Endocrine Disease and Dentistry

by Dr David Lim

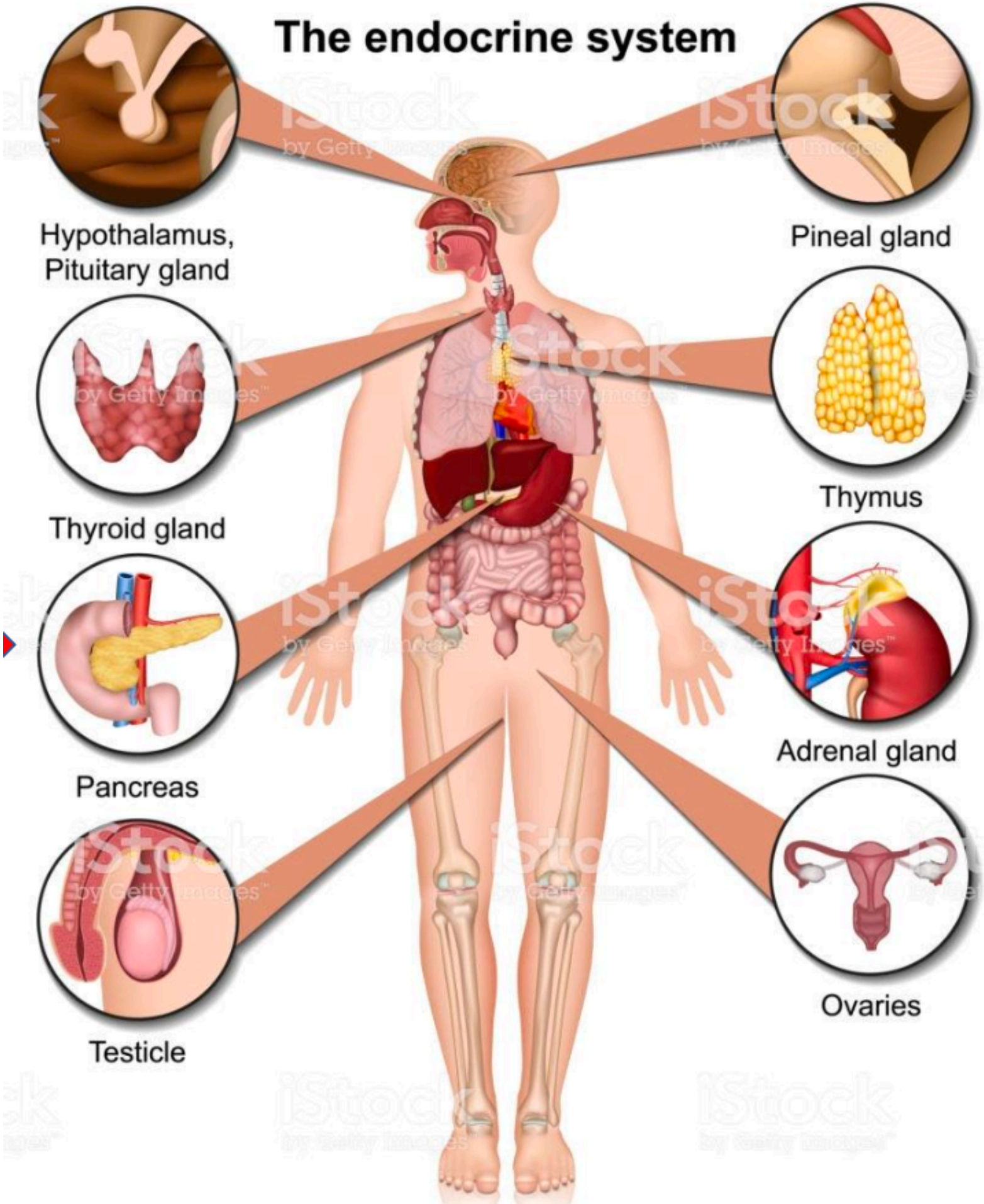
04 Aug 2026 (Tuesday) 10-11am

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Learning Objectives

1. Discuss the specific characteristics and dental needs of individuals with common endocrine conditions (e.g. diabetes, adrenal insufficiency, thyroid disorders)
2. Perform a risk assessment and make appropriate treatment adaptations to ensure safe and effective dental care.
3. Manage diabetic emergencies (hypoglycaemia and hyperglycaemia) in dental practice
4. Review the side-effects of corticosteroids and manage patients at risk of adrenal crisis in dental practice

