

Nausea and vomiting



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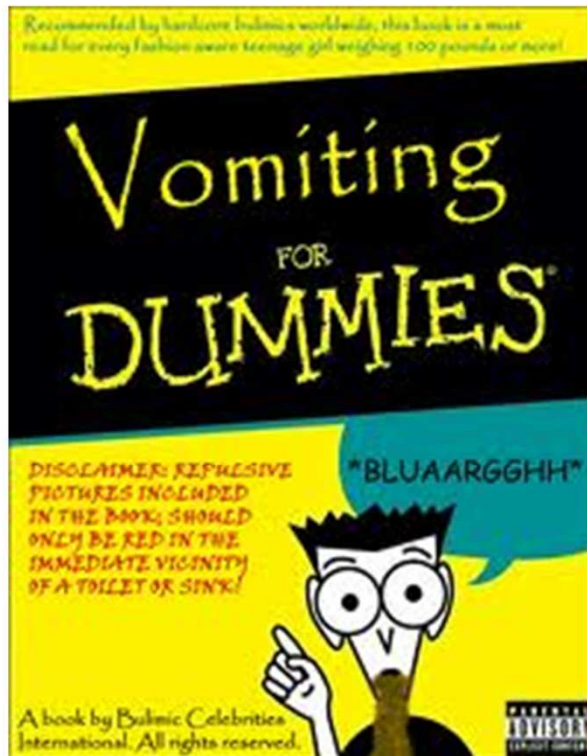


Prescribing information

- BNF
- Palliative Care Formulary
- The NHS Highland Formulary contains prescribing information and symptom control guidelines:

<http://www.nhshighland.scot.nhs.uk/Publications/Documents/Guidelines/formulary/highland%20formulary.pdf>.

“No treatment without diagnosis”



Treating symptoms depends on getting the right diagnosis

Nausea and vomiting are symptoms, not diagnoses

Getting the right diagnosis depends on
**HISTORY
EXAMINATION
INVESTIGATIONS**

Don't prescribe without diagnosing the cause of the problem!



Identifying the cause of sickness

Psychological

Intracranial

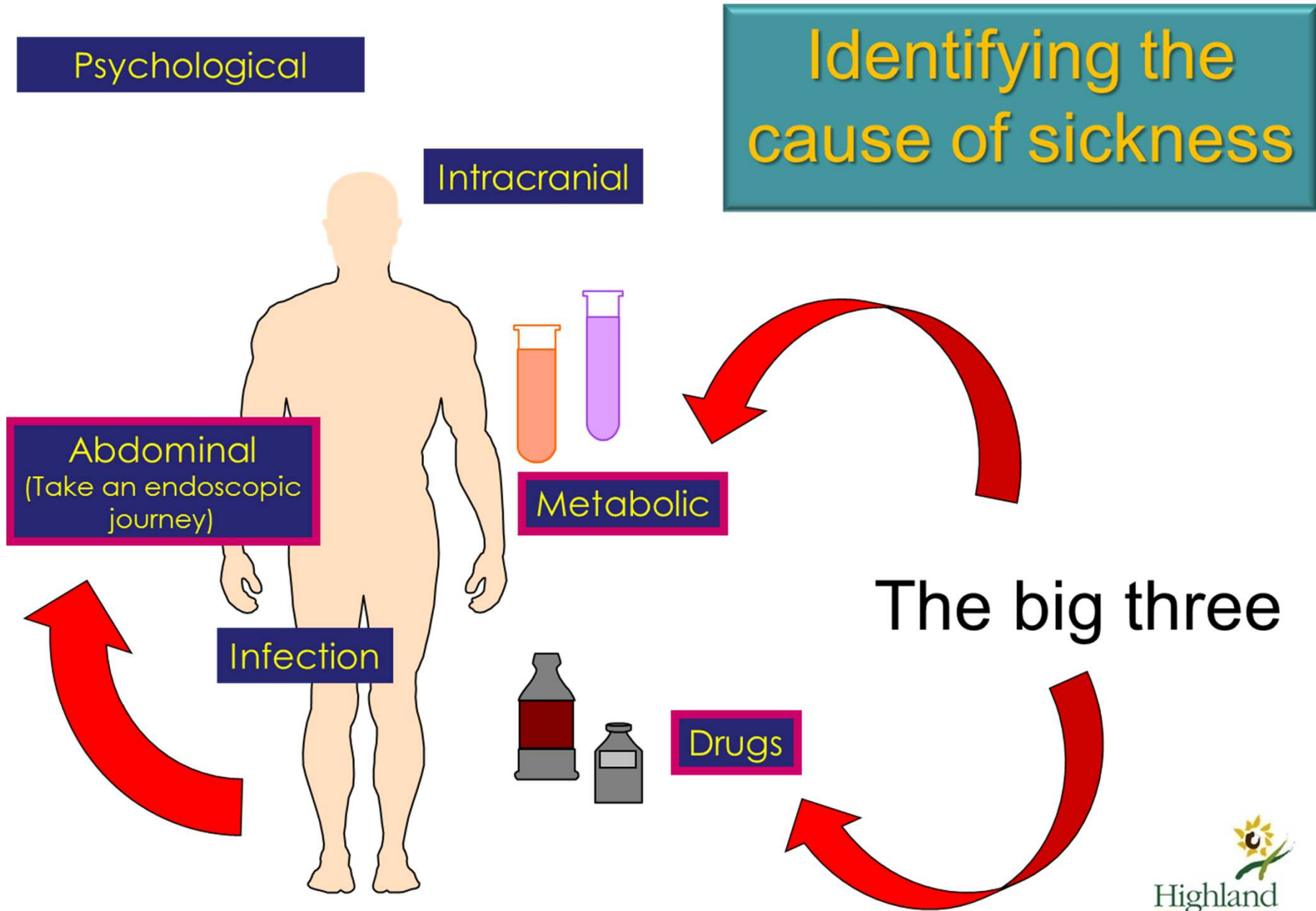
Abdominal
(Take an endoscopic journey)

