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paper Subject.
pharmacology. 1

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Ans:- (1)

Non selective cox Inhibitor:-

Salicylates:
Aspirin

propionic acid derivatives:-

Ibuprofen
naproxen
Ketoprofen
flurbiprofen
fenamate:
mephenomic acid.

Enolic acid derivatives:-
proxicam
tenoxicam

Acitic acid derivatives:-

Ketorolac - Indomethacin - Nabumetone

MOA of NSAIDS:

Normally-

we know that when a stimullis comes it disturb the cell membrane - cell membrane with release phospholipid phosphate with convert into Arachidonic acid with help of phospalipid enzyme Then arachidonic acid with the help of cyclo oxygenase enzyme produce prostoglandin and thromboxene which is Inflamintory mediator.

- prostoglandin cause alternation in the vascular permeability cause Bronchai construction and also increase the secreation of muscle which can lead to Bronchospasm congection and muscle pluging.

Stumulus

↓

Disturb the cell membrane

release phospholipids

↳ phospholipids A₂ enzym

Arachidonic acid

↳ cycloxoygence enzym

prostoglandin → alternation of vascular permbiality brochiol conduction increase muscle secreation Bronchospasm congestion muscle plugin

Thrumboxene → Inflammation

So when we give NSAIDS to patents it will block the cyclooxygenase enzyme so Archidonic acid will not release prostoglandin and ~~thromb~~ thromboxane and inflammation will not occure.

(7) Adverse effects of NSAIDS on GIT:

mediators.

(1) prostacyelint • it inhibit the synthesis of gastric acid secretions. • so when we give NSAID it will the synthesis increase of gastric secreation.	(2) prostaglandin. -It stimulate the synthesis of protective mucus in both the stomach and small intestine. • So when we give NSAID it will inhibit the synthesis of protectiv mucus and increase the risk of Ca bleeding and ulcantive.

In this situation we give PPI and H2 blocker to treat the condition.

Ans: (2)

Sedative	Hypnatics
• A drug that subdues excitement and calms the subject without inducing sleep. But drowsiness may produce.	- A drug that induces and/or maintance sleep similar to normal arousable sleep.
• sedation refers to responsiveness to any level of stimulation.	
• associated with some motor activity and ideation.	

(B) MOA of Benzodiazepines:

- it modulate the GABA effect by binding to a specific high affinity site located at the interface of the α and γ subunits and γ subunits on the GABA receptor.
- it increase the frequency of channel opening produced by GABA.

Side effects:

Dizziness, vertigo ataxic, disorientation, amnesia prolongation of reaction time.

impairines of Psychomotor skills.
Hangover weakness blurring of vision, dry mouth and urinary incontence.

MOA of Barbiturales:

it depress the neuronal activity in the mid brain reticular formation facilitating and prolonging the Inhibitory effects of GABA and glycine.

It also bind with multiple isoforms of the GABA receptor but at different sites from those with which benzodezipines intract.

- It increase the duration of GABA - medicted chloride ions channels opening.
- also block the excitatory transmitter glutaric acid and at high concentration sodium channels.

adverse effects:

Hangover mental confusion, impaxied performance and traffic accident.
excitement, idosyncratic reaction, also produce Hypersensitivity reatins like rashes swelling of eyelid etc.

Ans:- (3)

General anesthesia	local anesthesia.
-apply all over the body.	Apply on a specific part of the body.
- It induced the reversible lose of consciousness and all sensation.	-It blocks the nerve conduction in cause reversible lose of all sensation in the part supplied by the nerve.

Stages of Anesthesia.-

Stages (1) (Stage of analgesia.

In this stage patients is conscious but drowsy.

Stage II) (Stage of excitement)

sympathetic activity is increased
↑es Heart rate and BP.
↑es Muscle Tone
Irregular Breathing.

Stag III) (Stage of surgical anesthesia

→ Respiration becomes regular
Muscles relax
Reflexes are gradually loss.

Intercostal muscle are paralysed.

Stage IV) (Stage of medullary paralysis)

→ Respiration and vasomotor center are depressed.
→ Death occurs within few minutes.