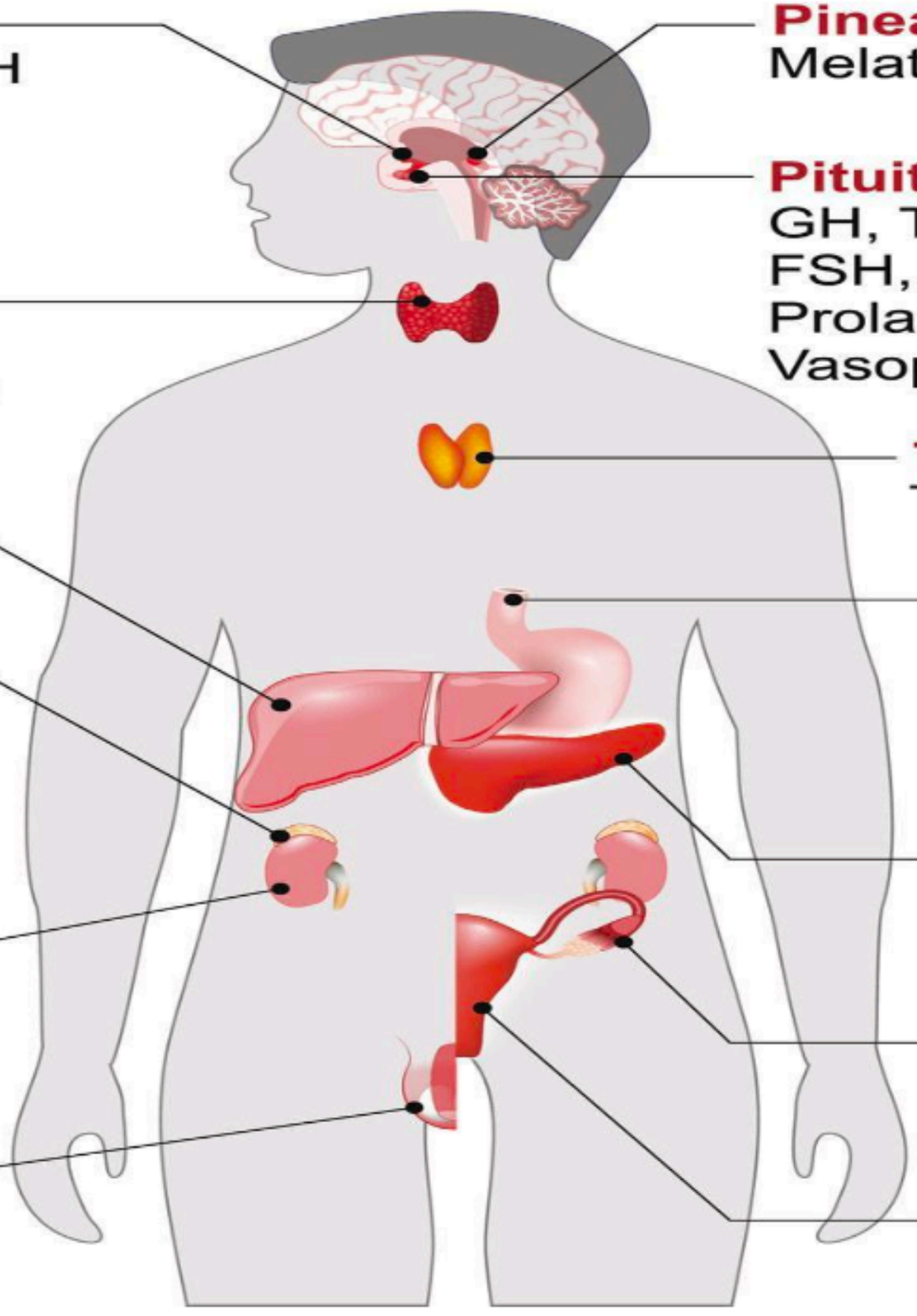
ADC: 5.1	iADH: 5A, 5B, 5C, 6Ci	5.1	Scientific and Clinical Knowledge	apply the social, cultural, biological, biomedical, physical and behavioural sciences in relation to oral health care provision and disease prevention



WHAT IS IT ABOUT XXX DISEASE/ CONDITION?

- Definition
- Aetiology
- Epidemiology
- General Manifestations/Clinical Features
- General Management/Treatment
- Oral Manifestations
- Oral Management = Risk Assessment and ACCESS acronym

HORMONES



Hypothalamus
TRH, CRH, GHRH
Dopamine
Somatostatin
Vasopressin

Thyroid and Parathyroid
T3, T4, Calcitonin
PTH

Liver
IGF, THPO

Adrenal
Androgens
Glucocorticoids
Adrenaline
Noradrenaline

Kidney
Calcitriol, Renin
Erythropoietin

Testes
Androgens
Estradiol, Inhibin

Pineal gland
Melatonin

Pituitary gland
GH, TSH, ACTH
FSH, MSH, LH
Prolactin, Oxytocin
Vasopressin

Thymus
Thymopoietin

Stomach
Gastrin, Ghrelin
Histamine
Somatostatin
Neuropeptide Y

Pancreas
Insulin, Glucagon
Somatostatin

Ovary, Placenta
Estrogens
Progesterone

Uterus
Prolactin, Relaxin

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Diabetes Mellitus

Definition

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent **hyperglycaemia (elevated blood glucose levels)** resulting from defects in insulin secretion, insulin action, or both. Prolonged hyperglycaemia can lead to damage of the eyes, kidneys, nerves, cardiovascular system, and oral tissues.