Metabolic

Infection

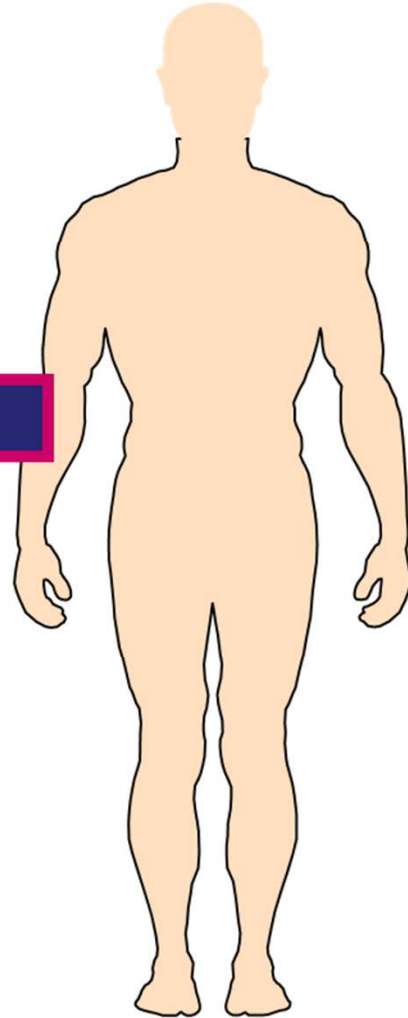
Drugs

The big three



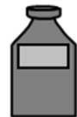
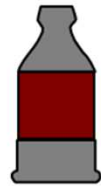
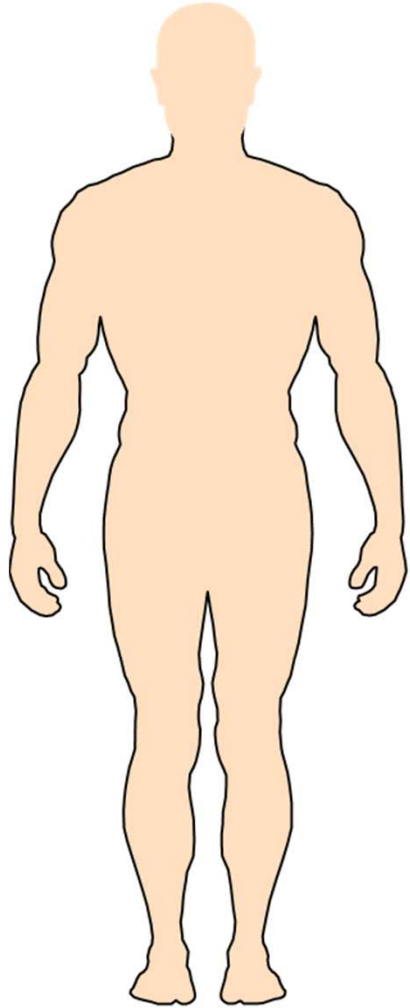
Identifying the cause of sickness

Abdominal



Take an imaginary endoscopic journey from top to bottom. Consider all structures en-route. e.g. oesophageal tumour, gastric outlet obstruction, small bowel mass, constipation, etc.

