Antihypertensive Drugs

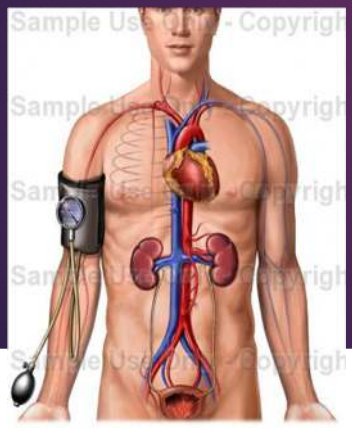

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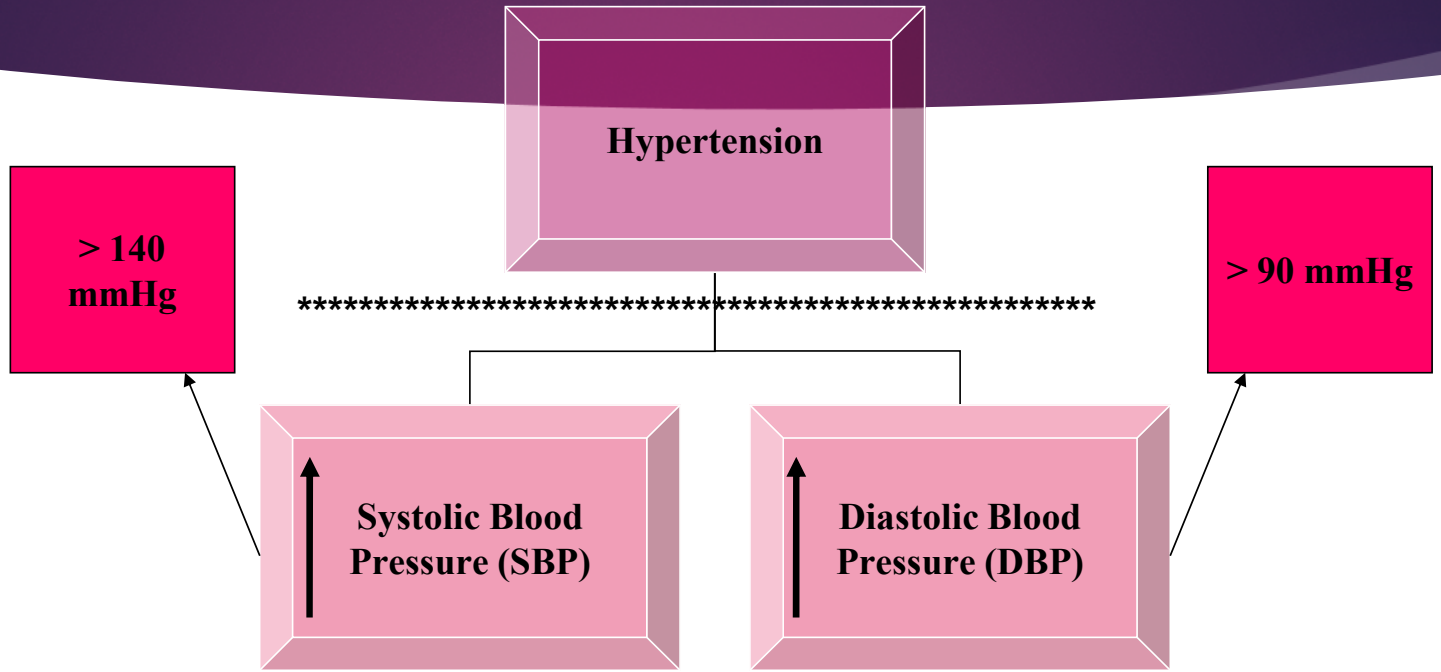
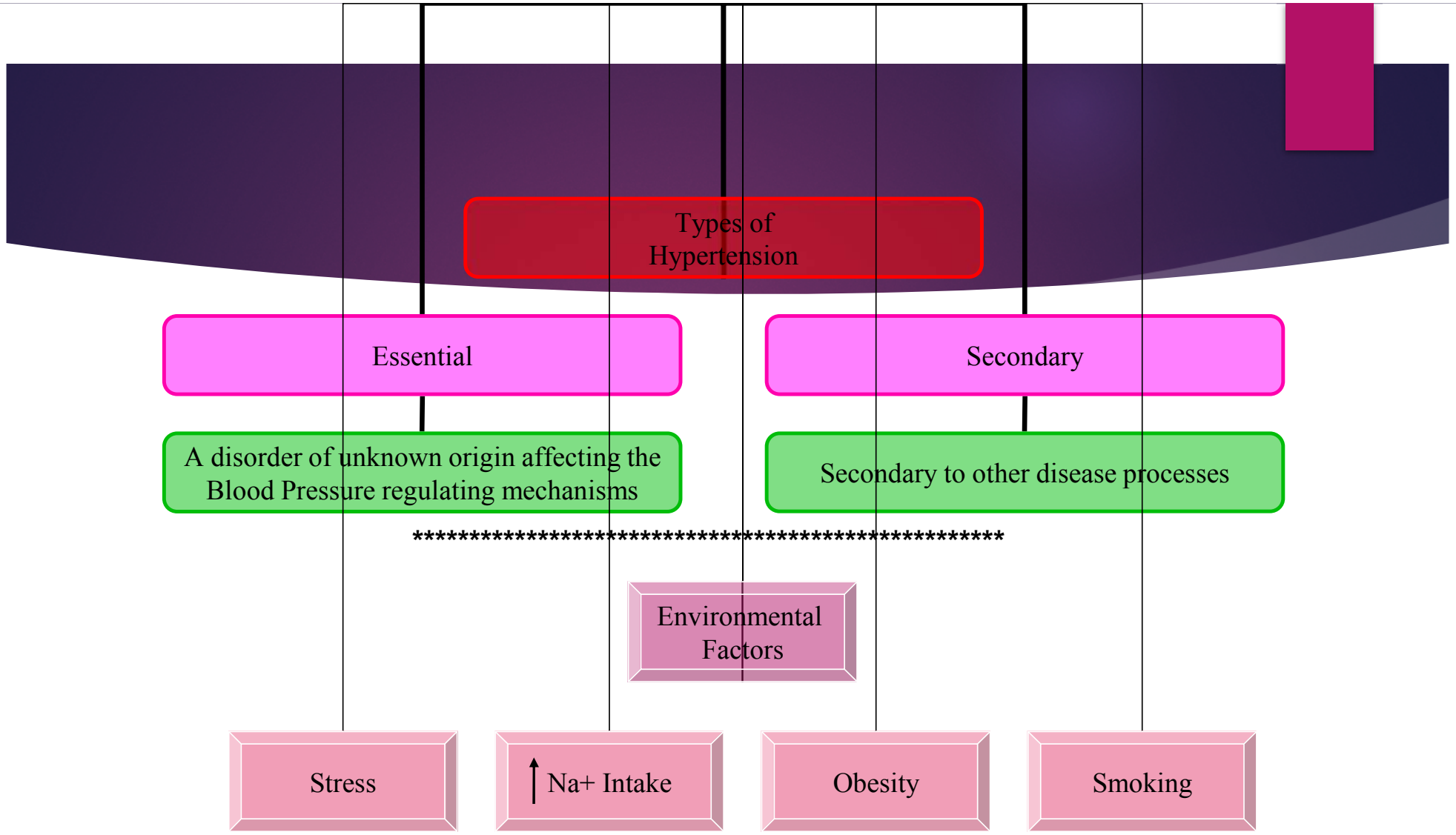
Table 3. Classification of Blood Pressure in Adults (age ≥18 years)			
Classification	**Systolic Blood Pressure (mmHg)**		**Diastolic Blood Pressure (mmHg)**
Normal	<120	AND	<80
Prehypertension	120-139	OR	80-89
Stage I HTN	140-159	OR	90-99
Stage 2 HTN	≥160	OR	≥100