Aetiology

Type 1 Diabetes

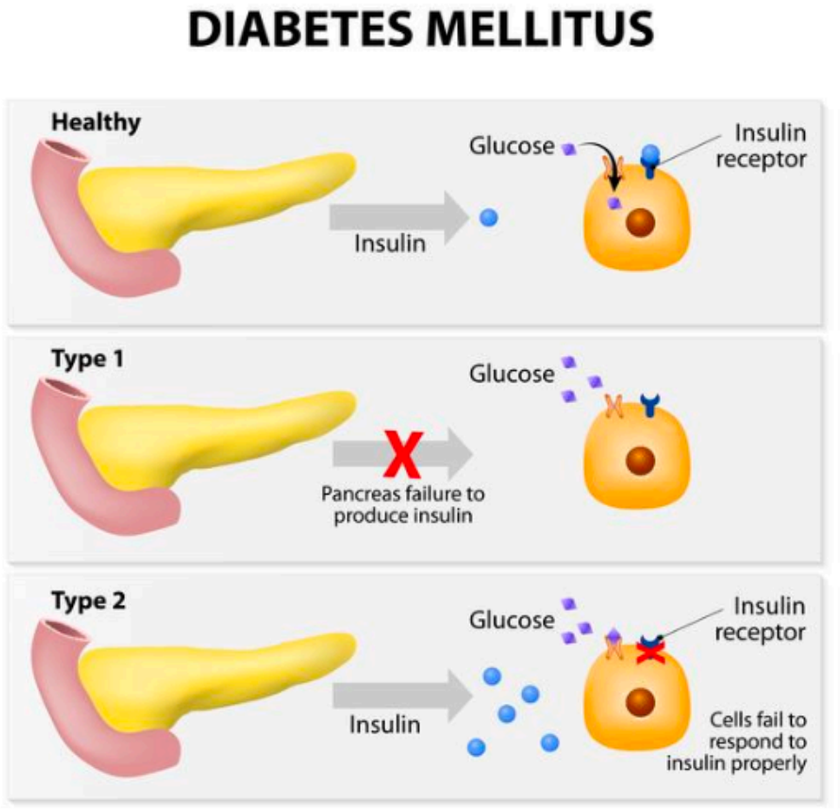
- Autoimmune destruction of pancreatic β-cells.
- Genetic predisposition (e.g., HLA-associated susceptibility).
- Environmental triggers such as viral infections may contribute.
- Results in little or no endogenous insulin production.

Type 2 Diabetes

- Combination of:
  - Insulin resistance.
  - Progressive β-cell dysfunction.
- Risk factors include:
  - GENETICS: Family history, ethnicity, .
  - LIFESTYLE: Physical inactivity. Unhealthy diet, Obesity
  - AGE
  - OTHERS: Previous gestational diabetes. Polycystic ovary syndrome.

Gestational Diabetes

- Pregnancy-induced insulin resistance exceeding pancreatic compensatory capacity.
- Usually resolves postpartum but increases future risk of Type 2 diabetes.



Epidemiology of Diabetes Mellitus

Worldwide

- Diabetes mellitus is one of the fastest-growing chronic diseases globally.
- According to the **2025 International Diabetes Federation (IDF) Diabetes Atlas**, an estimated **589 million adults (20–79 years)** are living with diabetes worldwide, representing approximately **1 in 9 adults**.
- Around **90–95% of cases are Type 2 diabetes**, while Type 1 diabetes, gestational diabetes, and other forms account for the remainder.
- Approximately **252 million people are unaware they have diabetes**, increasing the risk of delayed diagnosis and complications.

Australia

- Diabetes is a major public health issue in Australia and is among the leading causes of chronic disease and healthcare burden.
- The **Australian Institute of Health and Welfare (AIHW)** estimates that in **2022–2024**, around **1 in 15 Australians (approximately 1.3 million people, or 6.5%)** were living with diabetes based on national survey data.
- Data from the **International Diabetes Federation** estimate that in **2024**, approximately **1.69 million Australian adults aged 20–79 years** had diabetes, corresponding to a prevalence of **7.4%** in that age group.
- **Type 2 diabetes accounts for the vast majority of cases (around 90%)**.
- The prevalence increases markedly with age, with Australians aged **75 years and older** having the highest rates of disease.
- Diabetes disproportionately affects:
    - Aboriginal and Torres Strait Islander peoples
    - People living in rural and remote areas
    - Individuals from lower socioeconomic backgrounds
    - People with obesity and other metabolic risk factors

General Manifestations

Classic symptoms

- Polyuria (frequent urination)
- Polydipsia (excessive thirst)
- Polyphagia (increased appetite)
- Fatigue
- Unexplained weight loss (particularly in Type 1 diabetes)
- Blurred vision