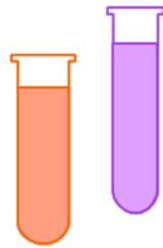
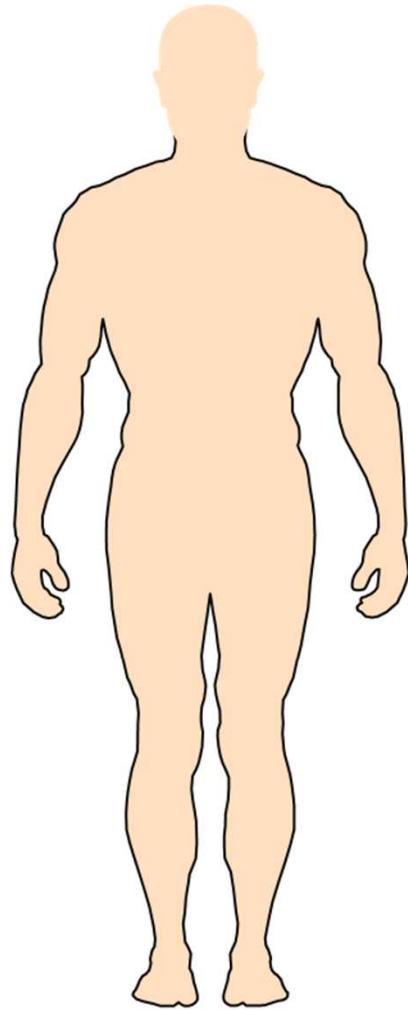
Identifying the cause of sickness



