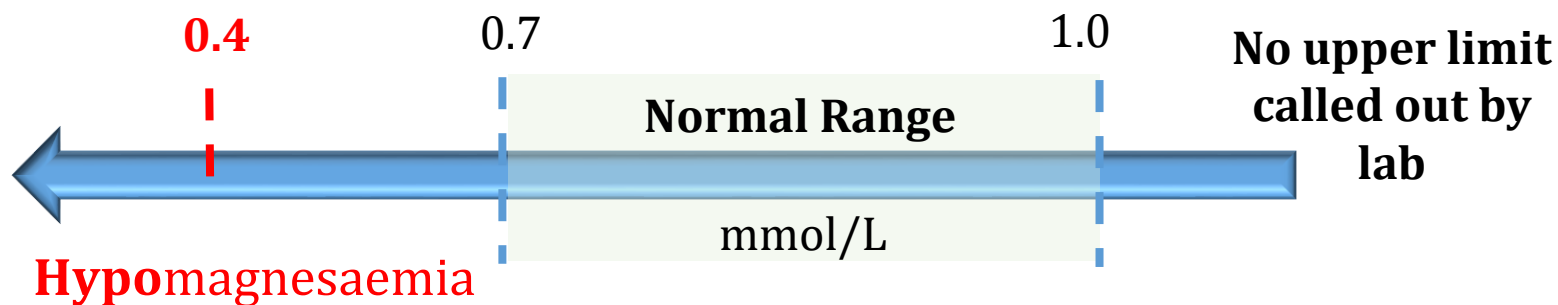


Ca<sup>2+</sup>

# HYPERCalcaemia



# MAGNESIUM



**Lab calls out:**

**Magnesium  $\leq 0.4$  mmol/L**

# HYPOMagnesaemia

- **ECG CHANGES**

Cardiac arrhythmias

- **SYMPTOMS & SIGNS**

Muscle weakness & tetany

Agitation, delirium

Ataxia, tremor, convulsions

- **CLINICAL CONTEXT**

Malabsorption

Chronic diarrhoea

Malnutrition

Refeeding syndrome

**ETOH excess** (withdrawal, chronic)

Cirrhosis

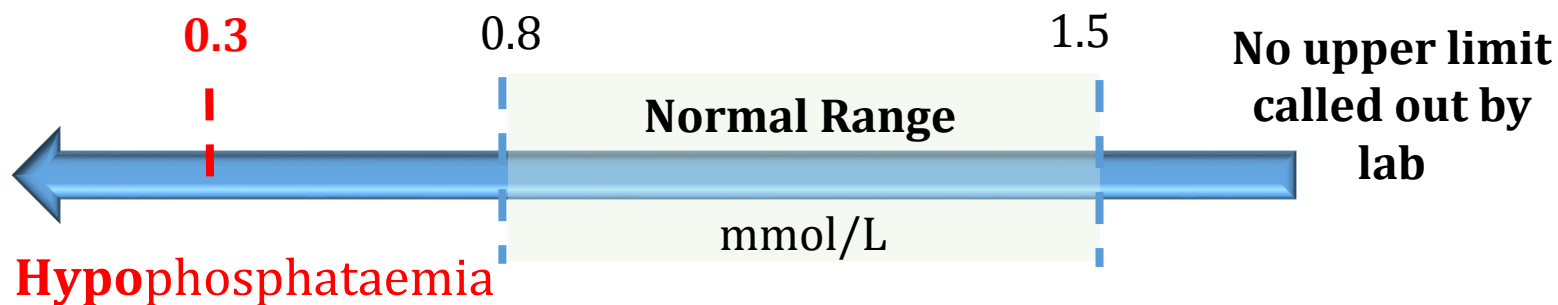
**Medication :**

PPIs, loop diuretics



**Mg<sup>2+</sup>**

# PHOSPHATE



Lab calls out:

