

GP BEST Event

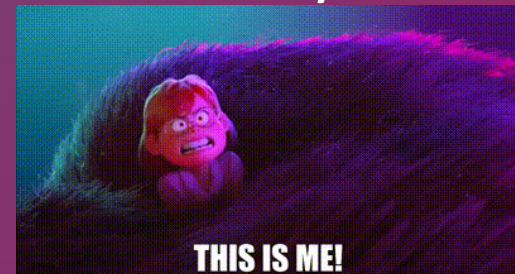
Wednesday 15th October 2025

BHF Priory Centre,
Barnsley

Dr Iain Woodrow
Consultant Clinical Scientist (Biochemistry)
South Yorkshire and Bassetlaw Pathology Network
Barnsley Hospital

A bit about me...

- Consultant Clinical Scientist (Biochemistry) based at Barnsley
- Trained in Salford, Liverpool, Wigan, worked for 12 years at Mid-Yorks
- Moved to Barnsley in August 2019 (just before the pandemic!)
- In April 2024, Barnsley Pathology subsumed by South Yorkshire and Bassetlaw Pathology Partnership (SYBP), part of STH
- Though Pathology is part of SYBP, we still serve the local community of Barnsley (primary and secondary care)



SYB Pathology Partnership

- South Yorkshire and Bassetlaw Covers S Yorks ICS:
 - 170 GP practices
 - 5 acute trusts
 - 9 hospitals (7 acute)
 - 3698 beds
 - 4 local authorities
 - 3 community/mental health trusts
 - 1 ambulance trust
 - Over 6000 VCSE organisations



SYB Areas

Barnsley: 329.2 km²
Pop 245,199
Known for: Michael Parkinson

Doncaster: 568 km²
Pop 310,000
Known for: Jeremy Clarkson

Sheffield: 367.9 km²
Pop 584,853
Known for : Michael Palin



Rotherham: 286.5 km²
Pop 264,671
Known for : Chuckle Brothers

Bassetlaw: 637.8 km²
Pop 116,839
Known for : Donald Pleasance

- Total area: 1005.7 km²
- Total Population: 1.522M

Disparate demographics across patch

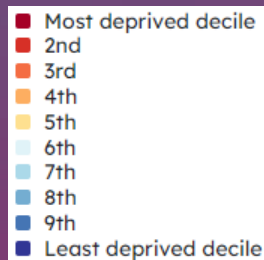
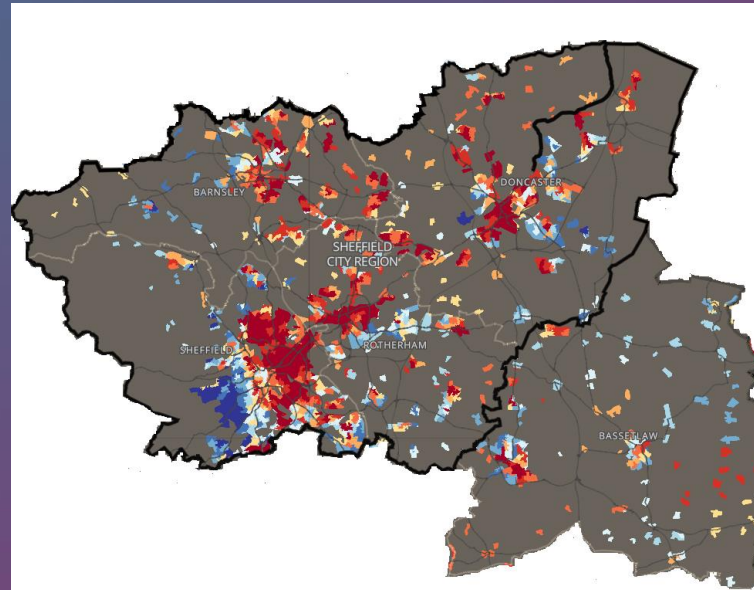
Comparing Barnsley and Sheffield

Team Parky v Team Palin

Barnsley



- Former mining town
- Not ethnically diverse (95.5% white British)
- Small town
- Outlying semi-rural former pit villages
- One of highest rates of people on incapacity benefit
- Median age 42
- 40-44% people live with chronic disease