Introduction




Types of Hypertension
Essential
Secondary
A disorder of unknown origin affecting the Blood Pressure regulating mechanisms
Secondary to other disease processes
****
Environmental Factors
Stress
↑ Na+ Intake
Obesity
Smoking

Treatment – Why?

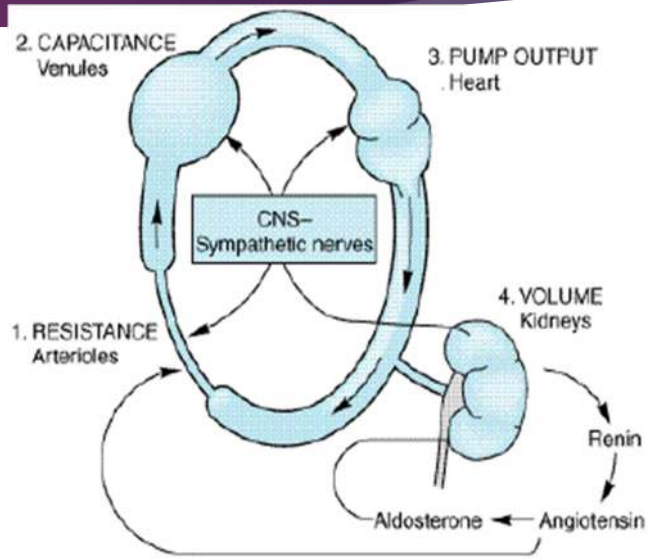
- Symptomatic treatment is Mandatory:
  - Damage to the vascular epithelium, paving the path for atherosclerosis (IHD, CVA) or nephropathy due to high intra-glomerular pressure
  - Increased load on heart due to high BP can cause CHF
  - Hypertension, even asymptomatic needs treatment

Normal Blood Pressure Regulation

- Hydraulic equation:

Blood Pressure = Cardiac output (CO) X Resistance to passage of blood through precapillary arterioles (PVR)

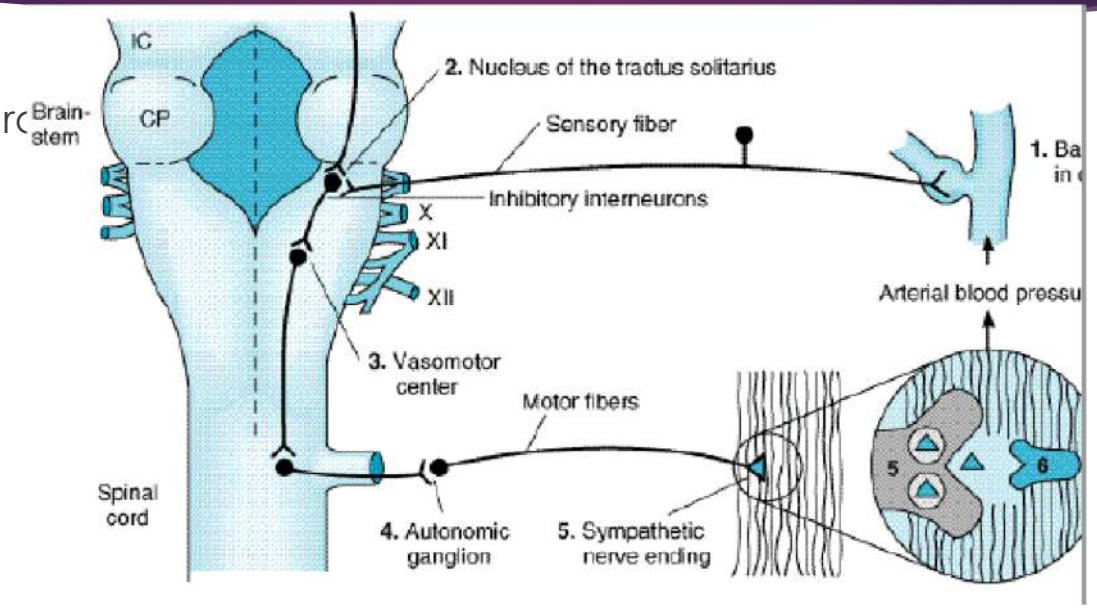
- Physiologically CO and PVR is maintained minute to minute by – arterioles (1) postcapillary venules (2) and Heart (3)
- Kidney is the fourth site – volume of intravascular fluid
- Baroreflex, humoral mechanism and renin-angiotensin- aldosterone system regulates the above 4 sites
- Local agents like Nitric oxide
- In hypertensives – Baroreflex and renal blood-volume control system – set at higher level
- All antihypertensives act via interfering with normal mechanisms