Chronic complications

Microvascular

- Diabetic retinopathy
- Diabetic nephropathy
- Peripheral neuropathy

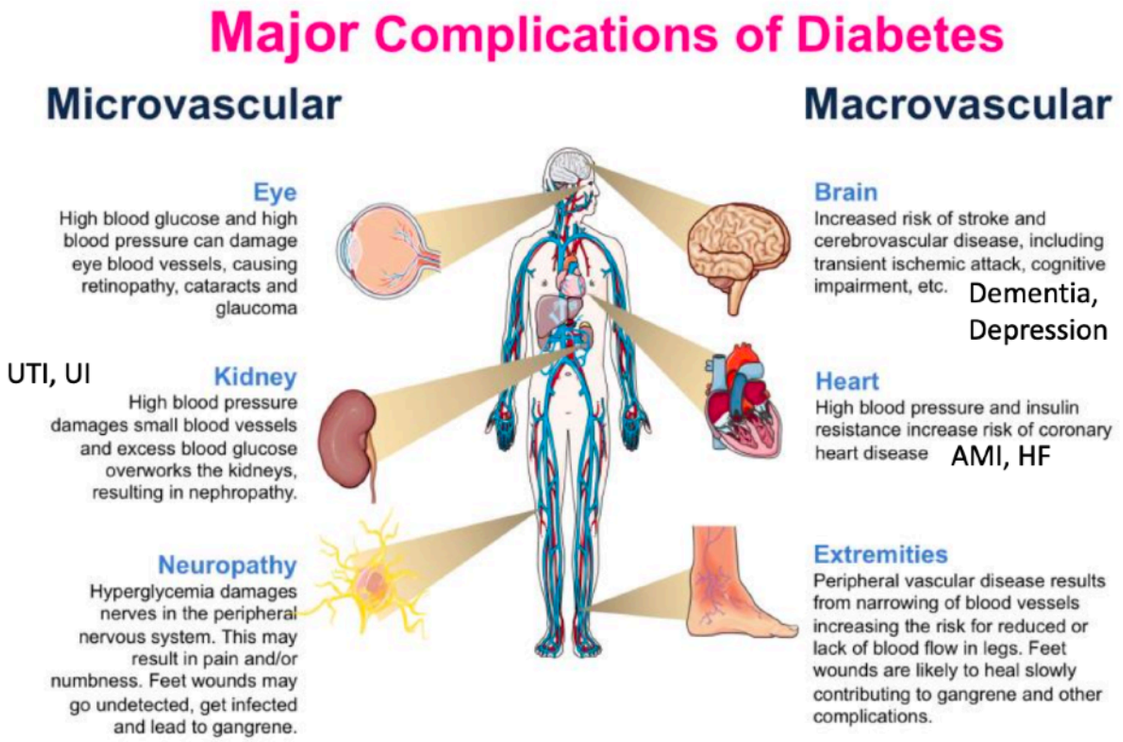
Macrovascular

- Coronary artery disease
- Cerebrovascular disease
- Peripheral arterial disease

Other features

- Delayed wound healing, susceptibility to infection
- Affects calcium metabolism - osteopenia
- Poor circulation
- Erectile dysfunction





General Management

Management aims to maintain glycaemic control and prevent complications through a multidisciplinary approach.

Increased prevalence in use of empagliflozin (and other SGLT2 inhibitors)

- Decrease oxidative stress but reducing load to proxiaml tubutles in kidneys, protecting kidney functions
- Mainly increase excretion of glucose through urine

Risk of Euglycaemic ketoacidosis, particularly after GA



Lifestyle interventions

- Healthy diet
- Regular physical activity
- Smoking and alcohol control

Medications (depending on type and severity)

- Insulin therapy (long and short)
- Oral hypoglycaemic agents: Sulfonylureas (glipizide, tolazamide), DDP-4 inhibits (sitagliptin), Biguanides (metformin), Alpha-glucosidase inhibitors (acarbose, miglitol) SGLT2 inhibitors
- Injectable non-insulin therapies

	Blood glucose
Glucose alert value	<70 mg/dL (3.9 mmol/L)
Clinically significant hypoglycemia	<54 mg/dL (3.0 mmol/L)
Severe hypoglycemia	No specific glucose level
Relative hypoglycemia	No specific glucose level

Surgery

- Amputation of gangrenous areas

Monitoring

- Self-monitoring of blood glucose and Regular HbA1c assessment.
- Blood pressure and lipid management.
- OTHER DISEASE: Screening for kidney disease, retinopathy, neuropathy, and cardiovascular disease.

Diabetes Mellitus

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Access	• Amputee - wheelchair user, fall risks • Morning appointments (without missing breakfast) - higher endogenous cortisol leading to decreased risk of hypoglycaemia
Communication	• Visual disturbances (cataract, diabetic retinopathy) • Patients might not be aware they have diabetes mellitus
Consent	• Usually no significant neurological impact.
Education	• Effective periodontal treatment can contribute to modest improvements in glycaemic control. • Fatigue might affect attendance and routine oral hygiene • Inform risk of failure of surgical implants, or poorer healing
Surgery (pre)	• check BG if needed prior to dental treatment (at least 5.0mmol/L) • Check that food and medications were taken • For GA fasting - place on glucose sliding scale • Appointment - short morning better
(peri)	• Watch for HypoG signs (sweat, confusion, tremors) • Suture, antibiotics, may help with managing post op infection
(post)	• Higher risk of oral infection/dry socket • Risk of denture stomatitis is higher • most NSAIDS, metronidazole and azithromycin may boost oral hypoglycaemic agents, • ciprofloxacin can compete with insulin.
Spread	Diabetes Mellitus is not contagious
Oral manifestation	• Increased prevalence and severity of periodontal disease. • Xerostomia (dry mouth). Burning mouth symptoms. • Delayed wound healing following dental procedures. Increased susceptibility to oral infections. Oral candidiasis. • Increased dental caries risk secondary to xerostomia. • Altered taste sensation Halitosis. • Enlarged parotid glands (occasionally).
Related conditions	• High BG increases risk of osteoporosis (MRONJ), Cardiovascular, Cerebrovasculr issues, • Those with late stage diabetes may have chronic renal failure, amputations, visual disturbances etc • Obesity

1. Definition

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Adrenal insufficiency (AI) is an endocrine disorder in which the adrenal glands fail to produce sufficient **glucocorticoids (primarily cortisol)**, and in some cases **mineralocorticoids (aldosterone)** and adrenal androgens. Inadequate cortisol production impairs the body's ability to respond to physiological stress, including illness and surgery.

The main types are:

- Primary adrenal insufficiency – due to destruction or dysfunction of the adrenal cortex.
- Secondary adrenal insufficiency – caused by reduced secretion of adrenocorticotropic hormone (ACTH) from the pituitary gland.
- Tertiary adrenal insufficiency – often results from suppression of the hypothalamic–pituitary–adrenal axis following prolonged corticosteroid therapy.

