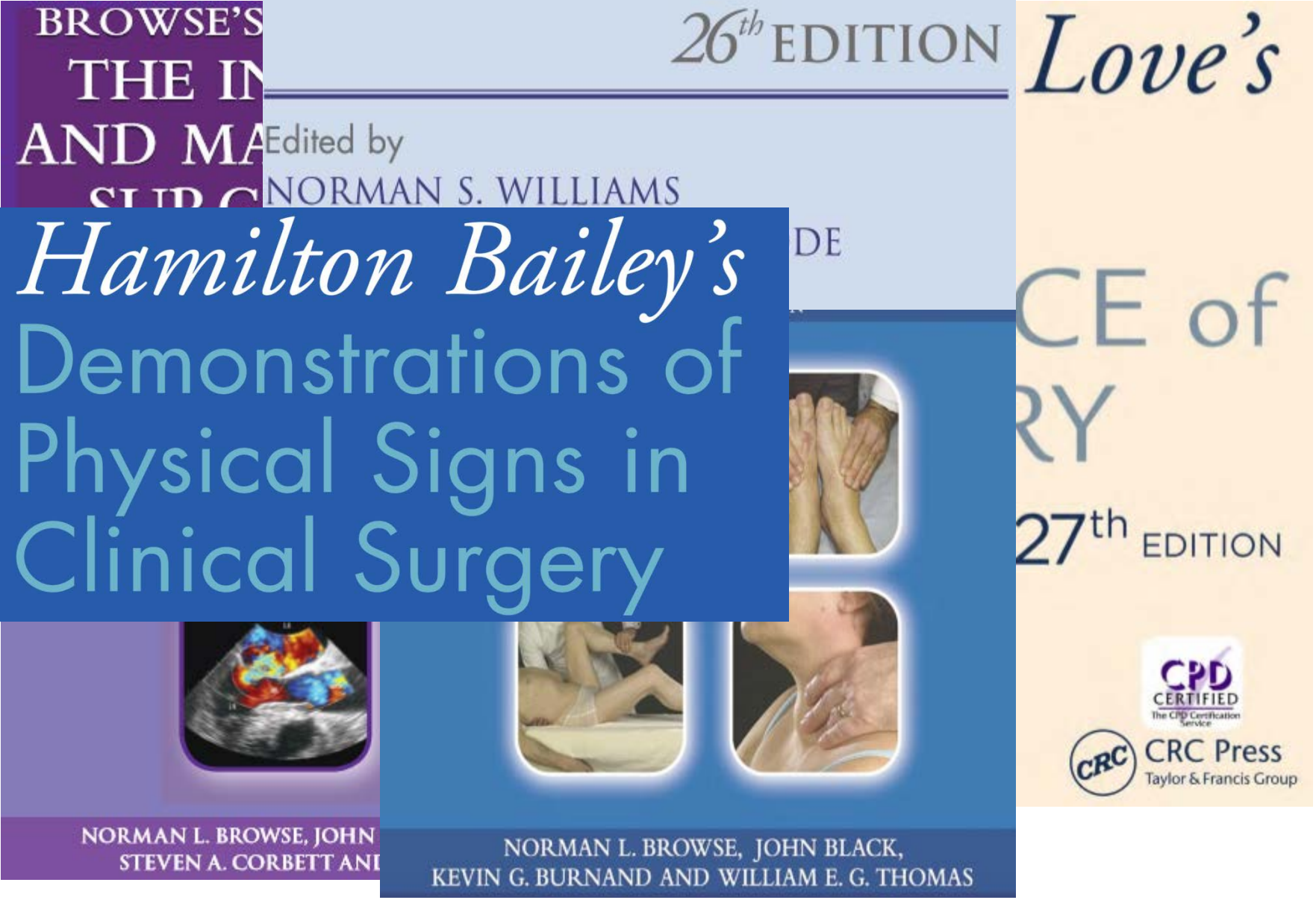
Ministry of Higher Education and Scientific Research
وزارة التعليم العالي و البحث العلمي
AL Mustansiriyah University
الجامعة المستنصرية
كلية الطب

كليه الطب فرع الجراحه

Parathyroids

الدكتور ذو الفقار حسن بيعي



Courtesy *Hamilton Bailey's* Demonstrations of Physical Signs in Clinical Surgery *19th Edition*

Courtesy BROWSE'S INTRODUCTION TO The Investigation and Management of Surgical Disease

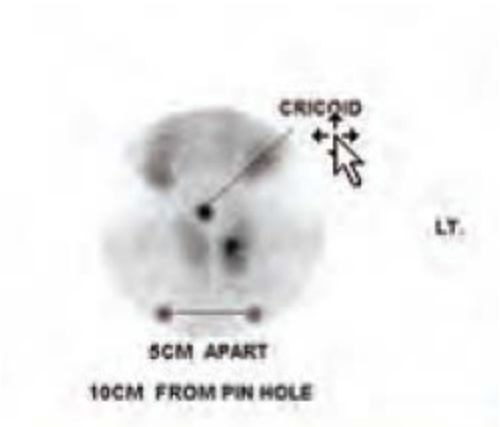
Courtesy Browse' s introduction to the symptoms & signs of surgical disease 4th edition

Courtesy Bailey & Love's Short Practice of Surgery

The parathyroid glands

الدكتور ذو الفقار حسن بيعي

DR.Thulfiqar Baiae
General surgeon
APR.2022



Sestamibi scan

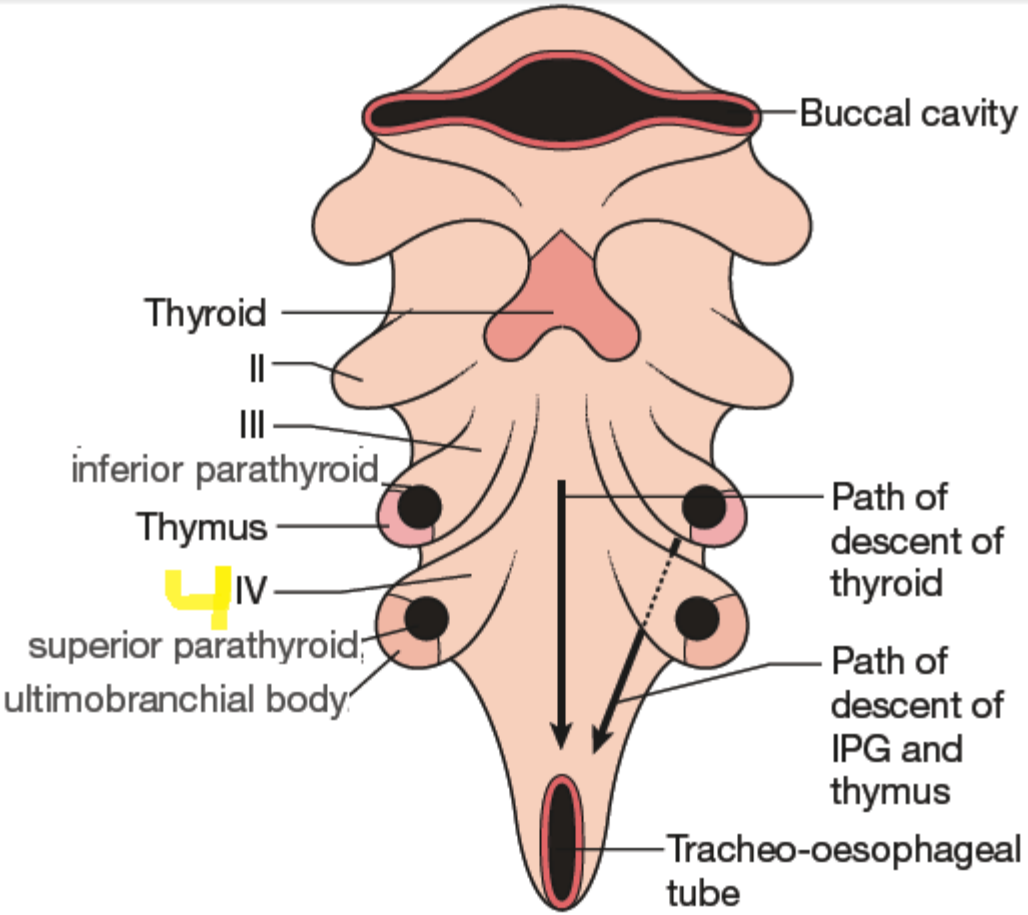
Courtesy A Bailey & Love short practice 27th edition.

