Gastrointestinal and Hepatic Diseases in Dentistry

by Dr David Lim

21 Jul 2026 (Tuesday) 10-11

IMPT!

Learning Objectives

1. Discuss the specific characteristics and dental needs of individuals with common gastrointestinal and hepatic disease (e.g. GORD, Crohn's disease, ulcerative colitis, viral hepatitis, liver cirrhosis, malnutrition)

2. Perform a risk assessment and make appropriate treatment adaptations to ensure safe and effective dental care.

ADC: 5.1	iADH: 5A, 5B, 5C, 6Ci	5.1	Scientific and Clinical Knowledge	apply the social, cultural, biological, biomedical, physical and behavioural sciences in relation to oral health care provision and disease prevention

GORD

Gastro-oesophageal reflux disease (GORD) is a chronic condition in which gastric contents reflux into the oesophagus, causing troublesome symptoms and/or complications. Reflux may contain hydrochloric acid, pepsin, bile salts and pancreatic enzymes that damage the oesophageal mucosa.

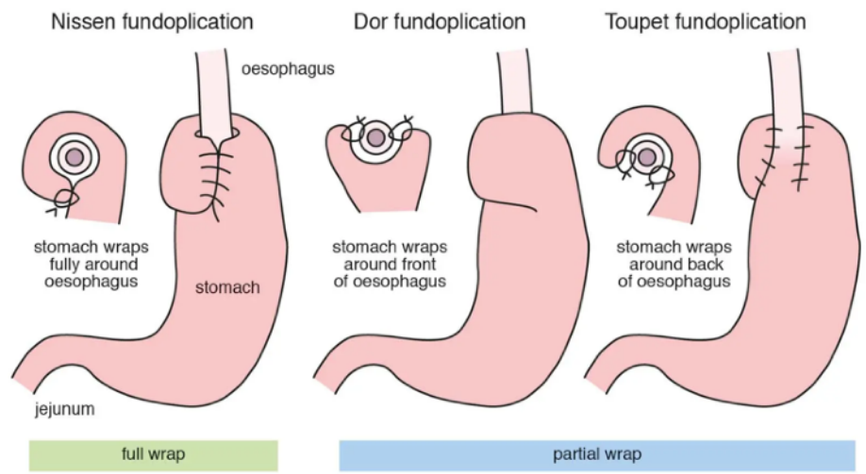
AETIOLOGY/RISKS
Dysfunctional lower oesophageal sphincter, hiatus hernia, obesity, pregnancy, smoking, alcohol consumption, high fat meals, certain medications

EPIDEMIOLOGY:
Worldwide

- Affects approximately **10–20%** of adults in Western countries.
- Prevalence is increasing globally due to rising obesity rates and ageing populations.

Australia

- Approximately **1 in 5 Australians** experience reflux symptoms at least weekly.
- One of the most common gastrointestinal disorders encountered in primary care.



tofs.org.uk

CLINICAL MANIFESTATIONS:
-TYPICAL - heartburn, acid regard, chest discomfort, sour or bitter taste
-OTHERS - Hoarseness, asthma exacerbation, chronic cough
-COMPLICATIONS (CX): Oesophagitis, Barrett's oesophagus, risk of Oesophageal adenocarcinoma

GENERAL MANAGEMENT:
1. Lifestyle Modifications: weight reduction, smoking cessation, avoid triggers, avoid eating 2-3hrs before bedtime
2. Medical Management: PPI, H2 receptor antagonists, antacids, etc
3. Surgical Management: Fundoplication for severe or refractory disease