Anatomic sites of blood pressure control.

Baroreceptor reflex arc

- Postural barc



If BP is high:
- ↓ CO and PR ⇨ ↓ BP

If BP is low:
- ↑ CO and PR ⇨ ↑ BP

The Renal response

- Long-term blood pressure control – by controlling blood volume
- Reduction in renal pressure - intrarenal redistribution of pressure and increased absorption of salt and water
- Decreased pressure in renal arterioles and sympathetic activity – renin production – angiotensin II production
- Angiotensin II:
  - Causes direct constriction of renal arterioles
  - Stimulation of aldosterone synthesis – sodium absorption and increase in intravascular blood volume

classification

- Diuretics
- Sympatholytic drugs
- ACE inhibitors
- ARBs
- CCBs
- vasoDilators

Antihypertensive Drugs

- Diuretics:
  - Thiazides: Hydrochlorothiazide, chlorthalidone
  - High ceiling: Furosemide
  - K+ sparing: Spironolactone, triamterene and amiloride

MOA: Acts on Kidneys to increase excretion of Na and H_2O – decrease in blood volume – decreased BP

- Angiotensin-converting Enzyme (ACE) inhibitors:
  - Captopril, lisinopril., enalapril, ramipril and fosinopril

MOA: Inhibit synthesis of Angiotensin II – decrease in peripheral resistance and blood volume

- Angiotensin (AT1) blockers:
  - Losartan, candesartan, valsartan and telmisartan

MOA: Blocks binding of Angiotensin II to its receptors

Antihypertensive Drugs

- Centrally acting:
  - Clonidine, methyldopa

MOA: Act on central a2A receptors to decrease sympathetic outflow – fall in BP

- ß-adrenergic blockers:
  - **Non selective:** Propranolol (others: nadolol, timolol, pindolol, labetolol)
  - **Cardioselective: Metoprolol** (others: atenolol, esmolol, betaxolol)

MOA: Bind to beta adrenergic receptors and blocks the activity

- ß and a – adrenergic blockers:
  - Labetolol and carvedilol
- a – adrenergic blockers:
  - Prazosin, terazosin, doxazosin, phenoxybenzamine and phentolamine

MOA: Blocking of alpha adrenergic receptors in smooth muscles - vasodilatation

Antihypertensive Drugs –

- Calcium Channel Blockers (CCB):
  - Verapamil, diltiazem, nifedipine, felodipine, amlodipine, nimodipine etc.

MOA: Blocks influx of Ca++ in smooth muscle cells – relaxation of SMCs – decrease BP

- K+ Channel activators:
  - Diazoxide, minoxidil, pinacidil and nicorandil

MOA: Leaking of K+ due to opening – hyper polarization of SMCs – relaxation of SMCs

- Vasodilators:
  - Arteriolar – Hydralazine (also CCBs and K+ channel activators)
  - Arterio-venular: Sodium Nitroprusside

Diuretics

- Drugs causing net loss of Na+ and water in urine
- Mechanism of antihypertensive action:
  - Initially: diuresis – depletion of Na+ and body fluid volume – decrease in cardiac output
  - 1)Subsequently after 4 - 6 weeks, Na+ balance is regained by 95%, but BP remains low!
  - Q: Why? Answer: 2)reduction in total peripheral resistance (TPR) due to deficit of little amount of Na+ and water (Na+ causes vascular stiffness)
  - Similar effect is seen with sodium restriction (low sodium diet)
  - 3)Also direct effect action on k+ channels causing hyperpolarization

Thiazide diuretics – adverse effects

- Adverse Effects:
  - Hypokalaemia – muscle pain and fatigue
  - Hyperglycemia: Inhibition of insulin release due to K+ depletion (proinsulin to insulin) – precipitation of diabetes
  - Hyperlipidemia: rise in total LDL level – risk of stroke
  - Hyperurecaemia: inhibition of urate excretion
  - Sudden cardiac death – tosades de pointes (hypokalaemia)
  - All the above metabolic side effects – higher doses (50 – 100 mg per day)
  - But, its observed that these adverse effects are minimal with low doses (12.5 to 25 mg) - Average fall in BP is 10 mm of Hg

