Mlt 2nd
General pharmacology 1
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Q1.
Q A.
Ans .

It is defined as “an alternation in the duration or magnitude of pharmacological effects of one drug produced by another drug, food, or
any other substance”.

Types

(drug-drug interaction) interactions between drugs come to mind

(drug-food interactions) interactions may also exist between drugs and foods

(drug-plant interactions) drugs and medicinal plants or herbs

(drug-disease interactions)

But there are essentially two types of drug interactions.

Pharmacokinetics DI

Pharmacodynamics DI

B.
Ans .Pharmacodynamics interactions:
Are those in which the activity of the object drug at its site of action is altered by the precipitant. Such interactions may be direct or indirect.
These are of two types

Direct pharmacodynamics interactions.

Indirect pharmacodynamics interactions.

Indirect pharmacodynamic interaction:
In which both the object and the precipitant drugs have unrelated effects. but the latter in Some way alerts the effects but latter in some way alerts the effects of the former.
Example: salicylates decrease the ability of the platelets to aggregate thus impairing the Homeostasis if warfarin induced bleeding occurs.
Direct pharmacodynamic interactions:
In which drugs having similar or opposing pharmacological effects are used concurrently.

The three consequences of direct interactions are
1. Antagonism (Antagonism : 1-1 = 0)
The interacting drugs have opposing actions
Example: Acetylcholine and noradrenaline have opposing effects on heart rate.
2. Addition or summation (Additive effect : 1 + 1 =2)
The interacting drugs have similar actions and the resultant effect is the some of individual drug responses
Example:CNS depressants like sedatives and hypnotics,…etc
3. Synergism or potentiation Synergistic effect : 1 +1 > 2 and Potentiation effect : 1 + 0 =2 :
It is an enhancement of action of one drug by another

Q2.

A.
Ans.1. Oral Hypoglycemics

orinase (chlorpropamide) Glucotrol (glipizide) Micronase (glyburide)

2. Hyperglycemic Agents

D50W is a sugar solution given intravenously for acute hypoglycemia.

Glucagon is indicated for emergency treatment when an IV is unobtainable.

B.

Ans.Drugs Used to Treat Emesis

Emesis, is the involuntary, forceful expulsion of the contents of an individual's stomach through the mouth.

Antiemetics:

Drugs

Serotonin Antagonists: Zofran.

Dopamine Antagonists: Compazine, Phenergan, Inapsine, Reglan.

Cannabinoids: Marinol, Cesamet.

C.
Ans
This combination medication is used to relieve coughs caused by the common cold, bronchitis, and other breathing illnesses. Guaifenesin belongs to a class of drugs known as expectorants. It works by thinning and loosening mucus in the airways, clearing congestion, and making breathing easier. Dextromethorphan belongs to a class of drugs known as cough suppressants. It acts on a part of the brain (cough center) to reduce the urge to cough.

Q3
A
Ans All such processes are targets for antibiotics; therefore, antibacterials, which interfere or disturb these processes in different ways, can be subdivided into four groups: such as cell wall synthesis inhibitors, inhibitors of membrane function, inhibitors of protein synthesis, and inhibitors of nucleic acid synthesis.

B
Ans Viral Replication

Adsorption.

Initially, the virus attaches or adsorbs to the surface of the host cell. Most viruses are attracted to the host cell because of the interaction between proteins on the outer surface of the virus and receptorlike proteins on the host cell membrane

Penetration and Uncoating.

The virus enters the host cell either by passing directly through the cell membrane or by fusing with the host-cell membrane and releasing the viral genetic material into the host cell

Biosynthesis.

When viral genetic material is released within the host cell, the virus takes control of the cell's molecular synthesizing machinery to initiate the biosynthesis of new viral enzymes and proteins

Maturation and Release.

The component parts of the virus (the genetic core and surrounding shell) are assembled into mature viruses and released from the host cell

Q4

A

Ans.There are many classes of antihypertensives, which lower blood pressure by different means. Among the most important and most widely used medications are thiazide diuretics, calcium channel blockers, ACE inhibitors, angiotensin II receptor antagonists (ARBs), and beta blockers.

