Target Organ Damage

• Heart: LV hypertrophy, heart failure, angina or MI, arterial aneurysm.
• Brain: Stroke or transient ischemic attack, cerebral atrophy and dementia
• Kidney: sclerosis and chronic kidney disease
• Peripheral arterial disease
• Eye: Retinopathy, hemorrhage, loss of vision.

HT Management

Goals of management of hypertension

✓ Treating SBP and DBP to targets that are <140/90 mmHg
✓ The goal of anti-HT therapy is reduction of mortality and morbidity to CVS and renal diseases.

I- Non-pharmacological treatment

✓ Weight reduction and Prevention of obesity.
✓ Stop smoking
✓ Dietetic modification (Increase Fruits, vegetables intake, Low free food, Intake of salt ... ≤ 5 – 6 g per day)
✓ Regular physical activity
✓ Reducing stress (relaxation therapy)

II- Pharmacological treatment

> Drug treatment should be started immediately after diagnosis in:

✓ Patients with stage 2 HT, (BP ≥ 160/100mm Hg)
✓ In patient with stage I, but with microalbuminuria, eGFR < 60, peripheral vascular disease (PVD), cerebro-vascular disease, hypertensive retinopathy, left ventricular hypertrophy (LVH) or ischemic heart disease (IHD), target organ damage.

> Pharmacological treatment should be influenced by the age, race and other clinical characteristics of the patient (e.g. pregnancy, obesity) or other clinical conditions (e.g. diabetes and coronary artery diseases).
>Hypertension and diabetes: HT co-exists with DM in about 40% at age 45 years. 70% of type II DM patients die from cardiovascular disease. At least 50% of DM patients will require 2 or 3 antihypertensive agents to achieve tight control.

> Long-acting drugs that need to be taken only once daily are preferred to drugs that require multiple doses.

1) Diuretics: (increase urination — decrease blood volume).

a) Loop diuretics (Furosemide, Bumetanide): strong short-lasting effect; usually used in heart failure and less commonly for hypertension due to its ability to excrete Na+ from filtrate block active re-absorption of Na+, Cl-, K+. But they aren’t suitable for long-lasting application.

b) Thiazide diuretics (hydrochlorothiazide, chlorthalidone) block re-absorption of Na+ and Cl. Weaker than loop diuretics — excrete 5% from Ná+ filtrate, most suitable diuretics for long—lasting treatment of HT. They increase anti HT activity of combined treatment.

   **Side effect** Decrease insulin sensitivity, not commonly used in DM.

c) K-sparing diuretics (spironolactone, aldosterone antagonist, amiloride). only assistant drugs to correct hypokalemia. Prescribed in combination with the other diuretic to prevent hypokalemia (commonly in HF).

   **Side effect**: Hyperkalemia, which risks potentially fatal arrhythmias.

2) B-blockers: block B-receptor

a) Non-selective (Bl &B2 affinity): Ex: Propranolol (Inderal), Carvedilol.

b) Selective “cardioselective” & beta-blockers (B1-effect): Ex: Metoprolol (Betaloc), Bisoprolol (Concor), Atenolol (Tenormin).

c) Hybrid substances (beside B-effect have also other effects, additional, B2-mimetic effect, through which they induce vasodilation) Ex: labetalol, carvedilol, nebulol,
**Side effects:**

- Broncho-constriction (contra-indicated in asthma) & vaso-constriction in the periphery
- Metabolic adverse effect: worsening of lipidogram; mask symptoms of hypoglycemia and can impair glucose tolerance.
- Sleep disturbances, bad dreams lead to depression
- Not stopped abruptly — rebound HT

3) **Calcium channel blockers:** Block the entrance of calcium to cell causes relaxation of smooth muscle in vessel wall — reduce systemic vascular resistance and arterial pressure. Two groups:

a) Dihydropyridins Nifedpine (adalate), amlodipine (lowvasc), felodipine.

b) Non- Dihydropyridins (Verapamil, Diltiazem)

Used to treat angina, & Raynaud’s disease, but the vasodilatation and hypotension effect can lead to reflex tachycardia. Other side effect gingival Hyperplasia, constipation, nausea, swelling in the feet and lower legs.