Thiazide diuretics – current status

- Effects of low dose:
  - No significant hypokalaemia
  - Low incidence of arrhythmia
  - Lower incidence of hyperglycaemia, hyperlipidemia and hyperuricaemia
  - Reduction in MI incidence
  - Reduction in mortality and morbidity
- JNC recommendation:
  - JNC recommends low dose of thiazide therapy (12.5 – 25 mg per day) in essential hypertension
  - Preferably should be used with a potassium sparing diuretic as first choice in elderly
  - If therapy fails – another antihypertensive but do not increase the thiazide dose
  - Loop diuretics are to be given when there is severe hypertension with retention of body fluids

Diuretics

- K+ sparing diuretics:
  - Thiazide and K sparing diuretics are combined therapeutically – DITIDE (triamterene + benzthiazide) is popular one
- Modified thiazide: indapamide
  - Indole derivative and long duration of action (18 Hrs) – orally 2.5 mg dose
  - It is a lipid neutral i.e. does not alter blood lipid concentration, but other adverse effects may remain
- Loop diuretics:
  - Na+ deficient state is temporary, not maintained round –the-clock and t.p.r not reduced
  - Used only in complicated cases – CRF, CHF marked fluid retention cases

Angiotensin Converting Enzyme (ACE) Inhibitors

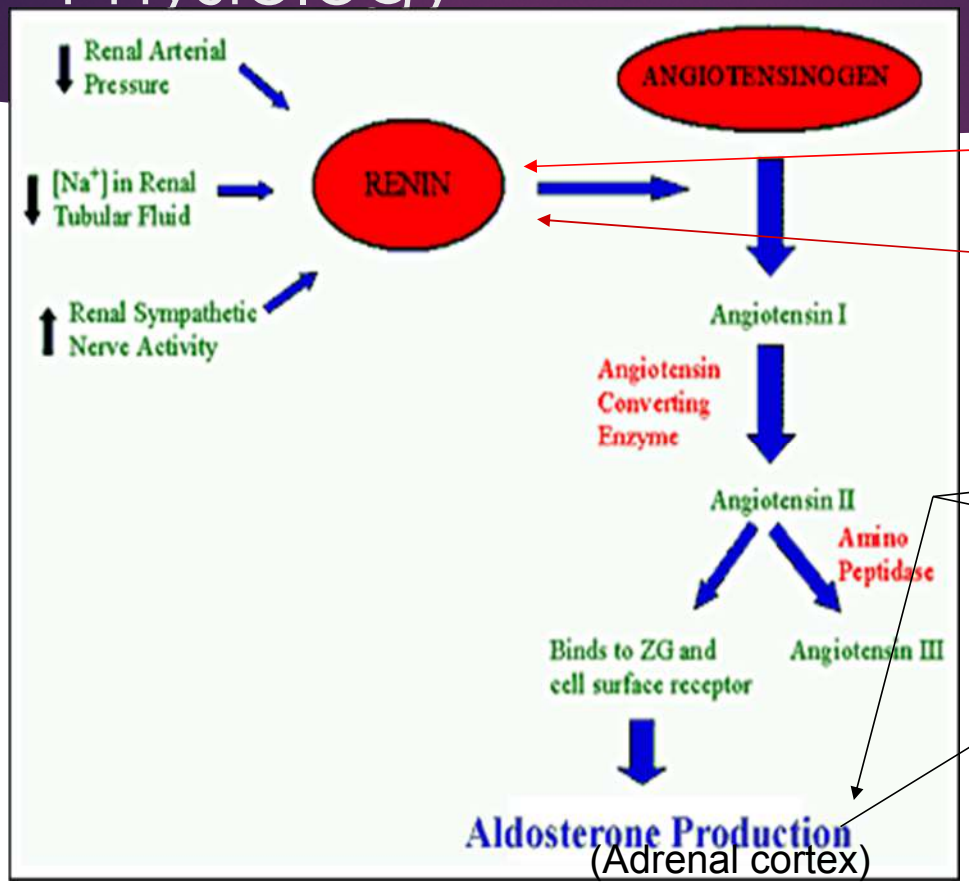
What is Renin - Angiotensin?

(Physiological Background)

RAS - Introduction

- Renin is a proteolytic enzyme and also called angiotensinogenase
- It is produced by juxtaglomerular cells of kidney
- It is secreted in response to:
  - Decrease in arterial blood pressure
  - Decrease Na+ in macula densa
  - Increased sympathetic nervous activity
- Renin acts on a plasma protein – Angiotensinogen (a glycoprotein synthesized and secreted into the bloodstream by the liver) and cleaves to produce a decapeptide Angiotensin-I
- Angiotensin-I is rapidly converted to Angiotensin-II (octapeptide) by ACE (present in luminal surface of vascular endothelium)
- Furthermore degradation of Angiotensin-II by peptidases produce Angiotensin-III
- Both Angiotensin-II and Angiotensin-III stimulates Aldosterone secretion from Adrenal Cortex (equipotent)
- AT-II has very short half life – 1 min

RAS - Physiology



Increased Blood Vol.