INTRODUCTION

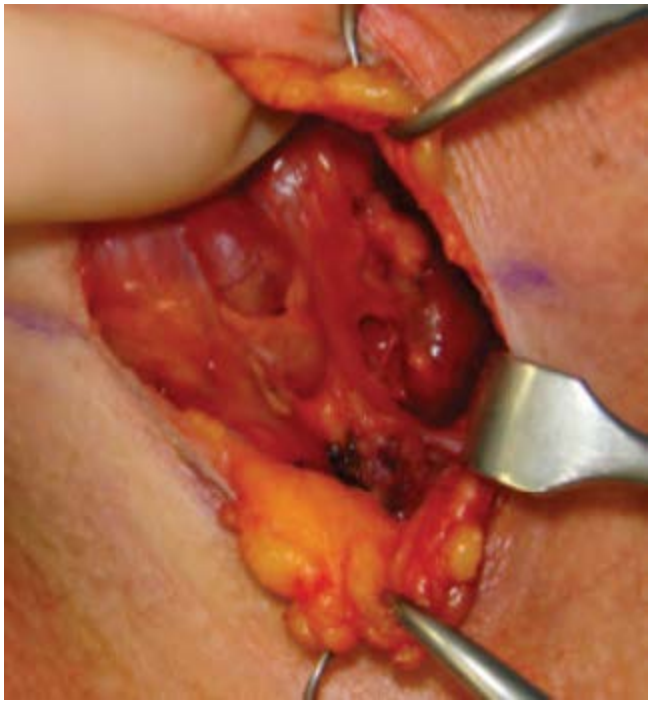
ANATOMY OF THE PARATHYROID GLANDS

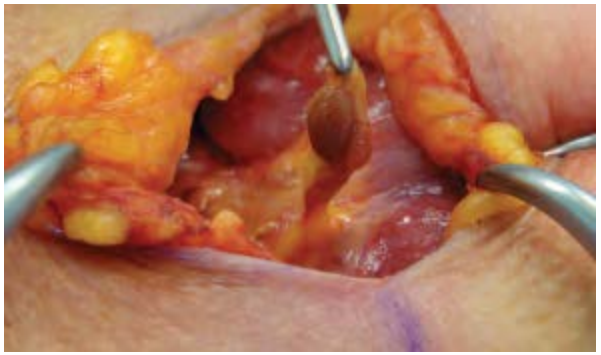
CALCIUM AND PARATHYROID HORMONE REGULATION

Courtesy A Bailey & Love short practice 27th edition.

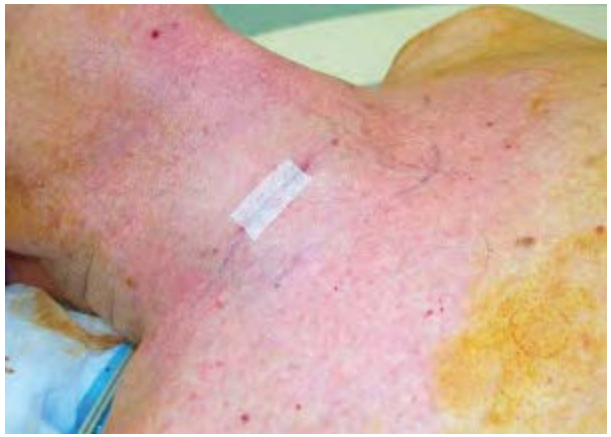


Courtesy Bailey & Love's Short Practice of Surgery

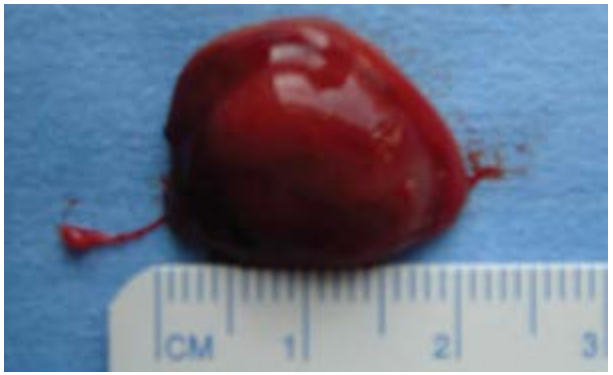




Courtesy Bailey & Love's Short Practice of Surgery



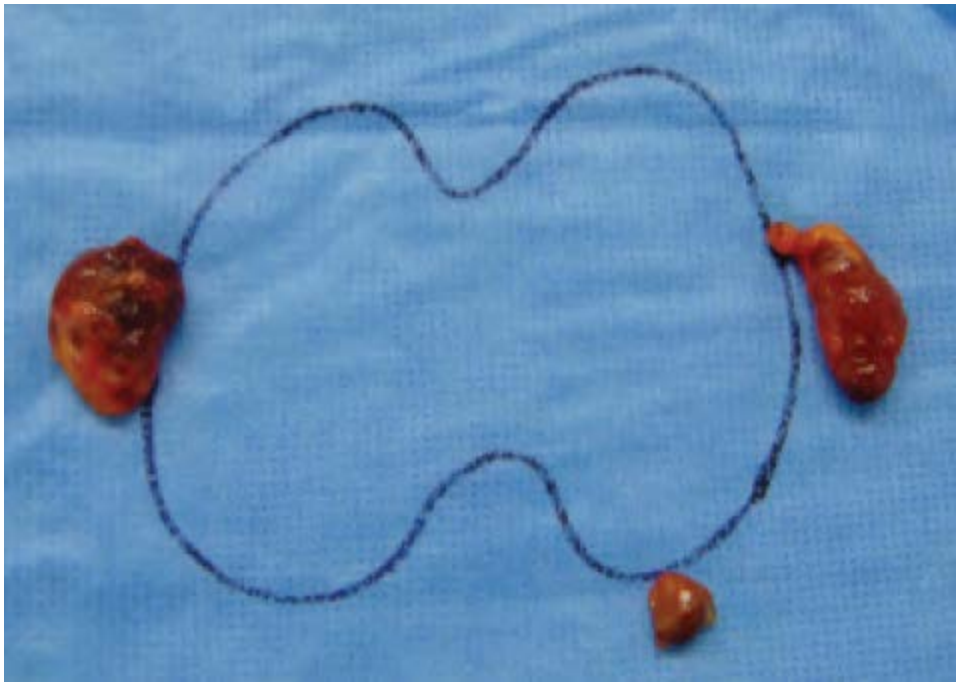
Minimally invasive parathyroidectomy



the excised parathyroid adenoma.