Sheffield

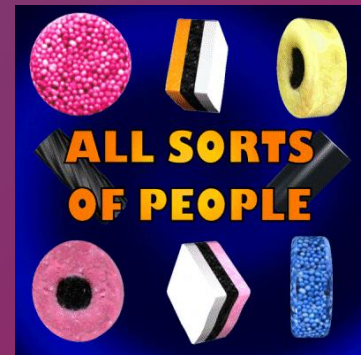


- Former industrial powerhouse (steel)
- Ethnically diverse (19% BAME)
- Big city
- High student population
- Inner city deprivation
- Suburban affluence
- Median age 37
- 19% have long-term health problem
- Destroyed in Threads



Problems with guidance across network

- Disparate populations
- Different priorities
- Also, individual labs across network use different equipment
- Results vary between manufacturer
- Though electrolytes *shouldn't* be radically different



SYBP Partnership

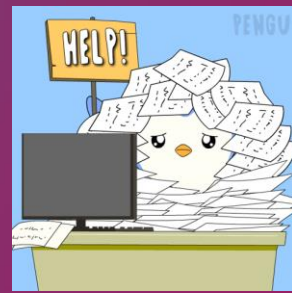
- Currently in process of procurement for new equipment
- Lots of work underway to implement new LIMS
- Very different regions of network
- Have different demographics and clinical priorities
- Providing a one size fits all Pathology service is difficult
- There remains the need to have standardised pathways/protocols

Guidance provided by STH

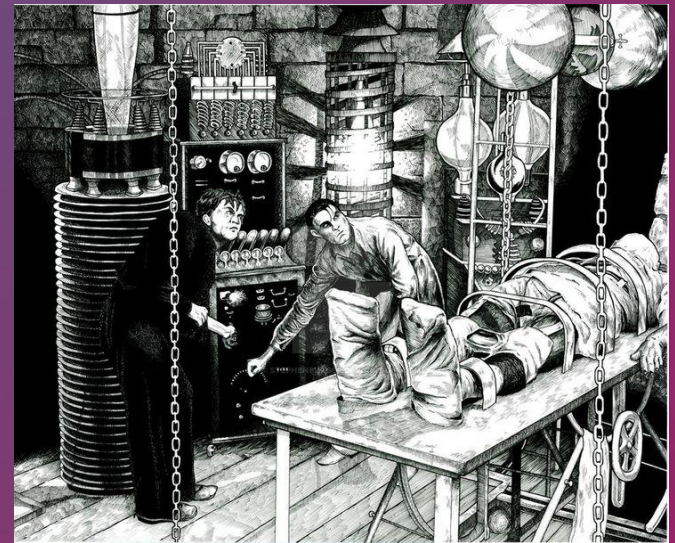
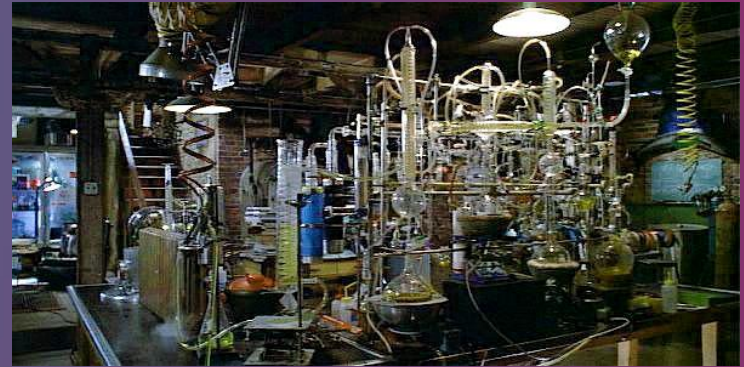
- Shared to BEST website (and equivalents in other areas)
- Based upon information, evidence and guidance from RCPATH, NICE, BMJ Best Practice etc
- Standardise practice across the ICS
- Documents for some of the most common abnormal lab findings
- Electrolytes, thyroids, LFTs
- Problem with ensuring documents are up to date and most current guidance is accessible

Documentation

- UKAS accreditation standards insist that all lab documentation maintained and reviewed
- A lot of resources go towards document control
- Task now bigger as it's across the Network
- Don't have standardised documents across Network yet
- Recently implemented a new document control package
- Cloud-based
- Accessible from anywhere via web (in theory!)