2. Aetiology

Primary Adrenal Insufficiency

- **Autoimmune adrenal destruction (most common in developed countries)**
- Infections (e.g., tuberculosis, fungal infections)
- Adrenal haemorrhage or infarction
- Metastatic cancer
- Genetic disorders
- Bilateral adrenalectomy

Secondary Adrenal Insufficiency

- Pituitary tumours or surgery
- Pituitary irradiation
- Pituitary infarction
- ACTH deficiency

Tertiary Adrenal Insufficiency

- Long-term systemic corticosteroid therapy with abrupt withdrawal
- Hypothalamic disease
- **Chronic suppression of the hypothalamic–pituitary–adrenal axis**

revision, not tested

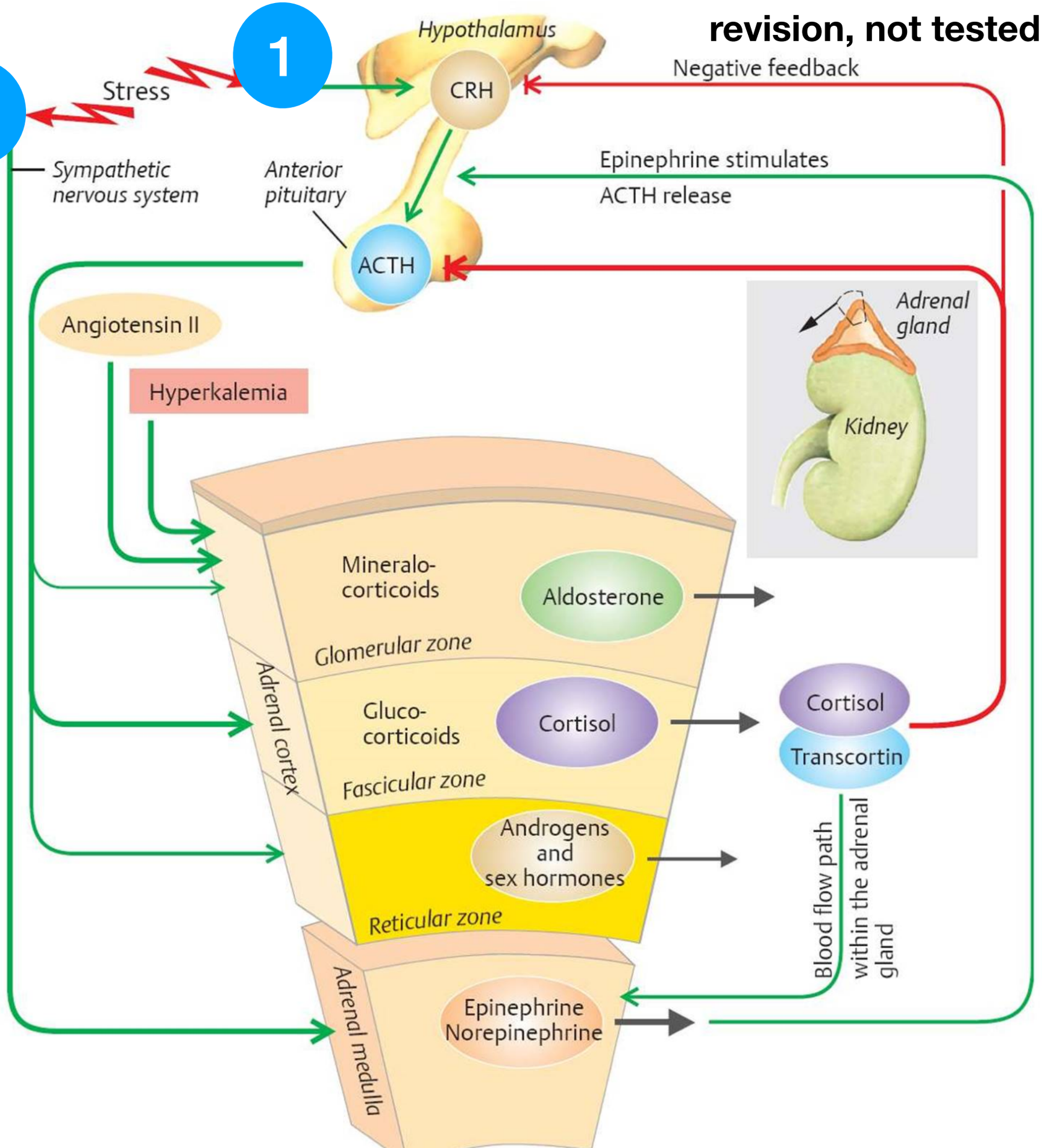
STRESS REGULATION CYCLE

Stress and Diurnal regulation (controlled by light-dark day pattern) affects endogenous steroid release

Corticotropin-Releasing Hormone (CRH)
Adrenocorticotropic Hormone (ACTH)

Adrenal Medulla (stimulated by SNS)

- adrenaline ~85% of secretion, rapid 'fight-flight' response
- noradrenaline ~15% of secretion, mainly vasoconstrict to maintain BP



3. Epidemiology

Global

- Primary adrenal insufficiency is **rare**, affecting approximately **100–140 people per million population** in developed countries.
- Autoimmune disease accounts for most cases in high-income countries, while infectious causes remain important in some low- and middle-income regions.
- Secondary/tertiary adrenal insufficiency is generally more common than primary adrenal insufficiency due to pituitary disease and widespread corticosteroid use.

Australia

- Adrenal insufficiency is an uncommon condition
- Addison’s disease (Autoimmune adrenalitis) is the leading cause of primary adrenal insufficiency in Australia.