GORD & Dentistry

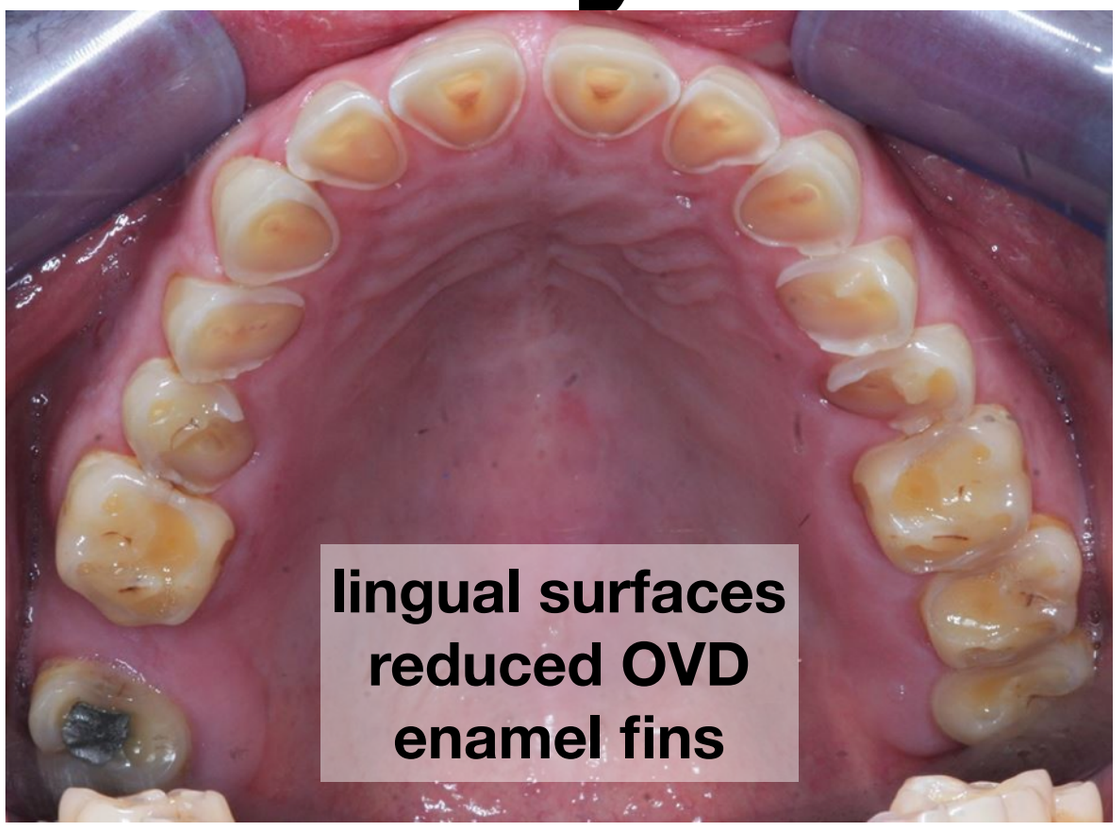
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ORAL MANIFESTATIONS:

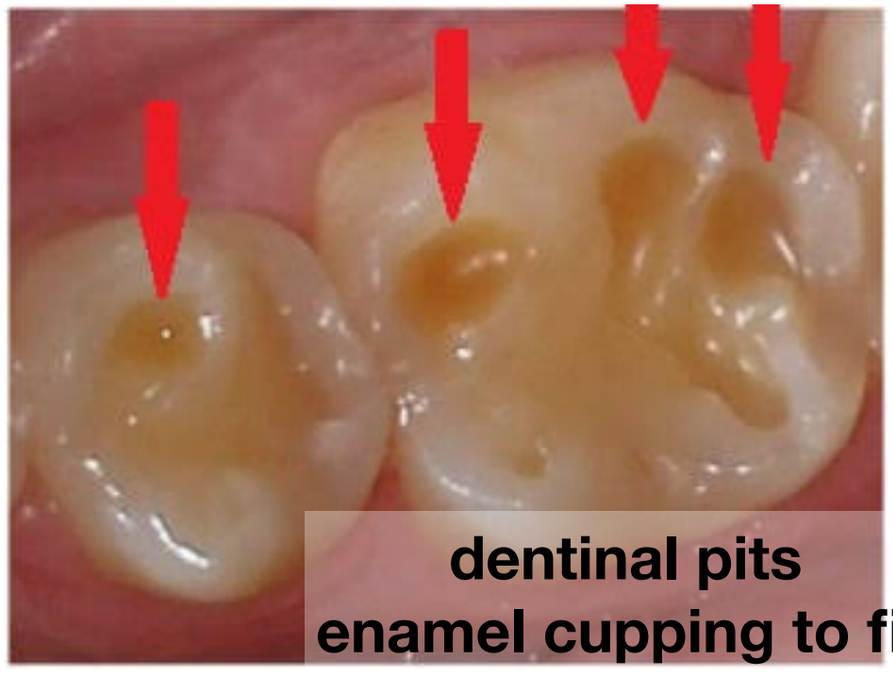
1. Erosion, sometimes on lingual, and related symptoms (hypersensitivity, reduced OVD)
2. Burning mouth