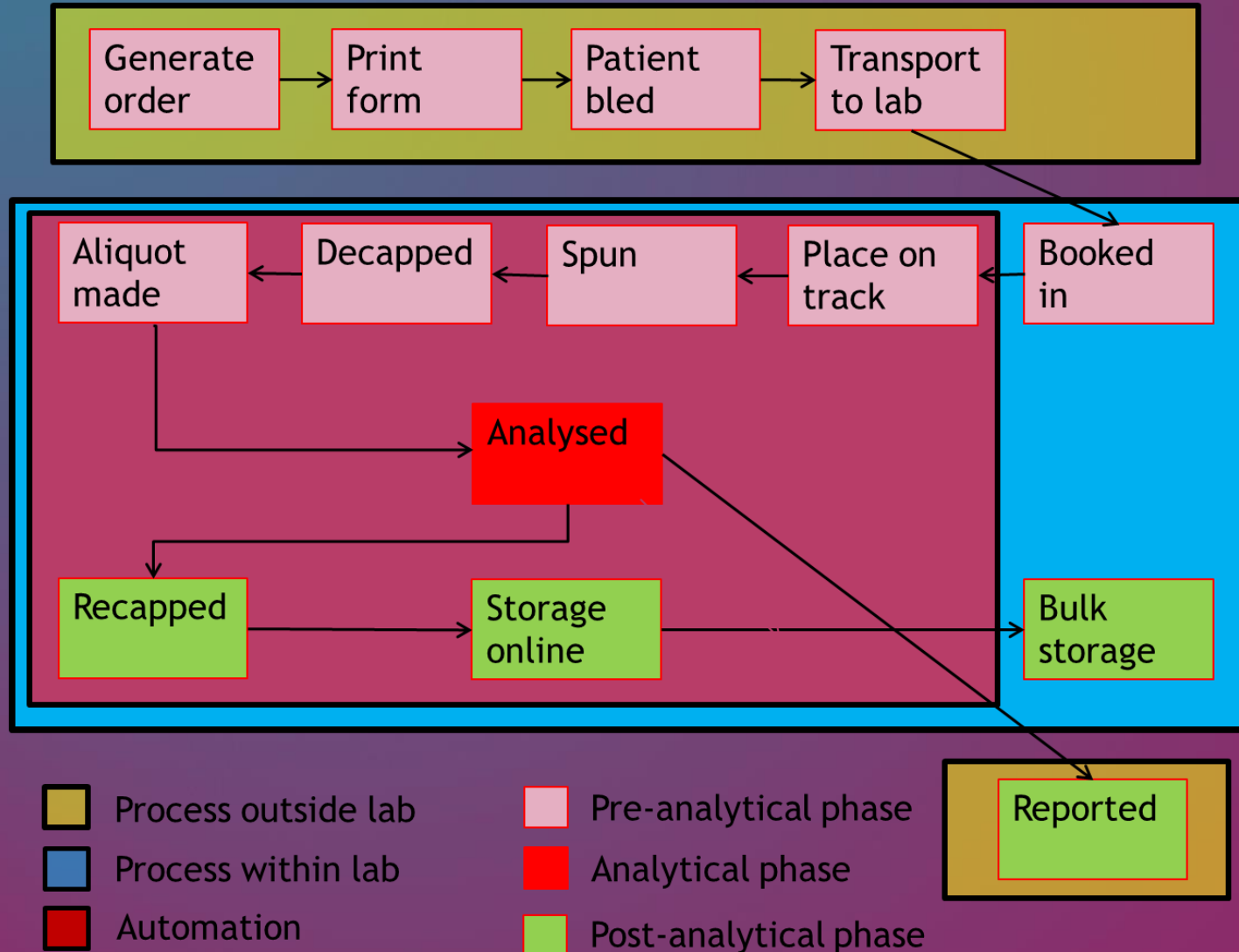
What does a lab look like?