MOA of local Anesthesia:

local anesthetic are weak basis

At tissue pH ~~at~~ 7.4

partly unionized.

partly ionized

↓

penetrate the nerve membrane

↓

Enter the axon

↓

Reionization of local anesthesia

↓

which gains access to its receptor in the open state of the channels

↓

Block the voltage Na+ channels from inside.

↓

(e)

↓

prevent entry of Na+ ions into the neuron

↓

prevent generation of action patient.

↓

No generation and conduction of impulses of CNS.

Ans- (4)

MOA of Morphine:

Morphine inhibit the release of many excitatory transmitter from nerve terminals which carry painfull stimulus.

Pharmacological action: on CNS:

It have depresent actions.

(a) Analgesia:

It cause ~~drug~~ analgesia by raising the pain threshold and by altering the brains preception of pain.

(b) sedation:

Morphine cause drowsiness and indifference to surrounding as well as to own body.

occurs without motor incoordination.

(c) Mood and subjective effects:

Morphine has a calming effect there is loss of apprehension feeling of detachment lack of initiative, limbs feel heavy and body warm inability to concentrate.

(d) Respiratory center:

Depress the respiratory center.

(e) cough center:

Morphine depress the cough center.

(f) Temperature regulating center.

depress the regulating center and decrease the temperature.

(g) vasomotor center:

depress the vasomotor center.

(h) CVCUS:

Morphine causes vasodilation due to.

(i) release of histamine.

(ii) depression of vasomotor center.

(iii) decreasing tone of blood vessels.

③ on GIT :-
↑es tone and segmentation but decrease propulsive movements
↑es GIT secretions due to reduction in movement of water and electrolytes from mucosa to the lumen.

④ Hormonal actions:
= It inhibits the release of gonadotropin releasing hormone, leuthnizing hormone follicle stimulating hormone adrenocorticotropic hormone.

- morphine ↑es growth hormones release and enhance prolactin secretion.
- it also ↑es antidiuretic hormones and thus leads to urinary retention.

Clinical uses of morphine-
- For pain (analgesic)
- for treatment of diarrhea
- for Relief of cough
- and for treatment of acute pulmunary edema.

ADRs:-
- sedation
- mental clouding.

- lethargy.
- Dysphoric in some subjects
- vomitting is occasional
- constipation
- Respiratory depression
- Blurring of vision
- urinary retention.

Idiosyncrasy and allergic reactions:

- urticaria.
- swelling of lips occurs Infrequently.
- A local reaction at injection site and generalized itching may occur due to histamine release.

Ans: (8)

(a) Neuronal systems:
There are two types.

Hierachial system	(a) Diffuse system
This system is a demilinated system.	it contain single cell which give branches to different areas.
It contain large myliniated fiber which are rapidly conduct.	It contain neuro-transmeter such as amines or peptide which comming

It have both excitatory and Inhibatory Neurotrammited.	exert action of metabotropic receptor.

Excitatory
- aspartate
- glutamate

Inhibitory Transmitt
- GABA
- glycine.

① Neurotransmitters:

it decrease the membrane permability to K^+

Example:
Drugs used in Alzheimer's disease e.g rivestigmine muscarnic blocking agent used in parkinsin e.g benztropine.

② Dopamine:

exerts slow inhibitory action at synapses in specific neuronal system.

③ Neuropinephrine:

Excitatory effects.
- are produce by activation of α_1 and β_2 receptor.

④ Serotonin:
cause excitation or Inhibition of CNS neurons depending on the receptor sub type activited.

⑤ Glutamic Acid:
appers to play a role in synaptic plasticity releated to learning and memory.

⑥ GABA and Glycine:
GABA receptor activities open Cl channels GABA$_B$ receptor are couple to G protein that either open K^+ channels or close Ca^+ channels

⑦ peptide Transmetter:
Two sub classes.

① opioid peptides
These are mediated via activation of receptor for these endogenous peptide.

Substance peptide is a mediator of slow EPSPs is neurons involved in nociceptive sensory pathways in the spinal card and brain stem.

(5) Endocannabinoids.

They are synthesized are released postsynaptically often ~~travel backward~~ depolarization but travel backward acting presynaptically to decrease transmitte release via their interaction with a specific cannabinoid receptors.