DENTAL MANAGEMENT:

1. REMINERALISE - 5000ppm, 22600ppm, CCP-ACP
2. RESTORATIVE ETC - full coverage, Essix trays, cover erosion pits
3. OTHER ADJUSTMENTS: sit chair up, rinse after reflux, general advise to reduce caries risk (brush, diet), avoid Bulimic habits
4. DRUGS: may prescribe COX-2 instead of NSAIDS, prescribe Antacide/PPI with these drugs
5. MEDICAL REFERRALS / MEDICATIONS



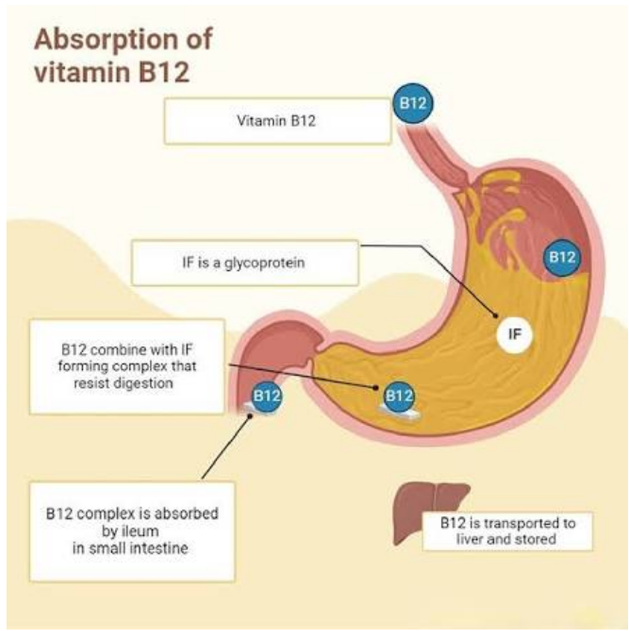
https://www.nature.com/articles/s41415-023-5677-0



https://allentxdentist.com/acid-reflux-disease-may-lead-to-erosion-of-teeth/

Crohn’s

Crohn's disease (CD) is a chronic inflammatory bowel disease (IBD) characterised by transmural (full-thickness) inflammation that can affect any part of the gastrointestinal tract, from the mouth to the anus. It most commonly involves the terminal ileum and proximal colon and follows a relapsing-remitting course.



https://scijournals.onlinelibrary.wiley.com/doi/10.1002/jsfa.13661

AETIOLOGY: is unknown, but Crohn's disease results from a combination of:

- Genetic susceptibility
- Immune dysregulation or Altered gut microbiota
- Environmental factors
    - Smoking (major risk factor)
    - Western diet
    - Certain infections

Worldwide Epidemiology

- Prevalence is increasing globally, particularly in industrialised countries.
- Most commonly diagnosed between **15 and 35 years** of age.
- A second smaller peak occurs after age 60.

Australia

- Australia has one of the highest incidences of IBD worldwide.

CLINCIAL MANIFESTATION

Gastrointestinal symptoms

- PAIN: Chronic diarrhoea, Abdominal pain with RECTAL BLEEDING
- MALNUTRITION: fatigue, reduced appetite, weight loss

Systemic manifestations

- Anaemia, Vitamin B12 deficiency, (Parietal cells destruction)
- Iron deficiency (bleeding, poor absorption)
- Osteoporosis
- Delayed growth in children

Extraintestinal manifestations

- Arthritis, Uveitis
- Erythema nodosum
- Primary sclerosing cholangitis (less common)

General Management

Medical therapy

- Corticosteroids (during flares)
- Immunomodulators (e.g., azathioprine, methotrexate)
- Biologic agents (e.g., anti-TNF therapies)
- Nutritional support
- Vitamin supplementation

Surgical management

Surgery is not curative but may be required for:

