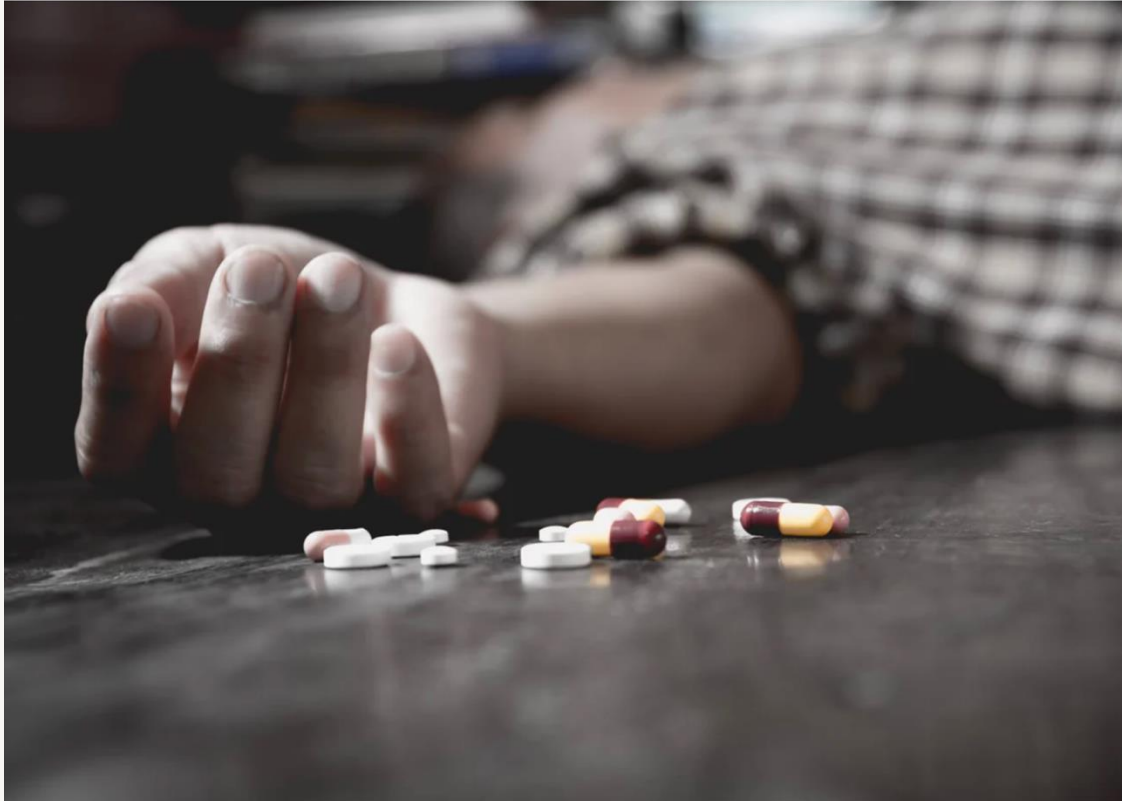


TOXIDROMES: RECOGNIZING PATTERNS IN POISONING



CHOLINERGIC

ANTICHOLINERGIC

SYMPATHOMIMETIC

SEDATIVE-HYPNOTIC

OPIOID

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SEP 29, 2026

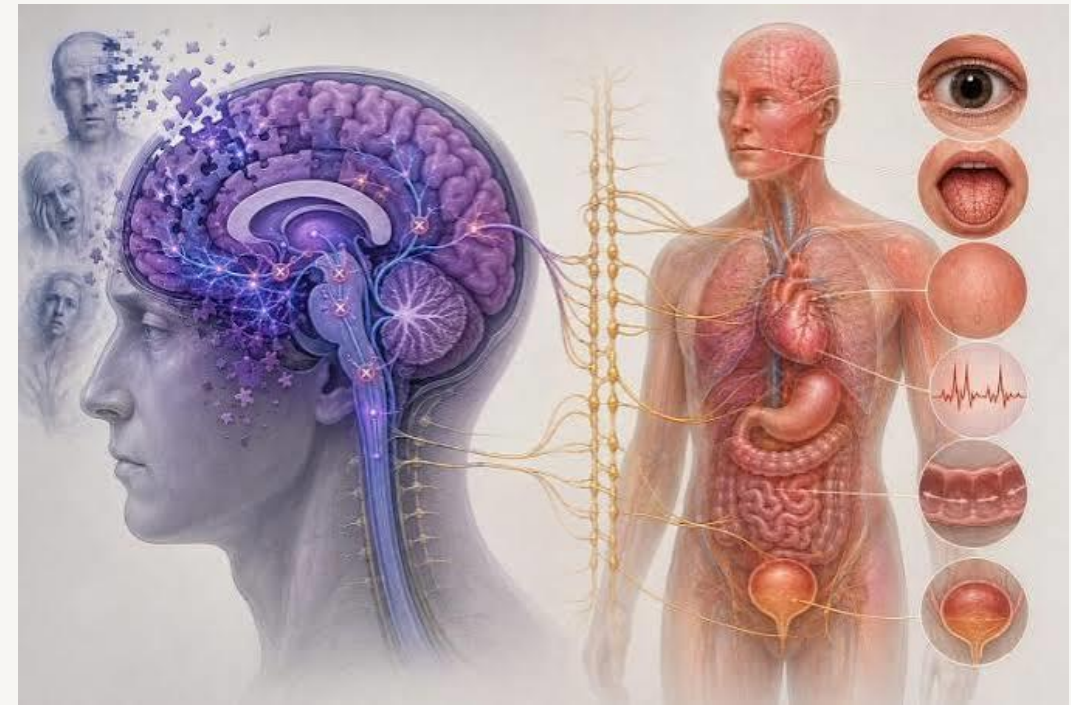
What is a toxidrome?

Definition:

- A group of clinical signs and symptoms produced by a drug or toxin class.
- It helps identify the likely exposure when the history is missing.

Best clues:

- Mental status
- Respiratory rate, pulse, blood pressure, temperature
- Pupils
- Skin moisture
- Bowel sounds
- Neuromuscular findings.



Clinical limit:

- Mixed ingestions, delayed effects and underlying disease can blur the pattern.
- Treat the immediate threat while testing alternate diagnoses.

First assessment: stabilize before naming the toxin

Airway and breathing



Circulation



Exposure and examination



History and testing: use the syndrome to choose tests

History sources

Ask patient, friends, family or EMS about substance, dose, route, timing, prescriptions, intent and possible co-ingestions.

Initial tests

Glucose and ECG commonly guide urgent care. Add electrolytes, renal function, blood gas, acetaminophen level or pregnancy testing when relevant.