Rise in BP

Vasoconstriction

Na+ & water retention

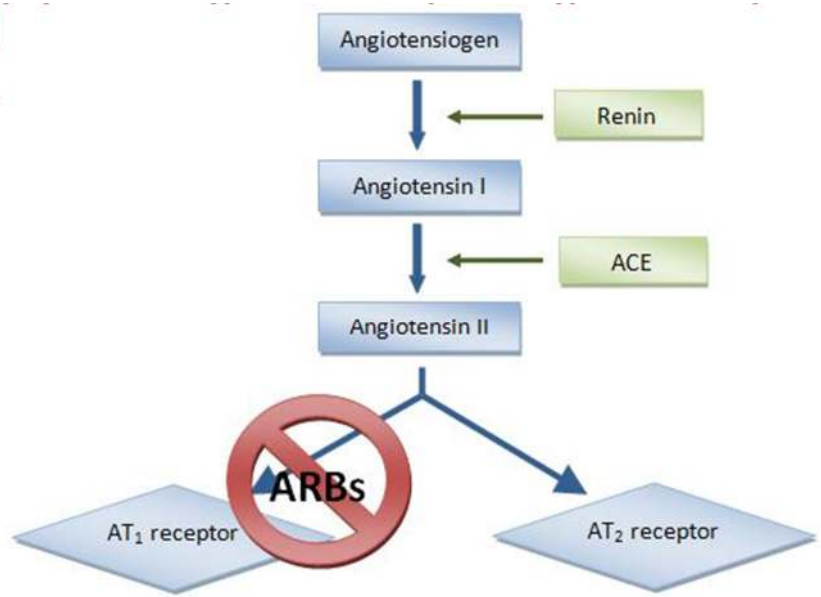
Kidney

Angiotensin-II

- What are the ill effects on chronic ?
  - Volume overload and increased t.p.r
    - Cardiac hypertrophy and remodeling
    - Coronary vascular damage and remodeling
  - Hypertension – long standing will cause ventricular hypertrophy
  - Myocardial infarction – hypertrophy of non-infarcted area of ventricles
  - Renal damage
  - Risk of increased CVS related morbidity and mortality
- ACE inhibitors reverse cardiac and vascular hypertrophy and remodeling

ACE inhibitors

- Captopril, l[illegible]il and fosinopril e[illegible]



Captopril – Adverse effects

- Cough – persistent brassy cough in 20% cases – inhibition of bradykinin and substanceP breakdown in lungs
- Hyperkalemia in renal failure patients with K+ sparing diuretics, NSAID and beta blockers (routine check of K+ level)
- Hypotension – sharp fall may occur – 1st dose
- Acute renal failure: CHF and bilateral renal artery stenosis
- Angioedema: swelling of lips, mouth, nose etc.
- Rashes, urticaria etc
- Dysgeusia: loss or alteration of taste
- Foetopathic: hypoplasia of organs, growth retardation etc
- Neutripenia
- Contraindications: Pregnancy, bilateral renal artery stenosis, hypersensitivity and hyperkalaemia

ACE inhibitors - Enalapril

- It's a prodrug – converted to enalaprilate
- Advantages over captopril:
  - Longer half life – OD (5-20 mg OD)
  - Absorption not affected by food
  - Rash and loss of taste are less frequent
  - Longer onset of action
  - Less side effects

ACE inhibitors and hypertension

- 1st line of Drug:
  - No postural hypotension or electrolyte imbalance (no fatigue or weakness)
  - Safe in asthmatics and diabetics
  - Prevention of secondary hyperaldosteronism and K+ loss
  - Renal perfusion well maintained
  - Reverse the ventricular hypertrophy and increase in lumen size of vessel
  - No hyperuraecemia or deleterious effect on plasma lipid profile
  - No rebound hypertension
  - Minimal worsening of quality of life – general wellbeing, sleep and work performance etc.

ACE inhibitors – other uses

- Hypertension
- Congestive Heart Failure
- Myocardial Infarction
- Prophylaxis of high CVS risk subjects
- Diabetic Nephropathy
- Schleroderma crisis

Angiotensin Receptor Blockers (ARBs) -

Angiotensin Receptors:

- Specific angiotensin receptors have been discovered, grouped and abbreviated as – AT1 and AT2
- They are present on the surface of the target cells
- Most of the physiological actions of angiotensin are mediated via AT1 receptor
- Transducer mechanisms of AT1 inhibitors: In different tissues show different mechanisms. For example -
  - PhospholipaseC-IP3/DAG-intracellular Ca++ release mechanism – vascular and visceral smooth muscle contraction
  - In myocardium and vascular smooth muscles AT1 receptor mediates long term effects by MAP kinase and others
- *Losartan* is the specific AT1 blocker

Angiotensin Receptor Blockers (ARBs) - Losartan

- Competitive antagonist and inverse agonist of AT1 receptor
- Does not interfere with other receptors except TXA2
- Blocks all the actions of A-II - vasoconstriction, sympathetic stimulation, aldosterone release and renal actions of salt and water reabsorption
- No inhibition of ACE

Losartan

- Theoretical superiority over ACEIs:
  - Cough is rare – no interference with bradykinin and other ACE substrates
  - Complete inhibition of AT1 – alternative remains with ACEs
  - Result in indirect activation of AT2 – vasodilatation (additional benefit)
  - Clinical benefit of ARBs over ACEIs – not known
- However, losartan decreases BP in hypertensive which is for long period (24 Hrs)
  - heart rate remains unchanged and cvs reflxes are not interfered
  - no significant effect in plasma lipid profile, insulin sensitivity and carbohydrate tolerance etc
  - Mild uricosuric effect

Losartan

- Pharmacokinetic:
  - Absorption not affected by food but unlike ACEIs its bioavailability is low
  - High first pass metabolism
  - Carboxylated to active metabolite E3174
  - Highly bound to plasma protein
  - Do not enter brain
- Adverse effects:
  - Foetopathic like ACEIs – not to be administered in pregnancy
  - Rare 1st dose effect hypotension
  - Low dysgeusia and dry cough
  - Lower incidence of angioedema
- Available as 25 and 50 mg tablets

Beta-adrenergic blockers

- **Non selective: Propranolol** (others: nadolol, timolol, *pindolol*, labetolol)
- **Cardioselective: Metoprolol** (others: atenolol, esmolol, betaxolol)

- All beta-blockers similar antihypertensive effects – irrespective of additional properties