**Phosphate  $\leq$  0.3 mmol/L**

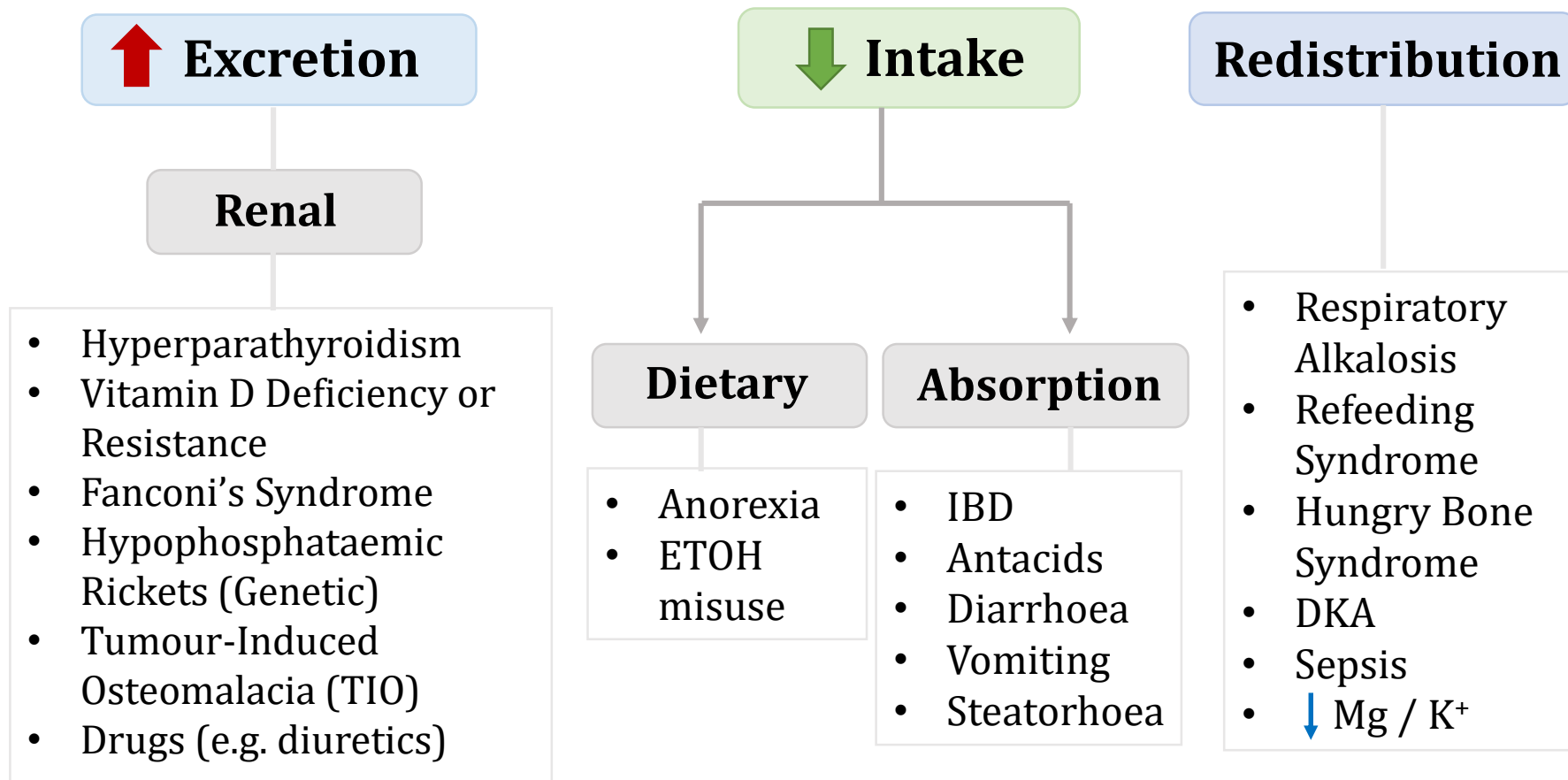


# HYPOPhosphataemia

## • SYMPTOMS & SIGNS

**Acute:** muscle weakness & dysfunction, rhabdomyolysis

**Chronic:** rickets & osteomalacia



# HYPERPhosphataemia

- **Renal Insufficiency - CKD**
- Tumour Lysis Syndrome
- Hypoparathyroidism
- Pseudohypoparathyroidism
- Acromegaly
- Excessive intake
- Vitamin D intoxication