Drugs

Opioids
NSAIDs
Chemotherapy
Antibiotics
SSRIs
Iron
Digoxin

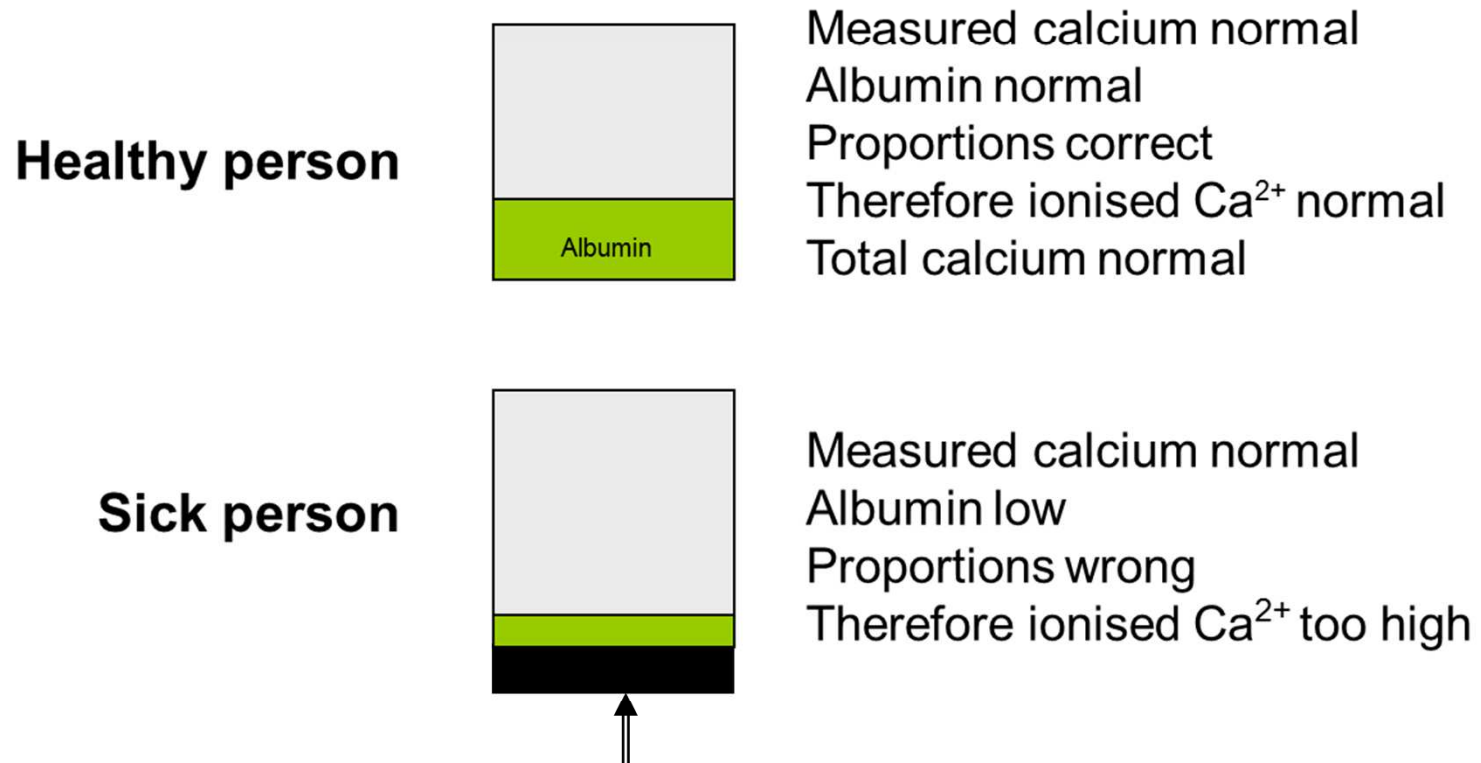
Identifying the cause of sickness



Metabolic

- Uraemia
- Hypercalcaemia
- Circulating immunological factors

Diagrammatic illustration of the reason for correcting calcium in the presence of low albumin.



The only way to 'see' the total calcium is by mathematically adding the missing albumin. Now we see that the total would be higher if the albumin was normal

Correcting plasma calcium

Example

A patient's calcium is reported as 2.6 mmol/l, and albumin as 16 g/l.

Normal albumin range in our lab is 36-52 g/l.

Correct against the lower limit of normal, i.e. 36 g/l

$36 - 16 = 20$ (i.e. the patient lacks 20g of albumin)

Each gram of albumin carries 0.02 mmol calcium.

So if the albumin was normal, there would be an additional ($20 \times 0.02 = 0.4$) mmol calcium

