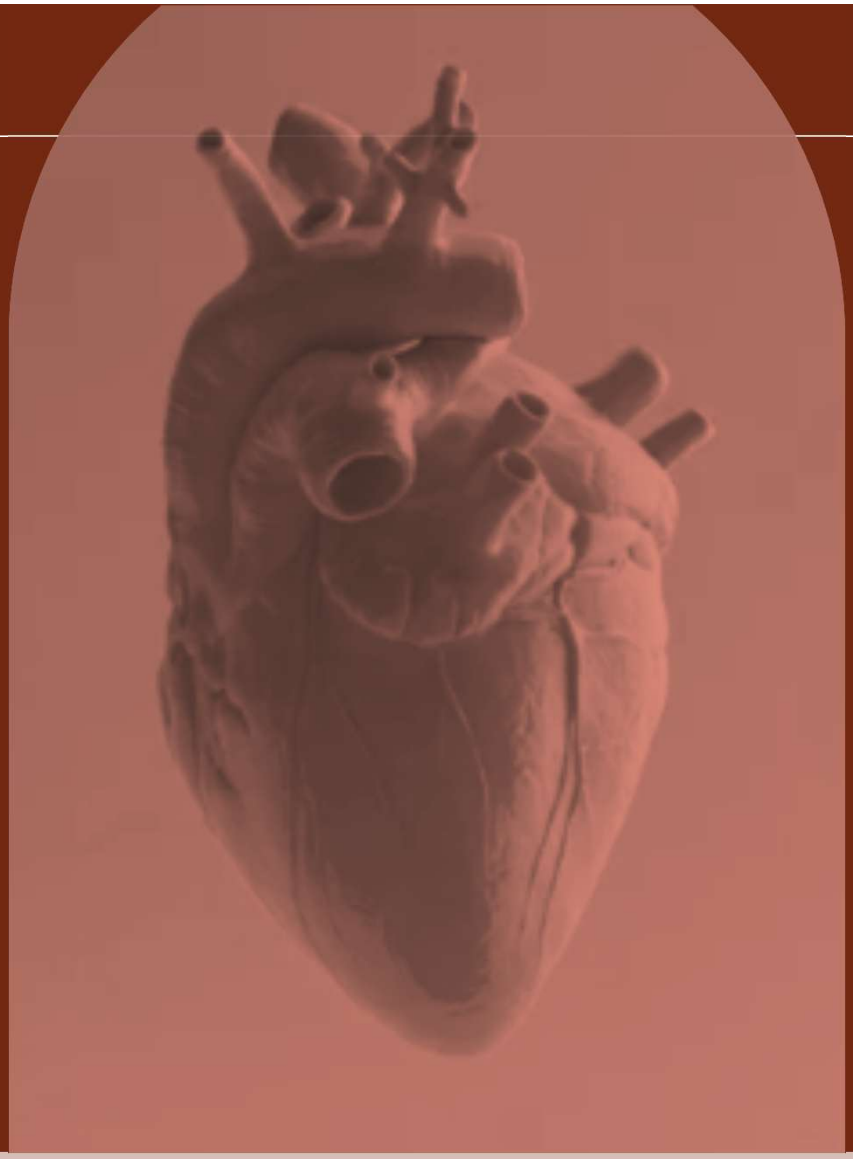


CARDIOVASCULAR DISEASE AND DENTISTRY

DR JEE-YUN LEUNG

LEARNING OUTCOMES

- PART 1
- 1. Review common vascular, valvular, electrical, muscular and congenital cardiac conditions and their management (e.g. hypertension, coronary artery disease, rheumatic heart disease, infective endocarditis, arrhythmias, congenital structural defects, heart failure)
- 2. Describe potential oral findings associated with cardiac conditions
- 3. Describe the oral manifestations and manage the risks associated with common treatments used to manage cardiac conditions
- 4. Assess the associated risks of common dental procedures and modify dental treatment appropriately to safely provide care

- PART 2
- 5. Work a patient up prior to cardiac surgery
- 6. Manage angina or an acute coronary syndrome in dental practice



COMMON CARDIAC CONDITIONS

Hypertension, Coronary Artery Disease, Rheumatic Heart Disease, Infective Endocarditis, Arrhytmias, Congenital Structural Defects, Heart Failure

WHO INTERNATIONAL CLASSIFICATION OF DISEASES - 11TH REVISION

- 11 Diseases of the circulatory system
  - Hypertensive diseases
  - Hypotension
  - Ischaemic heart diseases
  - Diseases of coronary artery
  - Pulmonary heart disease or diseases of pulmonary circulation
  - Pericarditis
  - Acute or subacute endocarditis
  - Heart valve diseases
  - BC20 Chronic rheumatic heart diseases, not elsewhere classified
  - Diseases of the myocardium or cardiac chambers
  - Cardiac arrhythmia
  - Heart failure
  - Diseases of arteries or arterioles
  - Diseases of veins
  - Disorders of lymphatic vessels or lymph nodes
  - Postprocedural disorders of circulatory system
  - Neoplasms of the circulatory system
  - Developmental anomalies of the circulatory system
  - Infections of the circulatory system
  - Symptoms, signs or clinical findings of the circulatory system
  - Cerebrovascular diseases
  - Functional vascular disorders of the skin

JB64.4 Diseases of the circulatory system complicating pregnancy, childbirth or the puerperium

BE2Y Other specified diseases of the circulatory system

BE2Z Diseases of the circulatory system, unspecified

TYPES OF CARDIAC CONDITIONS

Vascular / Ischaemic

- Hypertension
- Hypotension
- Ischaemic heart disease (Coronary artery disease) – angina, myocardiac infarction
- Thromboembolic complications of CVD

Valvular

- Rheumatic fever and Rheumatic heart disease
- Infective endocarditis
- Aortic and mitral valve stenosis, regurgitation and prolapse

Electrical (Dysrhythmias)

- Extrasystoles (ectopics)
- Tachycardia (including Atrial fibrillation)
- Bradycardias

Muscular (cardiomyopathies)

- Primary – genetic/inherited, acquired
- Secondary

Congenital

- Cyanotic
- Acyanotic

Heart Failure

VASCULAR / ISCHAEMIC

Hypotension

Hypertension

Ischaemic heart disease (Coronary artery disease) – angina, myocardial infarction

Thromboembolic complications of CVD

HYPOTENSION - DEFINITIONS

- **Hypotension** (chronic arterial hypotension) = systolic BP <100 mmHg
- **Pre-syncope ("near-fainting")** = condition in which a patient feels syncope is imminent
- **Syncope ("fainting")** = acute fall in blood pressure, resulting in transient global cerebral hypoperfusion, causing a loss of consciousness which usually leads to loss of postural tone and falling
  - **Vasovagal syncope** = reaction to pain, or to anxiety and fear before, during or after a dental procedure
  - **Orthostatic hypotension** = fall in blood pressure when standing up after sitting or lying down for an extended period of time in the dental chair

ORTHOSTATIC HYPOTENSION - AETIOLOGY

- **Cardiac conditions**: ability for heart to pump blood (e.g. abnormal heart rhythm, heart failure, MI)
- **Autonomic nervous system**: involved in returning BP to normal (e.g. Parkinson's disease)
- **Long-term bed rest** (e.g. hospitalised, pregnancy)
- **Low blood volume** (e.g. dehydration, anaemia, injury)
- **Medications** (e.g. antihypertensives, antidepressants)

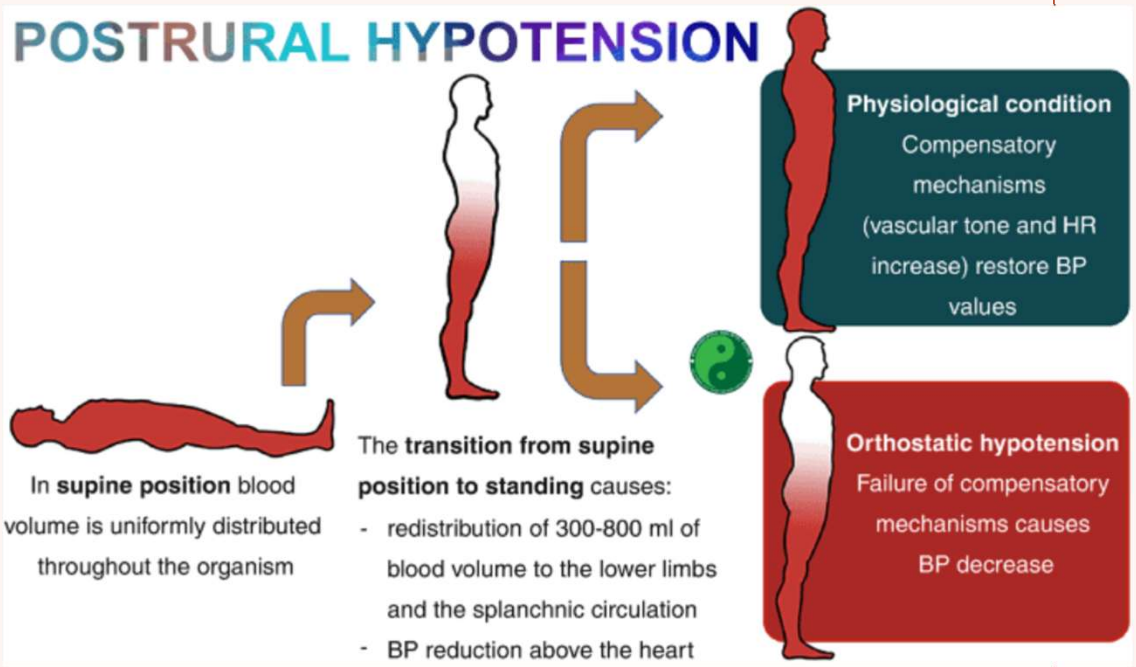
Table 5.12 *Causes of orthostatic hypotension*

Primary autonomic causes	Secondary autonomic causes	Non-autonomic causes
Familial dysautonomia (Riley–Day syndrome)	B vitamin deficiency	Hypovolaemia
Pure autonomic failure (idiopathic orthostatic hypotension)	Alcoholism	Ageing
Shy–Drager syndrome	Diabetes	Prolonged bed rest
Dopamine	Parkinsonism	Pregnancy
Beta-hydroxylase deficiency	Porphyria	Drugs (e.g. antihypertensives)

Scully C. Scully's medical problems in dentistry. 7th ed. Edinburgh: Churchill Livingstone/Elsevier; 2014

ORTHOSTATIC HYPOTENSION – GENERAL MANIFESTATIONS

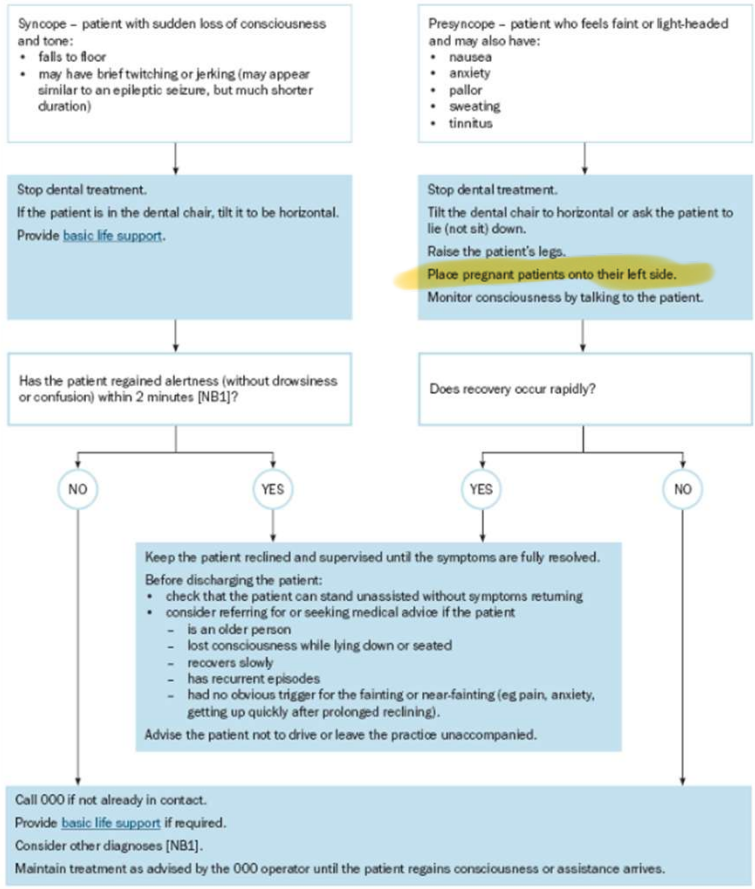
- **Early symptoms**: vertigo, dizziness, visual blurring, headache, syncope
- **Later**: tachycardia, paleness, cold extremities, sweating



HYPOTENSION – GENERAL MANAGEMENT

Figure 13.80 Management of presyncope and syncope in dental practice

Printable figure



NB1: Many people with chest pain do not have angina or a heart attack, but anyone with acute chest pain requires rapid medical evaluation for potentially life-threatening causes. The likelihood of angina or heart attack increases with the number of suggestive features.

NB2: Sublingual glyceryl trinitrate (GTN) dosing is a 400 microgram spray (or 300 to 600 microgram tablet) generally administered every 5 minutes to a maximum of 3 doses. Ask patients who use GTN to bring their GTN to their dental appointment so that it can be administered if chest pain occurs.

NB3: If symptoms (pain or other features that may indicate angina) last longer than the patient's usual angina symptoms, urgent medical evaluation of a possible heart attack or other urgent condition is required.

HYPERTENSION - DEFINITION

- **Essential (primary) hypertension (95%):** High blood pressure for which a secondary cause cannot be found.
- **Secondary hypertension (5%):** Systolic BP >140mmHg and diastolic BP >90mmHg with an identifiable cause.

National Heart Foundation of Australia – Classification of Blood Pressure

Table 2.1 Classification of clinic blood pressure levels in adults

Diagnostic category*	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High-normal	130–139	and/or	85–89
Grade 1 (mild) hypertension	140–159	and/or	90–99
Grade 2 (moderate) hypertension	160–179	and/or	100–109
Grade 3 (severe) hypertension	≥180	and/or	≥110
Isolated systolic hypertension	>140	and	<90

*When a patient's systolic and diastolic blood pressure levels fall into different categories, the higher diagnostic category and recommended actions apply.

