MFDS PART 1 · HUMAN DISEASE

Medicine Relevant to Dentistry 2

Dr Mohamed J. Abuasi · 14 May 2025 · 87-slide lecture deck

EXAM-DAY SUMMARY

everything on this page → detail in §§ overleaf

RESPIRATORY → §1

- URTI: strep most common; penicillin DOC → no co-prescribing; glandular fever + amoxicillin = rash; sinusitis mimics dental pain
- Asthma: avoid GA + NSAID; inhalers → dry mouth, ulcers, discoloration (salbutamol)
- COPD = bronchitis (cough >3 mo/yr for 3 yrs) + emphysema; avoid GA/sedation/rubber dam
- TB: haemoptysis, oral ulcers, CXR dx; aerosol → wrap unit, change water lines
- Bronchial ca: ↑ACTH → mucosal melanosis

LIVER → §2

- Cholestatic: intra-hepatic = bilirubin in plasma; extra-hepatic (stone/tumour/clot) = in urine
- Hep A food 3 wk · B blood/droplet 6 wk–6 mo · C blood-blood · D with B
- Anti-HBs alone = vaccinated (lecture: prior infection — corrected)
- Mgmt: coag screen, bleeding precautions, avoid GA, vit K days pre-op

DIABETES → §3

- I: dependent, fast, young, genetic/viral · II: independent, gradual, old, ± cortisone
- Oral: dry mouth, candida, BMS, perio breakdown
- Appoint 2 h after breakfast ; meds + food as normal; GA → stop meds night before
- Antibiotic prophylaxis; liaise with diabetes team

ADRENAL → §4

- Cushing: 1ry (steroids/adenoma) = syndrome ; 2ry (↑ACTH) = disease; moon face, buffalo hump, osteoporosis, HTN
- Addison: ↓cortisone, hyperpigmentation; steroid cover pre-surgery
- Crisis: shock, ↓BP, ↑K^+ ↓Na^+, confusion — emergency

THYROID · PARATHYROID → §4

- Hyper (Graves): wt loss, heat, tachy, exophthalmos — avoid epinephrine only if uncontrolled
- Hypo (Hashimoto/↓TSH): wt gain, cold, brady, hoarse
- HyperPTH: brown tumours, ↑Ca, stones · HypoPTH: tetany, Chvostek, delayed eruption, enamel hypoplasia

MRONJ DRUGS → §5

- Definition: exposed/probeable bone >8 wk + drug hx, no jaw RT, no jaw mets
- BPs bind hydroxyapatite; alendronate bone t½ ≈ 10 yrs ; jaw turnover 10× long bones
- Denosumab (RANKL mAb): no bone binding, effect gone in 9 mo; SC 6-monthly
- Anti-angiogenics: bevacizumab, aflibercept, sunitinib

MRONJ NUMBERS → §6

- Incidence: cancer 1%; osteoporosis 0.01–0.1%
- Post-extraction: 2.9% cancer · 0.15% osteoporosis
- Higher risk: BPs >5 yrs; + glucocorticoids any length; cancer Rx; previous MRONJ
- Past BP ever / denosumab ≤9 mo = still taking

MRONJ MANAGEMENT → §6

- Dentally fit pre-therapy; high-F toothpaste; low risk: extract in primary care
- No antibiotic/antiseptic prophylaxis; no drug holiday ; endo > extraction
- Higher risk: alternatives (retain roots), OS/OMFS letter; review 8 wk, refer if unhealed
- Denosumab: treat month before next dose; latent phase → sclerosis, wide lamina dura; CTX markers

CARDIOVASCULAR → §7

- HTN >140/>90; 95% essential; MAP = (2×dia + sys)/3
- 180/110 absolute cut-off ; emergency above it → 5 mg diazepam
- CCB → gingival hyperplasia; ACEI → angioedema; all → dry mouth, BMS, lichenoid
- Angina: L chest → L shoulder + jaw; MI: delay surgery 6 mo

GIT → §8

- Peptic ulcer: NSAIDs out, only paracetamol safe ; omeprazole → dry mouth, EM, ↑warfarin/diazepam
- GORD: palatal erosion; avoid night guards
- Antacids block absorption: fluoride, azoles, tetracycline
- Coeliac: gluten (wheat — not rice), oral ulcers, ± intestinal lymphoma

CROHN'S · IBD · IBS → §8

- Crohn's: ileal granulomatous; malabsorbs Fe/B12/folate; cobblestone tags, lip swelling, RAU ; cortisone
- IBD = Crohn's + UC (chronic bleeding); cortisone
- IBS: not a true disease — bruxism, wear facets, psychogenic orofacial pain

DYSPHAGIA → §8

- Oral: MAU, stomatitis, tonsillitis, herpes, malignancy, dry mouth, pharyngitis
- Systemic: Plummer–Vinson, oesophageal ca, foreign body, stricture
- Plummer–Vinson (Brown Kelly Paterson): premalignant — Fe-deficiency anaemia + post-cricoid web

§1 Respiratory disease

1.1 Upper respiratory tract infections

Upper respiratory tract infections (URTIs) include the common cold, pharyngitis, sinusitis and epiglottitis. They may be bacterial or viral, and streptococcal infection is the most common cause. Penicillin is the drug of choice, so the dentist should avoid co-prescribing — the patient is likely already covered by the physician's antibiotic. Glandular fever mimics these conditions, but amoxicillin given to a glandular fever patient may cause a sensitivity reaction, which is a classic prescribing trap.

Sinusitis mimics dental pain, so the working rule in dentistry is twofold: ★**exclude dental causes first, and avoid co-prescription of drugs**.

1.2 Bronchial asthma

Asthma is a reversible bronchoconstriction causing wheezing and breathlessness; patients complain of the chest feeling tight. There is often an allergic component — aspirin, non-steroidal anti-inflammatory drugs (NSAIDs) or penicillin can all act as triggers — and the condition is worsened by anxiety, which matters in the dental chair.

Asthmatic patients are usually treated with three inhaler classes: corticosteroid inhalers, beta-adrenergic agonist bronchodilators (salbutamol) and antimuscarinic bronchodilators (ipratropium). These inhalers usually cause oral complications: dry mouth, stomatitis, taste alteration, oral ulcers and teeth discoloration (the last attributed to salbutamol).

> **CAUTION**
> Before dental work on an asthmatic: the patient should bring their inhaler; decrease stress and anxiety; **avoid general anaesthesia (GA) and avoid NSAIDs**; and advise the patient to stop smoking.