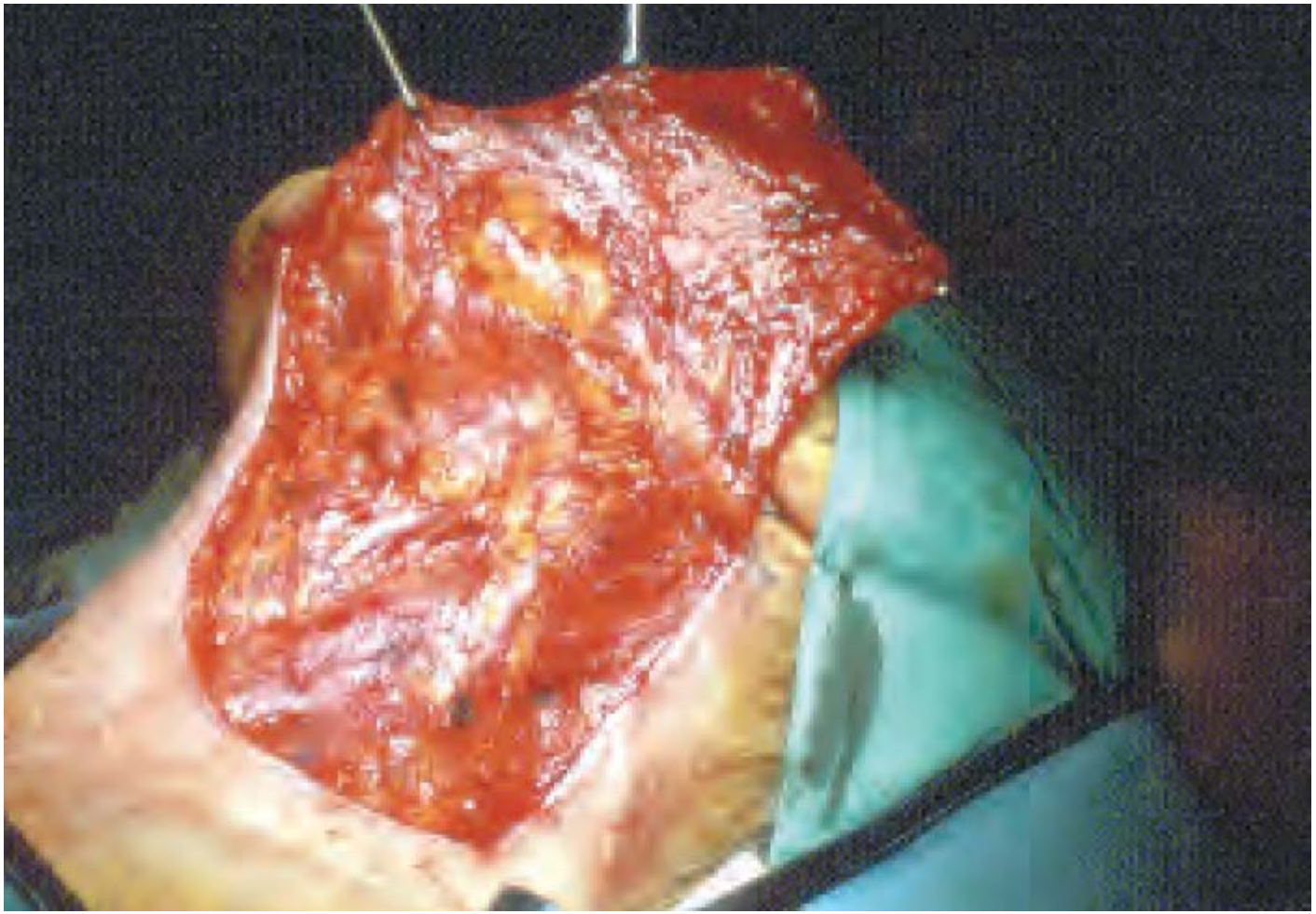
Courtesy Bailey & Love's Short Practice of Surgery

Subtotal parathyroidectomy for parathyroid hyperplasia.
Right inferior gland biopsied and half left *in situ*.



Courtesy Bailey & Love's Short Practice of Surgery

Skin flap developed for radical neck dissection.



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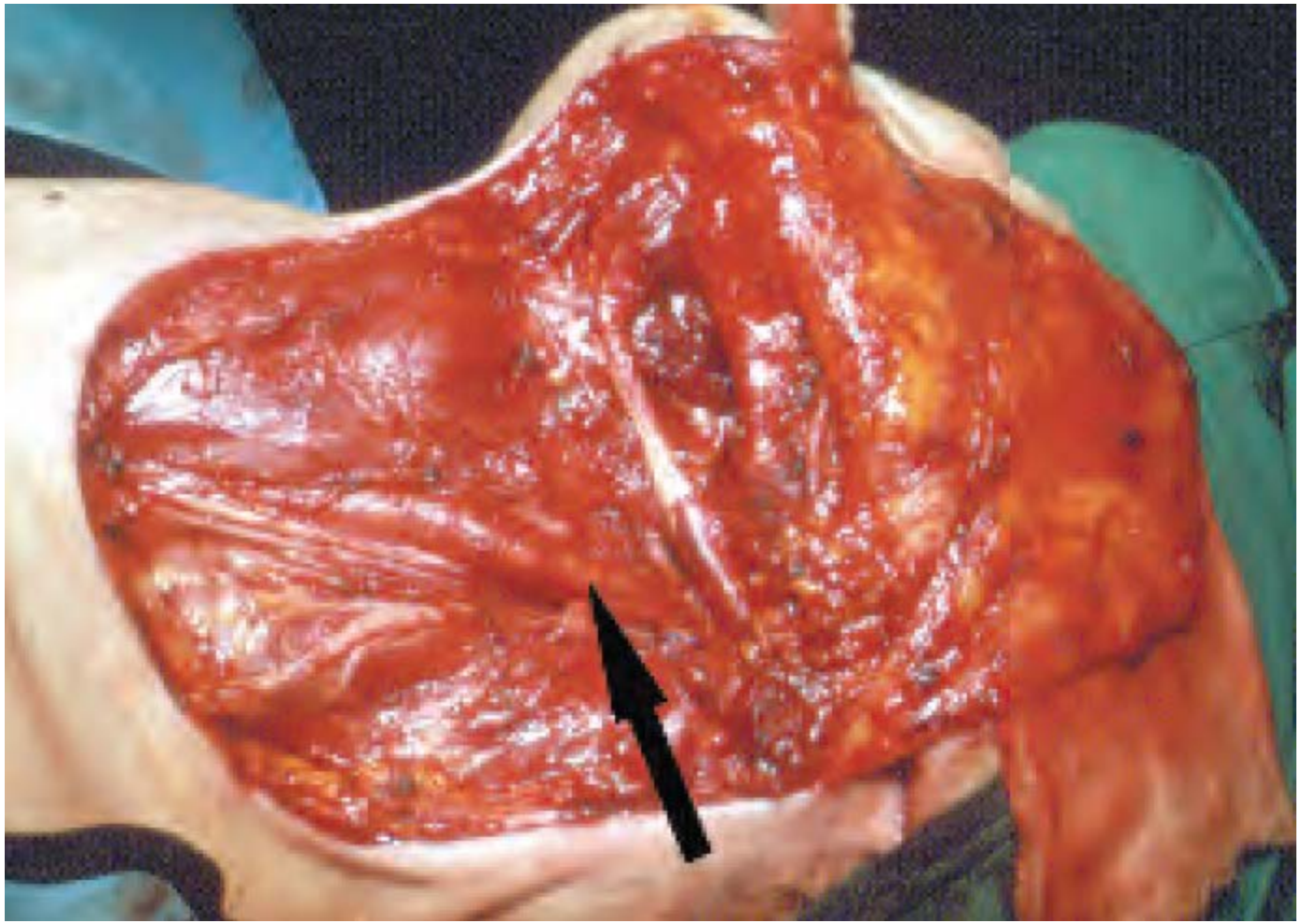
Completion of radical neck dissection revealing the great vessels of the neck