HYPERTENSION - AETIOLOGY

Essential hypertension	Secondary hypertension
• Genetic predisposition • High basal metabolic index • Sympathetic overactivity (40% have raised catecholamines and noradrenaline) • High alcohol, salt intake	• Renal disease (80%) • Endocrine conditions: pregnancy, Cushing syndrome, corticosteroids, hyperaldosteronism, acromegaly • Cerebral disease: cerebral oedema (mainly strokes, head injuries or tumours) • Coarctation of aorta • Sleep apnoea • Drugs: oral contraceptive pill, HRT, corticosteroids, NSAIDs, sympathomimetics (decongestants, diet pills, cocaine), stimulants (amphetamine) cyclosporine / tacrolimus, excess alcohol, SNRIs and MAOIs, haemopoietic drugs

HYPERTENSION - EPIDEMIOLOGY

Australian Institute of Health and Welfare

- 39% of Australians >18yo have hypertension (~7.2 million adults)
- Of adults with hypertension
  - 80-82% of those <54yo had uncontrolled hypertension
  - 65% had been dispensed antihypertensive medications
- Hypertensive disease contributed to 13% of all Australian deaths in 2023

- Nearly 1 in 20 (5%) Australian women aged 15-44 giving birth had new onset hypertension in pregnancy
- 24% of ATSI adults >18yo had hypertension 2022-2023

https://www.aihw.gov.au/reports/risk-factors/hypertension/contents/summary

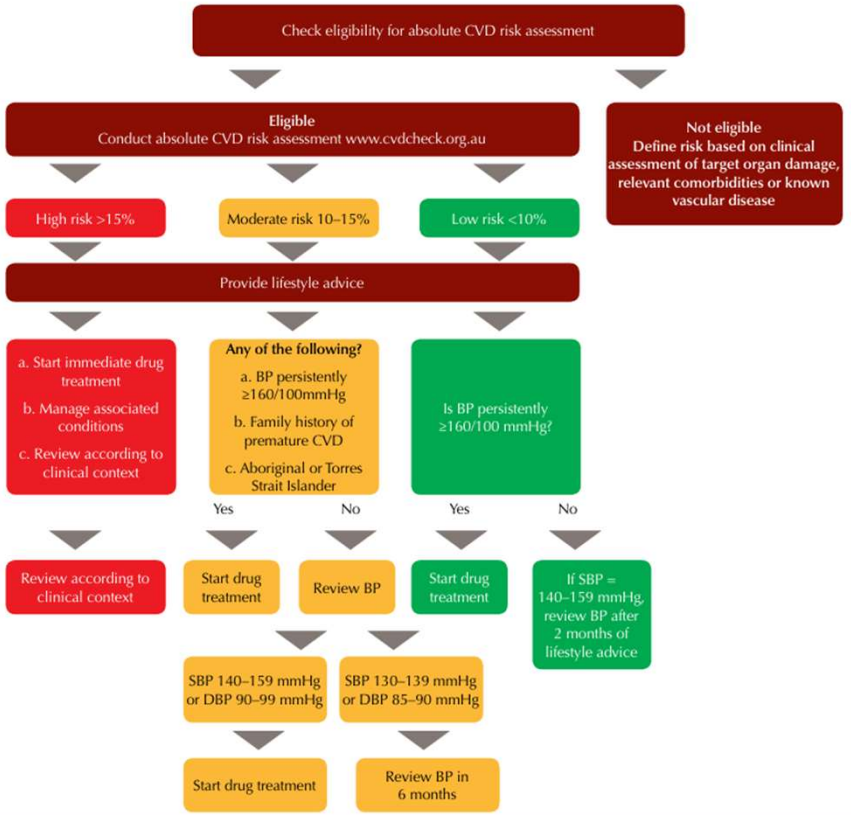
HYPERTENSION – GENERAL MANIFESTATIONS

Primary	Complications
Usually asymptomatic Advanced cases: headache, blurred vision, tinnitus, fatigue, dizziness, angina	Arteriosclerosis Cerebrovascular disease (stroke) Peripheral vascular disease Hypertensive retinopathy Hypertensive heart disease Hypertensive renal disease Hypertensive crisis

HYPERTENSION – GENERAL MANAGEMENT

- **Identify and manage risk factors**: physical activity, smoking cessation, weight control, diet, alcohol intake, etc.
- Assess presence/absence of organ damage
- **Pharmacologic strategies**: most widely used first-line ACEIs and ARBs

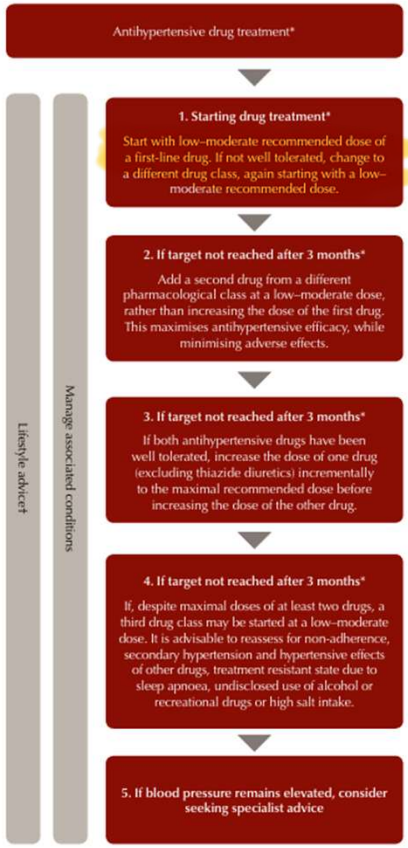
Figure 6.1 Treatment strategy for patients with newly diagnosed hypertension



https://www.heartfoundation.org.au/for-professionals/hypertension

HYPERTENSION – GENERAL MANAGEMENT

Figure 6.2 Drug treatment strategy to reach blood pressure target



*Maximum effect of drug likely to be seen in 4–6 weeks. If baseline blood pressure is severely elevated earlier reviews may be considered. For steps 1–4, review every 4–6 weeks for tolerance, efficacy and adverse effects. †All patients should receive lifestyle advice with follow-up based on clinical context.

Table 6.2 Effective drug combinations

First drug		Second drug	Comment
Effective combination			
ACE inhibitor or ARB*	plus	Calcium channel blocker	Particularly useful in presence of diabetes and/or lipid abnormalities[124]
ACE inhibitor or ARB*	plus	Thiazide diuretic	Useful in presence of heart failure or post stroke
ACE inhibitor or ARB*	plus	Beta-blocker	Recommended post myocardial infarction or in patients with heart failure†
Beta-blocker	plus	Dihydropyridine calcium channel blocker	Useful in presence of symptomatic coronary heart disease
Thiazide diuretic	plus	Calcium channel blocker	
Thiazide diuretic	plus	Beta-blocker	Not recommended in presence of glucose intolerance, metabolic syndrome or established diabetes
Combinations to use with care			
Diltiazem	plus	Beta-blocker	Due to risk of heart block, but risk is less than with verapamil
ACE inhibitor or ARB	plus	Potassium-sparing diuretic	Due to risk of hyperkalaemia
Combinations to avoid			
ACE inhibitor	plus	ARB	Increased risk of renal dysfunction[120]
Verapamil	plus	Beta-blocker	Due to risk of heart block

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker

*In head-to-head trials ACE inhibitors and ARB's are equally effective in blood pressure reduction and prevention of cardiovascular events overall, however may have important differences in their efficacy, so that they are not interchangeable, in some clinical conditions.

†Carvedilol; bisoprolol (beta-1 selective antagonist); metoprolol extended release (beta-1 selective antagonist); nebivolol.[125]

https://www.heartfoundation.org.au/for-professionals/hypertension

HYPERTENSION – ORAL MANIFESTATIONS

- No recognised oral manifestations from disease
- Hypertensive crisis – facial palsy may occur.
- Side-effects of antihypertensive drugs:

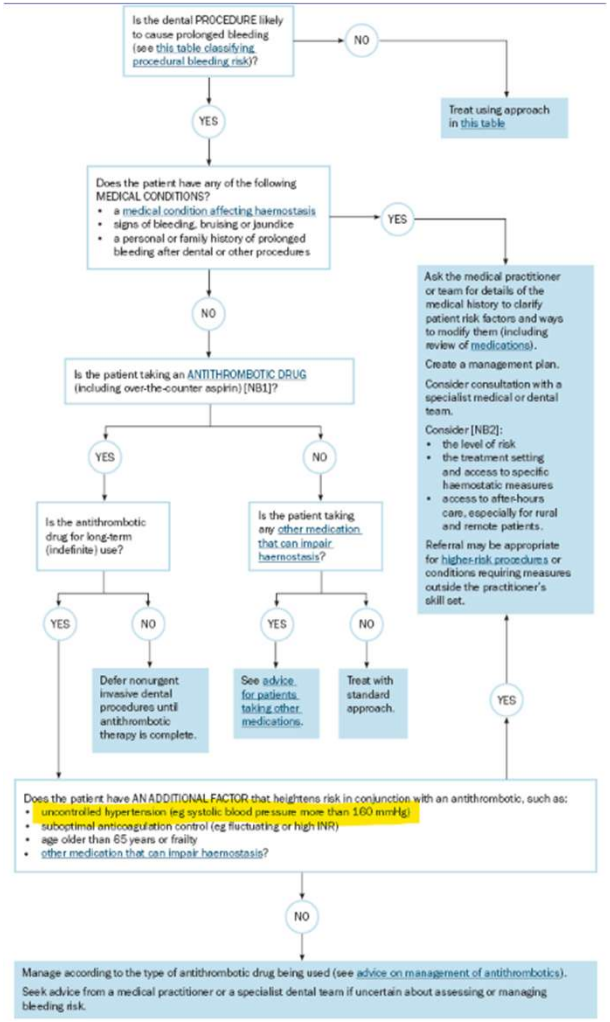
Drug Class	Examples	Oral Side Effects
ACEI	Captopril, Ramipril	Burning mouth syndrome, angioedema, ulceration, dysguesia, dry mouth, lichenoid reactions, coughing, taste disturbances
ARB	Candesartan, Irbesartan, Losartan, Valsartan	Facial flushing, taste disturbances, dry mouth, increased gag reflex, lupoid reactions
β-adrenergic blockers	**β_1 selective (not bronchioles):** Atenolol, Bisoprolol , Metoprolol **Non-selective:** Propranolol	Dry mouth, lichenoid lesions, paraesthesia, taste changes
Calcium channel blockers	**Dihydropyridines** (vascular selective): Amlodipine, Nifedipine, Nicardipine **Non-dihydropyridines** (heart rate lowering): Phenylalkylamine class: Verapamil, Gallopamil Benzothiazepine class: Diltiazem	Gingival hyperplasia (dihydropyridines), angioedema, facial flushing, xerostomia, metallic taste (diltiazem)
Diuretics	Hydrochlorothiazide, Furosemide, Spironolactone	Dry mouth

HYPERTENSION –ORAL MANAGEMENT - RISK ASSESSMENT

- General dental treatment possible if blood pressure is controlled
- Drug interactions and side effects
- Decrease endogenous catecholamine release: dental anxiety, "white coat syndrome", manage pain
- Underlying disease or problems (cardiac/renal/endocrine)
- Avoid elective dental treatment if >180/110 mmHg → refer for medical assessment
- Anaesthetic and sedation risks:

BP (Systolic/Diastolic)	Stage	ASA Grade
<140 and <90	-	I
140-159 and 90-99	1 Borderline/Mild	II
160-179 and 95-109	2 Moderate	III
>180 and >110	3 Severe	IV

HYPERTENSION – ORAL MANAGEMENT



Risk factor when evaluating peri-procedural bleeding risk

(Therapeutic Guidelines v4 2026 – Figure 13.73)

HYPERTENSION – ORAL MANAGEMENT

Access:

- Timing / Appointments: Short, minimally stressful, ideally late morning
- Positioning: orthostatic hypotension (ACEIs, ARBs)
- Setting: hospital if severe/uncontrolled hypertension and urgent dental care needed

Education/Prevention

- Common risk factor approach: smoking, alcohol, diet
- Antihypertensives: dry mouth management
- Calcium channel blockers (e.g. amlodipine) → gingiva enlargement
  - Minimise with good oral hygiene and perio debridement
  - Extensive enlargement → specialist periodontal and medical management

HYPERTENSION – ORAL MANAGEMENT

Surgery

- Check BP control prior to treatment
- Manage dental anxiety (exacerbates hypertension)
- Avoid NSAIDs = diuretic + either of:
  - Angiotensin converting enzyme inhibitor (e.g. perindopril)
  - Angiotensin II receptor blocker (e.g. candesartan)
  - → risk of acute kidney injury
- Sedation may be helpful to control stress/blood pressure
  - Pending nature and severity of underlying disease

HYPERTENSION – ORAL MANAGEMENT – VASOCONSTRICTORS IN LA

How much adrenaline-containing LA can we use?

- Profound anaesthesia for pain and anxiety control is important to ↓ endogenous catecholamine release
- LA with adrenaline:
  - Delays systemic absorption
  - Increases duration of anaesthesia
  - Provides local haemostasis
  - Reduces the dose required
- Poses a theoretical risk
  - Stimulates α and β adrenergic receptors (tachycardia, increase BP)
  - Irritates cardiac pacemakers cells (arrhythmias)

HYPERTENSION – ORAL MANAGEMENT – VASOCONSTRICTORS IN LA

How much adrenaline-containing LA can we use?

- Evidence regarding maximum safe dose of adrenaline is lacking
  - Malamed 2004: Most frequently quoted. Recommended max 0.04mg adrenaline per dental appt.
    - ~2 cartridges containing 1:100,000 adr (0.018mg per 1.8ml cartridge, or 0.022mg per 2.2ml cartridge).
  - Tolas et al 1982, Cioffi et al 1985: Evaluated healthy pts following 2% lignocaine 1:100,000 adr injection
    - After 1.8ml (1 cartridge): plasma levels incr 2-3x, no signif changes in BP or HR
    - After 5.4ml (3 cartridges): plasma levels incr 5-6x, signif incr HR and BP
  - Godzieba et al 2014: systematic review of 11 RCTs
    - Use of ≤4 cartridges of lignocaine with 1:100,000 adrenaline seems relatively safe for cardiovascular compromised patients
  - Serrera Figallo et al 2012: systematic review of RCTs
    - Possible to use a dose between 1.8ml and 3.6ml on a general basis in patients with CVD

HYPERTENSION – ORAL MANAGEMENT – VASOCONSTRICTORS IN LA

How much adrenaline-containing LA can we use?