What the lab actually looks like



Basic lab operation



Result authorisation

- Technical authorisation
 - Do the results make sense technically?
 - Quality control
 - instrument alerts
- Clinical authorisation
 - Do the results make sense clinically?
 - Check previous, clinical details
 - Add additional tests (if indicated)
 - Contact requesting clinician (if indicated)



Lab responsibility

- We can't look at every single blood test result
- Vast majority of results are normal and released
- Abnormal results flagged, reviewed (retrospectively, results generally released immediately)
- Mild abnormalities not commented upon
- Or else have automated comments appended
- Critically abnormal results are phoned to requestor (or surrogate eg I-Heart) directly from lab
- Some less abnormal results may be phoned later by clinical team

Lab critical phoning limits

- Informed by document from RCPATH
- See [The Communication of Critical and Unexpected Pathology Results 2017](#) (under review)
- Urgent results phoned from analyser immediately
- Refer to results that would usually require admission
- May depend on other factors too (eg renal patients)

Critical phoning limits in Biochemistry

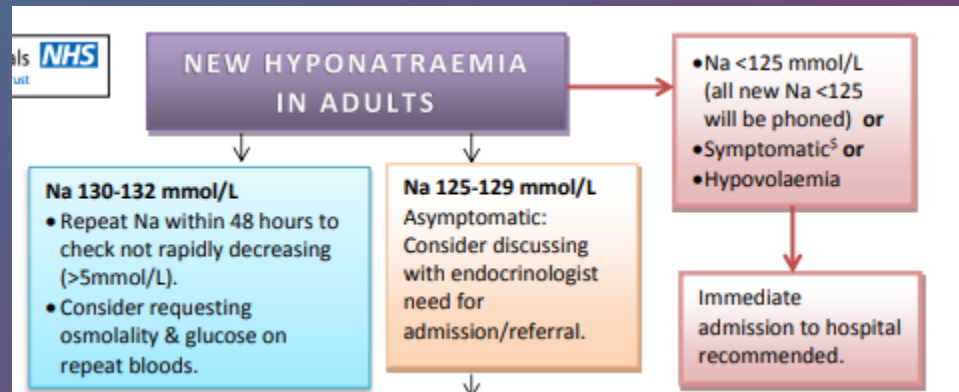
- Doesn't include eGFR, AKI, ammonia
- Results for more specialist tests may be phoned also (eg metabolics)
- Also delta checks
- May also vary according to local practice