  - Reduction in CO but no change in BP initially but slowly
  - Adaptation by resistance vessels to chronically reduced CO – antihypertensive action
  - Other mechanisms – decreased renin release from kidney (beta-1 mediated)
  - Reduced NA release and central sympathetic outflow reduction
  - Non-selective ones – reduction in g.f.r but not with selective ones
  - Drugs with intrinsic sympathomimetic activity may cause less reduction in HR and CO

Beta-adrenergic blockers

- Advantages:
  - No postural hypotension
  - No salt and water retention
  - Low incidence of side effects
  - Low cost
  - Once a day regime
  - Preferred in young non-obese patients, prevention of sudden cardiac death in post infarction patients and progression of CHF
- Drawbacks (side effects):
  - Fatigue, lethargy (low CO?) – decreased work capacity
  - Loss of libido – impotence
  - Cognitive defects – forgetfulness
  - Difficult to stop suddenly
  - Therefore cardio-selective drugs are preferred now

Beta-adrenergic blockers

- Advantages of cardio-selective over non-selective:
  - In asthma
  - In diabetes mellitus
  - In peripheral vascular disease
- Current status:
  - JNC 7 recommends - 1st line of antihypertensive along with diuretics and ACEIs
  - Preferred in young non-obese hypertensive
  - Angina pectoris and post angina patients
  - Post MI patients – useful in preventing mortality
  - In old persons, carvedilol – vasodilatory action can be given

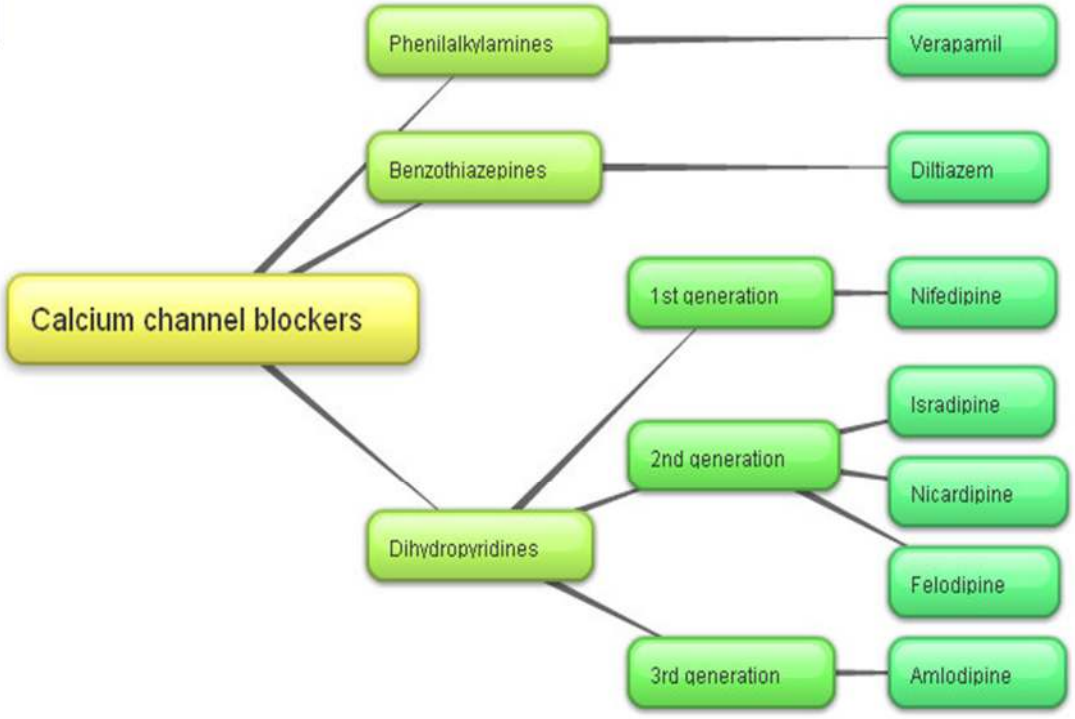
Alpha-adrenergic blockers

- Non selective alpha blockers are not used in chronic essential hypertension (phenoxybenzamine, phentolamine), only used sometimes as in phaechromocytoma
- Specific alpha-1 blockers like prazosin, terazosin and doxazosine are used
- PRAZOSIN is the prototype of the alpha-blockers
- Reduction in t.p.r and mean BP – also reduction in venomotor tone and pooling of blood – reduction in CO
- Does not produce tachycardia as presynaptic auto (alpha-2) receptors are not inhibited – autoregulation of NA release remains intact

Alpha-adrenergic blockers.

- Adverse effects:
  - Prazosin causes postural hypotension – start 0.5 mg at bed time with increasing dose and upto 10 mg daily
  - Fluid retention in monotherapy
  - Headache, dry mouth, weakness, dry mouth, blurred vision, rash, drowsiness and failure of ejaculation in males
- Current status:
  - Several advantages – improvement of carbohydrate metabolism – diabetics, lowers LDL and increases HDL, symptomatic improvement in BHP
  - But not used as first line agent, used in addition with other conventional drugs which are failing – diuretic or beta blocker
- Doses: Available as 0.5 mg, 1 mg, 2.5 mg, 5 mg etc. dose:1-4 mg thrice daily (Minipress/Prazopress)

Calcium Channel Blockers - Classification



Calcium Channel Blockers – Mechanism of action

- Three types Ca+ channels in smooth muscles – Voltage sensitive, receptor operated and leak channel
- Voltage sensitive are again 3 types – L-Type, T-Type and N-Type
- Normally, L-Type of channels admit Ca+ and causes depolarization – excitation-contraction coupling through phosphorylation of myosin light chain – contraction of vascular smooth muscle – elevation of BP
- CCBs block L-Type channel:
  - Smooth Muscle relaxation
  - Negative chronotropic, ionotropic and chronotropic effects in heart
- DHPs have highest smooth muscle relaxation and vasodilator action followed by verapamil and diltiazem
- Other actions: DHPs have diuretic action