- Conclusion:
  - Use of >2 cartridges of vasoconstrictor-containing LA should be considered a relative rather than absolute contraindication.
  - Current consensus seems to favour judicious use of local anaesthetics containing vasoconstrictors to ensure profound anaesthesia
- Recommendations:
  - If supplementation with plain LA is required, use vasoconstricting LA first
  - Aspirating syringe
  - Intraligamentary injection contraindicated
  - Some report felypressin has fewer haemodynamic effects
  - ?Adrenaline-containing retraction cord astringent

HYPERTENSION – ORAL MANAGEMENT – VASOCONSTRICTORS IN LA

Therapeutic Guidelines v4 2026

- Local anaesthetics containing adrenaline = limited effect on BP elevation
- Avoid in patients with uncontrolled hypertension
  - Consider felypressin (Citanest® = prilocaine + felypressin)
  - Also avoid: MI in previous 6 months, unstable angina, uncontrolled arrhytmias, uncontrolled hyperthyroidism

ISCHAEMIC HEART DISEASE (CORONARY ARTERY DISEASE) – DEFINITION / EPIDEMIOLOGY

- **Definition**: Group of syndromes resulting from myocardial ischaemia – an imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand.
- **Classification / Aetiology**:

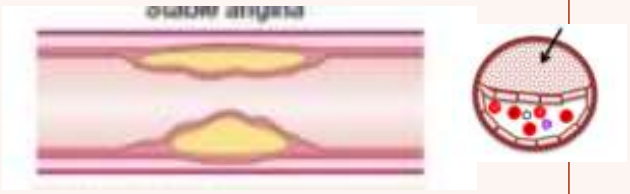
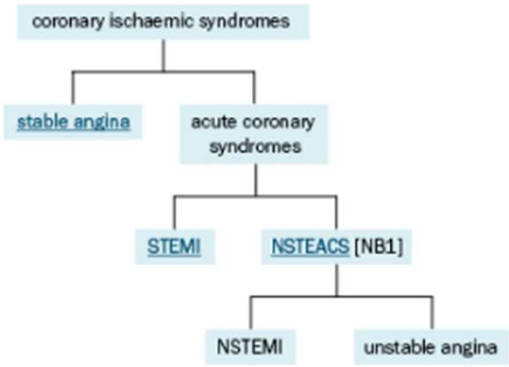


Figure 3.2 Classification of coronary ischaemic syndromes



NSTEACS = non–ST elevation acute coronary syndrome; NSTEMI = non–ST elevation myocardial infarction; STEMI = ST elevation myocardial infarction

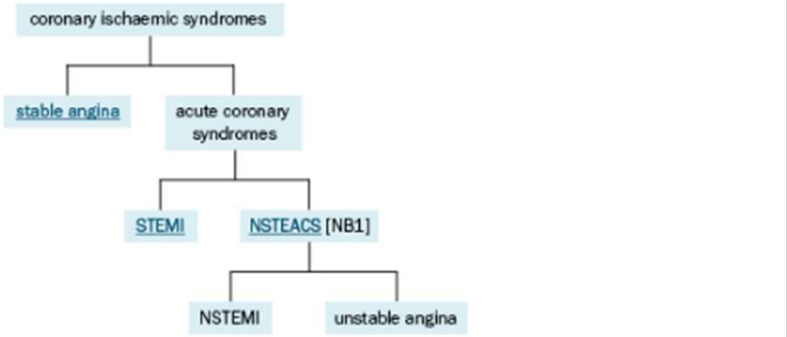
NB1: NSTEACS can progress to STEMI and acute coronary occlusion can occur without ST elevation, so ongoing monitoring is essential.

- **Stable angina**: chronic symptoms
  - Stable atherosclerotic plaque → Fixed coronary obstruction
- **ACS**: new or increasing ischaemic symptoms
  - Unstable atherosclerotic plaque or endothelial disruption → transient/permanent thrombotic occlusion → ischemia and infarction

ISCHAEMIC HEART DISEASE (CORONARY ARTERY DISEASE) – DEFINITION / AETIOLOGY

- **Classification / Aetiology**:

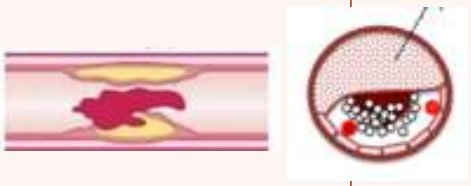
Figure 3.2 Classification of coronary ischaemic syndromes



NSTEACS = non–ST elevation acute coronary syndrome; NSTEMI = non–ST elevation myocardial infarction; STEMI = ST elevation myocardial infarction

NB1: NSTEACS can progress to STEMI and acute coronary occlusion can occur without ST elevation, so ongoing monitoring is essential.

Acute coronary syndromes:



- **Non-ST elevation acute coronary syndrome**
  - Unstable angina = without elevated cardiac biomarkers
  - Non-ST elevation myocardial infarction = pts with MI as determined by elevated cardiac biomarkers



- **ST-elevation myocardial infarction** = medical emergency

→ NSTEACs can progress to STEMI

Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol 2018;72(18):2231-64

1) **Type 1 myocardial infarction:**
    1) Atherothrombotic coronary occlusion compromises myocardial blood flow

2) **Type 2 myocardial infarction:**
    1) Secondary to an imbalance between myocardial oxygen supply and demand (arises from e.g. tachyarrhythmias, sustained bradyarrhythmias, anaemia, respiratory failure and hypotension)
    2) Coronary artery spasm
    3) Spontaneous coronary artery dissection
    4) Coronary embolism

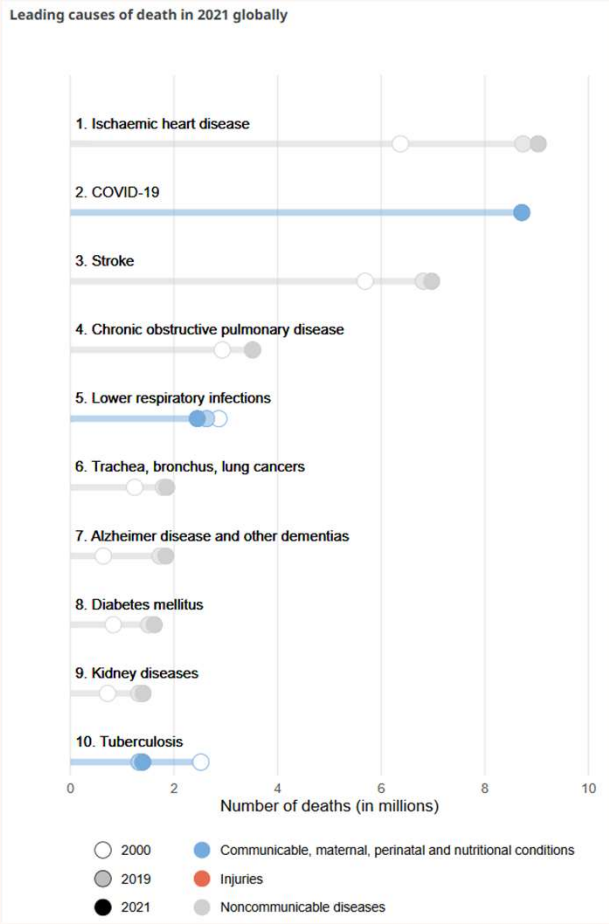
ISCHAEMIC HEART DISEASE (CORONARY ARTERY DISEASE) – AETIOLOGY

	Risk factors for ischaemic heart disease
H	Heredity
A	Age (older)
S	Sex (male)
L	Lipidaemia
I	Increased weight (obesity)
P	Pressure (arterial hypertension)
I	Inactivity (sedentary lifestyle)
D	Diabetes
S	Smoking tobacco

ISCHAEMIC HEART DISEASE (CORONARY ARTERY DISEASE) – DEFINITION / EPIDEMIOLOGY

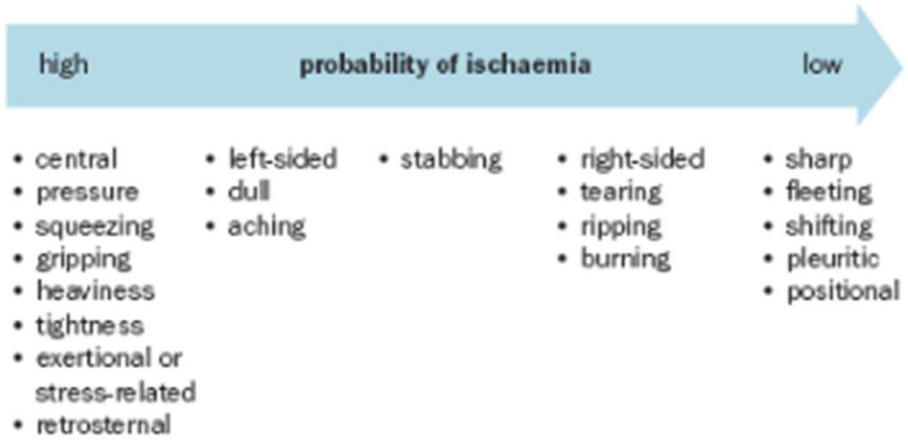
Epidemiology:

- Leading cause of death globally: 13%
- Prevalence: 3% in Australia (higher amongst males)
- Significant decrease in age-standardised IHD mortality rates
  - 1960s: 7/10 MI's fatal ; Today: 7/10 survive a MI
- CVD-related prescriptions increased 7-8 fold in this time



ISCHAEMIC HEART DISEASE (CORONARY ARTERY DISEASE) – GENERAL MANIFESTATIONS

Figure 13.81 Probability that chest pain is ischaemic in origin based on common descriptions of pain



Source: Gulati M, Levy PD, Mukherjee D, Amsterdam E, Bhatt DL, Birtcher KK, et al. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation 2021;144(22):e368-e454. URL

ISCHAEMIC HEART DISEASE (CORONARY ARTERY DISEASE) – GENERAL MANIFESTATIONS

Stable Angina	Acute Coronary Syndromes (eg. MI)	Cardiac Arrest
• Episodic retrosternal chest discomfort (pain, ache or tightness) • May radiate to arms, neck, back, shoulder, jaw • Commonly triggered by physical activity or emotional stress • Predictable, consistent over time • Lasts <10min if provoking stimulus removed • Subsides with rest or GTN	• "Crushing" or heavy central chest pain (more severe) • Shortness of breath, nausea, sweating, pale/clammy skin • Decrease pulse & BP, vomiting, "impending sense of doom" • May occur at rest • Lasts 20min to hours • Not relieved by rest or GTN • *NB: Silent Angina = some may have no pain due to psychological factors persistently inhibiting pain perception (elderly) or autonomic neuropathy (diabetics)*	• Sudden loss of consciousness • No pulse • Not breathing

ISCHAEMIC HEART DISEASE: GENERAL MANAGEMENT

Lifestyle

- ↓ Smoking
- Diet
- ↓ Alcohol
- Physical Activity
- Health weight
- Psychosocial

Pharmacological

- Beta-blockers
- Calcium-channel blockers
- Nitrates
- Nicorandil
- ACE inhibitors
- ARBs
- Antiplatelets
- Lipid-lowering drugs
- Ivabradine

Revascularisation

- Coronary artery bypass grafting (CABG)
- Percutaneous coronary intervention (PCI) = "coronary angioplasty and stent"

ISCHAEMIC HEART DISEASE: GENERAL MANAGEMENT

Therapeutic goal	Pharmacological strategy
Inhibit progression atherosclerosis	Statins - ↓lipids
↑coronary circulation	Nitrates Ca channel blockers
↓heart workload	B blockers
Prevent thrombus formation	Anti-thrombotic agents

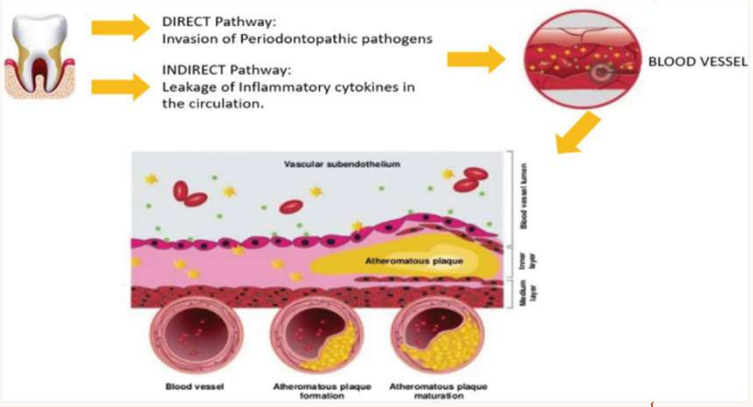
ISCHAEMIC HEART DISEASE: ORAL MANIFESTATIONS

- **Antihypertensive drugs**: as per previous section
- **Nicorandil**: *oral/skin/eye ulceration, facial flushing, lupoid lesions

- Possible association with **periodontitis** (growing evidence)
  - Direct pathway – bacteraemia
  - Indirect pathway – induces chronic systemic inflammatory state

- Possible association with **endodontic disease** (less investigated and inconclusive evidence)
  - apical periodontitis – comparable inflammatory and microbial profile (gram –ve anaerobes) to periodontal disease
  - pulp stones – similarity to calcified stones forming in coronary and renal arteries

- Rarely, **angina pain referred to lower jaw/teeth** mistakenly thought to be of dental origin (clue: onset with exertion)



ISCHAEMIC HEART DISEASE: ORAL MANIFESTATIONS



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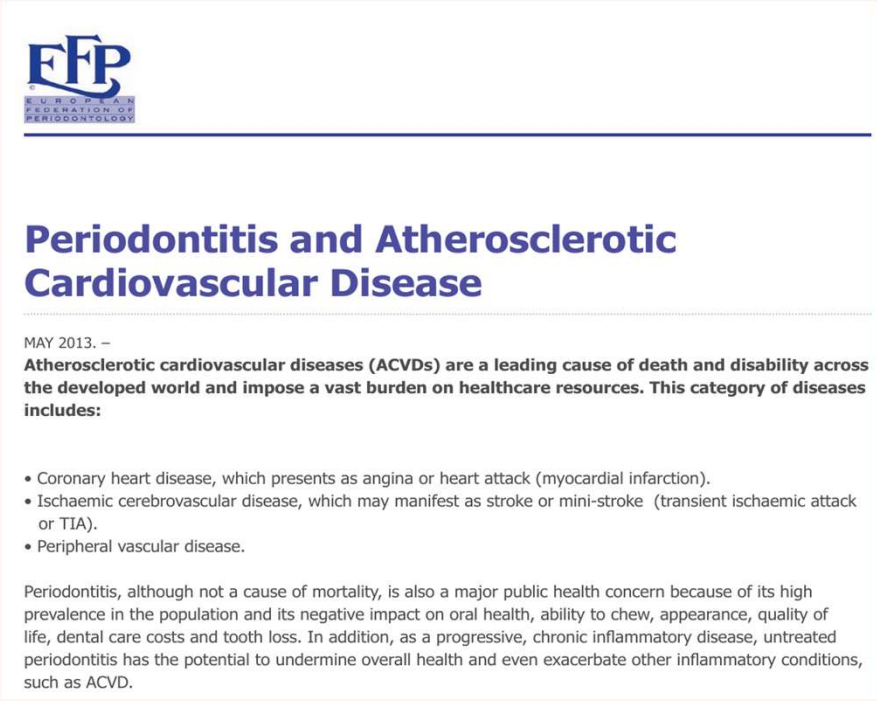
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Periodontal Disease and Atherosclerotic Cardiovascular Disease: A Scientific Statement From the American Heart Association