1.3 Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is a combination of chronic bronchitis — defined as a ★**productive cough for more than 3 months per year, for 3 years** — and emphysema. Smoking is the prime cause and must be stopped. These patients are usually treated with antibiotics, so again avoid co-prescription. In the dental setting, avoid GA, sedation and, if possible, rubber dam (it further restricts an already compromised airway); in cases of severe anxiety, IV sedation is the option.

1.4 Tuberculosis

Tuberculosis (TB) is a granulomatous infection presenting with chronic cough with haemoptysis, weight loss and oral ulcers, and is diagnosed by chest radiograph. Cross-infection is by aerosol, so it can spread through the whole clinic.

> **KEY POINTS**
> TB management in dental work: ask about antibiotics to avoid co-prescription; good infection control to reduce aerosol; wrap the whole unit and working surfaces; you may need to change your water lines; avoid GA.

1.5 Bronchial carcinoma

Bronchial carcinoma is mainly due to smoking. Its dental relevance is endocrine: increased secretion of adrenocorticotrophic hormone (ACTH) by the tumour causes ★**mucosal melanosis** — pigmentation of the oral mucosa can be the presenting sign.

> **★ EXAM PEARL**
> Two respiratory diagnoses hide in the mouth: oral ulcers point to TB, while new mucosal pigmentation (melanosis) points to ectopic ACTH from a bronchial carcinoma. Both are answers the examiner reaches via the oral sign, not the chest.

§2 Liver disease

2.1 Jaundice and its causes

Jaundice is the prime symptom of liver disease: a widespread tissue discoloration of the skin and mucosa that appears when the liver becomes unable to process bilirubin. Its causes fall into two groups: an excessive amount of bilirubin arriving for conjugation, as in haemolytic anaemia, or an inability to excrete bile — cholestatic jaundice.

Cholestatic jaundice itself divides into intra-hepatic and extra-hepatic forms, distinguished by where the bilirubin accumulates.

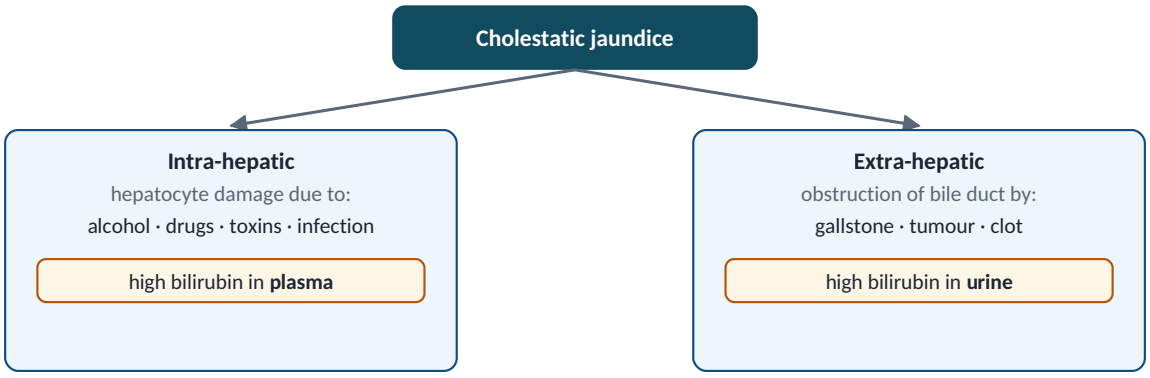

Diagram 1 — Classification of cholestatic jaundice, as given in the lecture. Intra-hepatic disease is characterised by high bilirubin in plasma; extra-hepatic obstruction by high bilirubin in urine.

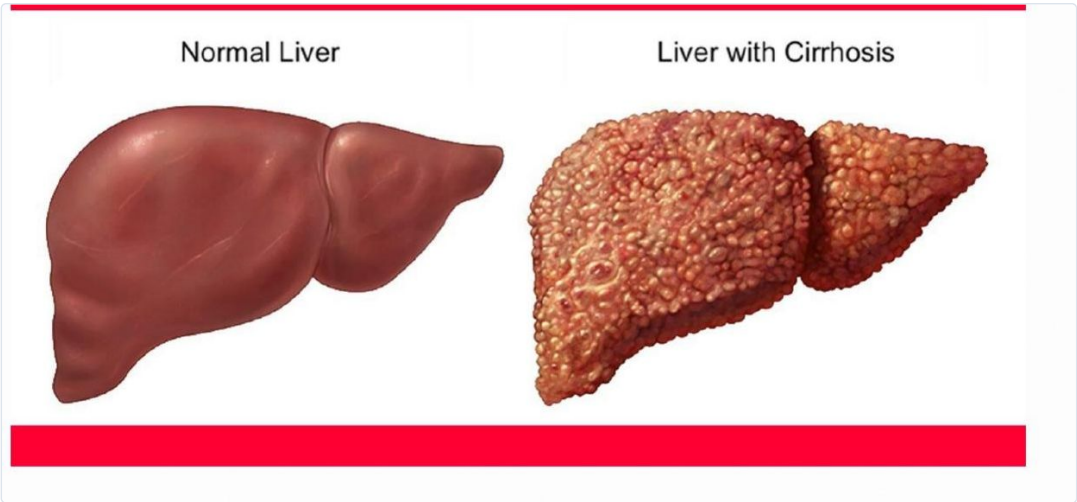

Fig. 1 — Normal liver (left) versus liver with cirrhosis (right): the smooth surface is replaced by regenerative nodules.