Calcium Channel Blockers

- Advantages:
  - Unlike diuretics no adverse metabolic effects but mild adverse effects like – dizziness, fatigue etc.
  - Do not compromise haemodynamics – no impairment of work capacity
  - No sedation or CNS effect
  - Can be given to asthma, angina and PVD patients
  - No renal and male sexual function impairment
  - No adverse fetal effects and can be given in pregnancy
  - Minimal effect on quality of life

Calcium Channel Blockers – current status

- As per JNC 7 CCBs are not 1st line of antihypertensive unless indicated – ACEI/diuretics/beta blockers
- However its been used as 1st line by many because of excellent tolerability and high efficacy
- Preferred in elderly and prevents stroke
- CCBs are effective in low Renin hypertension
- They are next to ACE inhibitors in inhibition of albuminuria and prevention of diabetic nephropathy
- Immediate acting Nifedipine is not encouraged anymore

Calcium Channel Blockers

- Contraindications:
  - Unstable angina
  - Heart failure
  - Hypotension
  - Post infarct cases
  - Severe aortic stenosis
- Preparation and dosage:
  - Amlodipine – 2.5, 5 and 10 mg tablets (5-10 mg OD) – Stamlo, Amlopres, Amlopin etc.
  - Nimodipine – 30 mg tab and 10 mg/50 ml injection – Vasotop, Nimodip, Nimotide etc.

Vasodilators

- Arteriolar : diazoxide, hydralaxine, minoxidil
- Areteriolar and veno : sodium nitroprusside

Vasodilators - Hydralazine

- Directly acting vasodilator
- MOA: hydralazine molecules combine with receptors in the endothelium of arterioles – NO release – relaxation of vascular smooth muscle – fall in BP
- Subsequenly fall in BP – stimulation of adrenergic system leading to
  - Cardiac stimulation producing palpitation and rise in CO even in IHD and patients – anginal attack
  - Tachycardia
  - Increased Renin secretion – Na+ retention
  - These effects are countered by administration of beta blockers and diuretics
- However many do not agree to this theory
- Uses: 1) Moderate hypertension when 1st line fails – with beta-blockers and diuretics 2) Hypertension in Pregnancy, Dose 25-50 mg OD

Vasodilators - Minoxidil

- Powerful vasodilator, mainly 2 major uses – antihypertensive and alopecia
- Prodrug and converted to an active metabolite which acts by hyperpolarization of smooth muscles and thereby relaxation of SM – leading to hydralazine like effects
- Rarely indicated in hypertension especially in life threatening ones
- More often in alopecia to promote hair growth
- Orally not used any more
- Topically as 2-5% lotion/gel and takes months to get effects
- MOA of hair growth:
  - Enhanced microcirculation around hair follicles and also by direct stimulation of follicles
  - Alteration of androgen effect of hair follicles

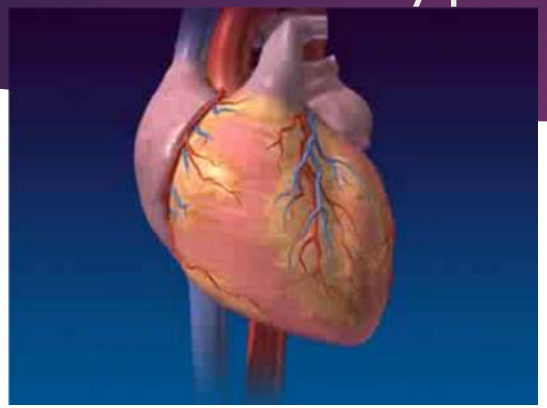
Sodium Nitroprusside

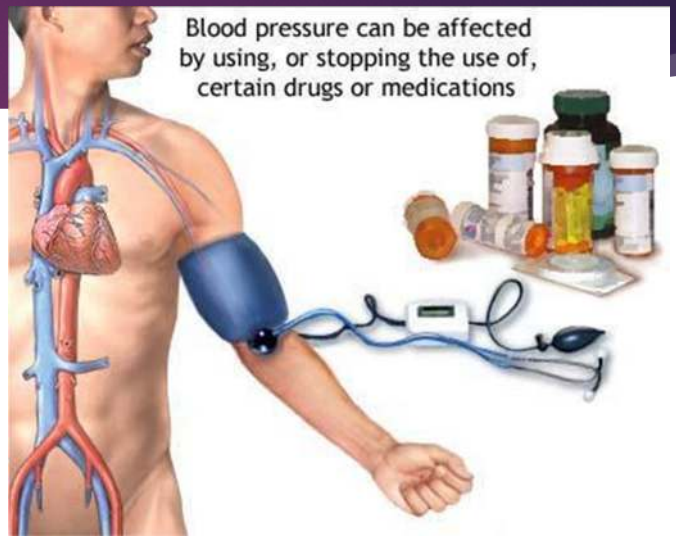
- Rapidly and consistently acting vasodilator
- Relaxes both resistance and capacitance vessels and reduces t.p.r and CO (decrease in venous return)
- Unlike hydralazine it produces decrease in cardiac work and no reflex tachycardia.
- Improves ventricular function in heart failure by reducing preload
- MOA: RBCs convert nitroprusside to NO – relaxation also by non-enzymatically to NO by glutathione
- Uses: Hypertensive Emergencies, 50 mg is added to 500 ml of saline/glucose and infused slowly with 0.02 mg/min initially and later on titrated with response (wrap with black paper)
- Adverse effects: All are due release of cyanides (thiocyanate) – palpitation, pain abdomen, disorientation, psychosis, weakness and lactic acidosis.