Andrew H. Tran, MD, MPH, MS, FAHA, Chair, Abbas H. Zaidi, MD, MS, Vice Chair, Ann F. Bolger, MD, FAHA, Oscar H. Del Brutto, MD, Rashmi Hegde, BDS, MS, Lauren L. Patton, DDS, Jamie Rausch, PhD, RN, and Justin P. Zachariah, MD, PhD, FAHA on behalf of the American Heart Association Cardiovascular Disease Prevention Committee of the Council on Lifelong Congenital Heart Disease and Heart Health in the Young; Council on Clinical Cardiology; Stroke Council; Council on Basic Cardiovascular Sciences; and Council on Cardiovascular and Stroke Nursing | AUTHOR INFO & AFFILIATIONS

Circulation • Volume 153, Number 6 • https://doi.org/10.1161/CIR.0000000000001390

[Periodontal Disease and Atherosclerotic Cardiovascular Disease: A Scientific Statement From the American Heart Association | Circulation]



EFP EUROPEAN FEDERATION OF PERIODONTOLOGY

Periodontitis and Atherosclerotic Cardiovascular Disease

MAY 2013. –

Atherosclerotic cardiovascular diseases (ACVDs) are a leading cause of death and disability across the developed world and impose a vast burden on healthcare resources. This category of diseases includes:

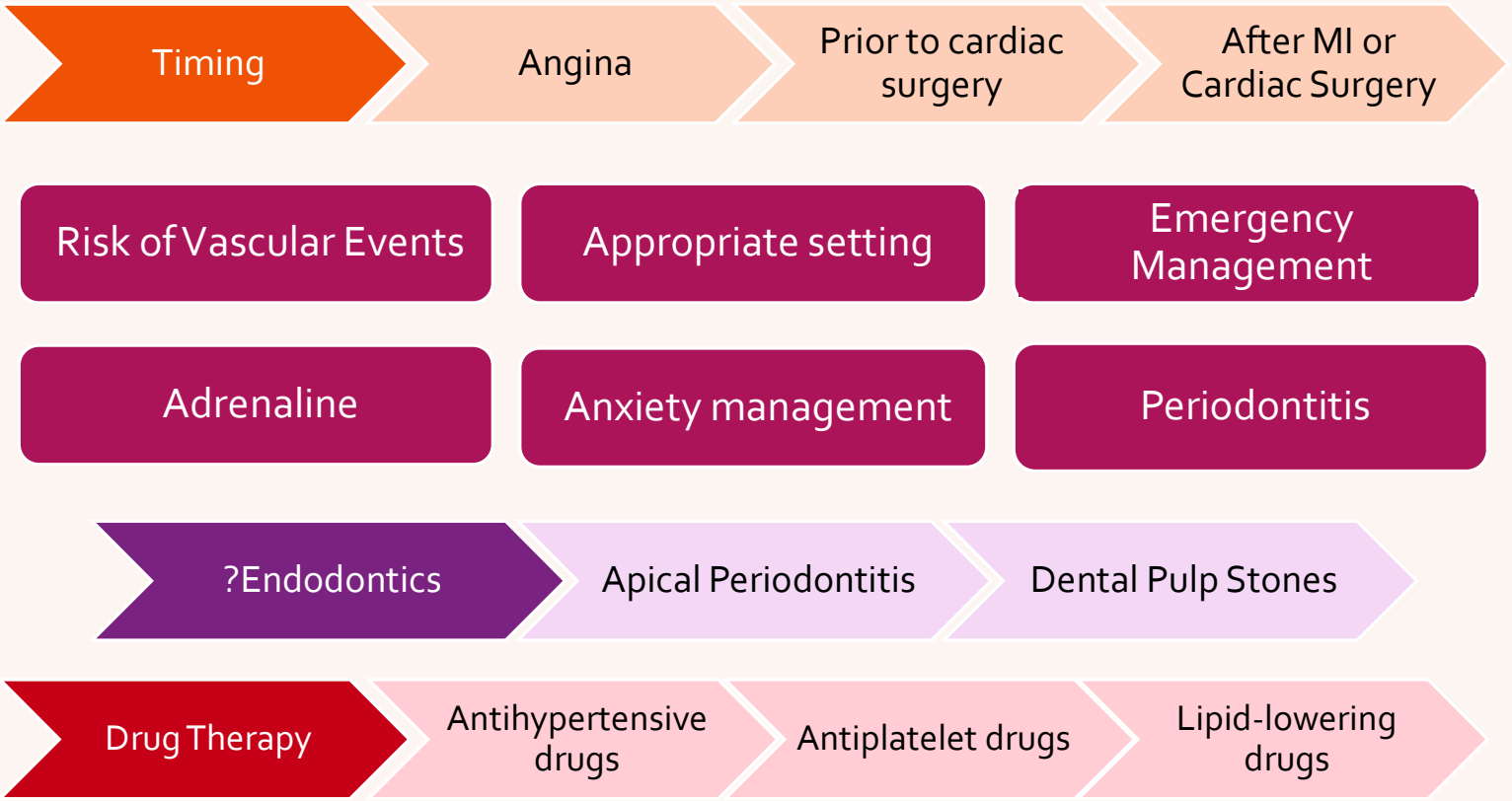
- Coronary heart disease, which presents as angina or heart attack (myocardial infarction).
- Ischaemic cerebrovascular disease, which may manifest as stroke or mini-stroke (transient ischaemic attack or TIA).
- Peripheral vascular disease.

Periodontitis, although not a cause of mortality, is also a major public health concern because of its high prevalence in the population and its negative impact on oral health, ability to chew, appearance, quality of life, dental care costs and tooth loss. In addition, as a progressive, chronic inflammatory disease, untreated periodontitis has the potential to undermine overall health and even exacerbate other inflammatory conditions, such as ACVD.

European Federation of Periodontology: [https://www.bsperio.org.uk/assets/downloads/EFP_cardiovascular-disease-gum-disease.pdf]

Careful medical history at first appointment. Ask about indicators of risk:

- History of chest pain at rest
- Triggers/frequency of episodes
- Carry GTN?
- How episodes are usually managed
- Number of anti-anginal medications
- Hx of CABG/stent placement with ongoing symptoms
- Hx of heart failure


Timing
Angina
Prior to cardiac surgery
After MI or Cardiac Surgery
Risk of Vascular Events
Appropriate setting
Emergency Management
Adrenaline
Anxiety management
Periodontitis
?Endodontics
Apical Periodontitis
Dental Pulp Stones
Drug Therapy
Antihypertensive drugs
Antiplatelet drugs
Lipid-lowering drugs

ACCESS

- **Access to clinic / Mobility**: Functional capacity (cardiopulmonary reserve)
- **Appointments:** Short, minimally stressful appointments. Late morning appointments.
- **Positioning:** Orthostatic hypotension (antihypertensives) → semi-supine and slowly upright
- **Location / setting:** Hospital setting may be required for severe cardiac disease when emergency dental treatment required.
- **Timing**:
  - Patients with history of angina (or MI >6 months earlier) can undergo dental treatment if medically stable
  - Defer elective dental care if unstable angina
  - Defer elective dental procedures for 6 months after MI, stent placement or CABG.
  - Pain and infection within 6 months of above:
    - provide adequate emergency treatment
    - avoid use of adrenaline-containing LA
    - consider specialist advice re: appropriate treatment and timing of procedures

ISCHAEMIC HEART DISEASE: DENTAL MANAGEMENT

CAPACITY
- Nil significant

CONSENT
- Nil significant

EDUCATION/PREVENTION
- Oral side effects of medications
- Possible association with periodontitis +/- endodontic disease

SURGERY

Pre-operative

- Instruct patients with angina to bring their medication (e.g. GTN spray or tablets), and have it readily accessible.
- Ensure GTN and oxygen readily available
- Monitor SpO_2, BP, HR

Peri-operative

- Use relaxation techniques and consider other measures for anxiety management
- Limit the duration of dental procedures
- **Bleeding**: may be on antithrombotics (see *Haematology – Bleeding Disorders* lecture)
- **LA :** Ensure effective analgesia → use of adrenaline (see *Hypertension)*
- **Sedation:** anxiety relief helpful (see *Hypertension)*

Post-operative

- Caution with NSAIDs

ISCHAEMIC HEART DISEASE – MANAGEMENT OF CHEST PAIN IN DENTAL PRACTICE

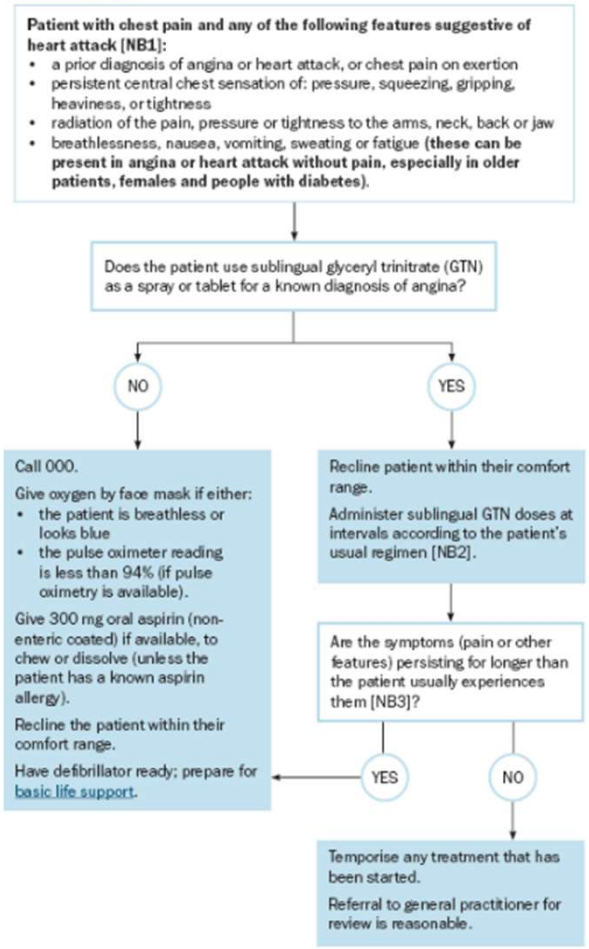
NB1: Many people with chest pain do not have angina or a heart attack, but anyone with acute chest pain requires rapid medical evaluation for potentially life-threatening causes. The likelihood of angina or heart attack increases with the number of suggestive features.

NB2: Sublingual glyceryl trinitrate (GTN) dosing is a 400 microgram spray (or 300 to 600 microgram tablet) generally administered every 5 minutes to a maximum of 3 doses. Ask patients who use GTN to bring their GTN to their dental appointment so that it can be administered if chest pain occurs.

NB3: If symptoms (pain or other features that may indicate angina) last longer than the patient's usual angina symptoms, urgent medical evaluation of a possible heart attack or other urgent condition is required.

Figure 13.82 Management of chest pain in dental practice

Printable figure



ISCHAEMIC HEART DISEASE – MANAGEMENT OF CHEST PAIN IN DENTAL PRACTICE

If administering nitrates, recline the patient because blood pressure can be reduced by administration. To manage chest pain in a patient with a history of angina, use:

1. glyceryl trinitrate spray 400 micrograms sublingually, repeat every 5 minutes if pain persists, up to a total of 3 doses if tolerated

OR

1. glyceryl trinitrate tablet 300 to 600 micrograms sublingually, repeat every 5 minutes if pain persists, up to a total of 3 doses if tolerated.

To manage chest pain if angina or heart attack is suspected, provided no contraindication (eg aspirin allergy) is present, use:

aspirin (non-enteric coated) 300 mg orally, chewed or dissolved before swallowing.

THROMBOEMBOLIC COMPLICATIONS OF CVD - AETIOLOGY

- **Arterial thrombi**: platelet aggregation + fibrin + erythrocytes
- **Venous thrombi**: slow & turbulent blood flow, ↑ risk small emboli detaching & blocking pulmonary arteries

THROMBOEMBOLIC COMPLICATIONS OF CVD – GENERAL MANIFESTATIONS AND MANAGEMENT

Arterial thrombi	Venous thrombi
TIA/stroke	DVT
Occlusion of coronary artery grafts	Pulmonary embolism
MI & death in unstable angina	
Systemic embolism with prosthetic heart valves/AF	

General management

- Antithrombotic agents: prevent thrombus formation (initiation)
- Anticoagulants: prevent coagulation (clot formation)

Dental management

- Antithrombotic drug use → bleeding (see *Haematology - Bleeding Disorders* lecture)

VALVULAR

Rheumatic fever and Rheumatic heart disease

Infective endocarditis

Aortic and mitral valve stenosis, regurgitation and prolapse

RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

DEFINITION

- Rheumatic Fever: An acute, immunologically-mediated, systemic inflammatory condition characterised by fever and arthritis 2-4 weeks after pharyngitis. Caused by certain strains of Group A beta-haemolytic streptococci (e.g. following impetigo and scabies).

EPIDEMIOLOGY

- 1.06 Acute rheumatic fever and rheumatic heart disease - AIHW Indigenous HPF
- RHD is a disease of disadvantage that is both preventable and treatable.
- First Nations people in Australia have one of the highest recorded rates of ARF and RHD in the world (119 per 100,000), with incidence rates increasing (50% increase 2012-2021)
- First Nations people accounted for 92% of the total number of ARF cases in Australia recorded over the 5-year period 2017–2021
- Otherwise rare disease in Western world, common in Indian subcontinent, Middle East, some Caribbean islands.

RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

AETIOLOGY

- Group A streptococci
- RHD: repeated episodes of RF
- Autoimmune reaction: antibodies produced to fight streptococci cross react with enzymes of the heart

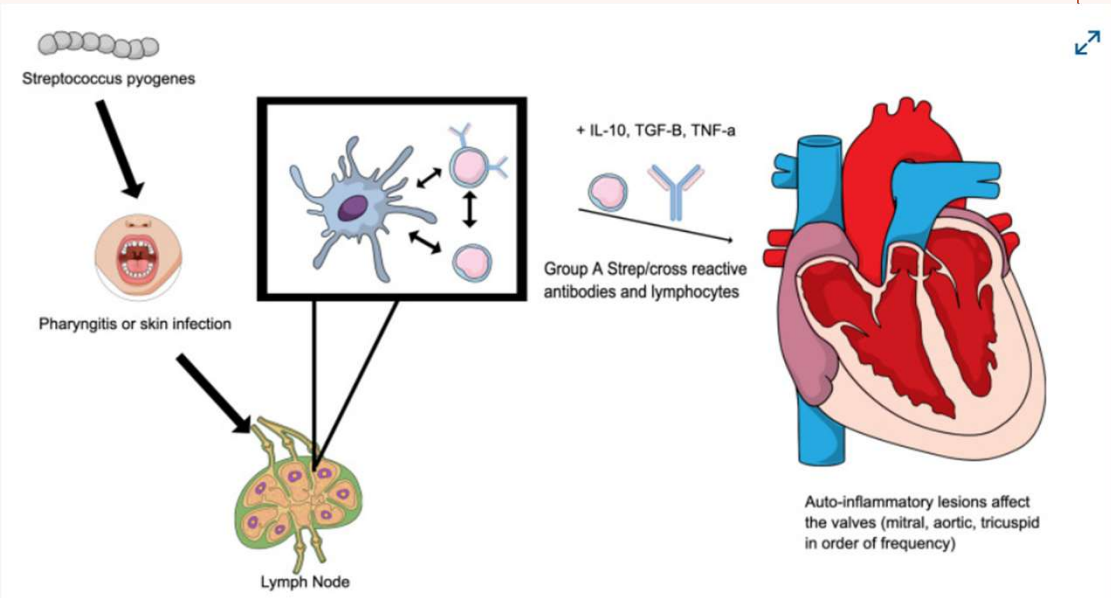


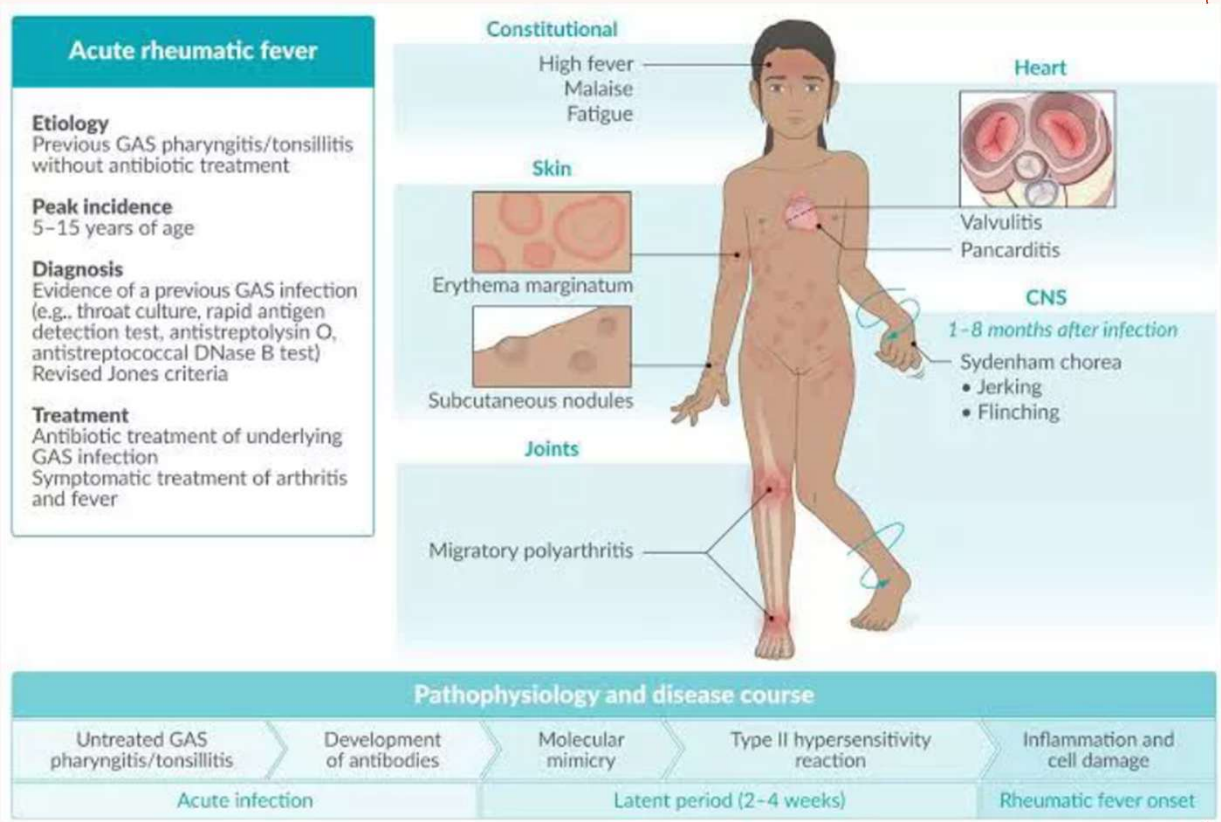
Figure 2 Pathophysiology of RHD. RHD occurs as a sequela of GAS infection, especially pharyngitis. Social determinants of health and genetic predisposition play a role in repeated infection as well as in the progression to RHD. Cross-reactive antibodies and immune cells form in response to GAS infection and attack endothelial cells throughout the body; however, damage to the endothelial surface of the valves is not repaired effectively.

Image: Simpson MT, et al. Rheumatic Heart Disease in the Developing World. *Structural Heart*. 2023; 7(6): 100219

RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

GENERAL MANIFESTATIONS

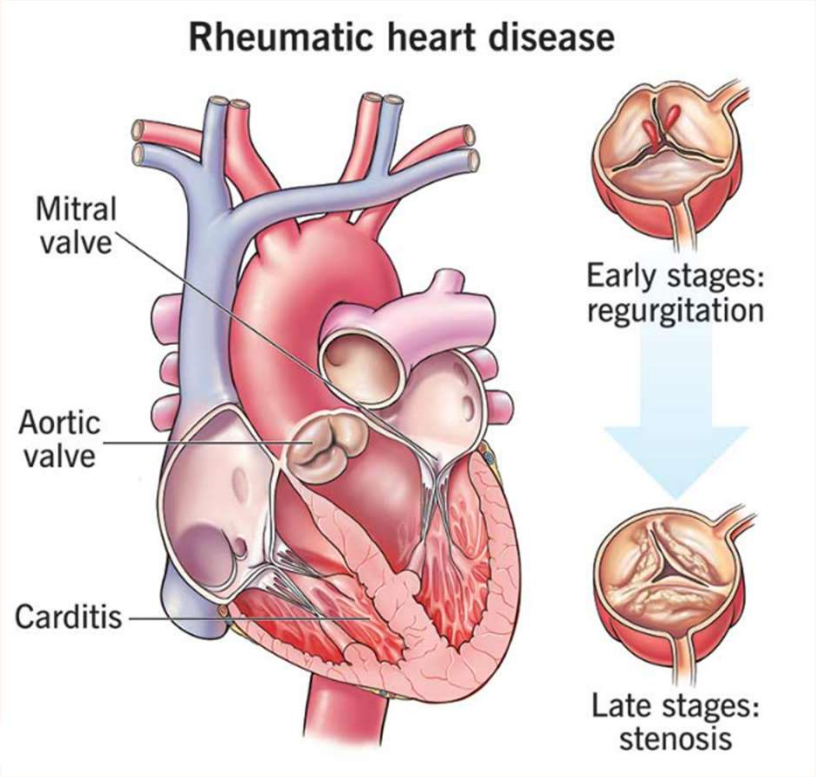
- Acute Rheumatic Fever
- Subacute/chronic Rheumatic Fever



RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

GENERAL MANIFESTATIONS

- Rheumatic Heart Disease
  - Valvular damage → regurgitation, stenosis → IE risk
  - Early heart disease: Heart murmur
  - Late effects: heart enlargement, cardiac failure
  - Other:
    - Shortness of breath
    - Chest pain
    - Fatigue
    - Swollen legs and feet



RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

Diagnosis	Treatment	Prognosis
Physical examination History collection Echocardiogram Electrocardiogram Blood tests	Antibiotic prophylaxis (benzathine benzylpenicillin G) Complications: • Anticoagulants • Balloon valvuloplasty • Heart valve repair or replacement • Heart failure management • Lifestyle and prevention	• Acute phase last 6-12 weeks, definitive cure up to 6 months • RF recurrence rate is high (50% in first 5 years) → prophylactic therapy with penicillin • Mortality rate: 0.5 per 100,000

INFECTIVE ENDOCARDITIS

DEFINITION:

A rare, but dangerous, infection of the endocardial surface of the heart. Predominantly affects one or more heart valves, especially where there is turbulent blood flow because of valve damage (typically left sided valves).

EPIDEMIOLOGY:

- Relatively uncommon illness with high morbidity and mortality
- 2010: Australian incidence 4.7 cases per 100 000 person-years
- In-hospital mortality variable – increases with age (up to 15% in 80-89 yos)

INFECTIVE ENDOCARDITIS - AETIOLOGY

- Endothelial sites with turbulent blood flow
- → Platelet and fibrin deposits accumulate (non-thrombotic endocarditis)
- → sterile "vegetations" produced
- → can be readily infected if a subsequent bacteraemia occurs

Causative organisms: Streptococci viridans (oral) 50-60%, Enterococci (faecal strep) 10%, Staphylococci (skin) 20-30%, Others 10% (Gram negative bacilli – mainly HACEK group, fungi)

INFECTIVE ENDOCARDITIS - AETIOLOGY

ASSOCIATION WITH DENTISTRY:

- Dental procedures involving bleeding can initiate a bacteraemia that may lead to endocarditis (usually transient and <15min, but occasionally up to 1 hour)

- Most common types of bacteraemia in IE:
  - S mutans and S sanguis (both Strep viridans) from dental plaque [40% of IE causative organisms due to complex attachment mechanisms enabling them to adhere to endocardium]
  - Gram negative bacilli (HACEK group, including AA) occasionally implicated

- Chance of dental extraction causing IE in patient with valvular disease may be as low as 1 in 3000, therefore bacteraemia not synonymous with IE (or would need antibiotic cover for brushing).

Dental treatment is not the only source of IE-bacteraemia

➔ Susceptible surface + high circulating bacterial loads

INFECTIVE ENDOCARDITIS - AETIOLOGY

- **High risk of IE**: previous IE, prosthetic heart valves or valves repaired with prosthetic material, unrepaired cyanotic congenital heart disease, or some repaired congenital heart detects (up to 6 months after the procedure or lifelong if residual shunt or valvular regurgitation remains), +/- cardiac transplantation with cardiac valvulopathy.
- **Moderate risk:** previous rheumatic fever, heart murmur, or evidence of native valve disease.

Table 5.32 *Main groups affected by infective endocarditis*

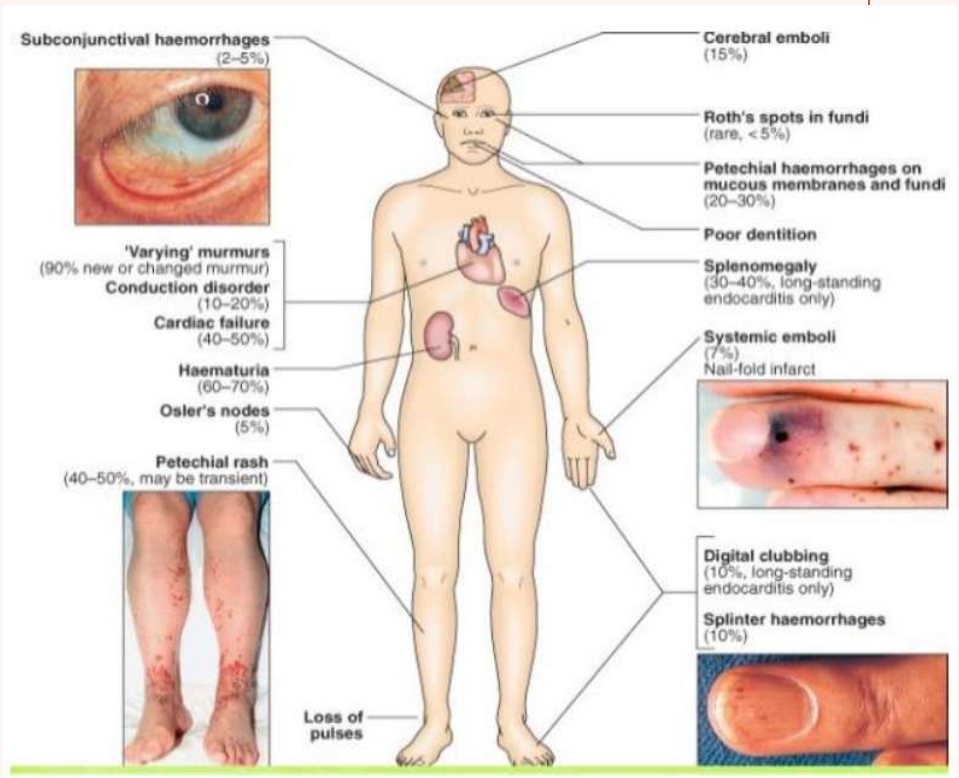
Aetiology	Approximate % of all cases of infective endocarditis
No obvious cardiac valve disease	40
Chronic rheumatic heart disease	30
Congenital heart disease	10
Prosthetic cardiac valves	10
Intravenous drug abuse	10

INFECTIVE ENDOCARDITIS – GENERAL MANIFESTATIONS

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Variable (flu-like) symptoms	Septic emboli (20-45%) – Systemic manifestations	Cardiac manifestations	Immune complex formation from antigens' resultant antibodies
Fever Weight loss Headache Malaise Joint pain SOB/cough Chest pain (esp IVDUs)	• Infarcts brain (→ stroke), limbs, spleen (→ splenomegaly), *kidney (→ haemuria), pulmonary. • Retinal necrosis and blindness • Coronary artery embolism • Mycotic aneurysms • Focal glomerulonephritis	Sepsis Tachycardia Hypotension Valve destruction Murmur Pericarditis	Vasculitis Arthritis Renal damage

Common signs: 90% fever, 85% heart murmurs



INFECTIVE ENDOCARDITIS – GENERAL MANAGEMENT

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Treatment

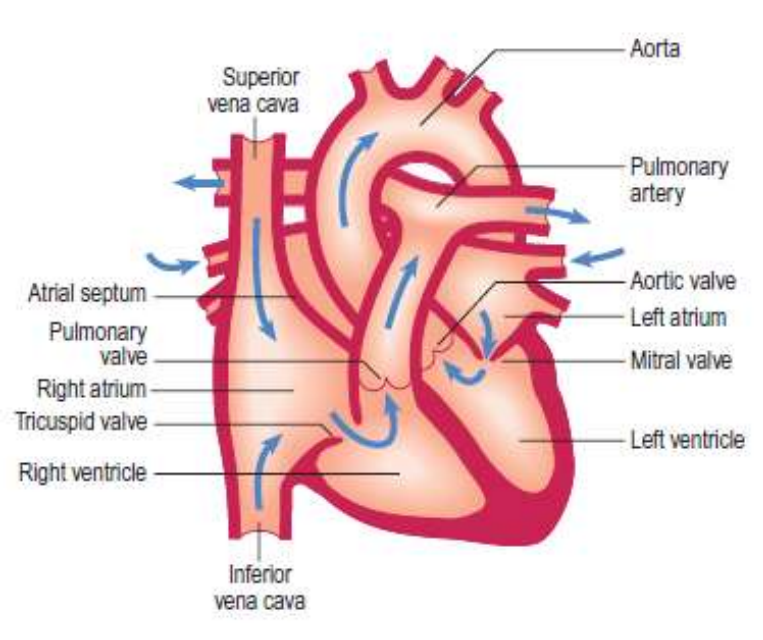
- Hospital admission for early treatment
- Antibiotic therapy
- Severe cases: valve replacement

Prognosis

- 15-20% die during initial hospital admission; further 10-15% die over the following year
- 40-45% require surgery during initial hospital admission; further 10% require surgery within 1 yr post-discharge
- Good prognostic factors: young, penicillin-sensitive isolates, early tx

AORTIC AND MITRAL VALVE STENOSIS, REGURGITATION AND PROLAPSE

- **Aortic stenosis:** narrowing of the aortic valve; this generates an increased pressure in the left ventricle to maintain the blood volume per beat
- **Aortic regurgitation**: blood propelled to the aorta during systole returns to the left ventricle during diastole due to a defective aortic valve
- **Mitral valve stenosis**: Narrowing of the mitral valve that generates an increasing pressure in the left atrium of the heart
- **Mitral valve regurgitation**: The blood propulsed to the left ventricle during systolic contraction returns into the left atrium during diastolic dilatation due to a valve closure defect
- **Mitral valve prolapse**: Protrusion of the valves into the left atrium during systole. May exist in isolation, but sometimes associated with mitral regurgitation. May be associated with atrial septal defect, patent ductus arteriosus.

