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VARIETY OF POISONS' EFFECTS

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Cases of poisoning due to overdose of painkillers and sedatives (opioids), antidepressants, cardiovascular drugs, pesticides and bites (snake, spider, ticks, bee) and some plants are common. Substance like cyanide, bee sting venoms, cobra bite, cytotoxin, hemotoxin, botulism toxin, tetanus toxin, paraquat poisoning, lead poisoning and mercury poisoning are the deadliest toxins against which there are no antidotes. In 2018 the number of poisoning deaths was 180,000, up from 200,000 in 1990, and the number of emergency room visits the United States was about 727,250. According to statistics in 2021 more than one person per 100000 population in India died due to unintentional poisoning.

The purpose of this work is to show the variety of the poison' effects and the need to deepen knowledge on the topic.

There are four main ways by which poison enters our body: ingestion, inhalation, cutaneous and injection. The path from fastest to slowest that can affect is I/V > inhalation > intraperitoneal > I/M > ingestion > topical absorption. Absorption through gas: poison in the body has to overcome or avoid the body natural defenses and move to the area in which it will act. When poison is inhaled, it enters the body through alveoli to the bloodstream. Intestinal absorption: when poison ingested through intestines, it damages the microflora. Absorption through skin: it first passes through the pores, then into the bloodstream. The damaging effect of a poison depends on the concentration of active components at the site of exposure. The liver, kidney and lungs have high blood flow, so they are more susceptible. Poisons can have many mechanisms by which they can cause harm or death; damage can occur atthe molecular (it can interact with protein, lipid or DNA), cellular or organ level. On cellular leverit interferes with the binding of receptors ligand, membrane function, cellular energy production, binds to biomolecules, and disrupt homeostasis.

Neurotoxin have a number of mechanisms by which they inhibit normal neuron cellular processes; these include membrane depolarisation and interneuronal communication pathways. It causes death by binding to nerve cells while performing normal activities, when cells never send signals that cause cell apoptosis, this can lead to complete shutdown of the nervous system and loss nerve signal to heart, leading to death. *Cytotoxin* contains digestive enzymes and molecules known as "spreading factor" that cause both local and systemic injury. Locally, the venom cause pain, swelling, bleeding and eventually necrosis. Systemically, anti-clotting proteins in the venom can cause massive bleeding and organ damage. *Hemotoxin***.** The poison itself is called as metalloproteinase. Proteinases are capable ofbreaking down protein and specifically act on the basement membranes of red blood cell and cause to lysis, and then lead to hemorrhages.

Reduction the level of free toxin can be achieved by using specific and non-specific agents that bind to the toxin. The most commonly used non- specific binding agent is activated charcoal. Specific binders may include chelating agents, biosquelching therapy, and immunotherapy. In some cases, enhanced excretion can be achieved through urine alkalization or hemadsorption. Other mechanisms of antidotes action include competitive inhibition of enzymes, enhancement of enzyme function and competitive receptor blockade. Drugs such as N-acetylcysteine and sodium thiocyanate reduce the formation of toxic metabolites in paracetamol and cyanide poisoning respectively. Drugs such as atropine and magnesium are used to counteract the end-organ effects in organophosphorus poisoning. Vitamins such as vitamin K, folic acid and pyridoxine are used to antagonize the effects of warfarin, methotrexate in the setting of toxicity or overdose.

Public health measures should include enforcement of regulations, establishing safety standards to increase public awareness, and increasing the knowledge level of medical personnel about the properties of poisons.