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Q No 3:

Answer:-

1) General Anesthesia:-

These are

the drugs that induced reversible loss of consciousness and all sensation.

feature of GA:-

- 1) Reversible loss of consciousness
- 2) Reversible loss of sensation
- 3) Analgesia and amnesia
- 4) muscle relaxation and abolition of reflexes.

Anesthetic protocols:-

- 1) premedication
- 2) Induction of Anesthesia
- 3) maintenance of anesthesia
- 4) Skeletal muscle relaxation
- 5) analgesia as pre-medication during and after operation.
- 6) use of other drugs
  - To reverse neuromuscular blockage.
  - To reverse the residual effect of opioids (naloxone) and benzodiazepines (flumazenil).

## 2) Local anesthetics :-

Local anesthetics are the drugs which when applied topically or injected locally, block nerve conduction and cause reversible loss of all sensation in the part supplied by the nerve.

### Order of blockage:

Nerve function is blocked in following manner:  
Pain - temperature - touch - pressure - Skeletal muscle power.

### Mechanism of Action of Local Anesthesia

Local Anesthetics act on voltage sensitive  $\text{Na}^+$  channel.

Local anesthetics are weak bases.

partly unionized  
penetrate the nerve membrane  
enter the axon (axonal pH is low)  
Reionization of local anesthetics.  
Local anesthetics gain access to its receptor in the open state of the channel.  
LAs block the voltage gated  $\text{Na}^+$  channel from inside, prevent entry of  $\text{Na}^+$  ion into the neuron (decreasing the rate of depolarization).  
prevent generation of action potential.  
No generation and conduction of impulses to CNS.

at tissue pH 7.4  
partly ionized

## Stages of Anesthesia :-

### Stage I (Stage of analgesia) :-

The patient is

Conscious but drowsy.

### Stage II: (Stage of excitement)

- Patient loses consciousness
- Sympathetic activity is increased
- Increased heart rate and increased blood pressure
- Muscle tone increased
- Breath is irregular.

### Stage III (Stage of Surgical anesthesia)

- Respiration become regular
- Muscles relaxes
- Reflexes are gradually lost
- Intercostal muscles are paralysed.

### Stage IV (Stage of medullary paralysis)

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

Q No 1:-

Answer: Anthelmintic :- These are group of antiparasitic drugs that expel parasitic worms and other internal parasites from the body by either stunning (immobility or unconscious) or killing them with out causing significant damage to the host.

Mebendazole :-

Mechanism of Action :-

Like albendazole, this drug selectively damage intestinal cell in these worms. Thus inhibiting the uptake and intracellular transport of glucose and other nutrient into these parasites. This activity leads to the destruction of the epithelial lining and subsequent death of the parasite.

Side effect :-

Mebendazole is a relatively safe drug although some mild, transient, gastrointestinal problems, may occur.

## Praziquantel :-

### Mechanism of Action :-

This drug's exact mechanism of action is unknown. Praziquantel may stimulate muscular contraction of the parasite, resulting in a type of spastic paralysis, which causes the worm to lose its hold on intestinal or vascular tissue. In higher concentrations, this drug may initiate destructive change in the integument of the worm, allowing the host defense mechanism (e.g. enzyme, phagocytes) to destroy the parasite.

### Side effect :-

Praziquantel is associated with a number of frequent side effects including gastrointestinal problems (abdominal pain, nausea, vomiting), CNS effects (headaches, dizziness), and mild hepatotoxicity.

## Piperazine Citrate:-

### Mechanism of action:-

This drug appears to paralyze the worm by blocking the effect of acetylcholine at the parasite neuromuscular junction. The paralyzed worm can then be dislodged and expelled from the host (human) intestine during normal bowel movement.

### Side effect:-

Side effect such as headache, dizziness, and gastrointestinal disturbances may occur during piperazine citrate administration but these effects are generally mild and transient.