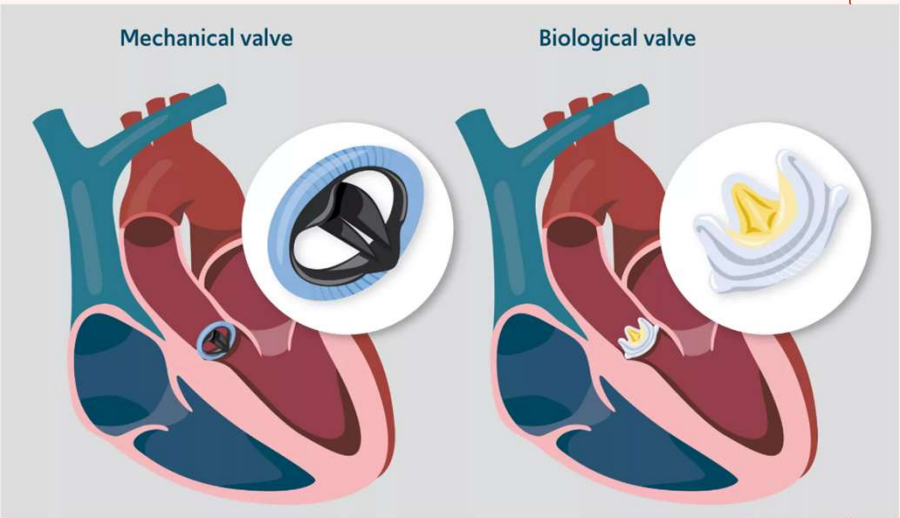


➔ DO NOT REQUIRE ANTIBIOTIC PROPHYLAXIS

VALVE REPLACEMENT

- **Tissue valve**
  - Material: porcine, bovine, human
  - Lifespan = 10-20 years
    - Usually for older patients
  - No turbulence = no anticoagulation required
    - Unless required for other reason e.g. AF, DVT
  - No noise

- **Mechanical valve**
  - Material: carbon, titanium
  - Lifespan = highly durable, can last lifetime (30+ years)
    - Usually for younger patients
  - Turbulence = anticoagulation required
  - Noisy



VALVULAR HEART DISEASE – RISK ASSESSMENT

- Bacteraemia to heart valves
- Oral hygiene
- Timing to perform dental treatment
- Anticoagulation
- Oral side effects of diuretics (e.g., furosemide), ACE inhibitors, and beta-blockers
- GA/sedation risks: myocarditis
- Concurrent antibiotic therapy

VALVULAR HEART DISEASE – DENTAL MANAGEMENT

DENTAL MANAGEMENT

- ACCESS:
  - Postpone elective dental care 6 months after episode of RF
  - RHD → IE or valve surgery = urgent management of dental infections (bacteraemia)
    - May be hospital in-patient setting
  - IVDUs: less reliable attendance unless in pain

- CAPACITY/CONSENT:
  - Nil significant

- EDUCATION/PREVENTION
  - Good oral hygiene most important factor in reducing risk in susceptible individuals
  - Preventive measures to avoid caries and periodontal disease
    - OHI for home maintenance, regular dental attendance, diet, etc
  - Patient educated to alert dental providers of RHD/IE history, and need for antibiotic prophylaxis

DENTAL MANAGEMENT

- SURGERY:
  - Antibiotic prophylaxis (see lecture *Antibiotic Prophylaxis and Dentistry*)
    - Liaise with Infectious Diseases physician if on concurrent antibiotics
  - Consult with physician re: anticoagulation (see lecture *Bleeding Disorders and Dentistry*)
  - LA: use adrenaline with caution, aspirating syringe (IV injection may precipitate arrhythmias)
  - Conscious sedation / GA: avoid when acute RF (myocarditis), hospital environment if required

- SPREAD
  - Infective endocarditis bacteraemia (antibiotic prophylaxis)
  - Possible antibiotic resistance issues

ANTIBIOTIC PROPHYLAXIS

Table 2.5 Indications for antibiotic prophylaxis for endocarditis

Printable table

Antibiotic prophylaxis against infective endocarditis is recommended **only** for people who meet **both** of the following criteria:

- are undergoing a procedure associated with a high risk of a bacteraemia and subsequent infective endocarditis
- have a cardiac condition associated with an increased risk of developing infective endocarditis and the highest risk of adverse outcomes from endocarditis.

Procedures for which endocarditis prophylaxis is recommended for patients with a cardiac condition listed below [NB1]

- Dental procedures – only those involving manipulation of the gingival or periapical tissue or perforation of the oral mucosa (eg dental extractions, implant placement, biopsy, removal of soft tissue or bone, subgingival debridement, replanting avulsed teeth) [NB2] [NB3] [12] [33] [44].

NB1: Endocarditis prophylaxis is not recommended for procedures other than those covered in this topic. However, surgical prophylaxis may be indicated even if endocarditis prophylaxis is not – see Surgical antibiotic prophylaxis recommendations for specific procedures.

NB2: For a list of dental procedures classified by invasiveness and their indications for endocarditis prophylaxis, see Table 13.29.

NB3: Evidence on the risk of bacteraemia and subsequent infective endocarditis following supragingival debridement is conflicting. Pending further evidence, endocarditis prophylaxis is only recommended for supragingival debridement that involves manipulation of the gingival tissue [49] [50].

Cardiac conditions for which endocarditis prophylaxis is recommended for patients undergoing a procedure listed above [NB5] [NB6] [12] [48] [56]

- prosthetic cardiac valve, including transcatheter-implanted prosthesis or homograft
- prosthetic material used for cardiac valve repair, such as annuloplasty rings and chords
- previous infective endocarditis
- ventricular assist devices [36] [56]
- congenital heart disease **but only** if it involves:
  - unrepaired cyanotic defects, including palliative shunts and conduits
  - repaired defects with residual defects at or adjacent to the site of a prosthetic patch or device (which inhibit endothelialisation) [33] [48]
- rheumatic heart disease [NB7].

NB5: Endocarditis prophylaxis is not recommended for patients with forms of valvular or structural heart disease not listed in this table, including patients with mitral valve prolapse, septal defects or cardiac implantable electronic devices.

NB6: Patients with a heart transplant who have developed cardiac valvulopathy may also be at high risk of adverse outcomes from endocarditis; consult the patient's cardiologist for specific recommendations.

NB7: Endocarditis prophylaxis was previously only recommended for rheumatic heart disease in Aboriginal and Torres Strait Islander peoples, or considered for people with rheumatic heart disease at significant socioeconomic disadvantage. However, studies have demonstrated that rheumatic heart disease is an independent risk factor for the development of and adverse outcomes from infective endocarditis [1] [3] [20].

Therapeutic Guidelines / Antibiotic / Infective endocarditis / Prevention of infective endocarditis [accessed July 2026]

ELECTRICAL (DYSRHYTHMIAS)

Extrasystoles (ectopics)

Tachycardia (including Atrial fibrillation)

Bradycardias

DYSRHYTHMIAS

- **Definition**: An alternation in the normal rhythm of the heartbeat.
- **Epidemiology:** 15% of the population
- **Aetiology**: disorders in heart automaticity (impulse formation) or conductivity (block or delay)

Abnormal impulse generation	Abnormal impulse conduction
Sinus bradycardia <60 bpm Sinus tachycardia >100bpm Atrial flutter Atrial fibrillation – acute/chronic Premature ventricular contractions Ventricular tachycardia Ventricular fibrillation Ventricular extrasystole	AV Block – delay/failure conduction from atria to ventricles

DYSRHYTHMIAS - AETIOLOGY

Causes

- **Cardiac disease**
- **Drugs**: caffeine, alcohol, smoking, β-2-agonists, digoxin, dopamine, tricyclics, tobacco
- **Metabolic changes**: catecholamines, autonomic/endocrine disease, hyperthyroidism, fever, hypoxia, surgery, persistent vegetative neurological states
- **Electrolyte imbalance**: e.g. secondary to CKD
- **Respiratory**: COPD

DYSRHYTHMIAS – GENERAL MANIFESTATIONS

Signs/symptoms

- Most frequent symptom: Palpitations.
- Others: sickness, syncope, dyspnoea, anxiety, fatigue, chest pain, dizziness.
- May have no symptoms

Classification:

- Rate:
  - Tachycardia (fast rate >100bpm)
  - Bradycardia (slow rate <60bpm day, <50bpm night)
  - Irregular
- Mechanism: automaticity, re-entry, fibrillation
- Origin: atrium, ventricle

DYSRHYTHMIAS – GENERAL MANIFESTATIONS

Types of Arrhythmias

Extrasystoles
Premature beats
Atrial extrasystoles
Ventricular extrasystoles

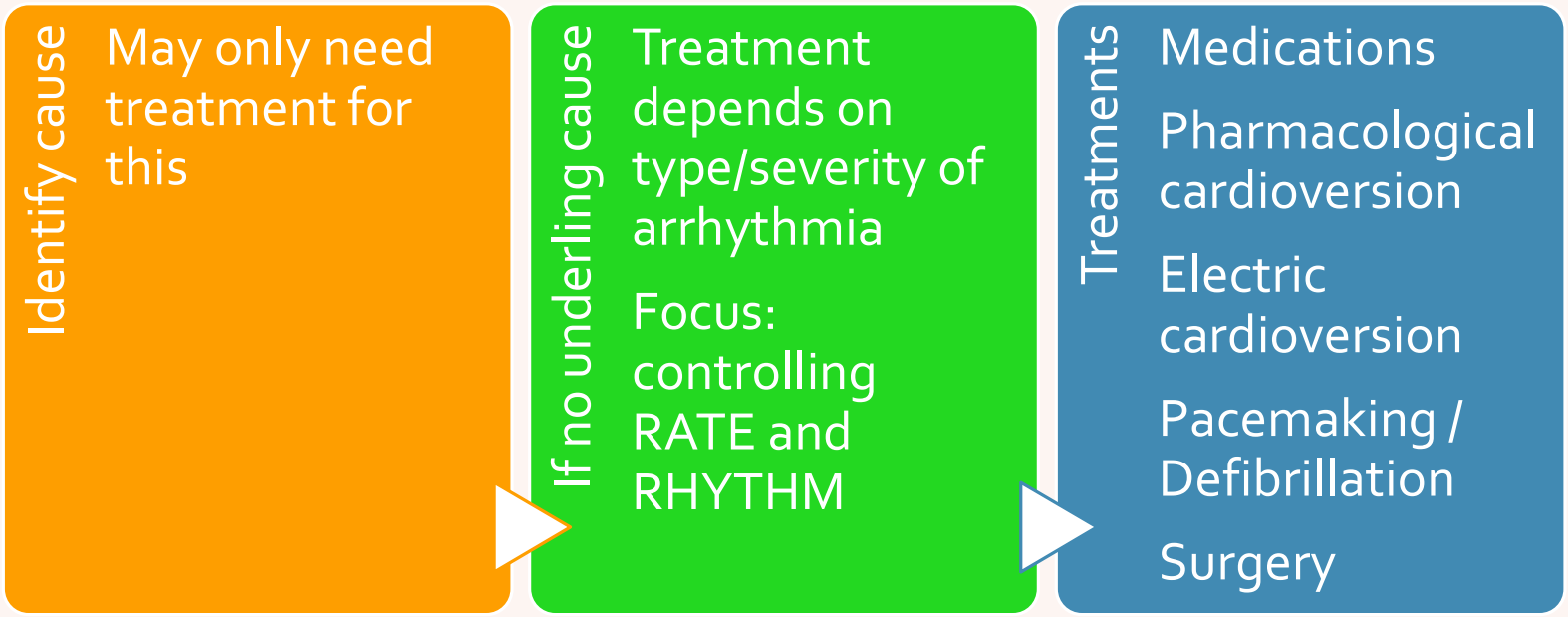
Inherited Disorders
Long QT syndrome
Brugada syndrome

Bradycardias
Sinus bradycardia
Sinus arrest
Atrioventricular block, bundle branch block, heart block

Tachycardias
Sinus tachycardia
Sinus node re-entrant tachycardia
Atrioventricular tachyarrhthmia
Supraventricular tachycardia
- Atrial flutter
- *Atrial fibrillation
  - Paroxysmal AF
  - Persistent AF
  - Permanent AF
- Paroxysmal atrial tachycardia
- Paroxysmal supraventricular tachycardia
- Wolff-Parkinson-White syndrome

Ventricular tachycardia
Ventricular fibrillation

DYSRHYTMIAS – GENERAL MANAGEMENT



DYSRHYTMIAS – GENERAL MANAGEMENT

Medications

- Control abnormal heart rhythm: anti-arrhythmic drugs (Vaughan Williams Classification)
- Treat related hypertension/CAD/heart failure: adenosine, digoxin
- Reduce stroke risk: DOACs