2.2 Viral hepatitis

Table 1 — Viral hepatitis A–D

Virus	Transmission	Incubation / association
Hepatitis A	Food	3 weeks
Hepatitis B	Blood and droplet	6 weeks – 6 months
Hepatitis C	Blood-to-blood contact	—
Hepatitis D	—	Associated with hepatitis B

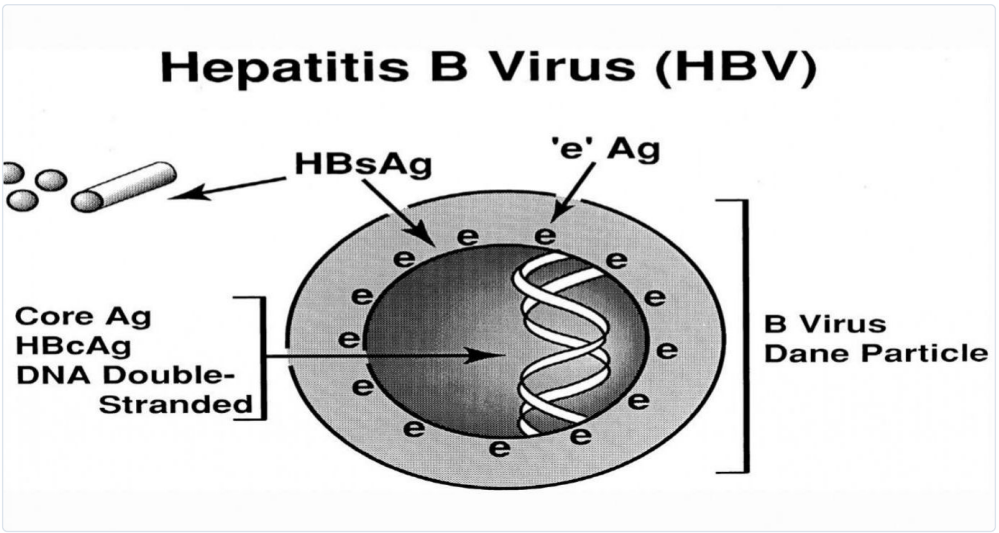

Fig. 2 — The hepatitis B virus (Dane particle): surface antigen (HBsAg) on the envelope, 'e' antigen (HBeAg), and the core antigen (HBcAg) enclosing double-stranded DNA.

Serological markers place a hepatitis B patient on a risk spectrum. Read the table by asking: is surface antigen still present (infection ongoing), and has 'e' antigen been cleared (infectivity falling)?

Table 2 — Hepatitis B markers and risk status

HBsAg	HBeAg	Anti-HBe	Anti-HBs	Risk status
+	Unknown	Unknown	−	High/low risk
+	+	−	−	High risk
+	−	+	−	Low risk
−	−	+	+	Immune due to previous infection
−	−	−	+	Immune (see pearl)

HBsAg, hepatitis B surface antigen; HBeAg, hepatitis B 'e' antigen; anti-HBe, antibody to 'e' antigen; anti-HBs, antibody to surface antigen.

★ EXAM PEARL

The lecture table labels the last row (anti-HBs alone, all other markers negative) "immune due to previous infection". The standard reading — and the single-best-answer the exam wants — is that **anti-HBs alone means immunity from vaccination**; previous infection additionally leaves anti-HBc (and often anti-HBe) behind. Verify against your textbook.

2.3 Hepatic oedema and stigmata

Fluid signs of chronic liver disease include ascites and ankle oedema, accompanied by itching and spider naevi on the skin.

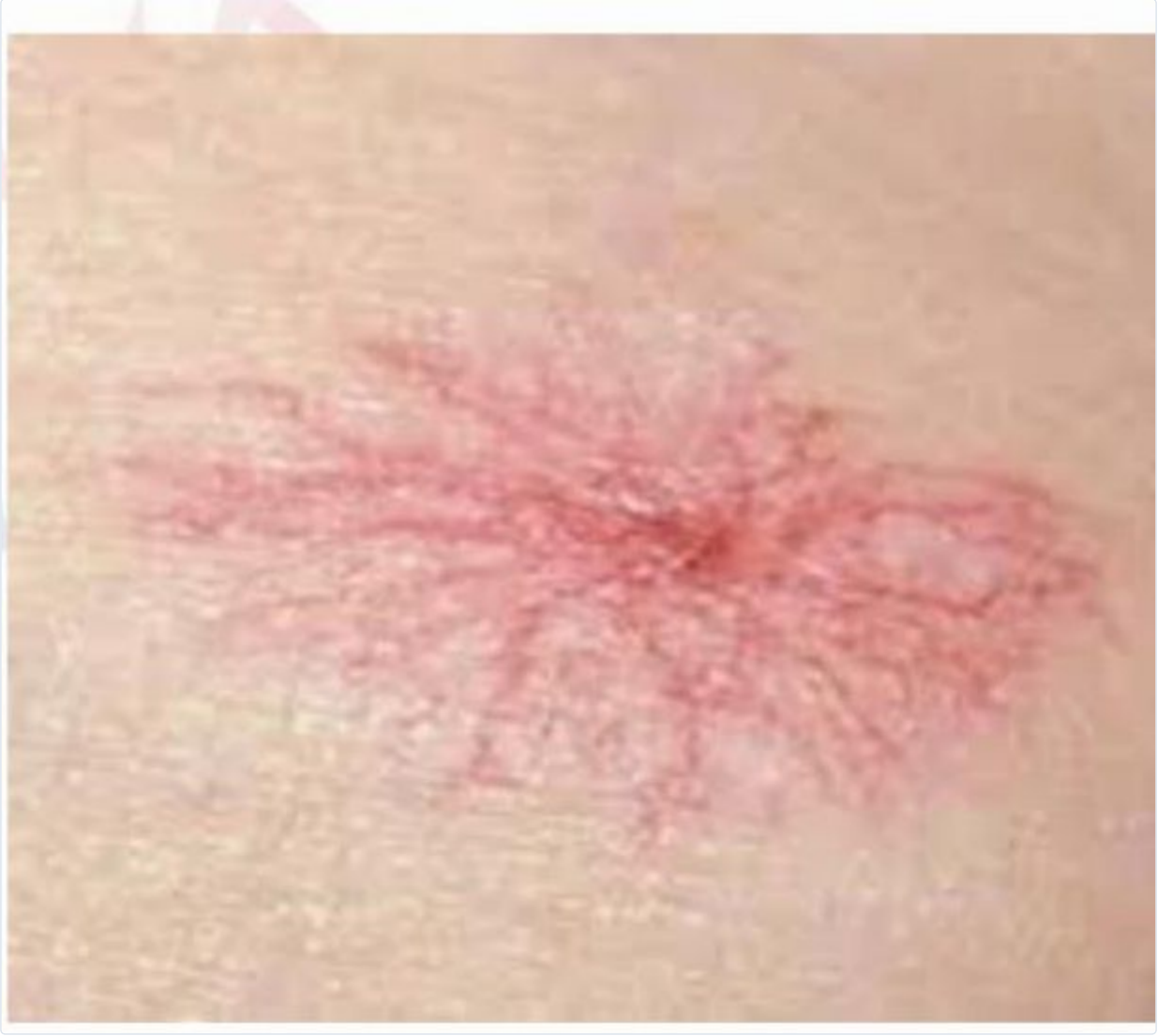

Fig. 3 — Spider naevus: a central arteriole with radiating vessels, a cutaneous stigma of liver disease.