4. General Manifestations

Common symptoms [stress symptoms]

- Chronic fatigue
- Muscle weakness
- Weight loss
- Loss of appetite
- Nausea and vomiting
- Abdominal pain
- Dizziness or postural hypotension

Features of Primary Adrenal Insufficiency

- Hyperpigmentation of the skin and mucous membranes
- Salt craving
- Hypotension Hyponatraemia Hyperkalaemia
- Dehydration

Adrenal Crisis (Medical Emergency)

5. General Management

Hormone replacement

- Lifelong glucocorticoid replacement (e.g., hydrocortisone or prednisolone)
- Mineralocorticoid replacement (e.g., fludrocortisone) for primary adrenal insufficiency

Stress dosing

- Patients may require increased ("stress-dose") glucocorticoids during illness, surgery, or major physiological stress.

Patient education

- Carry medical alert identification.
- Maintain an emergency steroid plan.
- Avoid abrupt discontinuation of prescribed corticosteroids.
- Have access to emergency injectable hydrocortisone when indicated.

Regular follow-up

- Endocrinology review
- Monitoring of blood pressure, electrolytes, and symptoms
- Adjustment of replacement therapy as needed
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6. Oral Manifestations

Patients with adrenal insufficiency may exhibit:

- Diffuse or patchy **oral mucosal hyperpigmentation**, particularly in primary adrenal insufficiency
- Delayed wound healing (especially in patients receiving corticosteroid therapy)
- Increased susceptibility to oral infections if immunosuppressed
- Oral candidiasis in patients using corticosteroids
- Fatigue that may affect tolerance of lengthy dental appointments
- oral pigmentation may involve the buccal mucosa, gingiva, tongue, palate, or lips and may be one of the earliest clinical signs of Addison disease.

Adrenal Insufficiencies

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Access	• Morning appointments - higher endogenous cortisol
Communication	• Pituitary tumor can compress optic chiasma
Consent	• Usually no significant neurological impact.
Education	• Corticosteroid use, infections, and oral health • Low-calorie, low fat, low sodium, protein rich, potassium rich, calcium rich diet for corticosteroid treatment may be helpful for caries risk.
Surgery (pre)	• Steroid Cover for invasive dentistry - **Gibson & Ferguson 2004** • Steroid Cover (IV) for GA - **Nicholson et al., 1998** • Appointment - short morning better
(peri)	• Look out for signs of Adrenal Crisis ~ Sometimes happens after treatment ▽
(post)	• Higher risk of oral infections • Azole antifunals and some macrolides can increase plasma glucocorticoid levels • NSAIDS and glucocorticoid use may increase risk of Gastrointestinal Injury
Spread	• Addison's disease is not contagious
Oral manifestation	• Oral pigmentation in buccal mucosa, gingiva, tongue, palate, or lip • Other risk of infection due to steroid use • Patient on long-term corticosteroids may be susceptible to infection and reactivation of Herpes virus, Varicella zoster, Hep B/C etc.
Related conditions	• Increased risk of osteoporosis with long-term steroid use • Cause of steroid prescription: SLE, Rheumatoid Arthritis etc.

Side effects of steroids

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Organ system	Complication	Considerations
Cardiovascular/ renal	Hypertension Sodium and water retention Peripheral edema	Use with caution in heart failure and renal disease Electrolyte and BP* monitoring
Endocrine/ metabolic	Cushingoid appearance and weight gain Elevated serum glucose	Monitor for signs of adrenal insufficiency Monitor serum glucose Diet and exercise education
Gastrointestinal	Peptic ulcer Abdominal discomfort	H2 blocker of proton pump inhibitors
Infectious	Immunosuppression Suprainfection: Fungal, parasitic Reactivation of infection	Monitor for signs of infection Consider stress dosing
Integumentary	Impaired wound healing	Monitor for signs and symptoms of infection
Musculoskeletal	Myopathy Increased calcium excretion	Falls risk evaluation Oral supplements or possibly bisphosphonates with prolonged use and life expectancy
Ophthalmologic	Cataracts Glaucoma	Safety Ophthalmological evaluation
Psychiatric	Anxiety Depression Mood disorder	Appropriate screening tools Psychotherapy Pharmacotherapeutics

*BP: Blood pressure.

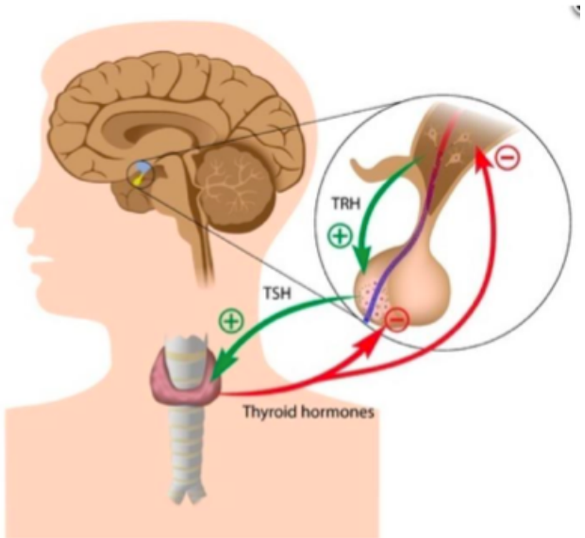
osteopenia
osteoporosis
↳ more anxiety depression
more disorder

Schwarzrock, Camille. (2016). Collaboration in the presence of cerebral edema: The complications o steroids. Surgical Neurology International. 7. 185. 10.4103/2152-7806.179228.