- Antiarrhythmic drugs (Vaughan Williams classification)

Class	Drug	Action
Class I	Membrane depressant drugs – quinidine, disopyramide	Sodium channel blockers – may slow conduction, delay recovery or reduce spontaneous discharge rate = VT or AF
Class II	Beta-adrenoceptor blocking drugs - propranalol	Anti-sympathetic nervous system. Decrease MI mortality, prevent recurrence of tachyarrhythmias
Class III	Drugs that prolong action potential	Potassium channel blockers. Amiodorone. Used for atrial flutter and AF
Class IV	Calcium-channel blockers – verapamil and diltiazem	Reduce ventricular rate in those with AF.
Other	Direct nodal inhibition - Adenosine and digoxin	Used for heart failure with AF

DYSRHYTHMIAS – GENERAL MANAGEMENT

Cardioversion

- Restores regular rhythm
- Pharmacological (chemical) cardioversion
- Electrical cardioversion

Cardiovascular Implantable Electronic Devices (CIEDs)

- Permanent pacemakers
- External cardiac defibrillators
- Ventricular assistive devices (VAD)
- Implantable Cardioverter Defibrillator (ICD)

Radiofrequency Ablation (Catheter Ablation)

DYSRHYTHMIAS – DENTAL MANIFESTATIONS

Anti-arrhythmic drugs

- Gingival swelling = verapamil/diltiazem (Ca-channel blockers), enalapril (ACE inhibitor)
- Lichenoid ulceration and oral ulceration related to agranulocytosis = some β-blockers
- Lupus-like reaction and mucosal ulcers due to agranulocytosis = procainamide
- Xerostomia = disopyramide

- Anticoagulation (see *Bleeding Disorders* lecture)
- Oral side effects of drugs (see *Hypertension* section)
- CIED interference

DYSRHYTMIAS – CARDIAC IMPLANTABLE ELECTRONIC DEVICES

- Patients with CIED can undergo most general dental treatment.
- Surgical diathermy can interfere with some CEIDs (especially monopolar)
- With modern CIEDs, interference from most other dental electronic and ultrasonic devices is usually not clinically significant
  - Consult cardiologist if in doubt
- CIED is not an indication for surgical nor IE antibiotic prophylaxis
- LVADs increase risk of thrombotic events ∴ increased risk of post-op bleeding
  - patients may be on antithrombotics
  - LVAD-induced blood pressure changes

DYSRHYTMIAS – CARDIAC IMPLANTABLE ELECTRONIC DEVICES

Further info on interference (American Dental Association):

https://www.ada.org/resources/ada-library/oral-health-topics/cardiac-implanted-devices-and-electronic-dental-instruments

Table 1. Tests of dental devices and reported electromagnetic interference.

Device	Reported Electromagnetic Interference	
	In-Vitro (laboratory testing)	In-Vivo (in people)
Ultrasonic dental scalers		
Piezoelectric	None,[15] Minor to Severe[6, 11]	None,[1] Minor[13]
Magnetostrictive	None,[15] Minor[4]	Minor[10]
Electronic apex locators	None,[14, 21, 22] Minor[2, 6, 11]	Minor[8, 13] to Severe[8]
Electric pulp testers	None,[4, 14] Minor,[6, 11, 18] Severe[6, 11]	None,[10] Minor[13]
Electrosurgery instruments	None,[4] Minor[6, 11, 18] to Severe[6, 11, 14, 18]	N.A.
Curing lights	Minor,[18] Severe[4]	None[10]
Gutta percha devices	None,[18] Minor[2]	Minor[8]
Ultrasonic cleaning systems	Severe[4]	Minor[10]
Electric toothbrushes	None[4]	Minor (rare)[10]
Osseointegration tools	Minor to Severe[6, 11]	N.A.

"Minor" refers to non-clinically significant interference (noise, telemetry); "Severe," to clinically significant outcomes (i.e., pacing disturbances or shock); "N.A." (Not Applicable), no studies available.

DYSRHYTHMIAS – DENTAL MANAGEMENT

Access

- Peak endogenous epinephrine levels in early morning → ideally appointments late morning or early afternoon
- High risk patients – treated in hospital

Education/Prevention:

- Oral side effects of medication

Surgery

- Liaise with cardiologist if concerned about CIED interference
- Pre-operative: anxiety management, check INR if warfarinated, defibrillator readily available
- Peri-operative:
  - Positioning: supine if pacemaker
  - LA: adequate provision, judicious use of vasoconstrictor, avoid intravenous injection
  - Sedation: oxygen saturation monitoring, may be helpful to minimise stress
- Post-operative: haemostasis if anticoagulated

MUSCULAR (CARDIOMYOPATHIES)

Primary – genetic/inherited, acquired

Secondary

CARDIOMYOPATHIES

Definition: broad term for diseases of the heart muscle that result in reduced ability for the heart to pump blood to the rest of the body.

Aetiology

Primary	Secondary
Genetic/Inherited • Hypertrophic cardiomyopathy (HCM) • Dilated cardiomyopathy (DCM) • Arrythmogenic R ventricular (ARVC) Acquired • Alcohol, chemotherapeutics *(most common acquired cause)* • Tako-tsubo cardiomyopathy • Tachycardia induced • Muscular dystrophies	• Storage: haemochromatosis, Fabry disease • Infiltrative: amyloidosis, Hurlers syndrome • Toxicity: alcohol, radiation • Nutrition: Beri-beri, Kwashiorkor • Autoimmune: SLE, RA • Neuromuscular: Friedrich's ataxia • Endocrine: Diabetes, thyroid disease, phaechromocytoma

CARDIOMYOPATHIES – GENERAL MANIFESTATIONS AND MANAGEMENT

Signs/Symptoms	Management
• Oedema (pulmonary, peripheral – ankles & legs, abdominal) • Thrombi/emboli/clot formation • Exertional and paroxysmal nocturnal dyspnoea (SOB) • Arrhythmias • Nausea • Chest pain • Palpitations • Fainting • Sudden cardiac death	• Lifestyle changes: diet, exercise, stress, avoid tobacco/alcohol • Medications: hypertension drugs, anti-arrhythmic drugs, oral anticoagulants. Often: beta blockers, potassium channel blockers. Other: CCBs (verapamil, diltiazem) +/- ACEIs, diuretics, digoxin. • Devices to correct arrhythmias: pacemakers, ICDs • Devices to improve blood flow: LVAD, cardiac resynchronisation therapy (CRT) • Procedures: catheter ablation, heart transplant

CONGENITAL

Cyanotic

Acyanotic

CONGENITAL HEART DISEASE

Definition

- Any congenital structural defects of the heart (including valve stenosis or septal defects) or adjacent great vessels that lead to malfunction (arrhythmias or flow problems). Can be alone or in combinations.

Epidemiology

- Is the most common type of heart problem in children (1% live births). 20% also have other congenital anomalies.
- Diagnosed in at least 1 in 180 births (1-2% of the population)

Aetiology

- Often unidentified, multifactorial, genetic, and environmental factors.
- Acquired causes:
  - maternal infections (Rubella, Cytomegalovirus)
  - maternal drug use (alcohol, anticonvulsants, lithium, thalidomide, warfarin, other drugs
  - maternal systemic disease (SLE, diabetes); maternal irradiation (rare).
- Genetic syndromes with cardiac detects:
  - Chromosomal: Downs, Edwards, Turners
  - Hereditary: Ehlers-Danlos syndrome, Marfan syndrome, osteogenesis imperfecta, mucopolysaccharidosis, Holt-Oram syndrome, Noonan syndrome, Williams syndrome

CONGENITAL HEART DISEASE – GENERAL MANIFESTATIONS

Classification

- Cyanotic (R to L shunting): shunting deoxygenated blood from the right ventricle into the left side of the heart and the systemic circulation (bypassing the lungs)
- Acyanotic (obstruction, or L to R shunting): blood contains enough oxygen but is pumped abnormally around body, forcing the heart to work harder

Cyanotic	Acyanotic, no shunt	Acyanotic with shunt
• Tetralogy of Fallot • Eisenmenger syndrome • Pulmonary atresia • Pulmonary valve stenosis • Total anomalous venous drainage • Transposition of the great vessels • Tricuspid atresia	• Aortic stenosis • Bicuspid aortic valve • Coarctation of the aorta • Dextrocardia • Mitral valve prolapse	• Atrial septal defect • Patent ductus arteriosus • Ventricular septal defect

CONGENITAL HEART DISEASE – GENERAL MANIFESTATIONS

Complications

- Arrhythmias
- Infective endocarditis
- Stroke (cerebral abscess)
- Pulmonary hypertension and oedema
- Polycythaemia/bleeding tendency/thrombotic tendency
- Heart failure

CONGENITAL HEART DISEASE – ORAL MANIFESTATIONS

- delayed eruption of both dentitions
- positional anomalies
- enamel hypoplasia
- gross vasodilatations in the pulps
- cleft palate
- fissure tongue
- greater caries and periodontal disease (associated to poor oral hygiene and lack in dental attention).

CARDIAC FAILURE

CARDIAC FAILURE

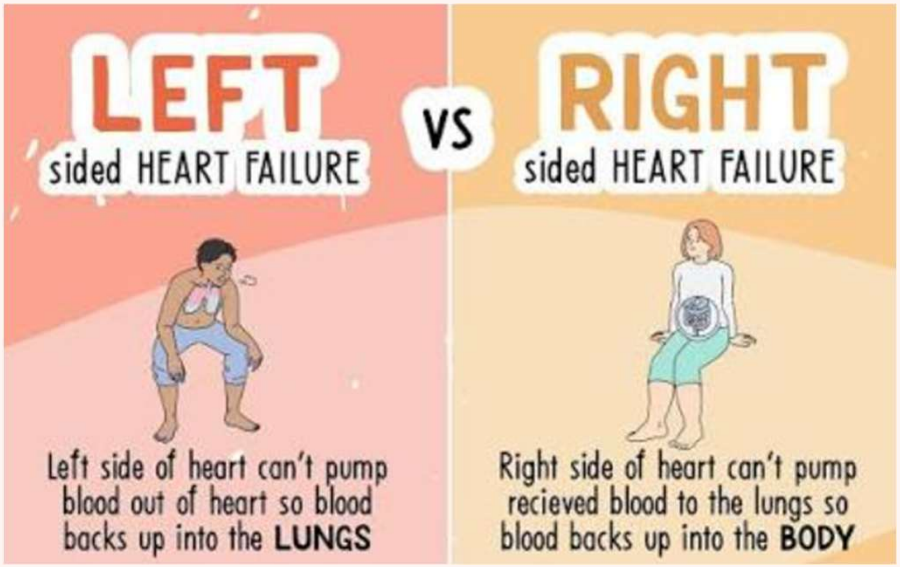
Definition: When structural or functional impairment of the pumping action of the heart leads to blood output insufficient to meet the body's demands → lack of tissue and organ perfusion

Aetiology: any structural or functional cardiac or non-cardiac disorder that impairs ability of heart to respond to physiological demands for CO

Table 5.34 *Main causes of heart failure*

Left-sided mainly	Right-sided mainly	Biventricular
Ischaemic heart disease	Chronic obstructive pulmonary disease	Ischaemic heart disease
Aortic valve disease	Pulmonary embolism	Aortic valve disease
Mitral valve disease		Mitral valve disease
Hypertension		Hypertension
		Cardiomyopathies
		Hyperthyroidism
		Chronic anaemias
		Arrhythmias

CARDIAC FAILURE – GENERAL MANIFESTATIONS



SYMPTOMS

- Pulmonary congestion:
  - Cough
  - Crackles
  - Pink-tinged sputum
  - Tachypnea
- Tachycardia
- Fatigue
- Cyanosis
- Exertional dyspnea

- Peripheral edema
- Ascites
- Enlarged liver & spleen
- JVD
- Weight gain
- Increased peripheral venous pressure

CARDIAC FAILURE – GENERAL MANAGEMENT

Lifestyle changes	Pharmacologic	Devices	Surgical intervention
○ Fluid & salt management (limit intake) ○ Minimise alcohol intake ○ Gentle exercise	○ Inotropes e.g. digoxin (AF) ○ Diuretics e.g. thiazide ○ ACE inhibitors ○ β blockers ○ Vasodilators ○ Aldosterone antagonists	○ Pacemaker ○ Cardiac resynchronisation device ○ ICD	○ Revascularisation procedures (CABG) ○ Valve replacement / repair (due to valve damage: CHD, IE, MI) ○ Heart transplantation (end stage)

HEART FAILURE – DENTAL MANAGEMENT

- Do not undertake dental treatment unless medical practitioner advised HF is stable
- Limit duration of dental procedures
- Patient may be unable to lie horizontally
  - Position dental chair with head higher than heart
  - Comfortable position for sleeping → "how many pillows do you use when sleeping?"
- Increased risk with sedation and GA → hospital environment with anaesthetist
- Avoid NSAIDs if possible
  - Especially if diuretic + (ACEI or A2RB) → risk of acute kidney injury
- Diuretics + drugs with diuretic effect (e.g. dapagliflozin, empagliflozin) → dry mouth
- May be candidate for heart transplant.
  - Require dental assessment before being waitlisted
  - Optimise oral health
  - May seek hospital dental specialist for advice for pre-transplant dental treatment plan

CARDIAC SURGERY WORK-UP

TYPES OF CARDIAC SURGERY

- Coronary angioplasty and stent (bare metal or drug-eluting) = Percutaneous coronary intervention (PCI)
  - Improve blood flow through occluded coronary arteries
- Coronary artery bypass grafting (CABG)
  - Redirect blood flow around occluded coronary arteries
- Valve replacement or Transcatheter aortic valve implantation (TAVI)
  - Replace faulty or damaged heart valves
- Congenital correction
  - Repair abnormal or damaged structures
- Pacemaker or Implantable Cardioverter Defibrillator (ICD) placement
  - Implant devices to control heart beat or support function / blood flow
- Cardiac Transplant
  - Replace a damaged heart, severe heart failure



CARDIAC RISKS OF DENTAL TREATMENT

- Risk stratification guidelines: Peri-operative risks for CVD patients undergoing non-cardiac surgical procedures

- H&N surgery 1-5%, dental surgery / superficial surgery <1%
  - European Society of Cardiology (ESC) and European Society of Anaesthesiology (ESA) (Halvorsen et al., 2022)
  - American College of Cardiology (ACC) and American Heart Association (AHA) (Thompson et al, 2024)

- Smith et al 2014:
  - Prophylactic dental extractions prior to cardiac surgery carries a 3% risk of death and 8% risk of major adverse outcome before cardiac operation

Smith MM, Barbara DW, Mauermann WJ, Viozzi CF, Dearani JA, Grim KJ. Morbidity and Mortality Associated with Dental Extraction Before Cardiac Operation. *Ann Thorac Surg.* 2014; 97: 838-44.