2.4 Dental management of the hepatic patient

KEY POINTS

Cross-infection precautions; coagulation screen; take additional precautions against post-operative bleeding; avoid GA as far as possible; and if surgery is required the patient may need **vitamin K for several days beforehand**.

§3 Diabetes mellitus

Diabetes is the first of the endocrine problems relevant to dentistry. The two types differ across every axis:

Table 3 — Type I versus Type II diabetes

	Type I	Type II
Insulin	Insulin dependent	Insulin independent
Onset	Fast	Gradual
Age	Young	Old
Cause	Genetic cause or viral infection	May be due to therapeutic cortisone

The underlying hormone pair is simple to state: **insulin** converts plasma glucose to glycogen stored in the liver, while **glucagon** converts liver-stored glycogen back to glucose.

Oral manifestations of diabetes: dry mouth, candida, burning mouth syndrome (BMS) and periodontal breakdown.

3.1 Management before dental work

KEY POINTS

Antibiotic prophylaxis; interfere least with mealtimes — appoint ★**2 hours after breakfast**; instruct the patient to take their medication and food as normal; in case of GA, medication stops the night before surgery; and keep in contact with the regional hospital diabetes team.

★ EXAM PEARL

The timing logic is the discriminator: medication and food continue *as normal* for routine dentistry (2 hours after breakfast puts the appointment at peak glucose cover), but for GA the medication **stops the night before**. The exam likes to swap these two rules.

§4 Adrenal, thyroid and parathyroid disorders

4.1 Cortisone level disturbance: Cushing versus Addison

Table 4 — Cushing versus Addison

	Cushing	Addison
Plasma cortisone	High	Low
Primary condition	Therapeutic steroids or adenoma — **Cushing syndrome**	Adrenal cortex atrophy
Secondary condition	High ACTH level — **Cushing disease**	Low ACTH level
Common features	Obesity (buffalo hump, moon face), osteoporosis, skin thinning, hypertension	Hyperpigmentation
Dental relevance	—	May need cortisone coverage before surgery

★ EXAM PEARL

Cushing **syndrome** = primary (therapeutic steroids or adrenal adenoma); Cushing **disease** = secondary to high ACTH. One word changes the diagnosis — a favourite single-best-answer switch.

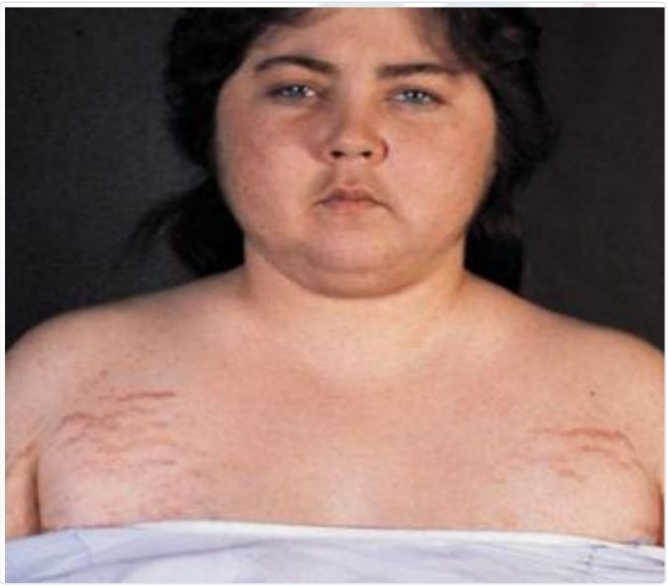

Fig. 4 — Cushing: moon face with truncal obesity and striae.

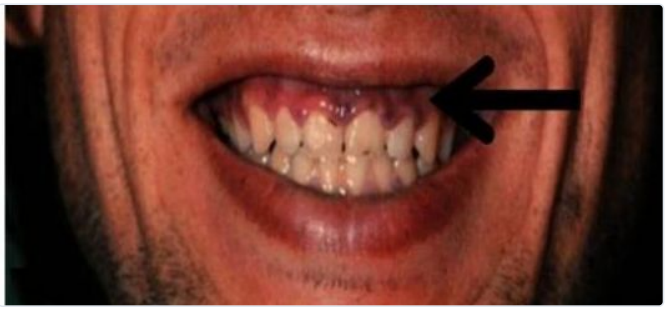

Fig. 5 — Addison: gingival hyperpigmentation (arrow) — the oral sign of low cortisone.

4.2 Addison disease and Addisonian crisis

Addison's disease symptoms usually develop slowly, often over several months — frequently so slowly that they are ignored until a stress such as illness or injury occurs and makes them worse. Signs and symptoms may include: extreme fatigue; weight loss and decreased appetite; darkening of the skin (hyperpigmentation); **low blood pressure, even fainting**; salt craving; **low blood sugar (hypoglycaemia)**; nausea, diarrhoea or vomiting (gastrointestinal symptoms); abdominal pain; muscle or joint pains; irritability; depression or other behavioural symptoms; and body hair loss or sexual dysfunction in women.

CAUTION

Addisonian crisis — sometimes the signs appear suddenly. Acute adrenal failure can lead to life-threatening shock; seek emergency medical treatment for: low blood pressure; high potassium (hyperkalaemia) and low sodium (hyponatraemia); severe weakness; confusion; pain in the lower back or legs; severe abdominal pain, vomiting and diarrhoea leading to dehydration; reduced consciousness or delirium.

4.3 Thyroid hormone disturbance

Table 5 — Hyperthyroidism versus hypothyroidism

	Hyperthyroidism	Hypothyroidism
Causes	Graves disease (autoimmune); adenoma	Primary: Hashimoto disease (autoimmune). Secondary: pituitary gland dysfunction with under-production of TSH
Features	Weight loss, heat intolerance, sweating, tachycardia, exophthalmos	Weight gain, cold intolerance, bradycardia, hoarse voice, loss of hair, loss of appetite, loss of memory

CAUTION

★**Avoid epinephrine in uncontrolled hyperthyroid cases only** — a controlled patient can receive adrenaline-containing local anaesthetic as normal.