Analyte (serum/plasma)	Units	Action Limits ^a		Communication Type ^b		Comments
		Lower	Upper	Primary Care	Secondary Care	
Na	mmol/L	120 (130 if < 16 yrs)	160	A	A	Note particular concern of risk of death in children with hyponatraemia.
K	mmol/L	2.5	6.5	A	A	Exclude haemolysis/old samples/EDTA contamination first. Agree, by local consensus, higher thresholds for phoning results in patients with known kidney disease including those on dialysis.
urea	mmol/L		30 (≥ 10 if < 16 yrs)	A	A	Agree, by local consensus, higher thresholds for phoning results in patients with known kidney disease including those on dialysis. Specific local cut points likely to be required for babies and neonates.
creat	umol/L		354 ^c (≥ 200 if < 16 yrs)	A	A	
glucose	mmol/L	2.5 ^d	25 (≥ 15 if < 16 yrs)	A	A	Exact cut points and response should be determined locally. ^d Glucose results < 2.5 mmol/L from primary care may be less crucial to phone immediately. For GPs and OPD, upper cut point of 30 mmol/L in known type 2 DM may be more appropriate.
Calcium (adj)	mmol/L	1.8	3.5	B ^e	A	^e Primary Care: If out of hours (OOHs) then communication next day to GP or GP OOHs service. Calcium levels ≥ 3.5 mmol/L may warrant more immediate communication with Primary Care as agreed by local consensus.
Mg	mmol/L	0.4		A	A	
PO4	mmol/L	0.3		B	A	
AST	U/L		15 x ULN	A	A	
ALT	U/L		15 x ULN	A	A	Agree specific cut points with key users locally (A&E, Liver Unit/Medical Admissions, GI Medicine).
Total CK	U/L		≥ 5000	A	A	
Amylase/Lipase	U/L		5 x ULN	A	A	
Digoxin	ug/L		2.5	B	A	Check timing >6hrs from last dose. More urgent if K+ < 3.0 mmol/L. Phone immediately to primary care if overdose suspected or K+ low.
Theophylline	mg/L		25	B	A	
Phenytoin	mg/L		25	B	A	
Lithium	mmol/L		1.5	B	A	
CRP	mg/L		300	A	-	
Troponin (I or T)			Local cut off for MI	A	-	Exact cut point should be discussed with local clinicians in cardiology and Primary Care.
AKI			AKI-3	A	A	All new occurrences
AKI			AKI-2	A	A	All new occurrences
AKI			AKI-1	B	A	Only if K > 6.0 mmol/L. Primary Care: If out of hours (OOHs) then communication next day to GP or GP OOHs service.
Ammonia	umol/L		100	-	A	
Bicarbonate	mmol/L	10		-	A	
Cortisol	nmol/L	50		B	A	Unless part of overnight dexamethasone suppression test
Cortisol (SST 30min)	nmol/L	250		B	A	As part of short synacthen test. Cut point used may need to be specific to assay being used.
Ethanol	mg/L		4000	-	A	or 400 mg/dL - consider much lower threshold in paediatrics.
Paracetamol	mg/L		f	A	A	f - All detectable levels - Agree specific thresholds locally with acute admissions/A&E - especially for paediatric samples.
Salicylate	mg/L		300	A	A	
Bilirubin (conj)			25	B	A	Neonates only
Urate	umol/L		340	B	A	Ante-natal indications only

Current Laboratory-Directed Guidance

- Various guidelines on STH Lab Med page
- [LabMed Homepage](#)
- Haematology and Biochemistry
- Source information
- *Should* be accessible via NHS device (though apparently isn't!)
- Run through some of the more salient information

Hyponatraemia



Hyperkalaemia

Author: Dr Hannah Delaney, Clinical Chemistry, NGH.
Version 4; Aug 2023
Sheffield Teaching Hospitals NHS Foundation Trust,
Laboratory Medicine Procedure LMGP004

HYPERKALAEMIA IN ADULTS (K > 5.3 mmol/L)

Patients with CKD, heart failure and/or diabetes, who are at risk of hyperkalaemia, should undergo regular monitoring at a frequency (2-4 times per year) dependent on level of renal function and degree of proteinuria.

MILD
5.4 – 5.9 mmol/L
Not usually a medical emergency.

MODERATE
6.0 – 6.4 mmol/L
Possible medical emergency.

SEVERE
≥ 6.5 mmol/L
Usually a medical emergency; admit
(unless spurious; see **BOX 1**)

BOX 1

Causes of spurious/artefactual:

1. Haemolysis. A comment will be added to the results if present.
2. Fist-clenching.
3. Contamination from FBC tube (K-EDTA).
4. Delayed arrival in lab (>6 hours).
5. Cold sample storage before arrival in lab e.g. put in fridge.
6. High platelet count.
7. High WCC.

If causes 1, 2, 3, 4 or 5 suspected, repeat, avoiding precipitating cause.
If causes 6 or 7 suspected, repeat using a green top sample tube sent straight to lab.

BOX 2

Some causes of true:

Drugs

- ACE inhibitors*
- Angiotensin receptor blockers (ARBs)*
- K-sparing diuretics/MRA (spironolactone, eplerenone, amiloride, triamterene)*
- Beta-blockers (non-selective)
- NSAIDs
- Trimethoprim
- Heparin
- Antifungals
- Ciclosporin
- Tacrolimus

**Serum K should be measured within 1-2 weeks of initiation or increase in dose of an ACEI, ARB, or MRA*

Renal Failure

- Chronic (usually stage 4 or 5)
- CKD and ingestion of foods high in potassium
- AKI
- Diabetic nephropathy (renal impairment may appear moderate)

