Cheatography

Hypertension Drugs Cheat Sheet

by Carm (Carmilaa) via cheatography.com/49544/cs/16839/

Introduction:

- > most common CVS disease
- > elevated arterial BP damages vessels in renal, heart and brain

Compli Renal failure,
cat- Coronary disease,
ions: Heart failure, Stroke,
Diamentia

Ciassilication.		
Classific-	Systol	

Classific-	Systolic	Diastolic
ation:	BP	BP:
Normal:	<120	<80
Pre-hy-	120-	80-89
perten-	139	
sion:		
Stage 1	140-	90-99
HTN:	159	
Stage 2	>= 160	>=100
HTN:		

Types of Hypertension:

Essential	Primary hypert-
Hypert-	ension, no identi-
ension	fiable cause
(85-90%):	(genetic), can't be
	cured, can be
	controlled
Secondary	Specific identified
Hypert-	cause (comorbid
ension	disease or drug),
(10-15%):	can be cured when
	cause is eliminated

Secondary Causes of HTN:

- > Genetic factors
- > Psychological stress

> Enviro-	high salt diet,
nmental	decreased calcium
and	and phosphate
Dietary	intake, sedentary
Factors:	lifestyle

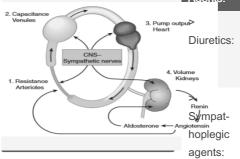
> renal, endocrine,
Diseases: vascular, renal
diseases

> Drugs: sympathomimetic amines, amphetamines, oral decongestants (eg. pseudoehedrine), corticosteroids, osteogens (C0Cs), NSAIDs, COX-2 inhibitors

Clinical presentation:

- Often incidental finding
- Adults should get BP checks once a year
- Headaches, visual
 Severe disturbances, target
 cases: organ damage
 (stroke, ischemic
 heart disease, renal
 failure)

Normal regulation of BP:



Factors Influencing BP:



Potential Mechanisms of Parthogenesis:

BP= CO x PVR

Increased Increased fluid
Cardiac volume from
output: excessive sodium
intake or renal
sodium retention
Venous constriction: due to
excess stimulation
of RAAS

Increased Peripheral resistance: Excess stimulation of RAAS Sympathetic nervous system over-activity

Classes of Antihypertensive

Reduce blood volume=Depletes the body of sodium, Venodilation

Reduce peripheral vascular resistance, Inhibit cardiac function, Increase venous pooling capacitance vessels

Reduce peripheral vascular resistance, Increase venous pooling capacitance vessels

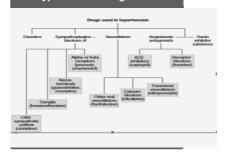
> Angiot- Reduction Reduct

Reduce peripheral vascular resistance, Reduce blood volume

Diuretics:

- Reduce blood volume and cardiac output
- Cardiac But peripheral
 output vascular resistance
 returns to declines
 normal
- Sodium Contributes to vascular resistance = Increase vessel stiffness

Antihypertensive Drugs:





By **Carm** (Carmilaa) cheatography.com/carmilaa/

Published 28th August, 2018. Last updated 28th August, 2018. Page 1 of 3. Sponsored by Readable.com Measure your website readability! https://readable.com



Hypertension Drugs Cheat Sheet

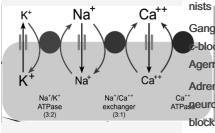
by Carm (Carmilaa) via cheatography.com/49544/cs/16839/

Diuretics: (cont)

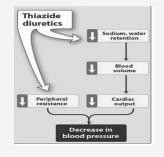
- Altered sodium-sodium

Venodi calcium exchange

Vasodilation mechanism by Diuretics:



Thiazide Diuretics:



Use of Diuretics:

- > Lower BP by 10-15 mmHg in most patients
- > Thiazide diuretics –mild to moderate HTN
- > Loop diuretics severe HTN and hypertensive emergencies

Adverse Effects (Thiazide Diuretics):

Potassium depletion – hypokalemia

Magnesium depletion

Hyperuricemia- gouty attacks

Glucose intolerance

Increase serum lipid concentrations

Sympathoplegic Drugs:

Centrally- Methyldopa, acting clonidine

Drugs:

Adreno- Beta-blockers, ceptor alpha-1 blockers antago-

Ganglionic-blocking Agents:

Adrenergic

Ca**
ATPasseuron
blocking
agents:

No longer used clinically; hexamethonium Block the release

of noradrenaline, Reserpine, guanethidine, debrisoquin, Not/rarely used clinically

Centrally-Acting Drugs:

- Methyl- rarely used dopa, except clondine clonidine
- Reduces sympathetic outflow
- Compen-salt retention satory response mechanism:
- Clonidine, Stimulate central guanabenz, alpha-2 adrenoguanfacine ceptors
 Results in the
- Methyldopa synthesis of a

 anologue false neurotranof L-dopa smitter

Centrally-Acting Drugs: (cont)