**Genuine?** Delayed separation or haemolysis of blood sample

# KIDNEY INJURY

**BASELINE**



**AKI**

No lower limit  
called out by  
lab

Lab calls out:

<b>UREA</b>	<b>≥ 30 (adult)</b>
	≥10 (<16yo)
<b>CREATININE</b>	<b>≥ 354 (adult)</b>
	≥ 200 (<16yo)

<b>AKI 2,3</b>	
<b>AKI 1</b>	<b>Only if K &gt; 6.0</b>

# AKI Staging – CKS NICE [5]

	CREATININE	URINE OUTPUT
STAGE 1	<p><b>≥ 1.5x</b> from baseline within 7 days</p>	<p><b>6 hours</b> of oliguria (<b>&lt;0.5mL/kg/hr</b>)</p>
STAGE 2	<p><b>≥ 2x</b> from baseline within 7 days</p>	<p><b>&gt;12 hours</b> of oliguria (<b>&lt;0.5mL/kg/hr</b>)</p>
STAGE 3	<p><b>≥ 3x</b> from baseline <i>Or</i> Rise to <b>≥354 μmol/L</b> with <b>≥50%</b> rise within 7 days</p>	<p><b>Anuria</b> for 12 hours <i>Or</i> Urine Output <b>&lt;0.3mL/kg/hr</b> for 24 hours</p>

# AKI - things to consider

- **Baseline**

What is 'normal' for patient

Rate of change – gradual progression vs acute change

- **Result +/- Complications**

Acute on chronic renal failure

pH, potassium level

Uraemia – pericarditis, encephalopathy

Urine output & risk of obstruction

Volume status – symptoms of overload

Evidence of hypovolaemia or sepsis

- **Medication Review**

Newly prescribed drugs e.g NSAIDs, ACEi,

Ensuring renal doses e.g antibiotics

- **Establish Cause**

# AKI

## Pre-Renal

- Sepsis
- Dehydration
- Hypotension
- Haemorrhage
- Burns
- Low cardiac output

## Intrinsic

- Glomerulonephritis
- Systemic Disease
  - SLE
  - Vasculitis
- Nephrotoxins
  - NSAIDs
  - Aminoglycosides
  - Contrast
- Intrarenal obstruction (BJP)

## Post-Renal

- Kidney Stones
- Prostate enlargement
- Urinary tract malignancy
- Retroperitoneal fibrosis
- Neurogenic bladder

**S**epsis

**T**oxicity

**O**bstruction

**P**arenchymal

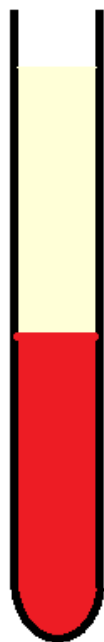
# Plasma Creatinine Conc<sup>n</sup>

= reliable & simple biochemical test of glomerular function.

Levels can fluctuate, independently of renal function, due to changes in muscle mass:

- Increased by: ingestion of meat, strenuous exercise, muscle bulk and with re-feeding.
- Decreased by: starvation & in wasting diseases, immediately post-surgery, treatment with corticosteroids.
- Plasma creatinine conc<sup>n</sup> decreases during pregnancy but rises again post-delivery.
- Interference with lab measurements (e.g. ketones, bilirubin).