E.g

Hydrochlorothiazide, furosemide

Propranolol, labetalol, prazosin

Clonidine

Hydralazine, minoxidil sodium

B

Ans. Angina pectoris is a syndrome characterized by sudden severe pressing substernal chest pain or heaviness radiating to the neck, jaw, back and arms.

It is often associated with diaphoresis, tachypnoea and nausea

Primary cause:

Imbalance between myocardial oxygen demand and oxygen supplied by coronary vessels

This imbalance may be due to:

a decrease in myocardial oxygen delivery

an increase in myocardial oxygen demand, or both

The discomfort abates when supply becomes adequate for demand. Typically angina lasts for seconds to minutes, up to 15 minutes.

Q5

QA.
ANS.Local anesthetics, which cause a reversible loss of sensation for a limited region of the body without necessarily affecting consciousness.

General anesthesia.
is a state characterized by unconsciousness, analgesia, amnesia, skeletal muscle relaxation, and loss of reflexes.

Stage 1: Analgesia
In stage 1, the patient has decreased awareness of pain, sometimes with amnesia. Consciousness may be impaired but is not lost

B. Stage 2: Disinhibition/ excitement
In stage 2, the patient appears to be delirious and excited. Amnesia occurs, reflexes are enhanced, and respiration is typically Irregular; retching and incontinence may occur.

Stage 3: Surgical Anesthesia
In stage 3, the patient is unconscious and has no pain reflexes; respiration is very regular, and blood pressure is maintained.

4: Medullary Depression
In stage 4, the patient develops severe respiratory and cardiovascular depression that requires mechanical and pharmacologic support.

Q
B .
Ans .
Non-opioid analgesics include nonsteroidal anti-inflammatory drugs (NSAIDs), selective COX-2 inhibitors, and acetaminophen. NSAIDs inhibit cyclooxygenases (COX-1 and COX-2), thereby disrupting the production of prostaglandin, an important mediator of pain and inflammation. Consequently, NSAIDs possess antipyretic, analgesic, and anti-inflammatory effects, and are particularly effective in the management of musculoskeletal pain (e.g., rheumatic disorders, inflammatory joint pain). Side effects include gastrointestinal ulcers and bleeding, increased risk of heart attacks, and renal function impairment. The severity of these side effects is often underestimated because most non-opioid analgesics are easily available OTC. Selective COX-2 inhibitors have similar effects to NSAIDs, but show a lower risk for gastrointestinal side effects. Acetaminophen possesses antipyretic and analgesic effects and is the most commonly used over-the-counter (OTC) oral analgesic drug. It is generally well tolerated, but overdose can result in significant hepatotoxicity with the risk of acute liver failure.

Common agents Activity profile Side effects
Nonsteroidal anti-inflammatory drugs (NSAID)
Ibuprofen
Diclofenac
Indomethacin
Naproxen
Ketorolac
Aspirin
Meloxicam
Analgesic
Antipyretic
Anti-inflammatory
Antiplatelet effect
Gastric and intestinal ulcers, bleeding, and perforation
Renal function impairment
Acute renal failure
Deterioration of chronic renal failure
Chronic analgesic nephropathy
Increased risk of heart attack and stroke (with the exception of aspirin and naproxen)

COX-2 inhibitors (selective NSAID)
Celebrex (celecoxib)
Analgesic
Anti-inflammatory
Increased cardiovascular risk
Renal side effects
Deterioration of chronic renal failure
Increase in blood pressure
Other non-opioid analgesics
Acetaminophen
Analgesic
Antipyretic
Hepatotoxicity
Acute liver failure in cases of intoxication
Limited nephrotoxicity
References:
Nonsteroidal anti-inflammatory drugs
Agents
Ibuprofen, diclofenac, indomethacin, naproxen, aspirin
Mechanism of action
Reversible inhibition of the enzymes cyclooxygenase 1 and 2 (COX-1 and COX-2) → decreased prostaglandin synthesis
Effects

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