Alpha-methylnoradrenaline = Stimulates central alpha-2 adrenoceptors

Clonidine:

Reduces cardiac output, PVR, relaxation of capacitance vessels

Rarely causes postural hypotension

Adverse effects:

- Dry mouth
- Sedation

Contraindication: Patients with depression

Caution: Abrupt discontinuation can lead to hypertensive crisis

Adrenoceptor antagonists: Betablockers

Non-selective: Propranolol

Beta-1 Betaxolol, bisoprselective: olol, esoprolol,

"BBEAM" atenolol, metoprolol,
Cardioselective

Vasodi- Also block alpha-1 lator receptors,Labetolol, beta-b- carvidelol, nebivolol lockers

Decrease cardiac output

Decrease peripheral vascular resistance

Inhibit stimulation of renin production by catecholamines

Adverse effects= Heart block, bronchoconstriction, diabetes, vivid dreams

Alpha-1 blockers:

Prazosin, terazosin, doxazosin Block alpha-1 receptors in arterioles and veins

Vasodilation Reduce

peripheral resistance

Compen- salt and water satory retention

mechanism:

More effective when used with other drugs

Calcium Channel Blockers:

> amlodipine, isradipine,
Dihydr nicardipine, nimodiopy- pine, felodipine, nisoldridines ipine, lacidipine

Verapamil, diltiazem,Non- hydralazine

dihydropyridines

> Benzothiazepine (diltiazem)

Mechanism of action:

- Inhibit calcium influx through voltage-dependent L-type calcium channels
- Relax arteriolar smooth muscle, reduce peripheral vascular resistance
- Cause coronary and peripheral vasodilation

CCB: Mechanisms of Action:

Dihydr Primary vasodilators
opyridine decrease cardiac
CCBs contractility except
amlodipine and
felodipine



Cheatography

Hypertension Drugs Cheat Sheet

by Carm (Carmilaa) via cheatography.com/49544/cs/16839/

CCB: Mechanisms of Action: (cont)

Non-dihydropyridines

AV node, decrease
heart rate, decrease
(diltiazem, cardiac contraction
verapamil)

Adverse effects:

1. Flushing, peripheral oedema, tachycardia, bradycardia, heartblock

- 2. Headache, flushing, dizziness, palpitations, hypotension occur within a few hours of dosing, Associated w high initial doses or rapid increase in dose, Common with short-acting preparations
- 3. Ankle oedema: Due to a rise in intracapillary pressure as a result of selective dilatation of precapillary arterioles, NOT due to sodium retention, Relieved by bed rest
- 4. **Gum Hypertrophy:** dihydropyridines
- 5. GIT: constipation (verapamil), nausea, and vomiting

Inhibitors of Angiotensin:

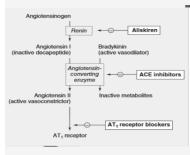
Angiotensin Captopril, converting enalapril, enzyme Ramipril, inhibitors fosinopril, (ACEIs) trandopril

Inhibitors of Angiotensin: (cont)

Angiot- Losartan,
ensin valsartan, telmisreceptor artan, irbesartan,
blockers candesartan
(ARBs)

Renin-inh- Aliskiren ibitors

Inhibitors of Angiotensin:



Adverse Effects

ACEIs =

Dry cough

Can cause hyperkalemia – potassium monitoring essential Angioedema (substance P?)

ARBs =

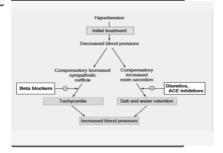
No dry cough

Hyperkalemia

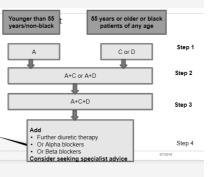
Angiodema is less common than ACEIs

Contraindicated in pregnancy

Combination Treatment: Vasodilators:



Management Approach:



First-line agents: "ACD" drugs:

- A: ACEIs and ARBs
- C: Calcium channel antagonists
- D: Diuretics (Thiazides)

Second-line agents:

- Beta-adrenoceptor blockers
- Aldosterone antagonists (spironolactone, eplerenone)
- Alpha-blockers (doxazosin, prazosin, terazosin)
- Loop diuretics (frusemide, torsemide)
- Direct vasodilators (hydralazine, minoxidil) [last-line of therapy]
- Central α-2 agonists (clonidine)
- Adrenergic antagonists (reserpine)

C

By **Carm** (Carmilaa) cheatography.com/carmilaa/

Published 28th August, 2018. Last updated 28th August, 2018. Page 3 of 3. Sponsored by **Readable.com**Measure your website readability!
https://readable.com