Therefore the corrected calcium is **$2.6 + 0.4 = 3.0$ mmol/l**

Principal mechanism of malignant hypercalcaemia, and its treatment



PTH

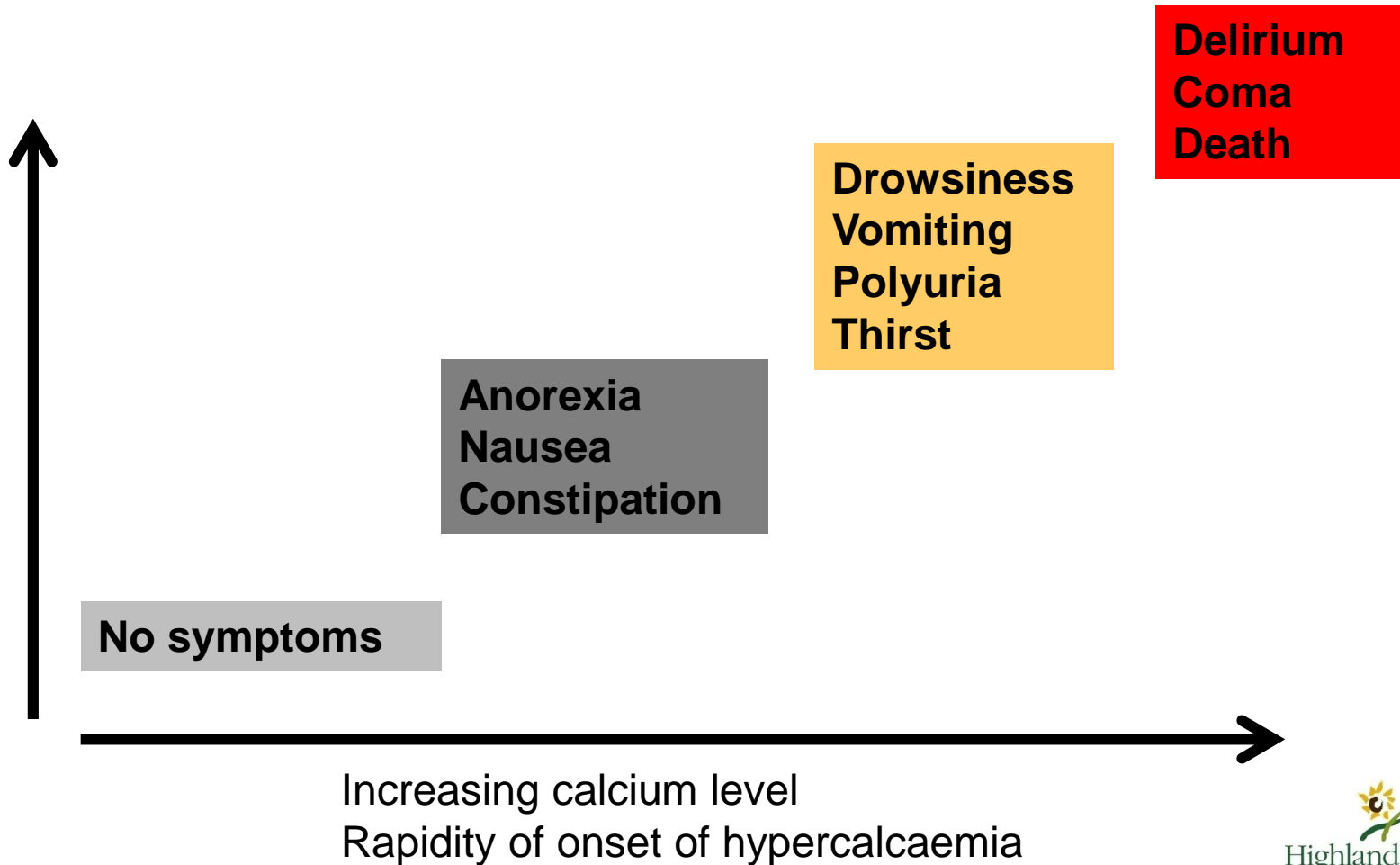
- Stimulates osteoclasts
- Inhibits osteoblasts
- Increases Ca^{2+} reabsorption
- Increases PO_4 excretion (Therefore low plasma PO_4)
- Increased free Ca^{2+} which can't bind to PO_4 to be deposited in bone