- Strictures
- Fistulas
- Abscesses
- Bowel obstruction
- Severe disease
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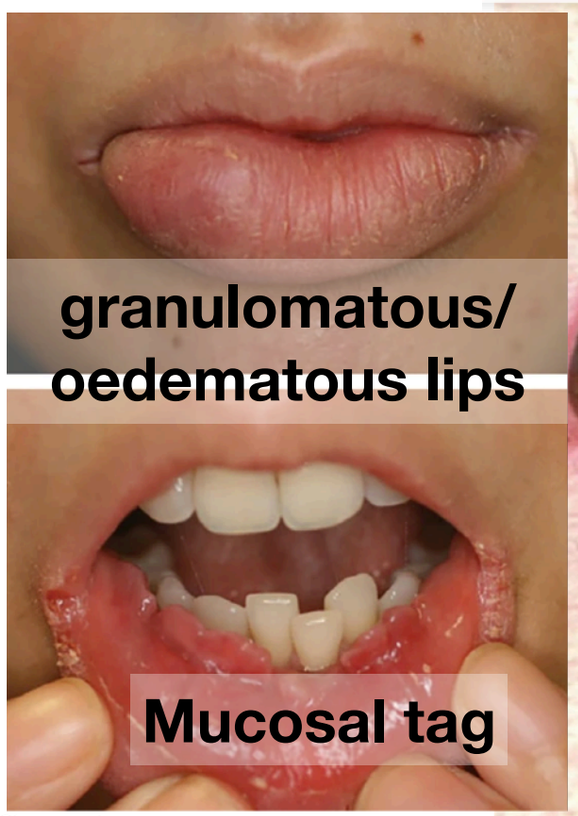
IBD & Dentistry

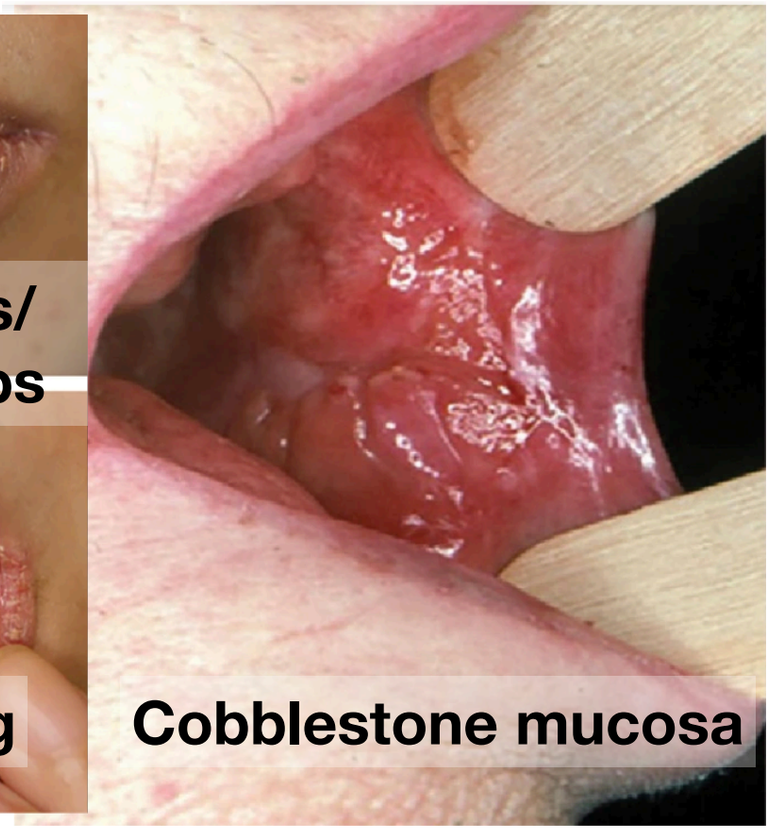
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ORAL MANIFESTATIONS:

1. Approximately **20–50%** of Crohn's patients develop oral manifestations, which may precede GI symptoms. **~5%** of UC patients have oral symptoms

2. Cobblestone mucosa, linear ulceration, mucosal tags (hyperplastic folds), granulomatous enlargement of lips, angular chelitis, recurrent aphthous-like ulcers, diffuse mucogingivitis, pyostomatitis vegetans





https://www.nature.com/articles/bdjteam201783

DENTAL IMPLICATIONS

ACCESS - schedule as appropriate (remission vs flare)

COMMUNICATION & CONSENT & SPREAD: nil

EDUCATION: manage ulcers (topical steroids, CHX, good OH)

SURGERY:
(pre) - check FBC for Hb, RBC, Steroid cover
(peri) - anaemia, thrombocytopenia (medications), neutropenia
(post) - prescription considerations (patients are on e.g. prednisolone, azathioprine, methotrexate infliximab etc)

Pyostomatitis vegetans

multiple small abscess on erythematous base





Ulcerative Colitis

Ulcerative colitis (UC) is a **chronic inflammatory bowel disease (IBD)** characterised by **continuous inflammation limited to the mucosa and submucosa of the colon and rectum**. Unlike Crohn's disease, ulcerative colitis affects **only the large intestine** and does **not involve transmural inflammation**. The disease follows a relapsing-remitting course.