Thyroid gland

- Thyroid gland secretes several thyroid hormones. Main hormone is thyroxine, also called T4. Calcitonin stops breakdown of bones and lowers body calcium level
- Thyroid hormones act throughout the body, influencing metabolism, growth and development, and body temperature.
- Thyroid function does not change greatly with ageing

- Breathing
- Heart rate
- Central Peripheral nervous systems
- Body weight
- Muscle strength
- Menstrual cycles
- Body temperature
- Cholesterol levels



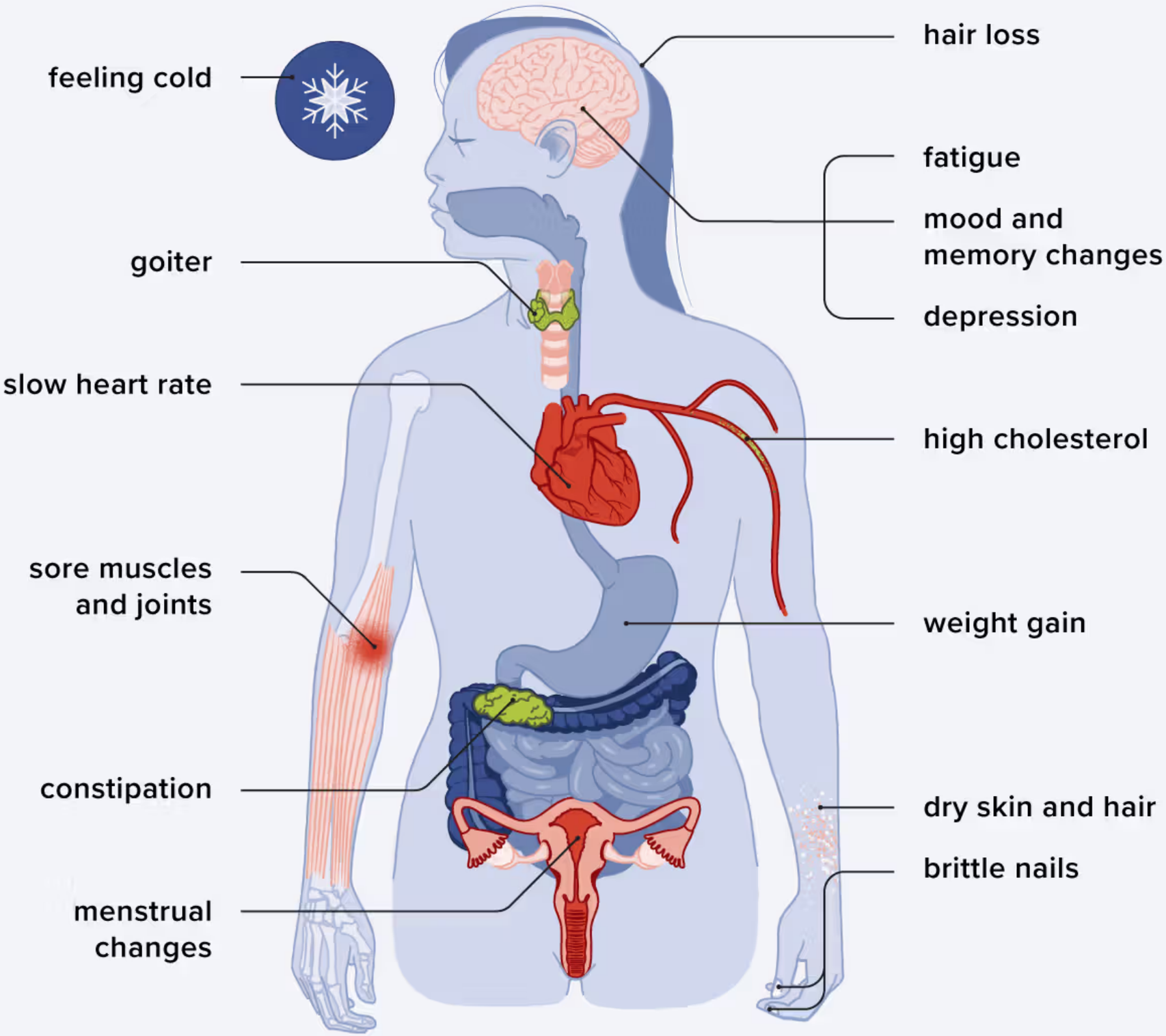
Causes of Hyperthyroidism	Causes of Hypothyroidism
Autoimmune: Graves' disease (most common cause) - TSH receptor antibodies secreted (TRAb and TMAb)	**Autoimmune:** Hashimoto's thyroiditis (most common non-iodine cause)
Toxic multinodular goitre	**Iodine deficiency** (most common worldwide)
Iatrogenic intake of excess hormones **TSH secreting pituitary adenoma** (rare) - increased TSH	**Iatrogenic:** thyroidectomy, radioactive iodine therapy **Secondary hypothyroidism:** pituitary disease causing low TSH
Prevalence 0.3% in Australia. affects women 10x more than men	Prevalence is 1-2% in countries with sufficient iodine. More common in women, older than 65 years, and 'white' individuals

