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The term "four stages of amphetamine" can have different interpretations depending on the context. Here are two possible interpretations:

1. Stages of action in the brain:

Release: Amphetamine enters the brain and blocks the reuptake of dopamine and norepinephrine, leading to increased levels of these neurotransmitters. This results in the initial euphoric and stimulating effects.
Reuptake inhibition: The amphetamine continues to block reuptake, prolonging the effects.
Metabolism: The body starts to break down and eliminate the amphetamine.
Depletion: Dopamine and norepinephrine levels fall back to normal or even lower, leading to a "crash" or comedown phase with symptoms like fatigue, depression, and irritability.
2. Stages of dependence and withdrawal:

Acute intoxication: This is the initial phase when the user experiences the desired effects like increased energy, focus, and euphoria.
Tolerance: Over time, the body adapts to the presence of amphetamine, requiring higher doses to achieve the same effects.
Dependence: The user relies on the drug to function normally and experiences withdrawal symptoms (anxiety, depression, fatigue) when trying to stop.
Protracted withdrawal: Even after acute symptoms subside, some individuals experience long-lasting effects like cognitive impairment and emotional dysregulation.

advantages of Nitrous Oxide:

Pain relief: Nitrous oxide, often called laughing gas, has mild analgesic properties, helping manage anxiety and pain during medical procedures, especially dentistry.
Sedation: It can induce a state of relaxation and euphoria, reducing anxiety and fear, making procedures more tolerable.
Fast onset and recovery: Effects are felt quickly when inhaled and wear off rapidly after discontinuing use, minimizing after-effects.
Fewer side effects: Compared to deeper sedation methods, nitrous oxide has fewer side effects like drowsiness or hangover.
Reduce of dose and side effects of another aesthetic when compound whit it

Disadvantages of Nitrous Oxide:

Limited effectiveness: It's not a strong pain reliever and may not be sufficient for all procedures or patients.
Adverse effects: Potential side effects include nausea, vomiting ,dizziness, tingling, and headache, though usually mild and transient.
Interactions with medications: Can interact with certain medications, so thorough communication with your healthcare provider is crucial.
Not suitable for everyone: People with certain medical conditions or pregnant women should not use nitrous oxide.

Adjuncts in anesthesia refer to medications or techniques used alongside the main anesthetic agent to enhance its effects, reduce side effects, and improve overall patient experience. They offer numerous benefits, including:

1. Improved Pain Management:

Opioids: Fentanyl, morphine, and sufentanil potentiate analgesia while reducing the need for the main anesthetic agent, lowering its associated side effects.
Non-steroidal anti-inflammatory drugs (NSAIDs): Ketorolac and tenoxicam provide additional pain relief and reduce inflammation postoperatively.
2. Enhanced Anesthesia and Sedation:

Clonidine: This alpha-2 agonist promotes relaxation, reduces anxiety, and improves hemodynamic stability.
Dexmedetomidine: This sedative agent provides anxiolysis, promotes premedication, and enhances the effect of regional anesthesia.
Midazolam: This short-acting benzodiazepine induces drowsiness and reduces anxiety.
3. Prolonged Block Duration:

Epinephrine: This vasoconstrictor slows the local anesthetic's absorption, extending its duration of action.
Dexamethasone: This steroid reduces inflammation and edema, prolonging the effect of nerve blocks.
4. Reduced Side Effects:

Anticholinergics: Glycopyrrolate and atropine prevent excessive salivation and bradycardia, particularly during neuraxial anesthesia.
Ondansetron: This antiemetic medication prevents nausea and vomiting after surgery.
5. Additional Benefits:

Ketamine: This medication exhibits analgesic and anti-inflammatory properties, potentially reducing chronic pain development after surgery.
Magnesium: This electrolyte promotes cardiovascular stability and reduces the risk of postoperative tremors and seizures.
Choosing the right adjuncts depends on several factors:

Type of surgery and expected pain level
Patient's medical history and comorbidities
Desired level of sedation and relaxation
Individual risk tolerance for potential side effects

definition and mechanism of action

Sugammadex, a cyclodextrin, is the first selective relaxant-binding agent; it exerts its reversal effect by forming tight complexes in a 1:1 ratio with steroidal nondepolarizing agents (vecuronium, rocuronium,). This drug has been in use in the European Union for the past few years, but is not yet commercially available in the United States.

The newer neuromuscular blocking agents, such as gantacurium, which are still under investigation, show promise as ultrashort-acting nondepolarizing agents; they undergo chemical degradation by rapid adduction with L-cysteine.

Clinical Considerations

dose ⟹

Sugammadex has been administered in doses of 4–8 mg/kg. With an injection of 8 mg/kg, given 3 min after administration of 0.6 mg/kg of rocuronium, recovery of TOF ratio to 0.9 was observed within 2 min. It produces rapid and effective reversal of both shallow and profound rocuronium-induced neuromuscular blockade in a consistent manner. Because of some concerns about hypersensitivity and allergic reactions, sugammadex has not yet been approved by the US Food and Drug Administration.