BISPHOSPHONATES

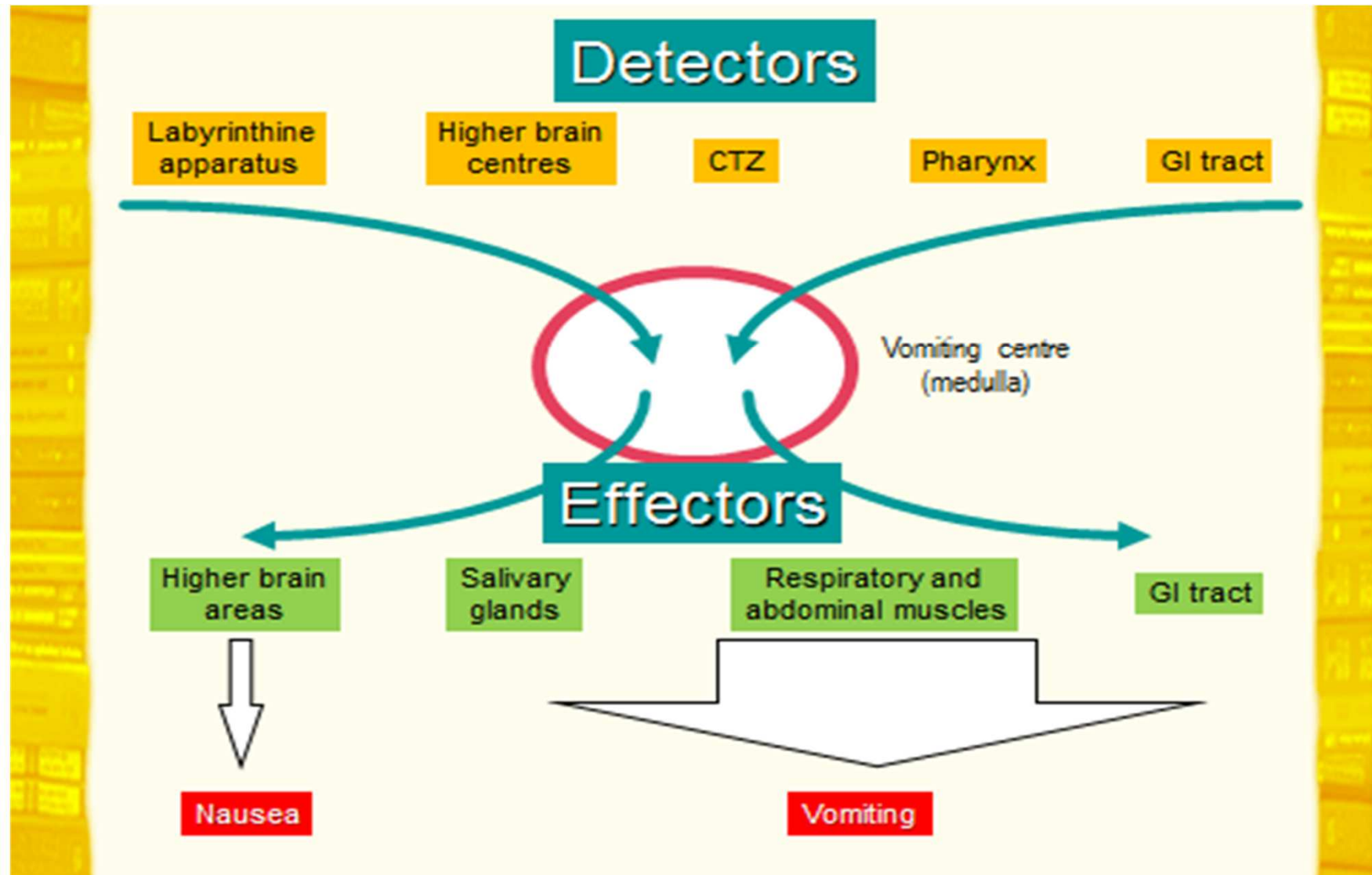
- Bind to calcium on surface of bone
- Bind to calcium ions in blood
- Inhibit osteoclast activity