AETIOLOGY: unknown, but UC is thought to result from a mix of Genetic/family, immune dysregulation, altered GI microbiota, environmental factors.

Epidemiology worldwide

- UC more common in industrialised countries, with millions worldwide affected.
- Most commonly diagnosed between **15 and 35 years** of age. A second smaller peak occurs after age 60.

Australia

- Australia has one of the highest incidences of inflammatory bowel disease worldwide.
- More than **180,000 Australians** are estimated to be living with IBD

Clinical Manifestations

- **Gastrointestinal symptoms : BLOODY DIARRHOEA, rectal bleeding, urgency to deface cramping**
- **Systemic manifestations: fatigue, anaemia, malnutrition, fever**
- **Extraintestinal manifestations**

General Management

Medical therapy

- Aminosalicylates (e.g. mesalazine)
- Corticosteroids (during flares)
- Immunomodulators (e.g., azathioprine, methotrexate)
- Biologic agents (e.g., infliximab)

Surgical management

- Total colectomy may be indicated for severe/refractory colitis, colorectal cancer, toxic megacolon, or perforation
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Comparison: Crohn's Disease vs Ulcerative Colitis (Dental Relevance)

Feature	Crohn's Disease	Ulcerative Colitis
Gastrointestinal involvement	Anywhere from mouth to anus	Colon and rectum only
Depth of inflammation	Transmural (full thickness)	Mucosa and submucosa only
Oral manifestations	Common (20–50%); may precede GI disease	Less common (5–10%)
Characteristic oral lesions	Cobblestone mucosa, linear ulcers, mucosal tags, lip swelling	Aphthous ulcers, **pyostomatitis vegetans**
Surgery	Not curative	Colectomy is generally curative for colonic disease
Dental considerations	Oral lesions may be the first sign of disease	Monitor for aphthous ulcers and medication-related complications

ChatGPT - only partially correct

Liver cirrhosis

Liver cirrhosis is the irreversible loss of liver structure due to necrosis and fibrosis. This results in increased resistance to portal blood flow (portal hypertension) and impaired liver function.

AETIOLOGY/RISKS
- In high income countries, Hepatitis C virus infection, alcohol abuse (and increasingly non-alcoholic fatty liver disease)
- In sub-Saharan Africa and most of Asia, it's Hepatitis B virus infection
- Involves inflammation, hepatic stellate cell activation, destruction of parenchyma as a result of vascular occlusion

EPIDEMIOLOGY:
Worldwide

- 185 million globally, HBV as leading ethology.
- HCV, alcohol, NAFLD dominates Western countries. HBV in Asia-Pacific
- Decompensation increase 1 year mortality by 80%

CLINICAL MANIFESTATIONS:
-Disease progress from (1) compensated (usually asymptomatic) to (2) decompensated stages. Clinical decompensation occurs due to events like ascites, sepsis (peritonitis), haemorrhage (oesophageal varicose), non-obstructive jaundice.
-GENERAL FINDINGS: fatigue, loss of appetite and weight, muscle wasting, disturbed sleep
- OTHER FINDINGS: Leuconychia, palmar erythema, dupuytren contractures, splenomegaly etc
COMPLICATIONS (CX): Hepatocellular Carcinoma, varicella bleeding (from portal hypertension), hepatic encephalopathy (from hyperammonemia)

GENERAL MANAGEMENT:
1. Lifestyle Modifications: alcohol abstinence, NAFLD prevention
2. Medical Management: antiviral therapies for HCV/HBV, Nutritional supplements, low sodium low protein diet, and prevention of other symptoms
3. Surgical Management: Liver transplant (for end-stage)

https://www.sciencedirect.com/science/article/pii/S2949752326000154

Case history

A woman, 37 years of age, has a mechanical mitral valve and is in atrial fibrillation. She has been on a daily dose of 7 mg of warfarin for 13 months with a stable INR of 3. She smokes 15–20 cigarettes per day and has a body mass index (BMI) of 31. She requires the removal of one molar tooth. Her GP directs her to stop warfarin for 3 days before the extraction. The tooth is removed and the warfarin recommenced 8 hours later. That evening she has a stroke that leaves her with a dense, right sided hemiplegia and expressive aphasia.