Q105 :-

Ans (A) :-

Role of vitamin K :-

Deficiency of vitamin

K a fat soluble vitamin is most common in older person with abnormalities of fat absorption and newborn, who are at risk of bleeding due to vitamin K deficiency. The deficiency is readily treated with oral or parenteral phytonadione (vitamin K) -

Blood Clotting :-

Inadequate blood clotting can result from vitamin K deficiency, genetically determined error of clotting factor synthesis (eg. hemophilia) a variety of drug induced condition and thrombocytopenia.

Treatment involve administration of vitamin K, proformed clotting factor or anti plasmin drugs.



## (B) Thrombolytic agent:-

Mechanism of action: Plasmin is an endogenous fibrinolytic enzyme that degrades clots by splitting fibrin into fragments. The thrombolytic enzyme catalyze the conversion of the inactive precursor plasminogen to plasmin.

1) Tissue plasminogen activator: PA is an enzyme that directly convert plasminogen to plasmin. It has little activity unless it is bound to fibrin. which is theory should make it selective for the plasminogen that has already bound to fibrin. (ie in a clot) should result in less danger of wide spread production of plasmin and spontaneous bleeding.

2) Streptokinase:- Streptokinase is obtained from bacterial although not itself is enzyme Streptokinase form a complex with endogenous plasminogen.

Q No 41

Ans:-

Alkylating agent:-

Mechanism of Action:-

The alkylating agent  
(R) Causes alkylation of guanine ~~nucleotides~~  
nucleotides located in the DNA strand.  
Cross link or then formed between two  
alkylated guanines. Thus creating strong  
bonds between or within the DNA strand  
in this case these cross link effectively  
tie up the DNA molecule reducing the  
ability of the DNA double helix to untwist.  
if the DNA double helix cannot unravel.  
the genetic code of cell cannot reproduce  
and cell reproduction is blocked.

Specific agent:-

- Alkylamine
- Busulfan
- Chlorambucil
- Cyclophosphamide
- Ifosfamide

Adverse drug reaction.

GI distress

Blood disorder

CNS Neurotoxicity.

## Antimetabolites :-

### Mechanism of action :-

Cell are able to synthesize genetic material - (DNA, RNA) from endogenous metabolites known as purin and pyrimidin nucleotides. Certain anticancer drugs are structurally similar to these endogenous metabolites and compete with these compound during DNA/RNA biosynthesis.

### Specific agent :-

### Adverse effect :-

Methotrexate

mercaptopurine

Thioguanine

Cytarabine

Fluorouracil

Flouxidine

- blood disorder (anemia, leucopenia, Thrombocytopenia)
- GI distress (including ulceration of GI tract)
- Skin disorder
- Hepatotoxicity
- CNS effect

## Plant alkaloids :-

### Mechanism of action :-

These agents are also called antimetabolic drugs. Because these agents (vincristine, vinblastine, vinorelbine) inhibit the formation of the mitotic apparatus.

### Specific agent :-

### adverse effect :-

- vincristine
- vinblastine
- vinorelbine
- paclitaxel
- Docetaxel

- blood disorder
- Hypersensitivity
- Joint/muscle pain
- peripheral neuropathies.
- GI distress.

Q No 2

Ans: (A)

Type I Diabetes mellitus:-

A form of chronic hyperglycemia caused by immunologic destruction of pancreatic beta cell.

→ Type I diabetes require treatment with insulin.

Type II Diabetes mellitus:-

A form of chronic hyperglycemia initially caused by resistance to insulin often progress to insulin deficiency.

→ The early stage of type 2 diabetes usually can be controlled with noninsulin antidiabetic drug.

(B) 1. Insulin:-

Effect:- Insulin has important effect on almost every tissue of the body - the major target organ for insulin action include.

P - T - O

① Liver:

Insulin Increase the storage of glucose as glycogen in the liver. This involves the insertion of additional GLUT2 glucose transport molecules.

② Skeletal Muscles:-

Insulin stimulate glycogen synthesis and protein synthesis. Glucose transport into muscle cell is facilitated by insertion of GLUT4 transporter into cell plasma membranes.

③ Adipose tissue:-

Insulin facilitate triglyceride storage by activating plasma lipoprotein lipase. Increasing glucose transport into cell via GLUT4 transporters and reducing intracellular lipolysis.