4.4 Parathyroid hormone disturbances

Table 6 — Hyperparathyroidism versus hypoparathyroidism

	Hyperparathyroidism	Hypoparathyroidism
Causes	Primary: adenoma. Secondary: low plasma calcium. Tertiary: follows on from secondary	Primary: autoimmune. Secondary: surgery to the thyroid gland
Features	Bone resorption, brown tumours of long bones, high serum calcium, renal stones, peptic ulcers	Tetany of facial muscles, Chvostek sign, delayed tooth eruption, enamel hypoplasia

★ EXAM PEARL

The dental features sit on the **hypo** side: delayed tooth eruption and enamel hypoplasia belong to hypoparathyroidism, while **brown tumours** belong to hyperparathyroidism. Chvostek sign (facial twitch on tapping) confirms the tetany of low calcium.

§5 MRONJ — the disease and the drugs

This section follows the SDCEP guidance *Oral Health Management of Patients Prescribed Bisphosphonates* (dental clinical guidance 2017, reviewed March 2024).

5.1 Definition and presentation

KEY POINTS

Medication-related osteonecrosis of the jaw (MRONJ) is a rare side effect of anti-resorptive and anti-angiogenic drugs. It is defined as **exposed bone, or bone that can be probed through an intraoral or extraoral fistula, in the maxillofacial region, persisting for more than eight weeks**, in a patient with a history of treatment with anti-resorptive or anti-angiogenic drugs, where there has been **no history of radiation therapy to the jaw and no obvious metastatic disease to the jaws**.

Signs and symptoms: delayed healing following a dental extraction or other oral surgery; pain; soft tissue infection and swelling; numbness; paraesthesia; exposed bone. Patients may also complain of pain or altered sensation in the absence of exposed bone — and some patients may be asymptomatic.

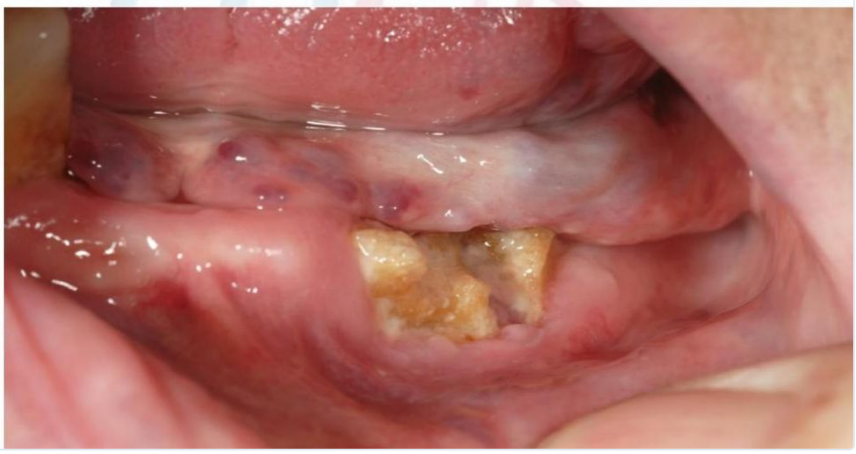

Fig. 6 — MRONJ: exposed necrotic bone in the mandible with surrounding soft tissue inflammation.

5.2 Bisphosphonates

Bisphosphonates are used most commonly in the management of osteoporosis but also in many other non-malignant and malignant conditions:

Table 7 — Conditions that may be treated with bisphosphonates

Non-malignancy	Malignancy
Osteoporosis	Multiple myeloma
Paget's disease	Breast cancer
Osteogenesis imperfecta	Prostate cancer
Fibrous dysplasia	Bony metastatic lesions
Primary hyperparathyroidism	Hypercalcaemia of malignancy
Cystic fibrosis	

In children, bisphosphonates are used for osteogenesis imperfecta, fibrous dysplasia, neuromuscular disorders, bone dysplasia, idiopathic juvenile osteoporosis, rheumatologic disorders and Crohn's disease; children on anti-resorptive drugs are likely to be managed by specialists in paediatric dentistry.

How they work. Bisphosphonates inhibit bone resorption and decrease bone turnover by hindering the formation, recruitment and function of osteoclasts, disrupting osteoclast-mediated resorption through inhibition of osteoclastic activity and apoptosis of osteoclasts. The result is production of dense bone by osteoblasts and the loss of normal bone physiology. They inhibit the enzymes essential to osteoclast formation, recruitment and function, have a **high affinity for hydroxyapatite** and persist in skeletal tissue for a significant period — ★**alendronate has a half-life in bone of around 10 years** — though it is unclear how this influences MRONJ risk once the drug is stopped. Bisphosphonates may also adversely affect soft tissue cells by inhibiting proliferation and increasing apoptosis, leading to delayed soft tissue healing, and there is some evidence they inhibit angiogenesis. They are used to reduce the

symptoms and complications of metastatic bone disease (particularly breast cancer, prostate cancer and multiple myeloma), usually delivered as regular high-dose intravenous infusions in that group.

Why the jaws? Bisphosphonates are not metabolised, and high concentrations are maintained within bone for long periods. They accumulate at sites of high bone turnover — and turnover in the maxilla and mandible is relatively high (★**10 times that of long bones and 5 times that of basal mandibular bone**), leading to greater accumulation there. These areas are easily traumatised by dental procedures, dentures or eating, allowing infection to enter; the reduced bone turnover and bone blood supply can then lead to death of the bone — osteonecrosis (BRONJ). The **mandible is a favoured site**.

Causes of the necrosis are likely multi-factorial, with both genetic and immunological elements: suppression of bone turnover; inhibition of angiogenesis; toxic effects on soft tissue; and inflammation or infection.

> **CAUTION**
> Advise patients prescribed an **oral** bisphosphonate not to hold the tablet in the mouth — direct mucosal contact risks damage to the oral mucosa.

Table 8 — Bisphosphonate drugs (three most commonly prescribed listed first)

Drug name	Trade name(s)	Indication
Alendronic acid	Binosto, Fosamax, Fosavance	Osteoporosis
Risedronate sodium	Actonel, Actonel Combi	Osteoporosis; Paget's disease
Zoledronic acid	Aclasta, Zometa	Osteoporosis; Paget's disease; treatment of cancer
Ibandronic acid	Bondronat, Bonviva, Iasibon, Quodixor	Osteoporosis; treatment of cancer
Pamidronate disodium	Aredia	Paget's disease; bone pain; treatment of cancer
Sodium clodronate	Bonefos, Clasteon, Loron	Bone pain; treatment of cancer

5.3 RANKL inhibitors and anti-angiogenic drugs

Denosumab (trade names Prolia and Xgeva; indications osteoporosis and treatment of cancer) is a fully human monoclonal antibody that inhibits osteoclast function and the associated bone resorption by binding to the receptor activator of nuclear factor κB ligand (RANKL). It is administered subcutaneously every six months in osteoporosis, with a higher dose given monthly in metastatic disease. Crucially, denosumab **does not bind to bone**, and its effects on bone turnover ★**diminish within nine months of treatment completion**.