Clinical features of hypercalcaemia are non-specific

Worsening
features

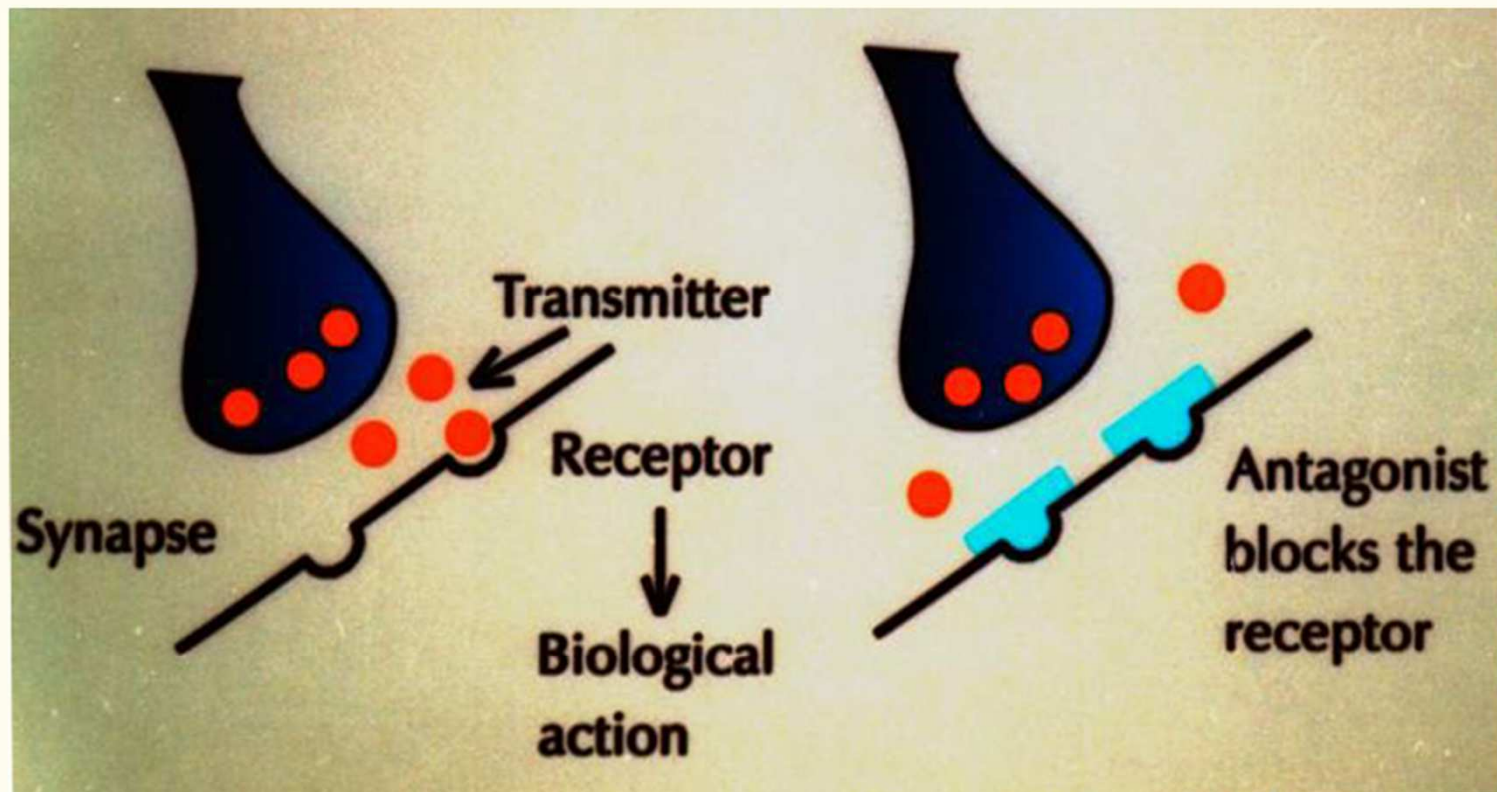


Pathophysiology of nausea and vomiting



Information is transmitted along these pathways via neurotransmitter chemicals

Neurotransmitter blockade reduces effect



Neurotransmitters in
emetic pathways



Dopamine

Histamine

Acetylcholine

Serotonin (5HT)

Antiemetics are
neurotransmitter blockers



Dopamine antagonists
*haloperidol; metoclopramide;
levomepromazine*

Anti-histamines
cyclizine; cinnarizine

Anticholinergics
hyoscine hydrobromide

5HT₃ antagonists
ondansetron; granisetron

Common doses

Metoclopramide: ORAL 10mg 8-hourly; PARENTERAL 30mg/24h csci^{*}
Levomepromazine: ORAL 3mg 12-hourly; PARENTERAL 12.5-25mg csci^{#*}
Haloperidol: ORAL 0.5-1.5mg daily or twice daily; PARENTERAL 1.25 – 2.5mg csci^{#*}

Cyclizine: ORAL 50mg 8-hourly; PARENTERAL 150mg csci^{*}
Cinnarizine: ORAL 15-30mg 8-hourly

Hyoscine hydrobromide: TRANSDERMAL Scopoderm patch 1mg over 72h

Ondansetron: ORAL 4-8mg 12-hourly; PARENTERAL 4-8mg iv[#]

****Continuous SubCutaneous Infusion***
#Bolus injection

Metoclopramide

Promotes gut peristalsis
(by stimulating peripheral 5HT₄ receptors)

Levomepromazine

- Hits lots of receptors – not just dopamine.
(A broad spectrum antiemetic)
- Often causes syringe driver infusion site reaction
- Sedative

5HT₃ antagonists

Good for post-op use

Great with emetogenic chemotherapy

BUT

No better than conventional drugs for longer-term control of sickness

AND

They are very constipating, and quite expensive

Think about what drugs would be appropriate for the following specific circumstances

1. Gastric stasis
2. Where sedation is appropriate
3. Raised intracranial pressure
4. Movement related sickness
5. Intestinal obstruction
(And what non-drug intervention might help a patient with intestinal obstruction?)

Route of drug administration

What goes down ...

...Will probably come up



Use the parenteral route until
sickness is controlled and the patient
can eat and drink again