Centrally acting Drugs

- Alpha-Methyldopa: a prodrug
  - Precursor of Dopamine and NA
  - MOA: Converted to alpha methyl noradrenaline which acts on alpha-2 receptors in brain and causes inhibition of adrenergic discharge in medulla – fall in PVR and fall in BP
  - Various adverse effects – cognitive impairement, postural hypotension, positive coomb`s test etc. – Not used therapeutically now except in Hypertension during pregnancy
- Clonidine: Imidazoline derivative, partial agonist of central alpha-2 receptor
  - Not frequently used now because of tolerance and withdrawal hypertension
  - Read it yourself

Treatment of hypertension





Treatment of Hypertension: 7 classification

Cat

BP	Systolic	Diastolic
Normal	>120	<80
Prehypertension	120-139	80-89
Stage1	149-159	90-99
Stage2	>160	>100

Risk factors

1. Age above 55 and 65 in Men and Woman respectively
2. Family History
3. Smoking
4. DM and Dyslipidemia
5. Hypertension
6. Obesity
7. Microalbuminuria

Treatment of Hypertension –

- *7 compelling Indications*:
  - Heart failure
  - Coronary artery disease
  - H/o MI
  - H/o stroke
  - Diabetes
  - Chronic Renal failure

Treatment of Hypertension

Table 1. Classification and management of blood pressure for adults*

BP Classification	SBP* mmHg	DBP* mmHg	Lifestyle Modification	Initial drug therapy	
				Without Compelling Indication	With Compelling Indications (See Table 8)
Normal	<120	and <80	Encourage	No antihypertensive drug indicated.	Drug(s) for compelling indications.‡
Prehypertension	120–139	or 80–89	Yes		
Stage 1 Hypertension	140–159	or 90–99	Yes	Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.	Drug(s) for the compelling indications.‡ Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed.
Stage 2 Hypertension	≥160	or ≥100	Yes	Two-drug combination for most† (usually thiazide-type diuretic and ACEI or ARB or BB or CCB).	

DBP, diastolic blood pressure; SBP, systolic blood pressure.

Drug abbreviations: ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; BB, beta-blocker; CCB, calcium channel blocker.

* Treatment determined by highest BP category.

† Initial combined therapy should be used cautiously in those at risk for orthostatic hypotension.

‡ Treat patients with chronic kidney disease or diabetes to BP goal of <130/80 mmHg.

Treatment of Hypertension – General principles

- Stage I:
  - Start with a single most appropriate drug with a low dose. Preferably start with Thiazides. Others like beta-blockers, CCBs, ARBs and ACE inhibitors may also be considered. CCB – in case of elderly and stroke prevention. If required increase the dose moderately
  - Partial response or no response – add from another group of drug, but remember it should be a low dose combination
  - If not controlled – change to another low dose combination
  - In case of side effects lower the dose or substitute with other group
- Stage 2: Start with 2 drug combination – one should be diuretic

Treatment of Hypertension – combination therapy

- In clinical practice a large number of patients require combination therapy – the combination should be rational and from different patterns of haemodynamic effects
  - Sympathetic inhibitors (not beta-blockers) and vasodilators + diuretics
  - Diuretics, CCBs, ACE inhibitors and vasodilators + beta blockers (blocks renin release)
  - Hydralazine and CCBs + beta-blockers (tachycardia countered)
  - ACE inhibitors + diuretics
- 3 (three) Drug combinations: CCB+ACE/ARB+diuretic; CCB+Beta blocker+ diuretic; ACEI/ARB+ beta blocker+diuretic

Treatment of Hypertension.

- Never combine:
  - Alpha or beta blocker and clonidine - antagonism
  - Nifedepine and diuretic synergism
  - Hydralazine with DHP or prazosin – same type of action
  - Diltiazem and verapamil with beta blocker – bradycardia
  - Methyldopa and clonidine
- Hypertension and pregnancy:
  - No drug is safe in pregnancy
  - Avoid diuretics, propranolol, ACE inhibitors, Sodium nitroprusside etc
  - Safer drugs: Hydralazine, Methyldopa, cardioselective beta blockers and prazosin

Hypertensive Emergencies

- Cerebrovascular accident or head injury with high BP
- Left ventricular failure with pulmonary edema due to hypertension
- Hypertensive encephalopathy
- Angina or MI with raised BP
- Acute renal failure with high BP
- Eclampsia
- Pheochromocytoma, cheese reaction and clonidine withdrawal
- Drugs:
  - Sodium Nitroprusside (20-300 mcg/min) – dose titration and monitoring
  - GTN (5-20 mcg/min) – cardiac surgery, LVF, MI and angina
  - Esmolol (0.5 mg/kg bolus) and 50-200mcg/kg/min - useful in reducing cardiac work
  - Phentolamine – pheochromocytoma, cheese reaction nd clonidine withdrawal (5-10 mg IV)

Desirable to know/learn

- Classification of Antihypertensive
- Antihypertensive mechanisms: Diuretics, ACE inhibitors, ARBs, Beta-blockers, alpha-blockers, CCBs, Vasodilators and central sympatholytics
- Present status of above mentioned group of Drugs
- Common Adverse effects of above groups of Drugs
- Pharmacotherapy of Hypertension
- Pharmacotherapy of hypertensive emergencies
- Preparation and dosage of commonly used drugs of above mentioned groups

- Learn the anti hypertensive drugs drugs used in pregnancy
- Treatment of pulmonary hypertension

The End