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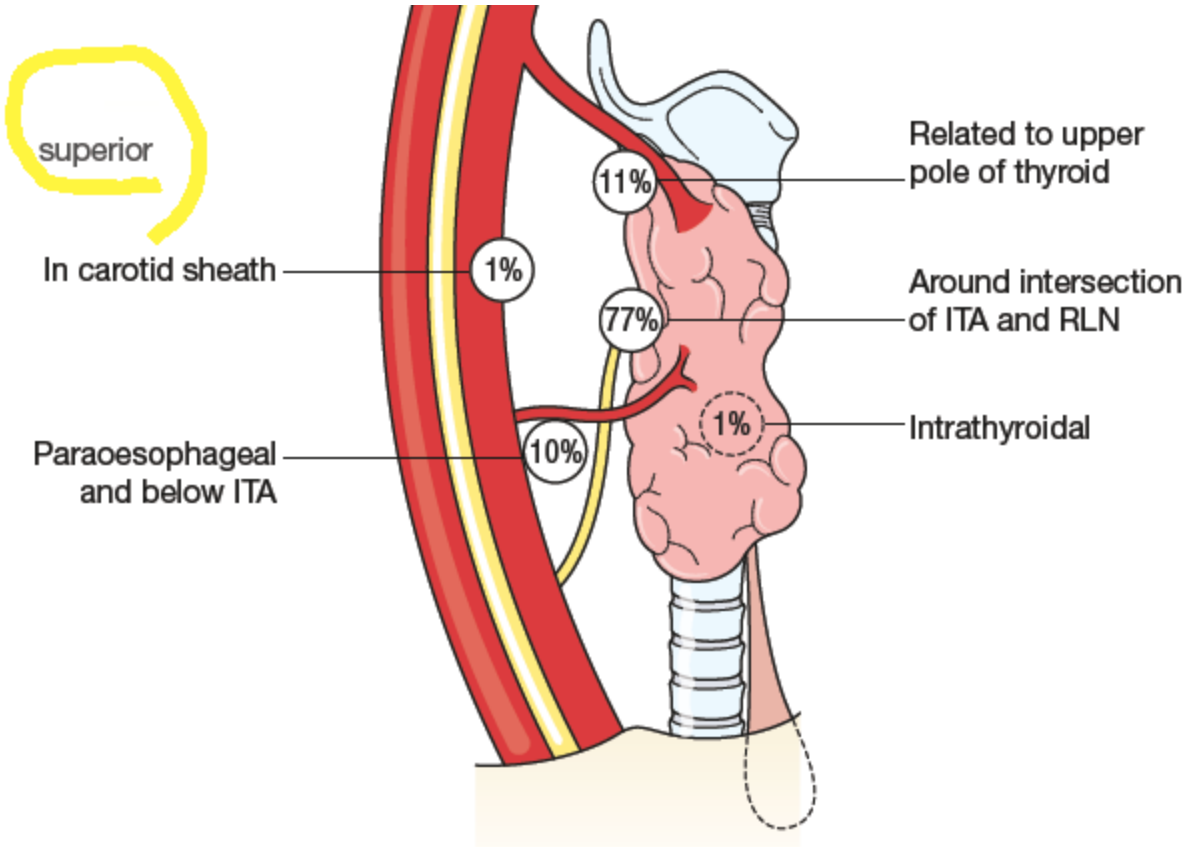

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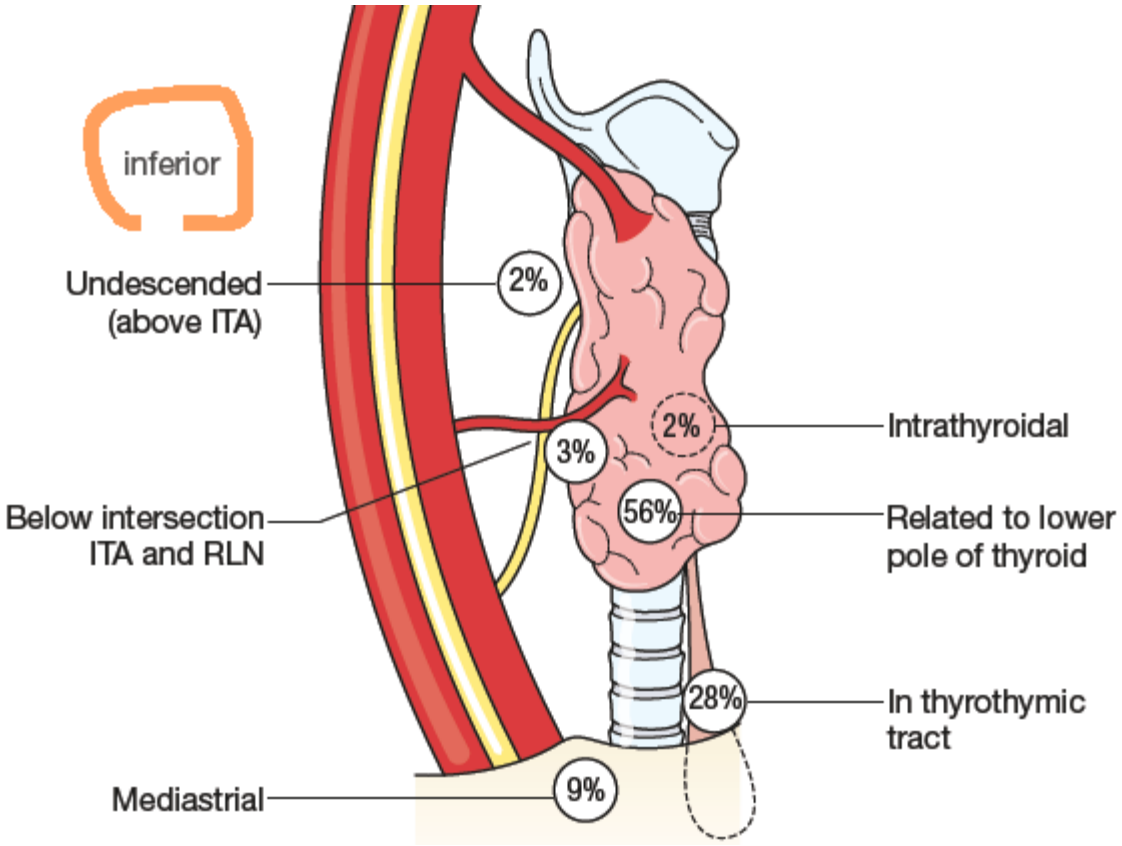

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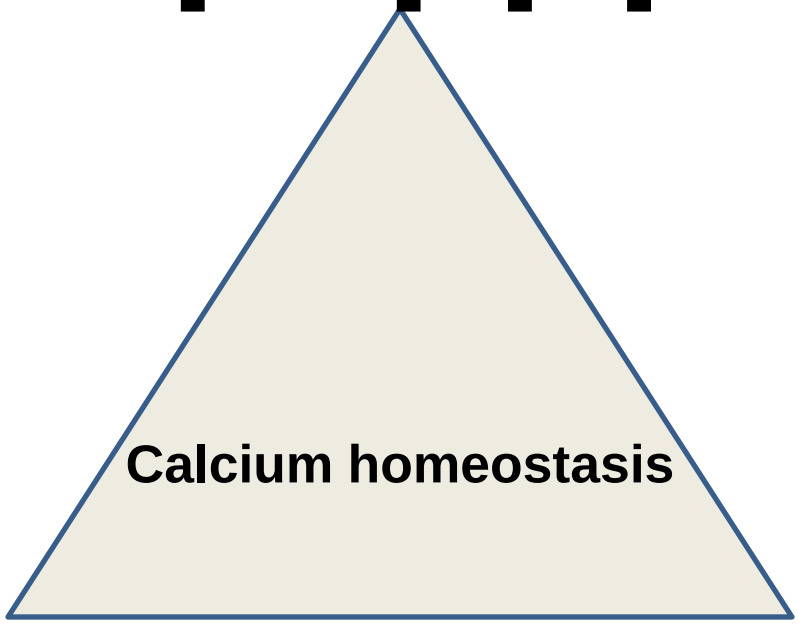
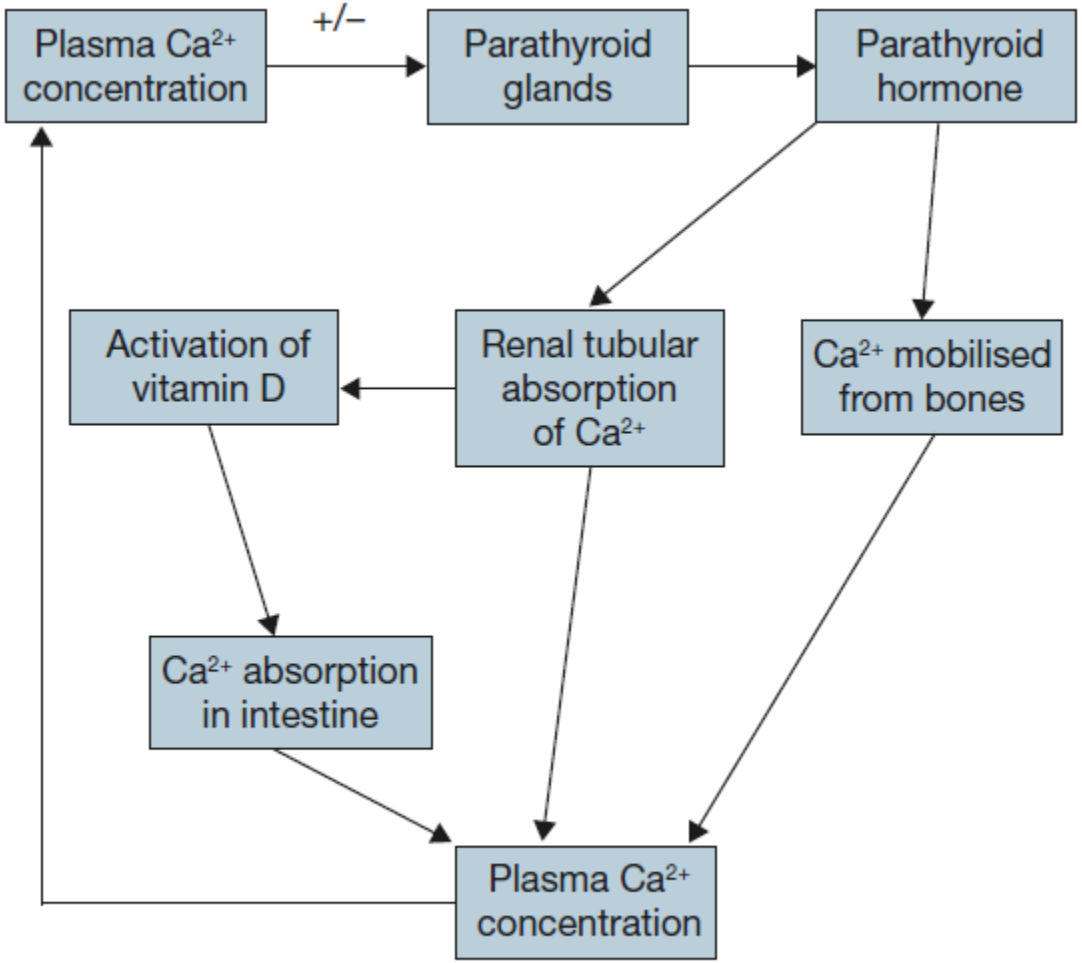
PTH