Anti-angiogenic drugs target the processes by which new blood vessels form and are used in cancer treatment to restrict tumour vascularisation. Not all are implicated in MRONJ, but the vascular endothelial growth factor (VEGF) inhibitors **bevacizumab** (Avastin) and **aflibercept** (Zaltrap) and the receptor tyrosine kinase (RTK) inhibitor **sunitinib** (Sutent) have been associated with osteonecrosis of the jaw — all indicated for treatment of cancer. Anti-angiogenics can be combined with bisphosphonates in cancer management, and there is some evidence this produces a greater MRONJ risk; the same may be true where anti-angiogenic drugs are used in patients with a previous history of bisphosphonate use.

Duration of effect. Bisphosphonates remain in the body long after the patient stops taking them, so a past user is allocated to a risk group as if still taking the drug. Denosumab's effect diminishes within nine months of completion, and anti-angiogenic drugs are not thought to remain in the body for extended periods.

> **★ EXAM PEARL**
> Contrast pair the exam loves: alendronate binds hydroxyapatite and persists ~**10 years** in bone; denosumab does not bind bone and washes out in **9 months**. This single difference drives the risk-allocation rules and the denosumab timing strategy in §6.

§6 MRONJ — risk assessment and dental management

6.1 How big is the risk?

Patients are not discouraged from taking bisphosphonate drugs or from undergoing dental treatment. The estimated incidence of MRONJ in **cancer** patients treated with anti-resorptive or anti-angiogenic drugs is ★**1% (1 case per 100)**; in **osteoporosis** patients treated with anti-resorptive drugs it is ★**0.01–0.1% (1–10 cases per 10,000)**. After tooth extraction, the incidence rises to ★**2.9% in cancer patients and 0.15% in osteoporosis patients**.

6.2 Risk categories

Table 9 — Patient risk categories (SDCEP Table 3.1); any one criterion is sufficient

Low risk	Higher risk
Treated for osteoporosis or other non-malignant bone disease (e.g. Paget's) with **oral bisphosphonates for less than 5 years**, **not** concurrently on systemic glucocorticoids	Treated for osteoporosis or non-malignant bone disease with oral bisphosphonates or quarterly/yearly IV bisphosphonate infusions for **more than 5 years**
Quarterly or yearly **IV bisphosphonate infusions for less than 5 years**, **not** concurrently on systemic glucocorticoids	Bisphosphonates or denosumab for **any length of time** while concurrently treated with **systemic glucocorticoids**
Treated with **denosumab**, **not** on systemic glucocorticoids	Anti-resorptive or anti-angiogenic drugs (or both) as part of the **management of cancer**
	Previous diagnosis of MRONJ

N.B. Patients who have taken bisphosphonates at any time in the past, and those who have taken denosumab in the last nine months, are allocated to a risk group as if they are still taking the drug.

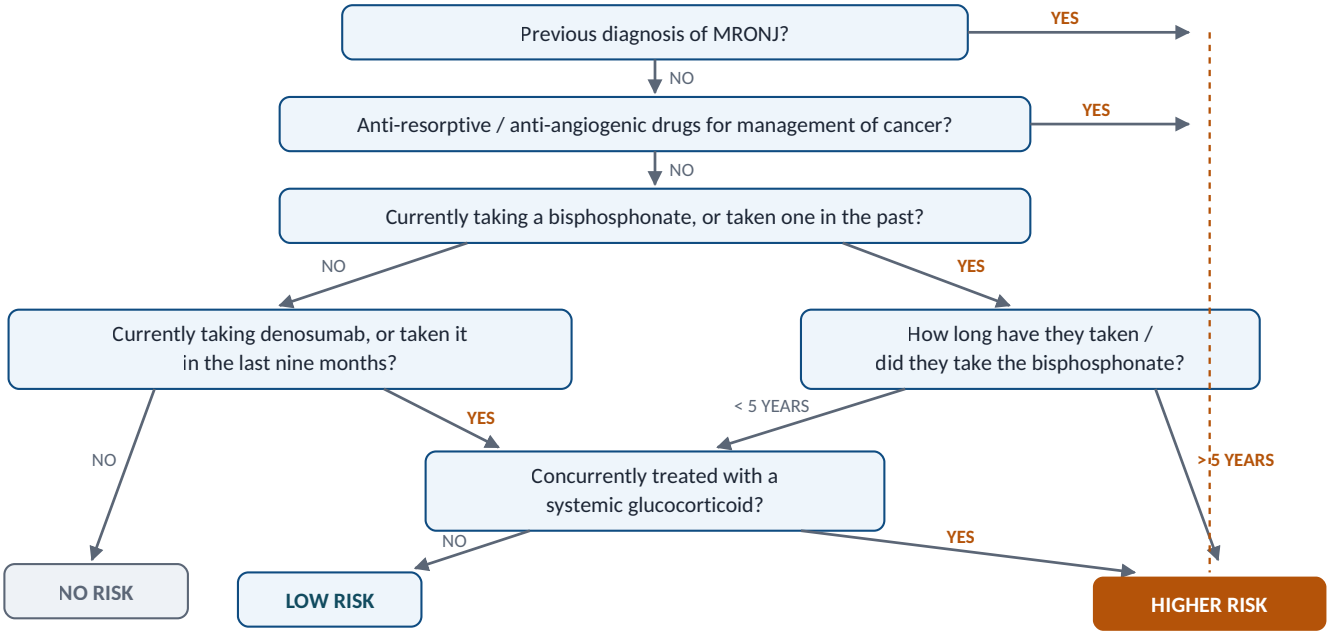

Diagram 2 — MRONJ risk assessment when taking or confirming a medical history (redrawn from SDCEP Figure 3.1). Any YES on the right-hand path leads to Higher Risk.