Key differences

Hyperthyroidism	Hypothyroidism
Raised pulse, hypertension, tremors, warm skin, weight loss Anxiety, irritability Exopthalamus, thyroid swell/lump (goitre), fine brittle hair Thyrotoxic periodic paralysis - weakness attacks (mild-severe) **THYROTOXIC CRISIS** - extreme anxiety, bomiting and abdominal pain, tachycardia, stupor, coma	Enlarged gland (goitre) due to overstimulation from TSH Asymptomatic forms **MYXEDEMA CRISUS/COMA** (encephalopathy, hypothermia, seizures, hyponatraemia, cariogenic shock, respiratory failure etc) Cretinism retardation (due to maternal iodine deficiency)
Symptomatic control e.g. beta-blockers Anti-thyroid drugs (carbimazole, propylthiouracil, methimazole for 18 months) Radioactive iodine, very effective, although results in long-term hypothyroid Surgery for large goitres or those where drugs are ineffective	Levothyroxine (synthetic T4), 1.5-1.8 microgram/kg Reduced in elderly or those with Atrial Fibrillation
Worldwide, autoimmune disease is the predominant cause where iodine intake is adequate	Worldwide, **iodine deficiency** remains the leading cause

Hypothyroidism

effects on the body



MEDICALNEWSTODAY

Emergency Management

Hypoglycaemia / diabetic crisis
Hyperglycaemia
Adrenal/Addisonian’s Crisis

Hyperglycaemic crisis

Patient can present with:
1. confusion, drowsiness, vomiting
2. fruity smelling breadth
3. unconscious (call 911)

Tests for Diabetic ketoacidosis:
1. Blood sugar levels over 300mg/dL (16.1mmol/L)
2. near-normal (**euglycemic**) blood sugar level may occur
3. Ketone level and blood pH

For dentistry, less common to see this. If the person is conscious,
- Do a blood glucose test.
- Drink plenty of water and
- Take their prescribed diabetic medications (e.g. Insulin, sulphnoureas)
- Send to medical clinics to manage **Diabetic Ketoacidosis** (DKA) or **Hyperosmolar hyperglycaemic state** (HHS), which is usually treated through
    1. Fluid replacement (dilute excess sugar)
    2. Electrolyte replacement (K and Na replaced to keep muscles, heart and nerves functioning)
    3. Insulin therapy (reverse ketone buildup and normalise blood sugar)

Managing hyperglycaemic emergencies: General advice (Royal Australia College of General Practitioners)

- Look for an underlying cause – for example, sepsis, cellulitis, myocardial infarction.
- If no causes are evident, or if you are unable to urgently assess ketones with the hyperglycaemia and the person is unwell, referral to the nearest emergency department may be prudent.
- After the event, review medications, dietary intake and hyperglycaemic and sick-day management.
- Re-evaluate for possible undiagnosed type 1 diabetes.

https://www.racgp.org.au/clinical-resources/clinical-guidelines/key-racgp-guidelines/view-all-racgp-guidelines/management-of-type-2-diabetes/managing-glycaemic-emergencies

Acute adrenal insufficiency
Adrenal/Addisonian's Crisis

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Patient can present with:
1. headache, confusion lethargy, vomiting
2. sudden hypotension / shock, or progress 6-12 hours after dental treatment
3. unconscious

Dx for Adrenal crisis:
1. History (high dose steroid long term / Addisonian)
2. Low BP, 20mmHg lower than usual. (Systolic <100mmHg)

For dentistry, rare. Manage by
- Calling 911/000
- Elevate legs, monitor BP
- Administer hydrocortisone 200mg intramuscularly (or other routes/doses)
- Supportive (hydration, provide oxygen, glucose)

Prevention (from AD Australia):

Minor surgery e.g. cataract surgery, hernia repairs, laparoscopy with local anaesthetic, endoscopy. Includes dental surgery >1hr and with local anaesthetic.	25mg hydrocortisone IV or 20mg oral with induction/immediately prior to onset procedure

Prevention in dentistry:
- Double dosage of steroid
- Avoid afternoon appointment in severe patients + stressful treatment (crisis can be fatal if occurring when sleeping)
- Avoid dentistry when patient has infection/fever/ diarrhoea/sick → stress+stress is really bad

Therapeutic Guidelines Pg 151

When to cover steroids?

Perioperative steroid is required to prevent an acute adrenal crisis in patients with HPA axis suppression. Patients taking

- ≥ 5 mg of prednisolone daily (or equivalent) for more than 1 to 4 weeks, or
- those who stopped a similar dose within the last 3 months

And for invasive or stressful procedures (e.g. GA, dental surgery or extraction, or if patient is highly anxious)

Name	Equivalent dose in milligrams
Hydrocortisone	20
Prednisone	5
Prednisolone	5
Methylprednisolone	4
Dexamethasone	0.75

Therapeutic Guideline, The Hospitalist

Vasovagal Syncope

Hypoglycaemia

Patient can present with:

1. Neuroglycopenic - hunger, confusion, coma, seizure
2. Adrenergic - sweating, shaking, palpitations
3. unconscious (call 911, BLS)

Indicators for hypoglycaemia:

1. Blood sugar levels under 3.5mmol/L (usually)
2. Missed meals
3. Overdosed diabetic medication
4. Unplanned exercise (dental anxiety, rush for bus)

For dentistry, it's common to see this. If the person is conscious,

- give 20-25g of glucose or fast acting food/drink (fruit juice, jelly) followed by lower glycemic load carbohydrate meal. Sugary substitutes do not work well (coke zero, xylitol, saccharin)
- Observe.
- Taking blood sugar is helpful but not mandatory.
- Reschedule, and ensure someone goes home with them (escort)

Therapeutic Guidelines Pg 178-179

thank you and all the best