"… raises the question whether we should in fact get on with our cardiac operations and deal with the dental work at some other time, if at all, or risk killing patients with good intent!"

Unsworth-White 2014 (commentary of Smith et al 2014)

Unsworth-White MJ. Invited Commentary - Morbidity and Mortality Associated with Dental Extraction Before Cardiac Operation. *Ann Thorac Surg* . 2014;97,844.

Management of perioperative acute pain and infection (pus, swelling, cellulitis) needs to be a joint decision between the dentist and the physicians caring for the patient planned for cardiothoracic surgery.

But when should we treat chronic, asymptomatic pathology?

Risks outweigh benefits



Benefits *may* outweigh Risks

✗ Ischaemic disease
(eg. CABG, PCI, stent, angioplasty)

✗ Rhythmic disease
(eg. ICD/pacemaker, ablation)

? Valvular disease
(eg. replacement, repairs, TAVI)

? Infective endocarditis
(often leads to valvular surgery)

? Heart transplant (heart failure)

ISCHAEMIC INDICATIONS – RISKS VS BENEFITS OF PRE-OP DENTAL TREATMENT

Potential benefits:

- **Decrease endogenous catecholamine** release (resulting in increased BP) if patient is in pain
- ?Reduce pre-operative **infection risk** or graft rejection
  - Limited literature regarding dental extractions prior to CABG or heart transplant surgery

→ **lack of clear evidence to support** the reduced risk of post-operative infection (Meyer *et al.*, 1999)

Management of perioperative acute pain and infection (pus, swelling, cellulitis) needs to be a joint decision between the dentist and the physicians caring for the patient planned for cardiothoracic

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eart failure)

Eberhard *et al. Journal of Cardiothoracic Surgery* 2013, **8**:73
http://www.cardiothoracicsurgery.org/content/8/1/73

JCTS JOURNAL OF CARDIOTHORACIC SURGERY

RESEARCH ARTICLE Open Access

The oral cavity is not a primary source for implantable pacemaker or cardioverter defibrillator infections

Jörg Eberhard[1*†], Nico Stumpp[1†], Fadi Ismail[1], Ulrike Schnaidt[1], Wieland Heuer[1], Maximilian Pichlmaier[2], Christian Kühn[2], Axel Haverich[2] and Meike Stiesch[1]

Abstract

Background: To test the hypothesis that the oral cavity is a potential source for implantable pacemaker and cardioverter defibrillators infections, the bacterial diversity on explanted rhythm heart management devices was investigated and compared to the oral microbiome.

Methods: A metagenomic approach was used to analyze the bacterial diversity on the surfaces of non-infected and infected pacemakers. The DNA from surfaces swaps of 24 non-infected and 23 infected pacemaker were isolated and subjected to bacterial-specific DNA amplification, single strand conformation polymorphism- (SSCP) and sequencing analysis. Species-specific primer sets were used to analyze for any correlation between bacterial diversity on pacemakers and in the oral cavity.

Results: DNA of bacterial origin was detected in 21 cases on infected pacemakers and assigned to the bacterial phylotypes *Staphylococcus epidermidis*, *Propionibacterium acnes*, *Staphylococcus aureus*, *Staphylococcus schleiferi* and *Stapyhlococcus*. In 17 cases bacterial DNA was found on pacemakers with no clinical signs of infections. On the basis of the obtained sequence data, the phylotypes *Propionibacterium acnes*, *Staphylococcus* and an uncultured bacterium were identified. *Propionibacterium acnes* and *Staphylococcus epidermidis* were the only bacteria detected in pacemeaker (n = 25) and oral samples (n = 11).

Conclusions: The frequency of the coincidental detection of bacteria on infected devices and in the oral cavity is low and the detected bacteria are highly abundant colonizers of non-oral human niches.
The transmission of oral bacteria to the lead or device of implantable pacemaker or cardioverter defibrillators is unlikely relevant for the pathogenesis of pacemaker or cardioverter defibrillators infections.

Keywords: Biofilm, Infection, Pacemaker, Defibrillator, Oral cavity

Management of perioperative acute pain and infection (pus, swelling, cellulitis) needs to be a joint dec[illegible] for the patient planned for cardiothoracic

But [illegible] **asymptomatic pathology?**

Risks

Original Contributions

Cover Story

Effect of dental treatment before cardiac valve surgery

Systematic review and meta-analysis

Peter B. Lockhart, DDS, FDS RCPS; Hillary R. DeLong, JD; Ruth D. Lipman, PhD; Elliot Abt, DDS, MS, MSc; Larry M. Baddour, MD; Monica Colvin, MD, MA; Craig S. Miller, DMD, MS; Thomas Sollecito, DMD, FDS RCSEd; Kelly O'Brien, MLIS; Cameron G. Estrich, MPH; Marcelo W.B. Araujo, DDS, MS, PhD; Alonso Carrasco-Labra, DDS, MSc

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Supplemental material is available online.

ABSTRACT

Background. The purpose of this systematic review was to determine the potential effect of dental treatment before cardiac valve surgery (CVS) or left ventricular assist device (LVAD) implantation on morbidity and mortality.

Types of Studies Reviewed. The authors included relevant studies from MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials, including randomized controlled trials and cohort studies, published from 1998 through 2019 and involving adults who received dental treatment before CVS or LVAD implantation. The authors assessed bias by using the Newcastle-Ottawa Quality Assessment Scale and evidence certainty by using the Grading of Recommendations Assessment, Development and Evaluation approach. The authors used a meta-analysis with a random-effects model to estimate dichotomous and continuous outcomes, expressed as relative risk (RR) and weighted mean difference.

Results. Six studies met the inclusion criteria for CVS but none for LVAD implantation. Very low certainty in the evidence suggested uncertainty as to whether health outcomes for patients undergoing dental treatment before CVS differed from those who did not. Postsurgical outcomes included all-cause mortality (RR, 1.00; 95% confidence interval [CI], 0.53 to 1.91), infective endocarditis (RR, 1.30; 95% CI, 0.51 to 3.35), postsurgical infection (RR, 1.01; 95% CI, 0.76 to 1.33), and length of stay in the hospital (weighted mean difference, 2.9; 95% CI, −2.3 to 8.1).

Conclusions and Practical Implications. From the available evidence, it is unclear whether postoperative outcomes differ in patients receiving dental treatment before CVS compared with outcomes in those who do not. Dentists and medical care professionals should collaborate on an appropriate course of action for each patient, weighing any potentially relevant care considerations.

Key Words. Cardiac; pretreatment; medically complex; presurgical; dental clearance.

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Benefits *may* outweigh Risks

- ? Valvular disease (eg. replacement, repairs, TAVI)
- ? Infective endocarditis (often leads to valvular surgery)
- ? Heart transplant (heart failure)

Dr Jee-Yun Leung

Management of perioperative acute pain and infection (pus, swelling, cellulitis) needs to be a joint decision between the dentist and the physicians caring for the patient planned for cardiothoracic surgery.

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ORIGINAL ARTICLE

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U. Joos

Heart transplants – assessment of dental procedures

Received: 25 September 1998 / Accepted: 26 February 1999

Abstract The object of this study was to evaluate the effects of dental foci on survival rates and rejection episodes in heart transplant recipients. Therefore, in a retrospective longitudinal study we studied 74 heart transplant recipients at the Department of Maxillofacial Surgery and Department of Thoracic and Cardiovascular Surgery, University of Münster. Study patients were divided into groups: those in which dental foci had been verified (n=31) and those without dental foci (n=43). Statistical analysis was performed using the chi-square test, Kaplan-Meier life table analysis, and the log-rank test. Before heart transplantation, patients were screened clinically and radiographically to determine the extent of dental foci. Postoperatively, patients were evaluated dentally and medically to identify the impact of dental foci on the incidence of systemic and oral infections, frequency and severity of rejection episodes, mortality, and complications arising during dental treatment. By comparing the mortality, infection and rejection rates in the various groups no statistically significant differences (P>0.05) were found between patients. Despite immunosuppression, extended inflammatory processes such as abscess formation or viral stomatitis were not found in the oral cavity. We therefore suggest that patients suffering from the symptoms of severe heart failure need not be subjected to rigorous preoperative dental treatment.

Key words Heart transplant · Dental treatment · Mortality

Introduction

The survival rate of patients after heart transplantation has improved due to recent advances in perioperative management and immunosuppression [7]. Preoperative evaluation includes assessment of dental foci and their subsequent eradication [13, 5, 8]. So far, no study has examined the need for such a radical approach.

Most microorganisms associated with bacteriemia in patients with heart transplantation are commensal organisms of the oral cavity [9]. Systemic infections and rejection dramatically decrease the survival rate in patients subjected to heart transplantation. Since odontogenic inflammation may favor transplant rejection [6], cause bacterial endocarditis and may lead to life-threatening postoperative infections [2], all existing or potential sources of dental infection should be eliminated before heart transplantation. To our knowledge, specific guidelines specifying the extent of such dental procedures do not exist. The purpose of this study was to analyze the complication rates in patients in whom dental foci had been verified as compared with patients without dental foci so that the role of these foci on the postoperative outcome of heart transplantation could be determined. The influence of various dental foci on differences in survival and rejection probabilities of heart transplant recipients was investigated to reevaluate our dental treatment protocol.

R

ıronic, asymptomatic pathology?



Benefits *may* outweigh Risks

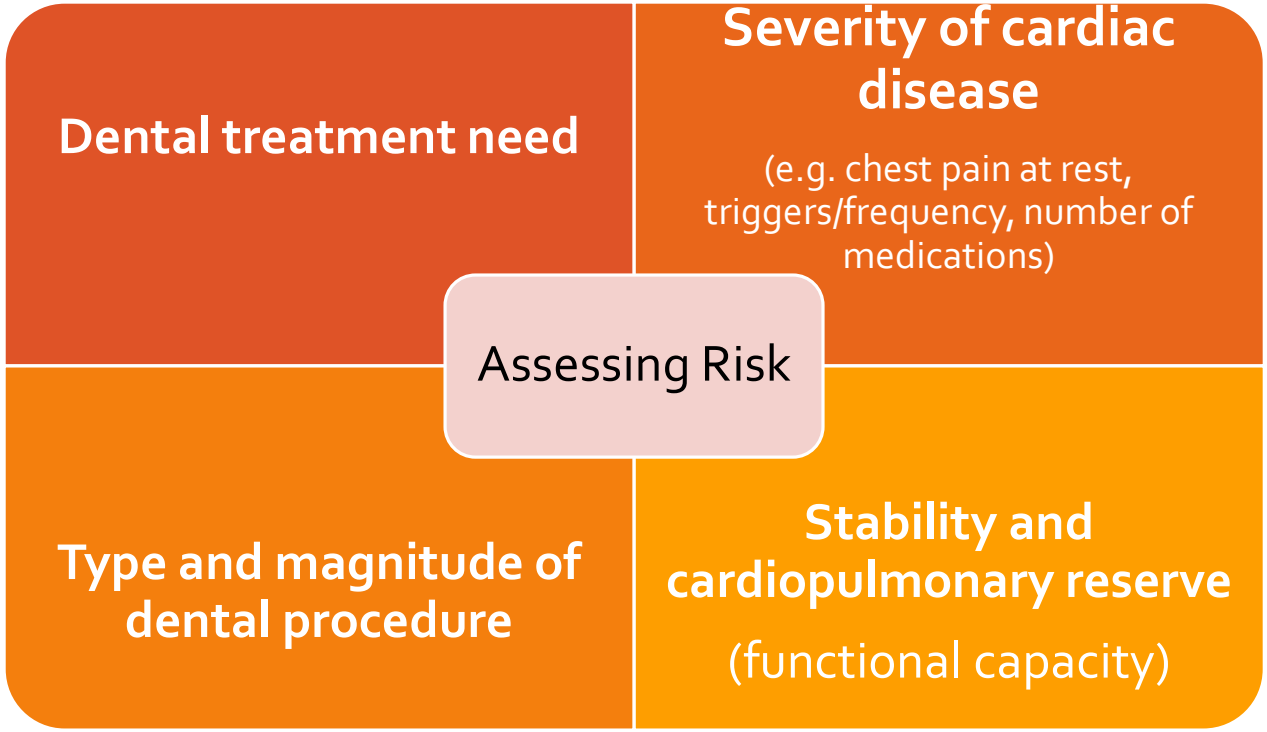
? Valvular disease
(eg. replacement, repairs, TAVI)

? Infective endocarditis
(often leads to valvular surgery)

? Heart transplant (heart failure)

Dr Jee-Yun Leung

TO PROCEED OR NOT TO PROCEED?



➔ Recommendation: integrated team decision based on individual situation of the patient (stable vs unstable condition)

How far ahead to future proof?

"IN THE 12 MONTHS AFTER MYOCARDIAL INFARCTION, STENT PLACEMENT, CORONARY ARTERY BYPASS SURGERY, OR VALVE PROCEDURES (INCLUDING TRANSCATHETER AORTIC VALVE IMPLANTATION [TAVI]), PATIENTS ARE AT INCREASED RISK OF A MAJOR ADVERSE CARDIAC EVENT (EG SUDDEN DEATH).

DEFER ELECTIVE DENTAL TREATMENT FOR 6 MONTHS AFTER MYOCARDIAL INFARCTION, STENT PLACEMENT, CORONARY ARTERY BYPASS SURGERY OR VALVE PROCEDURE.

IF DENTAL PAIN OR INFECTION OCCURS WITHIN 6 MONTHS FOLLOWING THE EVENT, PROVIDE ADEQUATE EMERGENCY TREATMENT BUT AVOID THE USE OF ADRENALINE-CONTAINING LOCAL ANAESTHETICS. CONSIDER SEEKING SPECIALIST ADVICE ABOUT APPROPRIATE TREATMENT AND TIMING OF PROCEDURES."

Oral & Dental Therapeutic Guidelines v4 2026

THINKING IT THROUGH… CARDIAC SURGERY

Rationale

- Does need for dental treatment outweigh the cardiac risks?
- Direct effect on cardiac outcome = Endocarditis risk

Medical, Dental and Social Considerations

- Bacteraemia risk of dental pathology
- Amount of treatment patient can tolerate in one go vs risks of performing over multiple visits
- Anticoagulation / antiplatelet drugs (prophylactic vs therapeutic)
- Antibiotic prophylaxis
- Focus on preventive oral hygiene to ↓ daily bacteraemia

Entire or localised areas of the mouth

- Entire dentition

Optimal Timeframes

- Urgency for cardiac surgery
- Future-proof for next 6 months (i.e. optimise for the short-term)

Treatment Approaches

- Aggressive (e.g. typically insufficient time to assess periapical response to RCT)

QUESTIONS?

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