⊕ effect ⟹

⊖

* هذا الدواء أجا لآخر
محروس منحج تقرفه

Ephedrine

- Mixed Action sympathomimetic (Direct + in direct) But mainly indirectly
- Activate $\alpha_1, \beta_1, \beta_2$

Action :- [1] local :- [I] vasoconstriction
[II] Mydrasis
[III] Nasal Decongestant

[2] systemic :- [I] CNS → stimulate cerebral cortex → insomnia, anxiety, tremor
Also cause sedation in ADHD
[II] Bronchodilation
[III] All probably of Adrenaline . . . . . نفسة نفس وظائف الادرينالين
fight and flight

Uses :- [1] Analeptic in CNS Depressant toxicity
[2] ADHD
[3] Nasal Decongestant
[4] Reversal of hypotension from spinal Anthesia (IV. ephedrine)

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grade of amphetamin الواجب
الجواب
In clinical practice, there isn't a standardized grading system for amphetamine intoxication with four distinct grades as you might find in some other medical conditions. However, amphetamine intoxication can indeed manifest across a spectrum of severity, ranging from mild to severe, and clinicians typically assess the level of intoxication based on observed symptoms and their impact on the individual's functioning and health.

Here's a general overview of the progression of amphetamine intoxication:

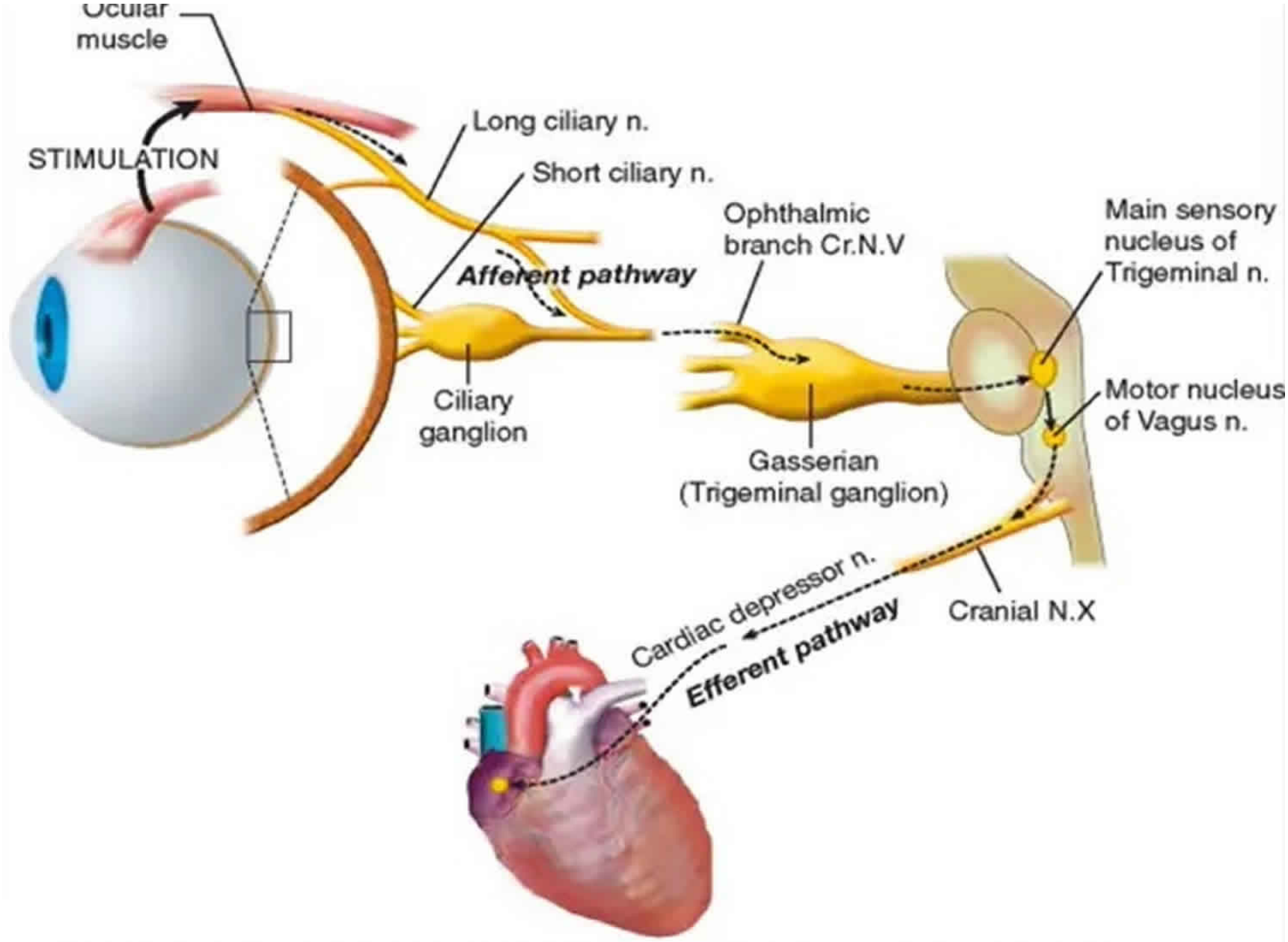
Mild Intoxication: In mild cases, individuals may exhibit increased energy, euphoria, talkativeness, heightened alertness, dilated pupils, and decreased appetite. They may also experience restlessness, insomnia, and increased heart rate. Functioning may be relatively intact, and the individual may not require immediate medical intervention.