Warfarin was approved for human use in 1954. It is the twentieth most frequently prescribed medication in Australia with approximately 80 000 prescriptions dispensed each month.[1,2] General practitioners are wary of the haemorrhagic potential of 'rat poison' and probably measure their patients' international normalised ratios (INR) too frequently.[3] Paradoxically, doctors and dentists are less aware of the greater risk of stroke when they stop a patient's warfarin before uncomplicated dental extraction.

Wafarin - stop?

- Stopping warfarin in high risk patients before endoscopy has been shown to result in a stroke rate of 3%.

- "not necessary, and indeed unwise, to cease warfarin prophylaxis for patients whose INR is within the normal therapeutic range of 2–4"

https://www.racgp.org.au/getattachment/b4155f9d-f3b8-4ff4-a554-817d933ae5c7/attachment.aspx

Alert Box 3.1 Platelet count values and restrictions/modifications for dental treatment.*

Risk level	Platelet count (cells/mm³)	Medical consultation recommended?	Interventions
Low	>100,000	No	None
Moderate	50,000–100,000	Yes	Consider localized measures
High	20,000–50,000	Yes	Localized measures indicated, consider platelet transfusion
Panic	<20,000	Yes	Requires platelet transfusion

*For procedures in which bleeding is anticipated.

Therapeutic indication	Medication class	Examples	Mechanism	Dose modifications?	Recommended diagnostic test(s)	Implications for dental care
Antithrombotic	Salicylates	Acetasylic acid	Irreversibly inhibits prostaglandin synthesis	None	None	Potential delay in *primary* hemostasis
	NSAIDs	Ibuprofen	Reversibly inhibits prostaglandin synthesis	None	None	Anticipate *early* oozing/bleeding
	Platelet aggregation inhibitors	Clopiogrel Prasugrel Ticlopidine	Inhibits ADP-induced platelet aggregation	None	None	Local measures only
		Dipyridamole	Inhibits thromboxane synthase, inhibits cellular reuptake of adenosine, inhibits adenosine deaminase and phosphodiesterase		None	
		Cilostazol	Inhibits cellular phosphodiesterase		None	
	Antiplatelet agents	Anagrelide	Reduces platelet count through inhibition of cyclic AMP and phosphodiesterase III	None	None	
Anticoagulant	Low-molecular weight heparins	Enoxaparin Dalteparin Tinzaparin	Binds to and increases activity of antithrombin III, inhibiting thrombin and Factor Xa	None	None	Potential delay in *secondary* hemostasis
	Coumarins	Warfarin	Inhibits vitamin K–dependent coagulation factor synthesis (II, VII, IX, X, proteins C and S)	None	PT/INR	Anticipate *late* oozing/bleeding
	Direct thrombin inhibitors	Dabigatran etexilate	Inhibits thrombin, blocks fibrin formation	None	None	Local measures only
	Factor Xa inhibitors	Rivaroxaban	Blocks active site of factor Xa	None	None	

https://pocketdentistry.com/3-potential-for-bleeding/

more details in lecture " Haematological conditions"…



https://www.sciencephoto.com/media/506683/view/bruising-of-the-mouth

LOCAL HAEMOSTATIC MEASURES

1. LA infiltration in palate/vestibule gently
2. Manipulate/retract gently
3. less-traumatic- avoid gingival tears, chop teeth rather than bone
4. bone wax
5. Diode lasers, electrocautery
6. Tranxeamic acid 5% mw (TXA)
7. Astringents Al2O3, FeSO4 etc
8. Haemostat / cellulose gel
9. Suture with tight apposition
10. Compress properly
11. Cold compress
12. Rest - dont pick