6.3 Oral and dental initial care

At the initial consultation, assess the patient's level of MRONJ risk, assign and **record a risk category**, and advise the patient that they are at risk of MRONJ — emphasising that the risk is small and discussing what they can do to reduce it (improving oral hygiene, reducing sugary snacks and drinks, limiting alcohol intake, stopping smoking). The programme when a patient starts (or is already on) bisphosphonate therapy:

1. Try to get the patient **as dentally fit as possible before commencement** of bisphosphonate therapy, or as soon as possible after.
2. Consider obtaining appropriate radiographs to identify possible areas of infection and pathology.
3. Prioritise care that will reduce mucosal trauma and avoid subsequent extractions or any oral surgery or procedure that may impact on bone. Carry out any remedial dental work, try to reduce periodontal/dental infection or disease, and adjust or replace poorly fitting dentures to minimise future mucosal trauma. Consider prescribing high-fluoride toothpaste.
4. Give preventive advice, emphasising: a healthy diet and reducing sugary snacks and drinks; maintaining excellent oral hygiene; using fluoride toothpaste and fluoride mouthwash; stopping smoking; limiting alcohol intake; regular dental checks; and reporting any symptoms — exposed bone, loose teeth, non-healing sores or lesions, pus or discharge, tingling, numbness or altered sensations, pain or swelling — as soon as possible.
5. Patients should have routine treatment for scale and polish, simple restorations, regular recall and radiological review.

6. **Orthodontic note:** orthodontic tooth movement during bisphosphonate therapy is possible but unpredictable, especially in low-risk patients. Bisphosphonate use is associated with longer orthodontic treatment times among extraction patients, increased odds of poor space closure, and increased odds of poor root parallelism.
7. If a patient has spontaneous or chronic bone exposure, refer to an oral surgery / oral and maxillofacial surgery (OS/OMFS) specialist.
8. An exception: consider removal of teeth of poor prognosis if this will avoid extractions or other bone-impacting treatments later during the patient's bisphosphonate therapy.

6.4 Treatment by risk group

Table 10 — Managing bone-impacting procedures by risk group

Low risk	Higher risk
Perform straightforward extractions and procedures that may impact on bone **in primary care**	**Explore all possible alternatives to extraction** where teeth could potentially be retained, e.g. retaining roots in the absence of infection
Do not prescribe antibiotic or antiseptic prophylaxis unless required for other clinical reasons	If extraction remains the most appropriate treatment, **proceed as for low-risk patients**

Low-risk patients in detail. Having made the patient as dentally fit as feasible, carry out all routine dental treatment as normal and continue personalised preventive advice. If an extraction or another bone-impacting procedure is required: discuss the risks and benefits with the patient (or carer, where appropriate) to ensure valid consent, and proceed as clinically indicated; do not prescribe antibiotic or antiseptic prophylaxis unless required for other clinical reasons; advise the patient to contact the practice if they have any concerns such as unexpected pain, tingling, numbness, altered sensation or swelling in the extraction area; and **review healing at 8 weeks** after any invasive treatment. If the extraction socket is not healed at ★**8 weeks** and you suspect MRONJ, refer to an oral surgery / special care dentistry specialist as per local protocols. Consider periodontal surgery in lower-risk cases to eliminate disease, but **non-surgical periodontal treatment only** should be considered in higher-risk cases. New dentures should be reviewed in at-risk patients to confirm comfort and stability, and soft linings can be considered in higher-risk patients.

Higher-risk patients: aim to avoid bone-impacting procedures by considering other treatment options; seek advice from an OS/OMFS specialist, **preferably by letter**, about whether to treat the patient in primary care for any extraction, oral surgery or bone-impacting procedure, or whether to refer. Medically complex patients merit specialist advice on clinical assessment, treatment planning and ongoing management. If you suspect spontaneous MRONJ, refer as per local protocols — refer **any** patient with evidence of spontaneous MRONJ. Consider reporting suspected MRONJ cases to the MHRA via the Yellow Card Scheme (www.yellowcard.mhra.gov.uk) and encourage the patient to do likewise.

6.5 Important rules

> **CAUTION**
>
> **Do not prescribe antibiotic or antiseptic prophylaxis** following extractions or other bone-impacting treatments *specifically to reduce the risk of MRONJ*. Routine restorations are not contraindicated, nor are superficial scaling and polishing. Endodontic treatment to retain teeth instead of extraction is indicated.

Drug holidays do not help. There is no supporting evidence that BRONJ risk is reduced if the patient temporarily, or even permanently, stops taking bisphosphonates before invasive dental procedures, since the drugs may persist in skeletal tissue for years. The denosumab exception is a matter of timing rather than cessation: osteoporosis patients on six-monthly subcutaneous denosumab can **delay non-urgent invasive dental treatment of an asymptomatic tooth until the month prior to the next scheduled drug administration**; resumption of denosumab should then be delayed until the soft tissues / extraction socket have healed. Close liaison with the patient's medical practitioner is necessary to coordinate this.

Allocating past users: if a patient has taken anti-resorptive drugs in the past but is no longer taking them (completed or discontinued the course, or on a drug holiday) — a past bisphosphonate user is allocated as if still taking the drug; a denosumab user within the past nine months likewise. Patients who previously took anti-angiogenic drugs **in combination with** anti-resorptives are allocated based on their anti-resorptive history. Patients may not know their medication is a bisphosphonate, but if they have one of the conditions listed in Table 7 then — in the United Kingdom — they are likely to have been prescribed one.

6.6 Investigations and the latent phase

Panoramic and periapical radiographs may show a **dense lamina dura**; 3D CT scans add detail. There is a phase called the **latent phase**, when the bone is not yet exposed or necrotic but the bisphosphonate effects are slowly affecting the jaws — at this stage

sclerotic bone and widened lamina dura may be visible, which could indicate a predisposition for BRONJ. Currently, newer investigations such as ★**CTX markers** are of higher value.

6.7 Dental implants

Inform patients with dental implants placed **prior to** commencement of anti-resorptive or anti-angiogenic drugs of the small risk of spontaneous MRONJ at those sites, and provide information on minimising the risk — for example, ensuring excellent oral hygiene at the implant site(s). Inform osteoporosis patients who are **considering** dental implants during or after anti-resorptive treatment of the risk of compromised bone healing and MRONJ following the procedure, and the additional small risk of long-term implant failure; provide those who choose to proceed with the same risk-minimisation information.