Iatrogenic

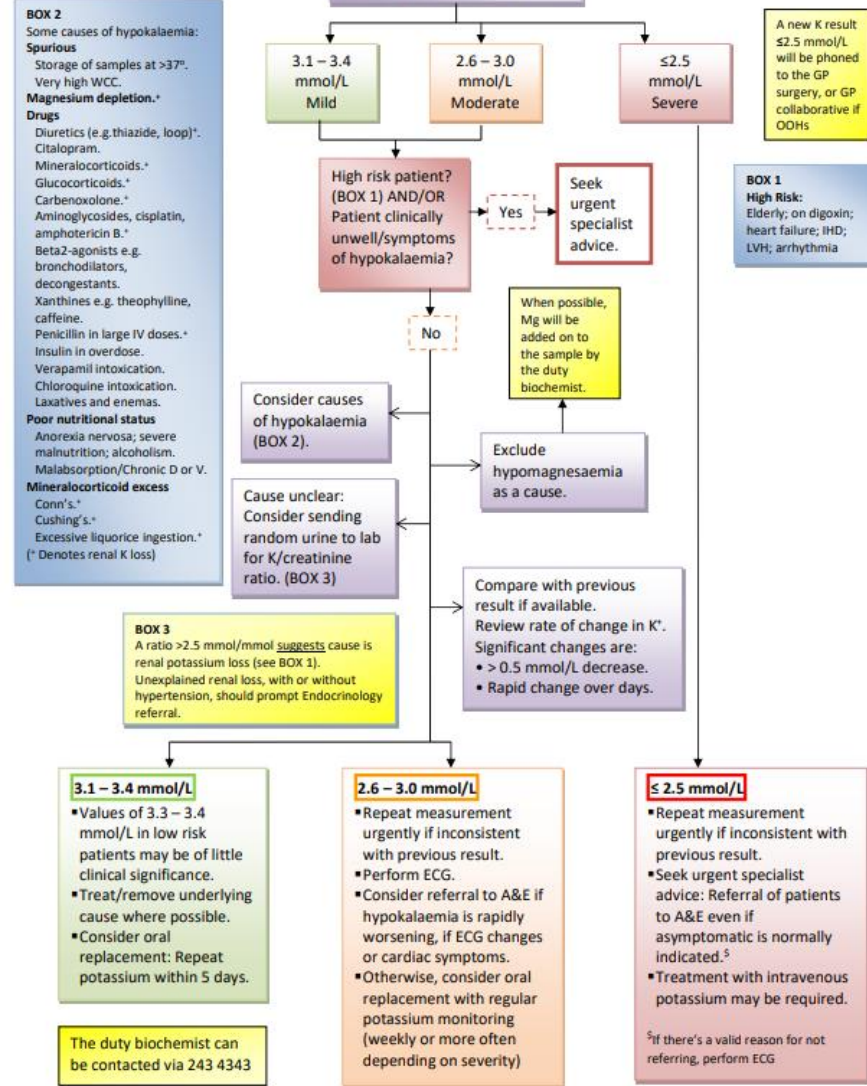
- K-supplements
- Salt substitutes e.g. LoSalt
- Herbal medicines

Metabolic Acidosis

Hypoadosteronism

Hypokalaemia

Author: Dr Hannah Delaney, Clinical Chemistry, NGH.
Version 3; Reviewed: 29/11/23 Next review by: 29/11/28
Sheffield Teaching Hospitals NHS Foundation Trust,
Laboratory Medicine Procedure LMGP0005



Hypercalcaemia

The duty biochemist can be contacted via switchboard (243 4343)

Author: Dr Hannah Delaney, Clinical Chemistry, NGH.
Version 5; 6/12/23
Review by; 6/12/28
Sheffield Teaching Hospitals NHS Foundation Trust,
Laboratory Medicine Procedure LMGRP003

BOX 1

Possible causes include:

1. PHPT: May have history of renal stones or #.
2. Malignancy: Bone mets (e.g. breast, prostate), lung ca and myeloma.
3. Drugs: See below
4. ESRF or transplant: Suggesting tertiary HPT.
5. Chronic immobilisation (particularly in Paget's).
6. Dehydration (may be a cause or a symptom).
7. Thyrotoxicosis (rare cause)
8. Addison's (v. rare cause)
9. Sarcoidosis or TB (both rare).

