***Name Saman Khan***

***ID 14957***

***BS DT***

***Submitted to ma’am Nadra***

***General pharmacology assignment***

***4th semester***

***Various drugs classes used for Hypertension:***

***1)Diuretics:***

***Mechanism of action:***

*Act on V-2 receptor in kidneys (vasopressin receptor) having an antediuretic function leads to diuresisIncreased secretion of Na & H2O decrease in blood volume thus Decreased CO Decreased Blood Pressure*

***Specific agents:***

*1)Thiazides diuretics:*

*chlorthalidone*

*2)Loop diuretics:*

*Furosemide*

*3)K+ sparing diuretics:*

*Spironolactone*

***Adverse effects:***

* *dizziness,*
* *electrolyte*
* *imbalance/depletion,*
* *hypokalemia,*

***2)Sympatholytics Drugs:***

* *Central Sympatholytics (a-2 Agonists)*
* *Sympatholytic drugs a-1 Adrenergic blockers (Antagonists) :*
* *adrenergic blockers ( Antagonists ):*
* *b-1 Dual Alpha and Beta receptor anatagonists:*

***1)Central Sympatholytics (a-2 Agonists)***

***Drugs:***

* *clonidine,*
* *methyldopa*

***Site of Action:***

*CNS medullary cardiovascular centers*

***Mechanism of action:***

*CNS a-2 adrenergic stimulation autoinhibitory feedback mechanism which decreased sympathetic outflow decreased norepinephrine release vasodilatationdecreased PR and decreased BP.*

***Adverse Effect:***

* *dry mouth*
* *sedation*
* *drowsiness*
* *nasal congestion*

***2)Sympatholytic drugs a-1 Adrenergic blockers (Antagonists):***

***Drugs:***

* *Prazocin*
* *Terazocin*

***Site of Action:***

* *peripheral arterioles*
* *smooth muscle*

***Mechanism of action:***

*Blocks a-1 receptor (in post synaptic neurone as well as in vascular smooth muscles) Which cause vasodialation due to relaxation of vascular smooth muscles decreased PRalso reduces preload by pooling of blood decreased CO and decreased BP.*

***Adverse Effects:***

*nausea,,Drowsiness ,postural*

***2)Vasodilators:***

***Arteriola:***

 *Hydralazine*

***Arterio-venular:***

*Sodium Nitroprusside used in emergency situation*

***Mechanism of action:***

*Releases NO stimulation of guanylyl cyclase more conversion of GTP to cGMP and activate protein kinase myosin phosphorylation & combination with actin inhibited relaxation of vascular inhibit \*MLCK phosphorylation-smooth muscles*

***3)Calcium channel blockers:***

***Drugs:***

*verapamil*

*nifedipine*

*diltiazem*

*amlodipine:felodipine*

***Site of Action:***

 *Vascular smooth muscle K+, Ca+, Na+*

***Mechanism of action:***

*Blocks long acting voltage sensitive calcium channels*

***Side effects:***

* *flushing*
* *headache*

***Various drugs classes used for cardiac heart failure:***

***1) Drugs That Increase Myocardial Contraction Force***

***Which Includes:***

***1)Cardiac glycosides:***

***Specific agents:***

* *Digoxin*
* *Digitoxin*

***Adverse effects:***

* *Gastrointestinal symptoms:*
* *Anorexia*
* *Nausea*
* *Abdominal pain*
* *Visual disturbances:*
* *Photophobia*
* *Scotomata*

***2)Phosphodiester inhibitors:***

***Mechanism of action:***

*Phosphodiesters agents cause a cAMP-mediated increase**in intracellular calcium*

 *which increases the force of contraction within the myocardial cell.*

***specific agents:***

* *Inamrinone*
* *Milrinone*

***3)Dopamine and Dobutamine:***

***Mechanism of action:***

*It exert a fairly specific positive inotropic effect, presumably through their ability to stimulate beta-1 receptors on the myocardium.*

*Other beta-1 agonists (epinephrine, prenalterol, etc.) will increase myocardial contractility,*

 *but most of these other beta-1 agonists will also increase heart rate or have other side effects that prevent their use in congestive heart failure*

***2)Miscellaneous drugs for chronic heart failure:***

1. ***ACE inhibitors:***

***Mechanism of action:***

*It supresses the enzyme that converts angiotensin I to angiotensin II in the bloodstream.*

*By inhibiting the formation of angiotensin II, ACE inhibitors limit peripheral vasoconstriction*

*Decrease in cardiac workload primarily by decreasing the pressure against which the heart must pump (cardiac afterload).*

*Decreased cardiac afterload eases the strain on the failing heart, resulting in improved cardiac performance and increased exercise tolerance.*

***Specific agents:***

* *Captopril*
* *Enalapril*
* *Nesirtide*

***2)Angiotensin II Receptor Blockers:***

***Mechanism of action:***

*These drugs prevent angiotensin II from binding to receptors on vascular tissues, thus inhibiting angiotensin II–induced damage of the cardiovascular system. It appears that ARBs are as effective as ACE inhibitors in treating heart failure and preventing mortality*

***Specific agents***

* *Candesartan,*
* *losartan*
* *Valsartan*

***Adverse effect:***

* *skin rashes,gastrointestinal discomfort, and dizziness.*

***4)Diuretics:***

***Mechanism of action***

*Diuretics work by inhibiting the reabsorption of sodium from the nephron*

 *decreases the amount of water that is normally reabsorbed with sodium*

*thus increasing water excretion*

*This effect reduces congestion which caused by fluids retained in the body and decreases cardiac preload by excreting excess fluid in the vascular system.*

***3)Vasodialation:***

***Function:***

*Its function is to Decrease workload on heart*

***Includes***

* *Nitroprussside*
* *Nitrates*
* *Hydralazine*

***Mechanism of action:***

*blocking alpha-1 receptors on vascular smooth muscle* ***and*** *vasodilators work by different mechanisms*

*decrease cardiac workload by decreasing peripheral vascular resistance*

***Specific agents:***

* *Prazosin,*
* *hydralazine,*
* *organic nitrates*

***Adverse effect:***

*headache ,dizziness ,hypotension ,orthostatic hypotension*

***TH***