§7 Cardiovascular disease

7.1 Hypertension — the silent killer

Hypertension is consistently raised blood pressure: ★**above 140 systolic or above 90 diastolic**. Mean arterial pressure is calculated as MAP = ((2 × diastolic) + systolic) / 3. In 95% of cases there is no definable cause; the remaining 5% are secondary to other diseases. Risk factors are age, family history, obesity and inactivity. The mechanism operates through increases in cardiac output (COP), total peripheral resistance (TAR), blood volume and heart rate; complications include ischaemic heart disease, cerebrovascular accident, renal failure, atherosclerosis and heart attacks.

Table 11 — Antihypertensive drugs and their oral manifestations

Class (example)	Action	Oral manifestations
Beta blockers (propranolol)	Decrease cardiac output	Dry mouth, BMS, lichenoid reaction
Calcium-channel blockers (nifedipine)	Minimise calcium influx into smooth muscle and heart	Dry mouth, BMS, lichenoid reaction, **gingival hyperplasia**
Diuretics (hydrochlorothiazide)	Reduce blood volume	Dry mouth, BMS, lichenoid reaction
ACE inhibitors (captopril)	Vasodilator	Dry mouth, BMS, lichenoid reaction, **angioedema**

★ EXAM PEARL

All four classes share dry mouth, BMS and lichenoid reactions — the discriminators are the extras: **gingival hyperplasia → calcium-channel blockers**; **angioedema → ACE inhibitors**.

CAUTION

Before dental work, ★**180/110 is the absolute cut-off**. In an emergency where treatment cannot wait and the BP is above the cut-off, use **5 mg diazepam**.

7.2 Angina pectoris, myocardial infarction and cardiac failure

Angina pectoris is a partial obstruction of a coronary artery: chest pain at the left side referring to the left shoulder and the left side of the jaw. Initially the attack takes place only with effort; with time, attacks continue even at rest. GA and sedation are risky.

Myocardial infarction (MI) follows rupture of the fibrous cap covering an atherosclerotic plaque; the exposed plaque triggers platelet adhesion and aggregation to form a thrombus. ★**Delay any surgical procedures at least 6 months after the last attack.**

Cardiac failure is the end point of cardiovascular diseases: the heart becomes unable to pump sufficiently to meet the needs of the body, producing peripheral oedema and breathlessness.

§8 Gastrointestinal disorders

8.1 Peptic ulcer, GORD and antacids

In **peptic ulcer** disease, avoid NSAIDs — **only paracetamol is safe** as an analgesic. The patient is usually treated with a proton pump inhibitor (omeprazole), amoxicillin and metronidazole (triple therapy), so avoid co-prescription. Omeprazole itself causes dry mouth and erythema multiforme (EM), and increases the effect of warfarin and diazepam.

Gastro-oesophageal reflux disease (GORD) is regurgitation of gastric acids causing heartburn and oesophagitis. In the mouth it causes palatal erosion, mucosal irritations and ulcers. Risk factors: pregnancy, obesity, fatty foods, smoking, alcohol and hernia. **Avoid night guards** in these patients — an appliance worn overnight pools refluxed acid against the teeth.

Antacids are used for GORD and peptic ulcers; instruct the patient to maintain them in the mouth for a few minutes to counteract acidity due to gastric fluids. Their dental side effect is pharmacokinetic: they interfere with the absorption of **fluoride, azole agents and tetracycline**.

8.2 Coeliac disease

The coeliac patient reacts to gluten, the protein in wheat. Features are the general features of anaemia plus oral ulceration, and the disease **may cause intestinal lymphoma**. Management is dietary withdrawal of gluten.

> ★ EXAM PEARL
> The slide advises withdrawing "wheat and rice" from the diet — **rice is gluten-free** and does not need to be withdrawn. Gluten is found in wheat, barley and rye; rice is in fact a staple of the gluten-free diet. Verify against your textbook before the exam.

8.3 Crohn's disease, IBD and IBS

Crohn's disease is a granulomatous inflammation of the gastrointestinal tract, especially the ileal region. Malabsorption problems affect iron, vitamin B12 and folic acid, producing general manifestations of anaemia, with abdominal pain. The oral manifestations are distinctive: **mucosal tags with a cobblestone appearance**, diffuse lip and cheek swelling, and recurrent aphthous ulceration (RAU). It is treated with cortisone.

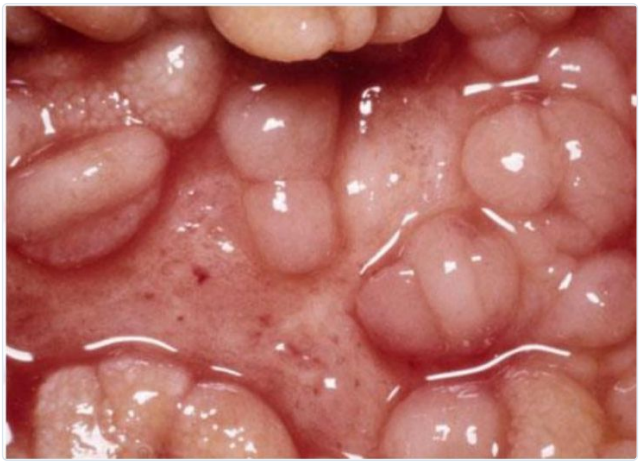

Fig. 7 — Crohn's disease: cobblestone appearance of the oral mucosa with mucosal tags.

Inflammatory bowel disease (IBD) was presented as an inflammatory condition of the small bowel combining Crohn's disease and ulcerative colitis: it shows the manifestations of Crohn's disease together with those of ulcerative colitis (chronic bleeding), and is treated with cortisone.

Irritable bowel syndrome (IBS) is not a true disease but a set of signs and symptoms: abdominal pain, cramps and excess gases, and constipation or diarrhoea. Its dental relevance is functional: psychogenic orofacial pain disorders, clenching and bruxism, and teeth attrition with wear facets.

8.4 Dysphagia and Plummer–Vinson syndrome

Table 12 — Causes of dysphagia

Oral causes	Systemic causes
Major aphthous ulceration (MAU)	Plummer–Vinson syndrome
Stomatitis	Oesophageal carcinoma
Tonsillitis	Foreign body
Herpetic infection	Benign stricture
Oral malignancy	
Dry mouth	
Pharyngitis	

Plummer–Vinson syndrome — also known as Brown Kelly Paterson syndrome — is ★**premalignant**: the combination of **iron deficiency anaemia and a post-cricoid web**.