Drugs: These include

1. Calcium supplements.
2. Thiazide diuretics.
3. Vitamin D analogues e.g. alfacalcidol, calcitriol.
4. Vitamin A toxicity.
5. Lithium^A.
6. Calcium-containing antacids (or calcium supplements co-prescribed with antacids).
7. Vitamin D replacement with ergo- or cole- calciferol (rare cause)

Hypercalcaemia in adults Adj. Calcium >2.56 mmol/L

Mild
2.6 – 3.0 mmol/L

Moderate
3.01 – 3.4 mmol/L

Severe*
≥3.5 mmol/L
or severe
symptoms of
hypercalcaemia

*Results ≥3.5 mmol/L will be phoned to the GP surgery or collaborative

Send repeat at 1 week to confirm.
Send EDTA^S as well for PTH.
Look for possible cause (BOX 1).
Consider other test results (BOX 2).

Arrange
emergency
admission
to hospital

PTH high-normal or raised

PTH low or low-normal

BOX 2

1. Raised ALP
Can occur in bone mets, PHPT, Paget's.
2. Raised globulin
Can occur in myeloma*.
3. Raised ESR
May suggest myeloma*.
4. Anaemia
Chronic disease.
5. Low phosphate
May suggest PHPT.
6. TFT
Exclude thyrotoxicosis.

Causes include:

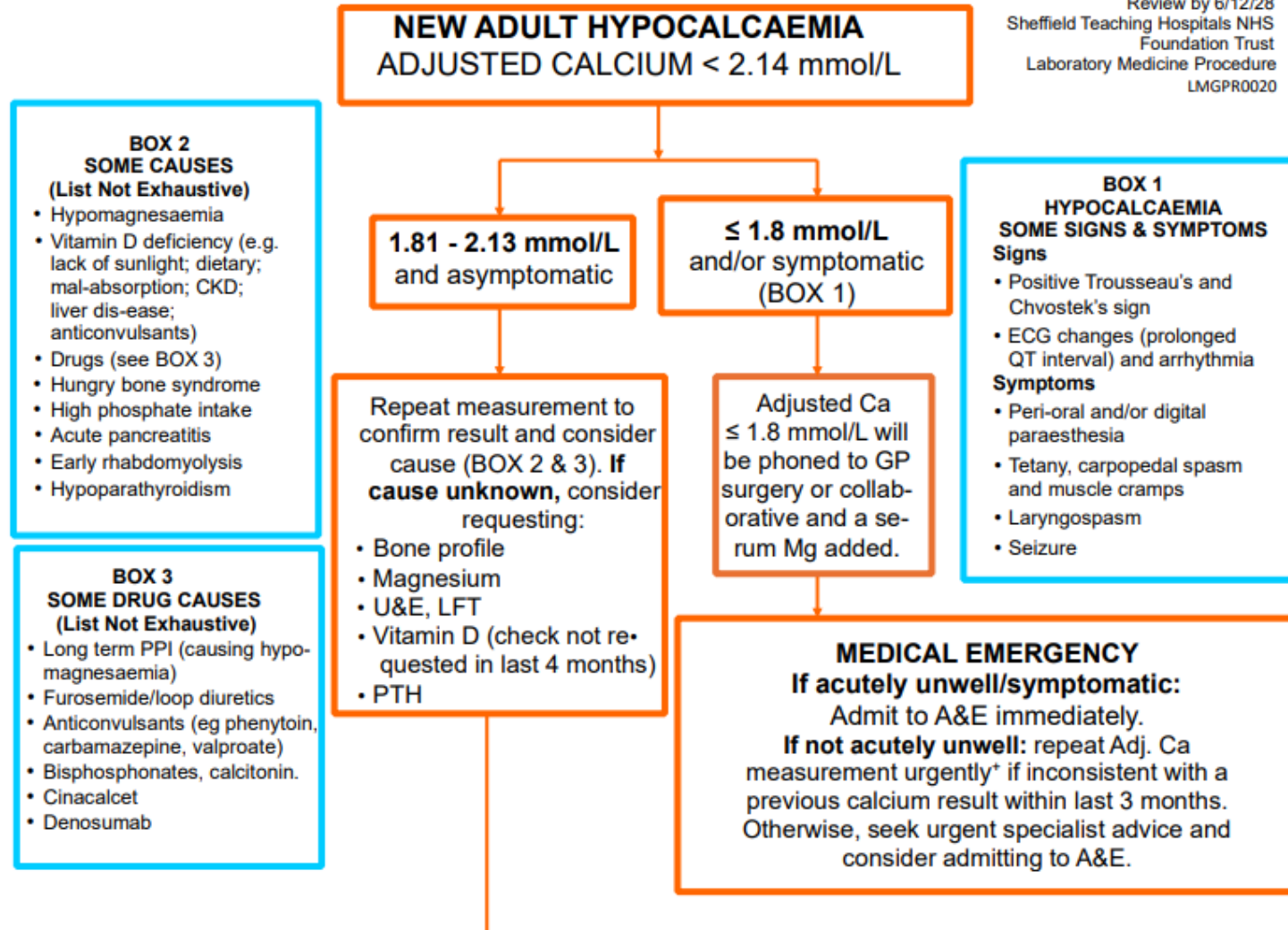
- Primary HPT
- Tertiary HPT
- Familial hypocalciuric hypercalcaemia (FHH)