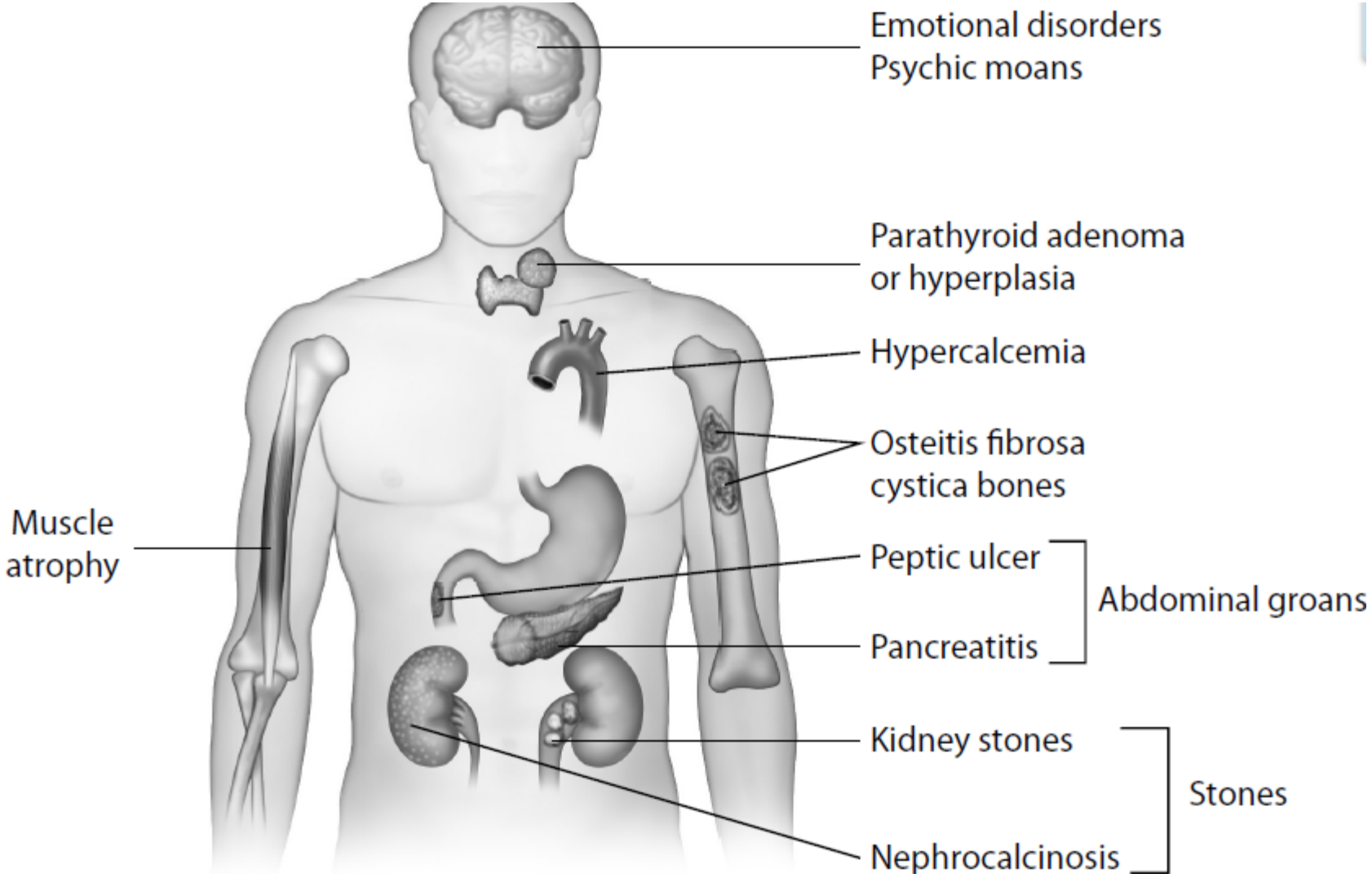
Calcitonin

Calcitriol



Schematic diagram of actions of parathyroid hormone.

Courtesy Bailey & Love's Short Practice of Surgery



Courtesy MCQs and EMQs in Surgery - Datta, Pradip K.