# Interference



Normal



Hemolysed



Lipemic

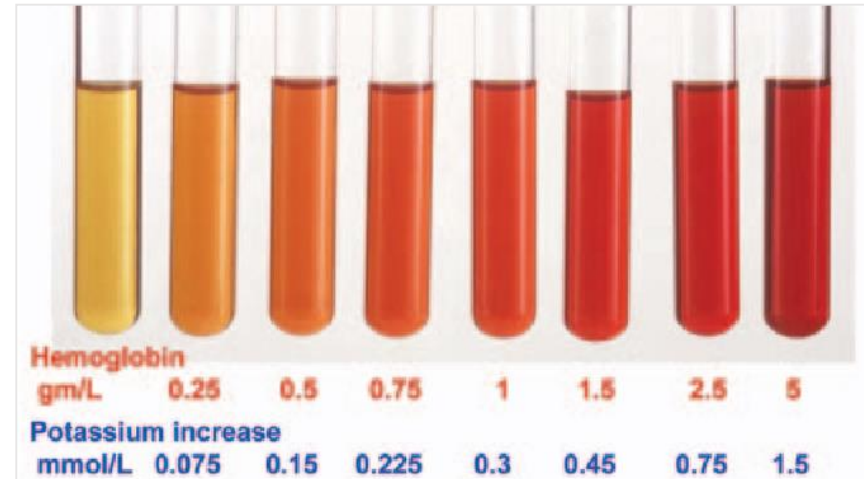


Icteric



# 1. Haemolysis

- Can be *in vivo* or *in vitro* (incorrect sample processing)
- Lab can identify haemolysed samples
- Source of error in chemical analysis



# 2. Icterus

- Elevated levels of bilirubin can also cause endogenous interference

# 3. Lipaemia

= turbidity in samples which is visible to the naked eye

- Most common cause is increased triglycerides
- Increased concentration of lipids in diabetes, pancreatitis, ETOH

# Sources of Error

## 1. Pre-analytic

- Sample collection – haemolysed, clotted, insufficient
- Incorrect tube type
- Incorrect / missing patient identification
- WBIT
- Contamination (EDTA, IV drip)
- Delay
- Inappropriate transport or storage

## 2. Analytic

- Within the lab

## 3. Post-analytic

# Summary

**Interpret electrolyte disturbances with:**

- Clinical context
- +/- symptoms and ECG changes
- Severity of result
- Rate of change and baseline result

Be aware of the **artefactual & erroneous results** that can occur due to pre-analytic and analytical errors

**Action Limits** - critical results phoned out by the Lab within hours & OOH

A **Duty Biochemist** is always available for clinical advice, interpretation of results and further guidance.

# Biochemistry Advice 24/7

## Within hours (Monday to Friday 09.00 – 17.30):

- Duty Biochemist

Direct Line: 0203 313 0348

Email: [ICHC-tr.biochemistryadvice@nhs.net](mailto:ICHC-tr.biochemistryadvice@nhs.net)

## Out of Hours

- Oncall Metabolic Medicine Registrar and Consultant

Switchboard: 0203 311 1234



OOH Chemical Pathology (Duty Biochemist) via mobile.

Critical results as per the [Action Limits](#) are phoned out by the Lab

# References & Resources

- [1] Ball S., Barth J., Levy M., 2016. Society for Endocrinology endocrine emergency guidance: Emergency management of severe symptomatic hyponatraemia in adult patients. *Connect*, **5**: G4-G6.
- [2] Smellie, S., McNulty C., Galloway M., 2010. Primary Care and Laboratory Medicine. Frequently Asked Questions. *ACB Venture Publications*.
- [3] McGhee M., 2008. A Guide to Laboratory Investigations, 6<sup>th</sup> Edition. *Radcliffe Publishing*.
- [4] Marshall W. J., Lapsley M., Day A., 2017. Clinical Chemistry, 8<sup>th</sup> Edition. *Elsevier*
- [5] Acute Kidney Injury, clinical knowledge summary, NICE. Revised April 2018. <https://cks.nice.org.uk/acute-kidney-injury#!scenario>
- [6] Serum Indices: Reduction of clinical errors in laboratory medicine, 2007. Roche.