4) **Angiotensin Converting Enzyme (ACE) Inhibitors:**

Inhibit the change of angiotensin I to angiotensin II lead to vasodilation. Indicated in HT with heart failure (HF), and as vasodilating therapy after MI. Also in HT with DM and different forms of diabetic nephropathy starting with microalbuminuria (nephroprotective).

Treatment should be started from the lowest doses, excessive initial fall in BP lead to postural hypotension or syncope.
Ex: Captopril (Capoten), Enalapril (Enap), Lisinopril (Zestril). Effect to reduce BP is in the whole group similar.

**Side effects** dry cough, impaired renal function, hyperkalemia, hypotension, angioneurotic edema.

**Contra-indications**: Pregnancy, hyperkalemia, impaired renal function.

5) **Angiotensin II Receptor Blockers (ARBs):**

Often replacement of ACEI in case of intolerance or cough. But clinical studies indicate that they have among patients with HT and DM 2 slightly better protective effects than ACEI (now first choice). Losartan (Cozaar), valsartan (Diovan), candesartan (Atacand), irbesartan (Aprovel), telmisartan (Micardis). Have a relatively low incidence of side effect, but contra-indicated in pregnancy.

6) **Combination Treatment**

Some cases one drug cannot control HT. Combination treatment of two or three medications is indicated. Different combinations of drugs in varying dosages are used. ACE inhibitors or ARBs are often effective when combined with other classes of medications (thiazide diuretic, CCB). Amlodipine & valsartan (Exforge), Amlodipine and telmisartan (Twynsta). Having a combination of drugs in one pill may better control HT by improving compliance of patient.

**Hypertensive emergencies**

Severely elevated BP (>180/120 mmHg), with progressive target organ dysfunction. Ex: severely elevated BP with

- Hypertensive encephalopathy,
- Acute LVF with pulmonary edema
- Acute MI or unstable angina
- Dissecting aortic aneurysm

Require emergent management in ICU (intensive care unit). Immediate intravenous (IV) parenteral therapy. Acutely lowering BP should be avoided, the blood pressure be lowered smoothly, not too abruptly.
Oral manifestations caused by the adverse effects of anti HT drugs:

- Xerostomia: ACEIs, thiazide diuretics, loop diuretics, and clonidine
- Gingival Hyperplasia: CCB, majority of cases are associated with nifedipine.
- Lichenoid Reaction: thiazide diuretics, methyldopa, propranolol, captopril, furosemide, spironolactone.
- Loss of taste (ageusia) or taste alteration (dysgeusia): ACE•
- Most anti-HT drugs have interactions with local anesthesia & may increase LA toxicity.

Implications of CVD on dental practice

> Patients with cardiac disease should receive dental treatment in minimal stressful environment. Anxiety, exertion and pain should be minimized.

> The blood pressure may preferably be measured before any dental procedure to ensure that this is normal or controlled. A high blood pressure may result into precipitation of other related problems such as angina or heart failure.

> Clinical general examine should be done. Irregular pulse, engorged jugular veins and tachypnea may indicate the presence of cardiac disease.

> Angina may present as pain in the mandible, teeth and other oral tissues.

> A history of hypertension, ischemic heart disease or any other cardiac problem particularly congenital heart disease and drug intake (anticoagulant, aspirin) should be sought.

> Epinephrine in the local anesthesia may raise the blood pressure and precipitate arrhythmias.

> In patients with IHD, facilities for medical help, oxygen and nitroglycerine should be available.

> Elective dental surgery should be deferred for months following acute MI.
Prophylaxis for infective endocarditis is mandatory.

Cardiac patients on anticoagulant drugs or aspirin are at increased risk of bleeding following dental procedures. Hence, these drugs should preferably be stopped a week before the procedure.

Calcium channel blockers may cause gingival swelling and lichenoid lesions in the oral cavity. ACE inhibitors can cause loss of taste, burning sensation in oral cavity, and angio-edema.

Dry mouth can result due to antihypertensive drugs such as diuretics, beta blockers and clonidine.

Sudden change in the posture from supine to standing following dental procedures may cause postural hypotension and syncope, particularly in patients using diuretics and calcium channel blockers.

Oral abnormalities such as enamel hypoplasia, delayed eruption of dentitions, positional anomalies, bluish white “skimmed milk” appearance of teeth and vasodilatation in the pulps may be associated with cyanotic congenital heart disease.

Patient with left ventricular failure should be managed in partially reclining or erect position. Supine position may worsen dyspnea.