Stones,

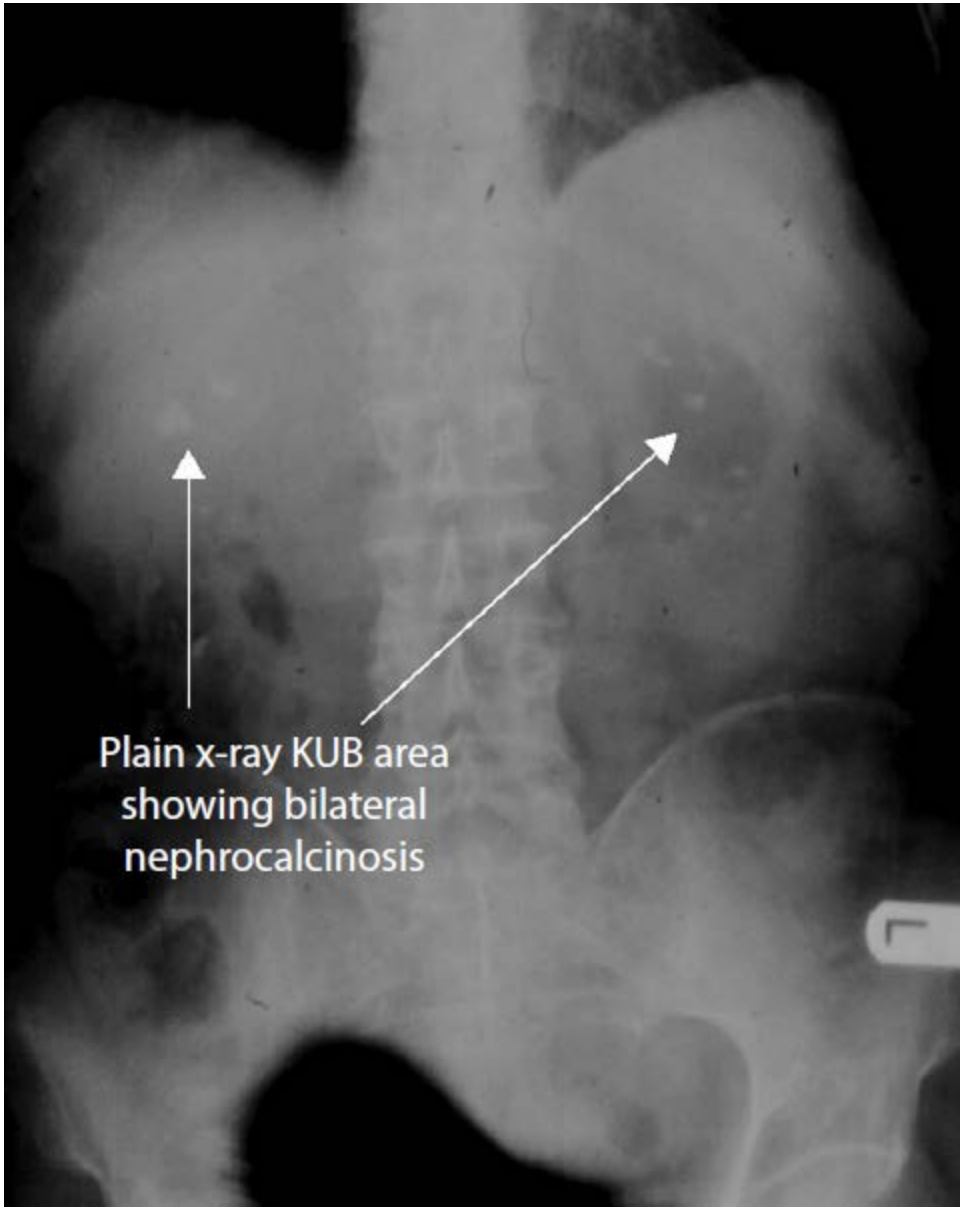
Bones,

Psychic moans

Abdominal groans

Courtesy MCQs and EMQs in Surgery - Datta, Pradip K.

Primary hyperparathyroidism.



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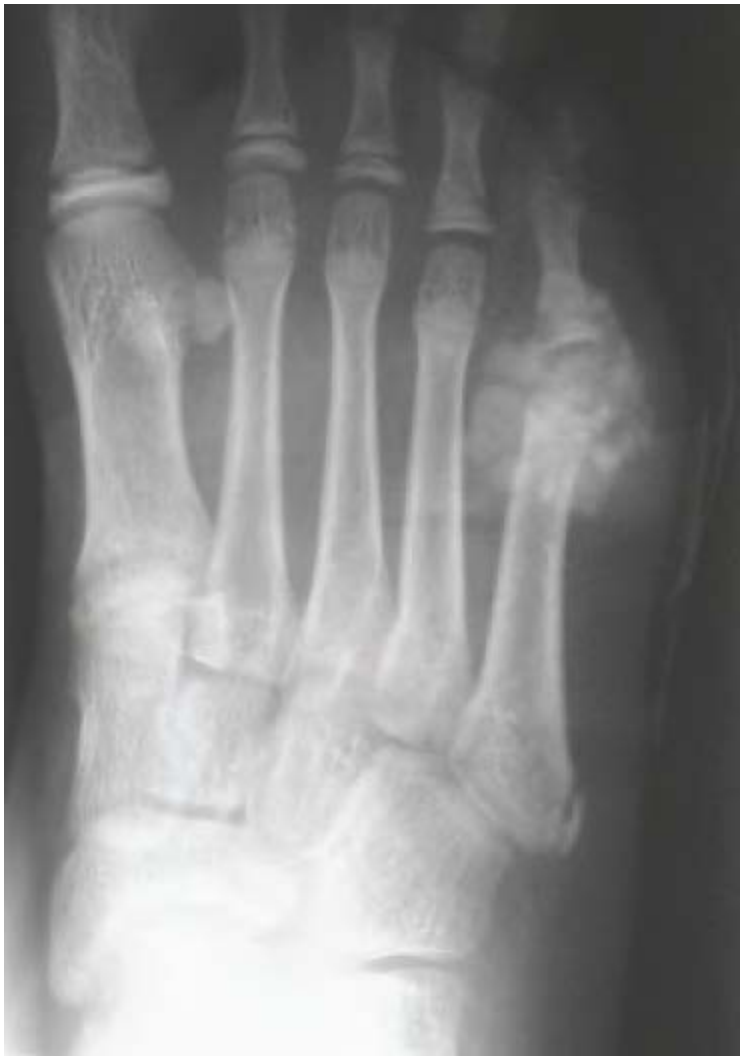
Secondary hyperparathyroidism. X-ray showing ectopic calcification.



Courtesy Bailey & Love's Short Practice of Surgery

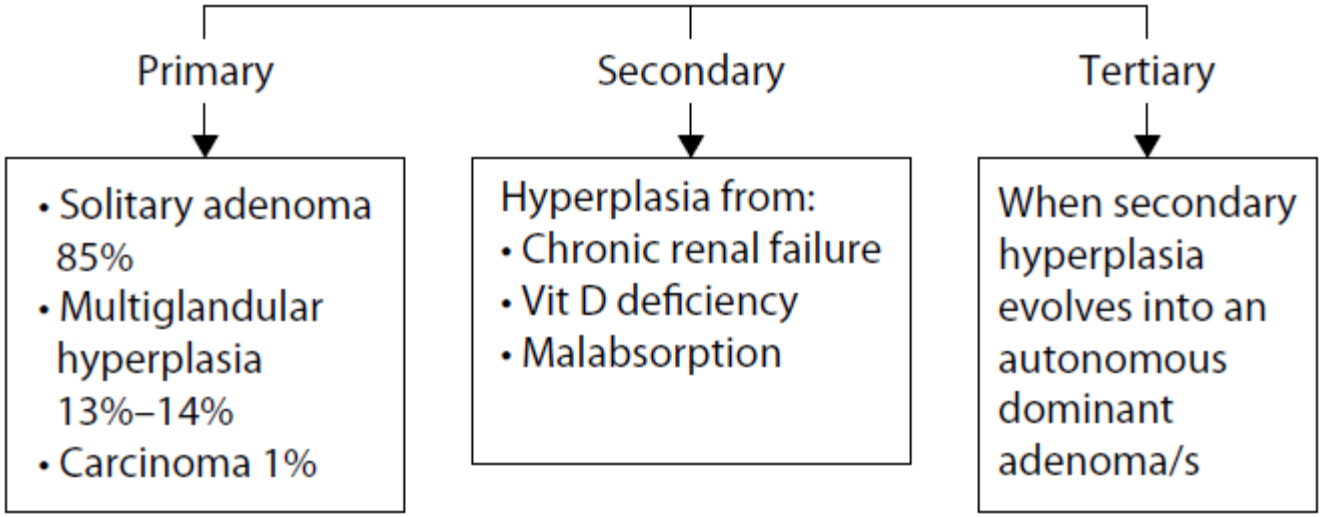
Causes of hypercalcaemia

Parathormone-induced

- Primary hyperparathyroidism
  - Adenoma (85%)
  - Hyperplasia (13%–14%)
  - Carcinoma (1%)
- Tertiary hyperparathyroidism

- Malignancy
  - Multiple myeloma
  - Skeletal secondaries from breast, thyroid, kidney, prostate, lungs
- Excess intake of Vitamin D
- Excess intake of calcium
  -'Milk-alkali' syndrome
- Thyrotoxicosis
- Hypoadrenalism
- Paget's disease of bone
- Sarcoidosis
- Familial hypocalciuric hypercalcaemia

Courtesy MCQs and EMQs in Surgery - Datta, Pradip K.



Summary of types of hyperparathyroidism.

Courtesy MCQs and EMQs in Surgery - Datta, Pradip K.

Hyperparathyroidism	Calcium	PTH	Vitamin D	Phosphate
Primary	↑	↑ →	↑	↓
Secondary	↓ →	↑	↓	↑ or ↓
Tertiary	↑	↑ ↑	↓	↑

Key: ↑Elevated, ↓decreased, →normal.

