Pharmacology Mechanism of Action (Part-1)

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MECHANISM OF BENZODIAZEPINES

- BZD is a CNS depressant drug. When BZD bind to $GABA_A$ receptor, it facilitates the inhibitory neurotransmitter GABA to its receptor surface. By this frequency of cl^- channel opening increases and it leads to hyperpolarization. The neuronal excitement decreases.

MECHANISM OF MORPHINE

- Morphine is an opioid analgesic, when it binds to its specific receptor (k -receptor) surface. At pre-synaptic neuron, it inhibits the Ca^{2+} channel results decrease in neurotransmitter release and decrease the pain perception.
- In post synaptic neuron, morphine binds to μ and δ receptor and improves k^+ efflux which show hyperpolarization and pain is reduced.

MECHANISM OF ACETYLCHOLINE

- Ach when binds to cholinergic receptor (i.e., muscarinic and nicotinic). It causes vasodilation and release of nitric oxide.
- Muscarinic action- decrease heart rate (bradycardia)
    - Increase glandular secretions
    - Contraction of smooth muscle
- Nicotinic action- stimulant effect

MECHANISM OF ATROPINE

- It is a protype drug obtained from *Atropa belladonna*. Atropine blocks the effects of acetylcholine on muscarinic receptor.
- It is non-selective and blocks all type of muscarinic receptors.

MECHANISM OF ADRENALINE

- It is a catecholamine, which is secreted mainly by adrenal medulla. It is direct acting adrenergic agonist. It binds to adrenergic receptor (alpha and beta) by stimulating nor epinephrine or epinephrine.

MECHANISM OF SALBUTAMOL

- Salbutamol is selective short acting beta2 -agonist. It increased cAMP concentration and inhibit release of histamine, leukotriene from the mast cells It has smooth muscle relaxant properties, which allow the inhibition of bronchial smooth muscle contraction and leads to bronchodilation.

MECHANISM OF CHLORPROMAZINE

- Chlorpromazine is a neuroleptic drug comes under phenothiazines. It blocks dopaminergic receptors in cortical and limbic areas of the brain, by preventing the excess of dopamine in the brain. It reduces psychotic symptoms such as hallucination and delusion.

MECHANISM OF PHENYTOIN

- It is an anti-epileptic drug comes under hydantoin derivative. It blocks the voltage gated Na^+ channel and this inhibits the generation of repetitive action potential. At higher dose it reduces the influx of Ca^{2+}during depolarisation and supress repetitive firing neurons. Both the action decreases glutamate release.

MECHANISM 0F LOACAL ANAESTHETICS

- These are the agent which upon local administration or local injection, loss of sensation on reversible loss of sensory perception especially of pain in a localised area.
- LAs (which are in unionised form) enter to axoplasm transversely. It gets ionised and binds to its receptor (which are present in intracellular domain). It blocks the Na^+ channel and no entry of Na^+ ions into axoplasm. So sensory nerve impulse fails to reach CNS and pain can't produce.

MECHANISM OF PROPRANOLOL

- It selectively blocks the beta1adrenergic receptor in heart and decreases force of contraction, heart rate, cardiac output and blood pressure.
    - Selectively blocks the beta 1 receptor of kidney
    - Then decrease renin secretion

- Decrease effect of renin angiotensin system
- Decrease BP

MECHANISM OF LEVODOPA

- Levodopa when administered; it is converted to dopamine by DOPA decarboxylase enzyme and maintain the dopamine level in the receptor site (D_2 receptor) and decrease parkinsonian effects.
- It is metabolised by peripheral tissue and GIT ,95%, so along with carbidopa it is given.

MECHANISM OF CENTRALLY ACTING MUSCLE RELAXANT

- The drug inhibit transmission in poly-neuronal pathway, control muscle tone.
- So, they inhibit spinal interneurons and promote inhibitory influence of the brain stem reticular formation on spinal motor neurons.
- They reduce spasm of skeletal muscle without affect voluntary movement.
- Mephenasin act on the spinal cord and inhibit polysynaptic reflex without affect the monosynaptic (knee jerk) tendon reflex.

MECHANISM OF SSRIs (Selective serotonin reuptake inhibitors)

- SSRIs block the reuptake of serotonin from the synapse into serotonergic nerve endings. This effect is due to inhibitor of serotonin transporter.

MECHANISM OF TCAs (Tricyclic antidepressants)

- TCAs block the reuptake of both noradrenalin and serotonin into presynaptic terminals.
- This effect is due to binding with
  - 1.Nor epinephrine transporter (NET)
  - 2.Serotonin transporter (SERT)
- So, the synaptic levels of these amines increase. So, their action on receptor is increased. This produced anti-depressant effect.