Moderate Intoxication: Moderate intoxication may involve more pronounced symptoms such as agitation, irritability, paranoia, confusion, and hallucinations. Physical signs such as tremors, sweating, palpitations, and increased blood pressure may also be present. Functioning may be impaired, and medical evaluation may be necessary to manage symptoms and ensure safety.

Severe Intoxication: Severe cases of amphetamine intoxication can lead to life-threatening complications. Symptoms may include extreme agitation, psychosis, delirium, hyperthermia (elevated body temperature), seizures, and cardiovascular instability. Individuals may be at risk of harming themselves or others, and immediate medical attention in an emergency setting is essential.

Overdose: Amphetamine overdose occurs when an individual consumes a toxic amount of the drug, leading to severe and potentially fatal complications such as cardiovascular collapse, respiratory depression, stroke, and coma. Overdose requires prompt medical intervention, including supportive measures to stabilize vital signs and administration of antidotes or medications as appropriate.

While these descriptions provide a general framework, it's important to recognize that the severity of amphetamine intoxication can vary widely among individuals and may not neatly fit into distinct categories. Prompt medical assessment and intervention are crucial in managing amphetamine intoxication to prevent complications and ensure the individual's safety and well-being. Additionally, substance abuse treatment and support may be necessary to address underlying issues related to amphetamine use.


Ocular muscle
STIMULATION
Long ciliary n.
Short ciliary n.
Afferent pathway
Ophthalmic branch Cr.N.V
Main sensory nucleus of Trigeminal n.
Ciliary ganglion
Motor nucleus of Vagus n.
Gasserian (Trigeminal ganglion)
Cardiac depressor n.
Efferent pathway
Cranial N.X

Afferent Limb

Trigeminal Nerve (ciliary ganglion to ophthalmic division of trigeminal nerve to gasserian ganglion to the main trigeminal sensory nucleus). Also afferent tracts from maxillary and mandibular divisions of trigeminal nerve have been documented.

Efferent Limb

Vagus Nerve (afferents synapse with visceral motor nucleus of vagus nerve located in the reticular formation and efferents travel to the heart and decrease output from the sinoatrial node).

Triggering Stimuli

Triggered by traction on the extraocular muscles (especially medial rectus), direct pressure on the globe, ocular manipulation, ocular pain.

Can also be triggered by retrobulbar block (pressure associated with local infiltration), ocular trauma, or manipulation of tissue in orbital apex after enulcleation.

Cocaine

is a tropane alkaloid that acts as a central nervous system (CNS) stimulant. As an extract, it is mainly used recreationally, and often illegally for its euphoric and rewarding effects. It is also used in medicine by Indigenous South Americans for various purposes and rarely, but more formally, as a local anaesthetic or diagnostic tool by medical .practitioners in more developed countries

Cocaine crosses the blood–brain barrier via a proton-coupled organic cation antiporter and (to a lesser extent) via passive diffusion across cell membranes. Cocaine blocks the dopamine transporter,inhibiting reuptake of dopamine from the synaptic cleft into the pre-synaptic axon terminal; the higher dopamine levels in the synaptic cleft increase dopamine receptor activation in the post-synaptic neuron, causing euphoria and arousal. Cocaine also blocks the serotonin transporter and norepinephrine transporter, inhibiting reuptake of serotonin and norepinephrine from the synaptic cleft into the pre-synaptic axon terminal and increasing activation of serotonin receptors and norepinephrine receptors in the post-synaptic neuron, contributing to the mental and physical effects of cocaine .exposure

Cocaine stimulates the reward pathway in the brain. Mental effects may include an intense feeling of happiness, sexual arousal, loss of contact with reality, or agitation.Physical effects may include a fast heart rate, sweating, and dilated

pupils. High doses can result in high blood pressure or high body temperature. Onset of effects can begin within seconds to minutes of use, depending on method of delivery, and can last between five and ninety minutes. As cocaine also has numbing and blood vessel constriction properties, it is occasionally used during surgery on the throat or inside of the .nose to control pain, bleeding, and vocal cord spasm

Cocaine increases the minimal alveolar concentration (MAC) of anesthetic gases because it enhances the sensitivity of the central nervous system to those gases. This means that higher concentrations of the anesthetic gas are required to achieve the same level of anesthesia when cocaine is present in the system.

Top of Form

2