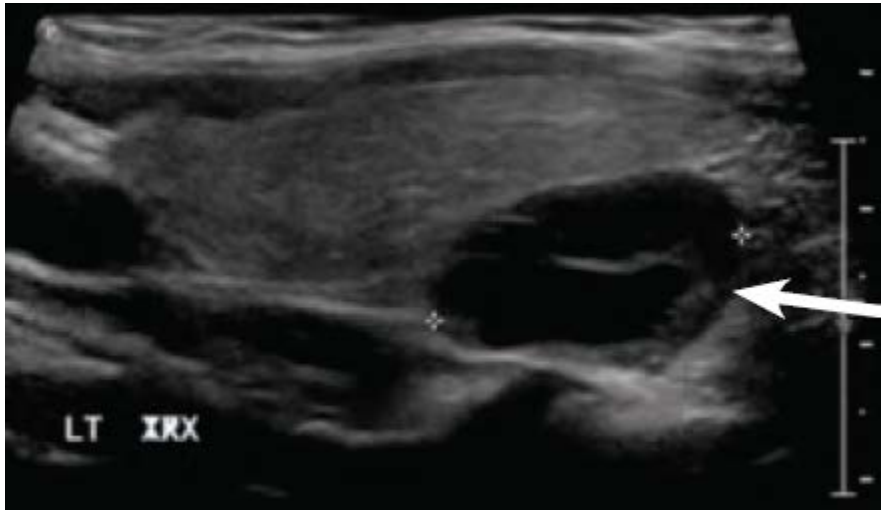
Source: Brashers. *Pathophysiology.* 2015.[6]

Courtesy https://www.mdedge.com/clinicianreviews/article/136564/endocrinology/primary-hyperparathyroidism-case-based-review/page/0/3

Sestamibi scan

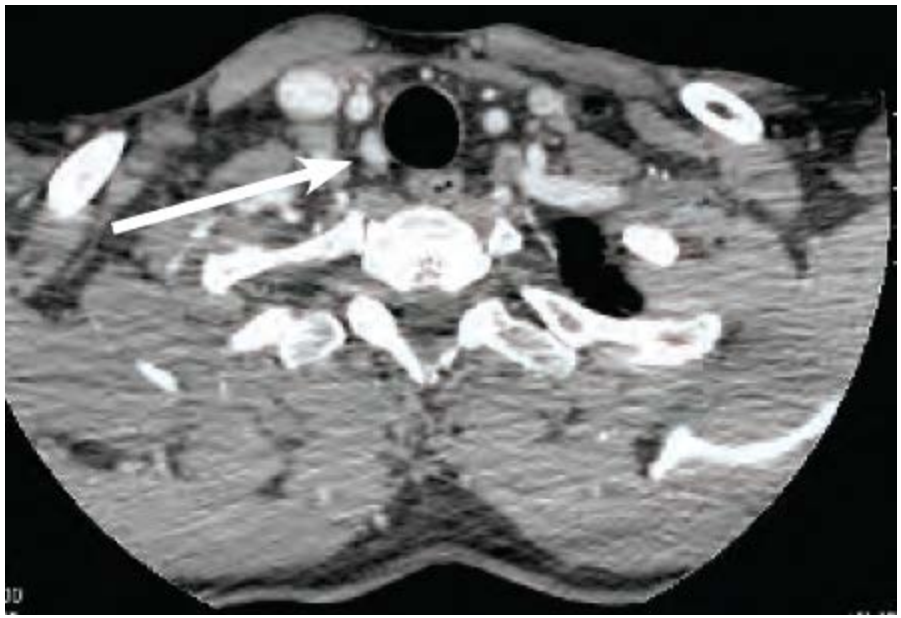
Courtesy Bailey & Love's Short Practice of Surgery

Right inferior adenoma,
with ultrasound



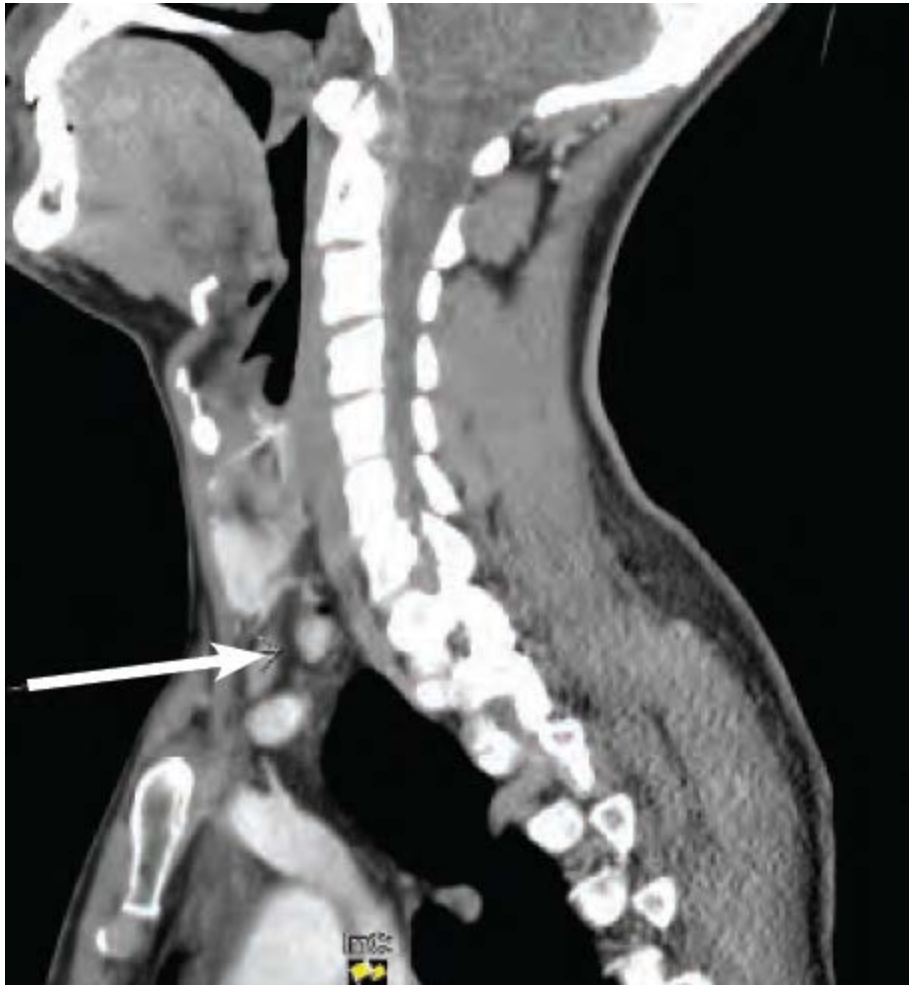
Courtesy Bailey & Love's Short Practice of Surgery

4D Computed tomography scanning demonstrating a right inferior parathyroid adenoma.



Courtesy Bailey & Love's Short Practice of Surgery

4D Computed tomography scanning demonstrating a right inferior parathyroid adenoma.



Courtesy Bailey & Love's Short Practice of Surgery

Calcium 8.6–10.0 mg/dL 2.15–2.50 mmol/L

CALCIMIMETICS

cinacalcet

Courtesy Bailey & Love's Short Practice of Surgery

Primary hyperparathyroidism

Primary hyperparathyroidism is usually sporadic

Hyperparathyroidism can be associated with a pituitary adenoma.

Biochemical diagnosis of primary hyperparathyroidism?

Raised total calcium and elevated PTH levels.

Raised ionised calcium and elevated PTH levels.

Low serum phosphate levels.

Low urine calcium level.

Primary hyperparathyroidism

Primary hyperparathyroidism is associated with raised serum calcium and low phosphate levels.

The PTH levels are not suppressed despite the hypercalcaemia.

The condition resulting from an excessive secretion of PTH, is caused by a solitary parathyroid adenoma in 85% of patients; in 13%–14%, the cause might be multiglandular hyperplasia, whilst the remainder might be due to a carcinoma or multiple adenomas. The most common presentation of primary hyperparathyroidism from an adenoma is when it is discovered in an ‘asymptomatic’ patient who undergoes a routine biochemical screening. When hypercalcaemia is found routinely and the cause is an adenoma and it is removed, patients admit to a huge sense of well being. In the past they had ascribed their insidious symptoms to ageing. Thus, strictly speaking, in retrospect the patient was not ‘asymptomatic’ but had symptoms that could not be pinpointed.

