Name:shahid noor Section: B Semester: 2<sup>nd</sup> I'd: 16119 Department: MLT

## ÷= Q No: 1 ( drug interaction )

Drug interaction is defined as the pharmacological activity of one drug is altered by the concominant use of another drug or by the presence of some other substances.

(types)

- Drug- drug interactions.
- Drug-food interactions.
- Chemical- drug interactions.
- Drug- laboratory test interactions.
- Drug disease interactions.

*Part: B ( pharmacodynamic drug interaction )* 

Pharmacodynamic drug interaction are those in which the activity of the object drug at its site of action is altered by the preciptant. Such as interactions may be direct or indirect.

1: indirect pharmacodynamic interactions In which both the object and the preciptant drug have unrelated effects. But the letter in some way alter the effect but latter in some way alter the effect of the former.

**Example:** salicylates decrease the ability of the platelets to aggregate this impairing the homeostasis if warfarin induced bleeding occurs.

2: Direct pharmacodynamic interactions In which drugs having similar or opposing pharmacological effects are used concurrently.

• Antagonism

The interacting drugs opposing actions.

- Example: acetylcholine and noradrenline have opposing effects on heart rate.
- Addition or summation

The interacting drugs have similar actions and the resultant effect is the same of individual drug responses.

- Example : cns depressants like sedatives and hypnotic etc.
- Synergism or potentiation

*It is an enhancement of action of one drug by another.* 

• Example: alcohol inhensces the analgesic activity of aspirin.

÷= Q No:2 1: hypoglycemic

- Hypoglycemia refers To a deficiency of glucose in the blood.
- Blood sugar level drops less than 7omg/d1
- It can be caused by excessive intake of anti glycemic agents beyond the prescribed dose
- Commonest complications is diabetic ketoacidosis .
- Example: sweating, pallor, irritability, hunger, lack of coordination and sleeping.
- 2: hyperglycemic
  - Hyperglycemia refers an excess of glucose in the blood
  - Blood sugar level rises more than 130mg/dL
  - Can be caused by non- compliance of anti glycemic agents

• Commonest complications is hyperosmolar hyperglycemic nonkettic syndrome.

**Example:** dry mouth, increased thirst, weakness, headache, blurred vision and frequent urination.

=Part: B ( Emesis )

It is the action or process of the vomiting

#### • Antiemetic drug example

- 1. Antihistamine
- 2. Dopamine
- 3. Neurokinin receptor antagonists

#### =Part: c

Tab claritek 500mg Tab baydal Tab nuberal fort

Syp hydralin

#### ÷= Q No:3

=Part: A

#### • Antibiotics targets:

- 1. The inhibitions of cell wall synthesis
- 2. The disruption of cell membrane function
- 3. The inhibitions of translation
- 4. The inhibitions of metabolism
- 5. The inhibitions of transcription
- 6. The inhibitions of DNA replication **=Part: B** 
  - Viral replication

As virus are obligate intracellular pathogens they cannot replicate with out the machinery and metabolism of a host cell. Although the replicative life cycle of viruses differ greatly between species and category of virus, there are six basic stages that are essential for viral replication. 1. Attachment: viral proteins on the capsid of phospholipid envelope interact with specific receptors on the host cellular surface. This specific determines the host range of a virus.

#### 2. Penetration

The process of attachment to a specific receptors can induce informational changes in viral capsid proteins, or the envelope, that results in the fusion of viral and cellular membrane.

## 3. Uncoating

The viral capsid is removed and degraded by viral enzymes or host enzymes releasing the viral genomic nucleic acid.

## 4. Replication

After the viral genome has been uncoated transcription or translation of the viral genome is initiated. It is this stage of viral replication that differs greatly between DNA and RNA viruses and viruses with opposites nucleic acid polarity.

## 5. Assembly

After synthesis of viral genome and protein which can be post transcriptionaly modified viral proteins are packaged with newly replicated viral genome into new visions that are ready for release from the host cell.

## 6. Release

÷= Q No:4 classification of antihypertensive drug?

1. Diuretics

- Thiazides and congeners
- Loop diuretics
- Potassium sparing diuretics
- 2. Sympatholytic
- Centralling acting antidrenergic agents
- Alpha adrenergic blockers
- Beta adrenergic blockers
- Alpha- Beta adrenergic blockers
- 3. Vasodialators
- Nitric oxide releases
- Potassium channel openers
- Calcium channel blockers
- D1 dopamine receptors agonist
- 4. Angiotensin inhibitors and antagonists
- Angiotensin converting enzyme inhibitors.
- Angiotensin receptor antagonists.

#### *÷= Q No:4*

# Part: B causes and drug therapy of angina pectoris

- Coronary atherosclerosis
- Coronary artery spasm
- Transient platelets aggregation and coronary thrombosis
- Endothelial injury causing the accumulation of vasoconstriction substances.
- Coronary vasoconstriction following adrenergic stimulation.

Kinds

- Stable angina
- Unstable angina
- Variant angina
- Microvascular angina

#### ÷= Q No:5

## Difference between general and local anesthetics and various stages of anesthesia?

- 1. *General anesthetics:* which result in a reversible loss of consciousness.
- Local anesthetics: which cause a reversible loss of sensation for a limited region of the body with out necessary affecting consciousness.
  Stages of general anesthesia
- 1. Analgesia: the patient has decreased awareness of pain, some times with amnesia. Consciousness may be impaired but is not lost.
- 2. Disinhibition/ excitement : the patient appears to be delirious and excited. Amnesia occurs reflexes are enhanced, and respiration is typically irregular retching and incontinence may occur.

- 3. Surgical anesthesia: the patient unconscious and has no pain reflexes respiration is very regular, and blood pressure is maintained.
- 4. *Medullary :* the patient develops severe respiratory and cardiovascular depression that requires mechanical and pharmacological support.

*=Part : B: mechanism of action of narcotic analgesic* 

- Opioid have an onset of action that develop on the rout of administration.
- Opioid causes hyper polarisation of nerve cells, inhibitions of nerve firing and pre synaptic inhibitions of transmitter release.
- Cellular effect of these drugs involves enhancement of neural potassium efflux and inhibitions of calcium influx.

- Brainstem opioid receptor mediate respiratory depression produced by opioid analgesic.
- Constipation results from activation of opioid receptor in the CNS and in the GIT.

#### =Non- narcotic analgesic

- Depression of cyclooxygenases activity
- Decreasing of prostaglandins synthesis in the peripheral tissues and in the central nervous system.
- Decreasing of sensitivity of nervous endings and depression of Transmission of nociceptiv impulses on the level of CNS structures.
- Pain relieving action of non- opioid analgesic is partly connected with their anti- inflammatory activity.

The end of the paper