LIVER CIRRHOSIS

Access	• Hospital dentistry if patient has severe comorbidities, or decompensation is severe
Communication	• Fatigue, tiredness • Stigma with excessive alcohol, or diseases such as Hep B/C
Consent	• Usually no significant neurological impact, unless drunk or encephalopathy
Education	• Informed of bleeding prevention protocols, and drug prescription's considerations
Surgery (pre)	• Bleeding tests e.g. FBC (platelet) and clotting screen: PT, PTT, INR as required • Consult physician • Systemic haemostatic measures: platelet transfusion, fresh blood, desmopressin (IN/IV/SQ), Vitamin K 10mg/day 5-7 days prior
(peri)	• Amide LA undergo biotransformation in liver • Sedatives (e.g. benzodiazepines, propofol) utilise liver enzymes. Nitrous oxide tend to be safe • Suture, antibiotics, may help with managing post op infection • Optimise local haemostat measures
(post)	• Impaired drug metabolis, hepatotoxicity of drugs (high doses of paracetamol) • Delayed healing or increased infection • Avoid NSAIDS if cirrhosis or portal hypertension • Limited studies on survival of dental implants in liver cirrhosis
Spread	Hepatitis B and C - standard universal precautions
Oral manifestation	• Jaundiced mucosa • Sialosis (chronic bilateral asymptomatic inflammation of parotids) occasionally • salivary abnormalities in electrolytes and protein composition; diuretics used reduce salivary flow • Fetor hepaticus in advanced liver failure • Bleeding - petechiae, ecchymosis, bleeding gums • Periodontal disease, likely associated with immunosuppression • Gastric regurgitation and erosion • Oral cancer risk if excessive alcohol
Related conditions	• Comorbidities (e.g. hepatitis etc)

Hepatitis A to D

	Epidemio	Symptoms	Vaccine	Infection	Curative treatment	Drugs used
Hep A (RNA, Oror-faecal)	~1.5mil cases/ year	jaundice, fever, dark urine	yes	Self-limiting	Supportive	rest, nutrition
Hep B (DNA, Blood, sex perinatal)	254 mil; ~1% in Aust	Fever etc, Chronic infection is asymptomatic / HCC	yes	Acute Chronic	No, control only	tenofovir,entecavir, transplant
Hep C (RNA, blood)	~50mil; 0.5% in Aust		no	Acute Chronic	DAA cure >95%	sofosbuvir/ velpatasvir
Hep D (RNA, blood,	~12 mil	increase risk of fulminant hepatitis	with Hep B	Co-infect with HBV	No, control only	same as hep B
Hep E (RNA) Oro-faecal, water, game meat)	~20mil/yr	similar to Hep A	Limited options	Self-limiting	Supportive, Ribavirin	rest, nutrition

more details in lecture " HIV Hepatitis B Hepatitis C"…

Malnutrition

- Malnutrition is a condition resulting from an imbalance between nutritional intake and the body's requirements. It includes **undernutrition** (protein-energy malnutrition and micronutrient deficiencies), **overnutrition** (obesity), or deficiencies/excesses of specific nutrients.
- **Aetiology:**
    - inadequate intake - poverty, eating disorders (anorexia nervosa, bulimia), poor appetite (elderly, chronic illness) alcohol/ substance misuse, restrictive diets, disabilities (dysphagia, edentulous, dementia, depression, apathy etc)
    - impaired absorption - celiac's, Crohn's and UC, pancreatitis, bariatric surgery
    - increased nutrient requirements - infection, pregnancy, cancer
    - increased nutrient loss - diarrhoea, vomiting, nephrotic syndrome (protein leak)
- **Epidemiology:**
    - Globally, ~148 mil children under 5 are stunted, micronutrient deficiencies (Fe, Fit A, Iodine, Zn)
    - Australia, malnutrition affect 20-50% of hospitalised adults, 30-60% of residential aged care residents.
- **FEATURES**: Weight loss, low BMI, muscle wasting (sarcopenia), tired, depressed, delayed growth, Poor immune function, brittle nails, hair thinning anaemia
- **MANAGEMENT**
    - Nutritional assessment: dietary assessment, weight and BMI monitoring, screening tools, Blood Tests
    - Treat underlying cause - e.g. GI diseases, infection, psychology/psychiatry for eating disorders, dysphagia (speech pathologist NG, PEG feed)
    - Nutritional support: high caloric, high protein diet, food fortification (milk), supplements

Malnutrition

Access	• Frail persons may require hospital or domiciliary care • Homelessness - difficulty in return appointment and contacting
Communication	• Oral health might not be high priority
Consent	• For those with cognitive impairment or delirium
Education	• importance of balanced nutrition, • alcohol cessation
Surgery	• delayed healing, infection risk • anaemia
Spread	nil
Oral manifestation	• Glossitis, angular chelitis, apthous ulcers can happen with deficiencies in Iron, Folate, Vitamin B12 and C • candidiasis
Related conditions	• alcoholism • anorexia, bulimia • dysphagia, end of life