Causes include:

- Malignancy
- Drugs (see BOX 1)
- Chronic immobilisation (+/- Paget's)
- Thyrotoxicosis, adrenal insufficiency
- Sarcoid or TB

Hypocalcaemia

Author: Dr Edmund Rab, Clinical Chemistry
 NGH Version 2, 6/12/23,
 Review by 6/12/28
 Sheffield Teaching Hospitals NHS
 Foundation Trust
 Laboratory Medicine Procedure
 LMGPR0020



Hypomagnesaemia

Sheffield Teaching Hospitals NHS
Foundation Trust, Laboratory
Medicine
Version 2
Author: Dr Hannah Delaney
Pharmacist review: Kirsty Dale,
Clinical Practice Pharmacist,
Medicines Management, Sheffield
CCG
Active Date: January 2018

HYPOMAGNEAEMIA (new diagnosis in adults) (Mg <0.7 mmol/L)

A new Mg result
<0.4mmol/L will be
phoned to the GP
surgery, or GP
collaborative if OOHs

0.40 – 0.69 mmol/L
Moderate

<0.40 mmol/L
Severe

BOX 1

Some causes of hypomagnesaemia:

Drugs: – see BOX 2

GI causes:

Malnutrition (esp alcoholics).
Malabsorption.
Diarrhoea – chronic.
Intestinal fistula.
Short bowel syndrome.

Renal loss:

Hypercalcaemia
Diabetes mellitus (poorly controlled).
Alcoholism.
Rare inherited tubular disorders e.g.
Gitelman or Bartter syndrome.
Tubular dysfunction (recovery from
acute tubular necrosis or
postobstructive diuresis).

Iatrogenic:

Long term Mg-free IV fluids or TPN.

Other:

Refeeding syndrome in chronic
malnutrition/anorexia.

Clinical reason for low magnesium? (see BOX 1&2)
Assess if patient clinically unwell/symptomatic (BOX 3)?

Cause unclear?

Consider sending
24hr urine in
acidified bottle to
lab for magnesium
– see BOX 4

Check renal function,
calcium and potassium
levels –

- Low Mg can cause
secondary hypocalcaemia
and hypokalaemia

BOX 3

Symptoms include:

- Weakness
- Apathy
- Tremor
- Paraesthesia
- Tetany
- Nystagmus

At <0.4:

- Seizures
- Drowsiness
- Confusion
- Coma

BOX 4

Urinary Mg loss of
>1.0mmol/24h in a
patient with normal
renal function
suggests renal
magnesium wasting

Take home message

- Most guidance is no different now to how it was years ago
- Any changes should be reflected in the available documents
- Documents should be in cycle of periodic review
- There may be technical variation from different equipment
- This should be standardised in short- to mid-term
- Guidance should be accessible on new document system
- Available on any browser

Any questions?