Women are more often affected. It is said that 1% of the adult population is found to have hypercalcaemia on routine biochemical screening.

The condition may be familial when it is a part of multiple endocrine neoplasia (MEN) syndrome.

When symptomatic, the clinical features reflect hypercalcaemia and hypophosphataemia: bone pain with x-ray changes in the hands (osteitis fibrosa cystica), skull (pepper-pot skull) and abdominal pain from peptic ulcer, recurrent pancreatitis and constipation. In almost one-third of patients there might be psychiatric symptoms. Renal disease in the form of nephrocalcinosis and recurrent ureteric colic may be a presentation when a raised serum calcium alerts one to the diagnosis.

These protean manifestations have been traditionally summarised as ‘stones, bones, psychic moans and abdominal groans’, although such a florid presentation is rarely seen.

Rarely, patients might present as a metabolic emergency due to acute hyperparathyroid hypercalcaemic crisis.

The patient’s corrected serum calcium should be carried out. For this the serum albumin is done .

The next step is to do the serum PTH.

Elevated levels of PTH with hypercalcaemia and hypophosphataemia and increased excretion of calcium in the urine confirms a diagnosis of primary hyperparathyroidism.

The next step is to localise the site of the adenoma. This is done by technetium-99 m labelled sestamibi scan and ultrasound to include the mediastinum to exclude an adenoma in an ectopic gland.

In primary hyperparathyroidism, systematic bilateral neck exploration is done in cases of parathyroid hyperplasia where removal of three-and-a half glands is carried out with the remaining gland being marked with a metal clip or nonabsorbable suture or transplanted into the forearm.

However, targeted parathyriodectomy through a small unilateral incision for an adenoma is the procedure of choice in the vast majority as solitary adenoma is the most common cause of primary hyperparathyroidism.

Hypoparathyroidism

Serum calcium levels can come down when the parathyroid glands are inadvertently removed.
Levels must always be checked within 24 hours of any thyroid surgery.

This may be due to injury, removal, or ischaemia during major neck surgery. The level of calcium at which symptoms of tetany develop can vary and is unpredictable.

Hypoparathyroidism

This is a medical emergency, particularly if the level is less than 1.9 mmol/lL An intravenous drip of 10% calcium gluconate might be necessary. This is supplemented by oral calcium and Vitamin D; magnesium supplements may also be required. If the hypocalcaemic symptoms are minimal with the serum level in the region of 2.0 mmol/L, oral calcium supplements of 1 g in divided doses is given. Less than 1% of patients undergoing such surgery will have such a complication permanently.

The normal serum calcium is 2.2 to 2.6 mmol/L. Of the total calcium in the body, 50% is free ionised; of the rest 40% is bound to plasma proteins, whilst the remainder is bound with anions – citrate, phosphate and sulphate.

Hypoparathyroidism

When an abnormal serum calcium level is obtained from the laboratory, it is important to estimate the corrected serum calcium level, particularly in the presence of hypoalbuminaemia where the corrected serum calcium will be higher.

The regulation of calcium homeostasis is the result of an interplay between parathormone (PTH) secreted by the parathyroids, the kidneys, the absorption of Vitamin D and calcitonin secreted by the parafollicular ‘C’ cells of the thyroid.

Hypoparathyroidism

When there is a drop in serum calcium, there is increased secretion of PTH acting in the form of a feedback mechanism .

PTH acts in concert with 1,25- dihydroxycholecalciferol (1,25-DHCC, 1,25 vitamin D) and calcitonin on kidney and gut to maintain a constant blood ionised calcium, which is necessary for nerve and muscle excitability and blood coagulation.

PTH has a greater role in the regulation of serum calcium than calcitonin.

Hypoparathyroidism

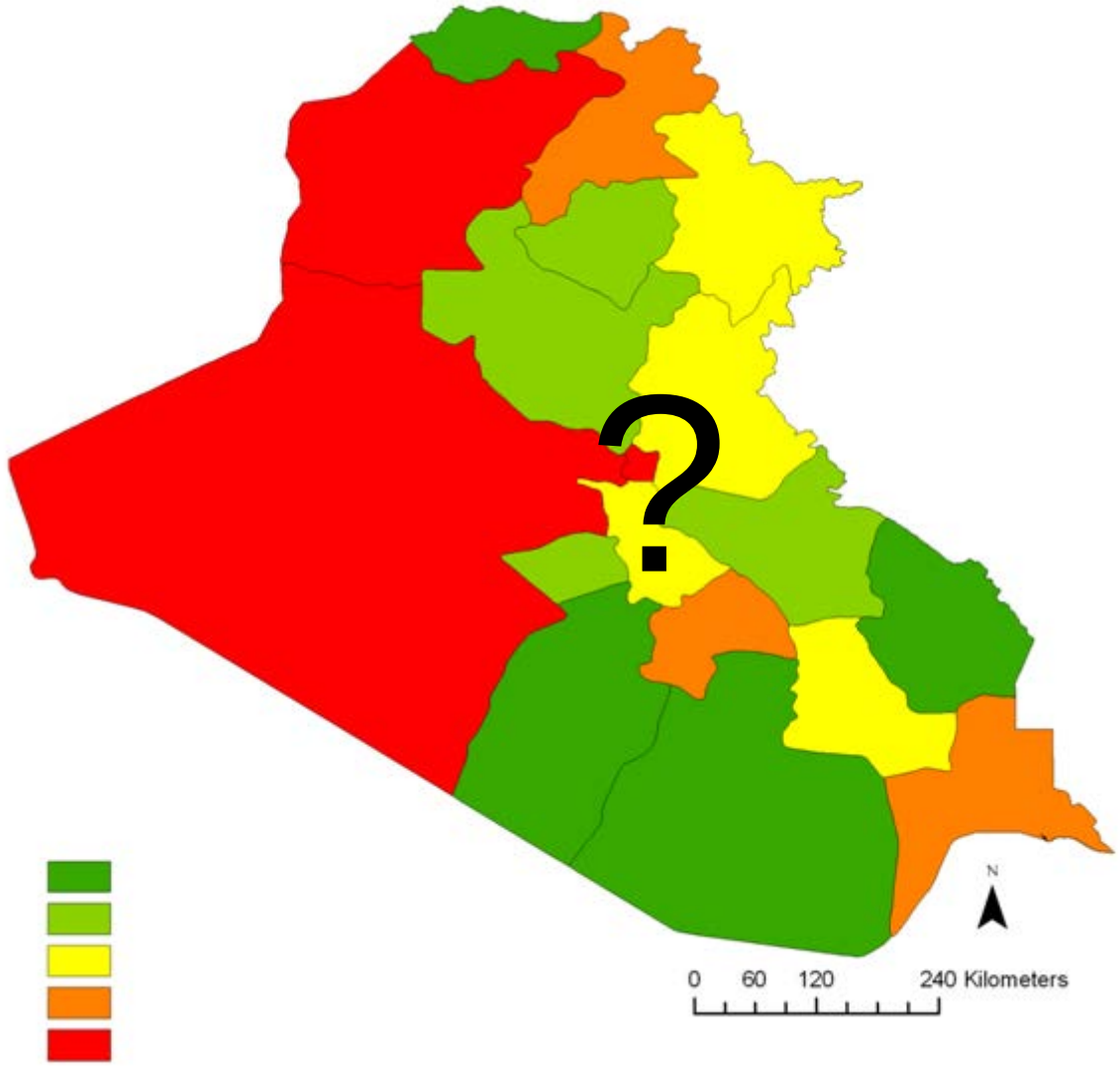
The role of calcitonin is to cause a reduction in serum calcium .

Serum calcium regulation is also linked to plasma phosphate (PO) levels.

The normal PO level is 0.8 to 1.4 mmol/L; PTH increases the excretion of PO while increasing renal tubular reabsorption of calcium.

Variations in plasma PO levels affect serum calcium.

In renal failure, there is increase in plasma PO that stimulates the parathyroids causing secondary hyperparathyroidism.



courtesy أ.م.د جمال الخضيري

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