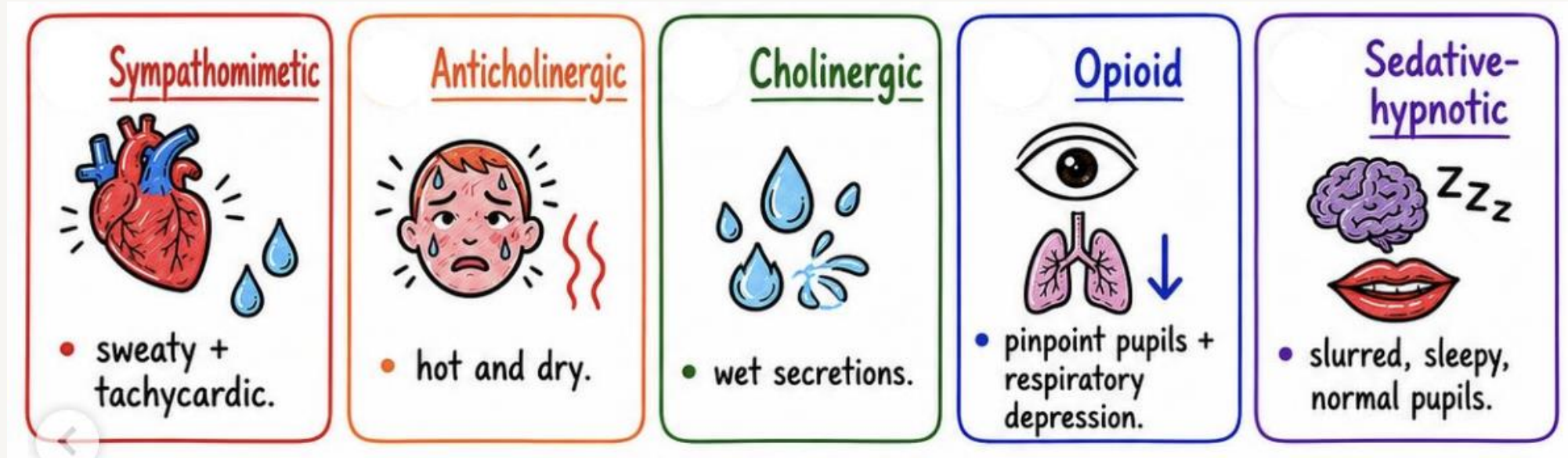


Interpretation

A routine urine drug screen may miss agents and cannot quantify impairment. A negative result does not override the bedside exam.



Five major toxidromes



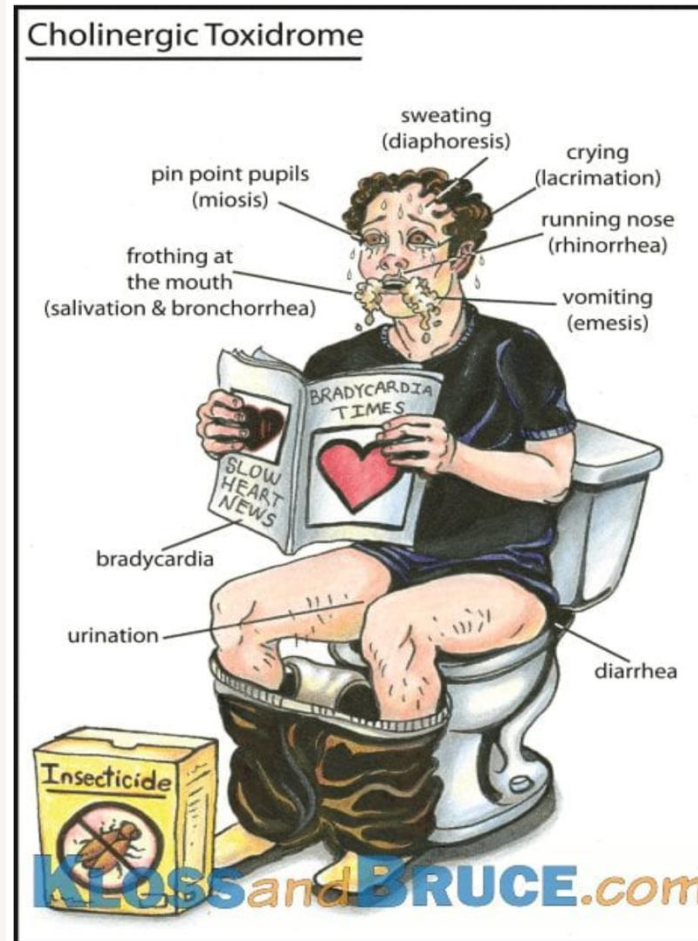
Cholinergic toxidrome: too much acetylcholine

Mechanism and causes

Organophosphates inhibit acetylcholinesterase → increased Acetylcholine levels

Clinical signs: DUMBELLSS

Diarrhea
Urination
Miosis
Bradycardia
Bronchospasm
Emesis
Lacrimation
Lethargy
Salivation
Seizures



Treat the threat

Protect staff and decontaminate.

Suction and support airway.

Atropine treats bronchial secretions.

Pralidoxime is used for organophosphate poisoning.

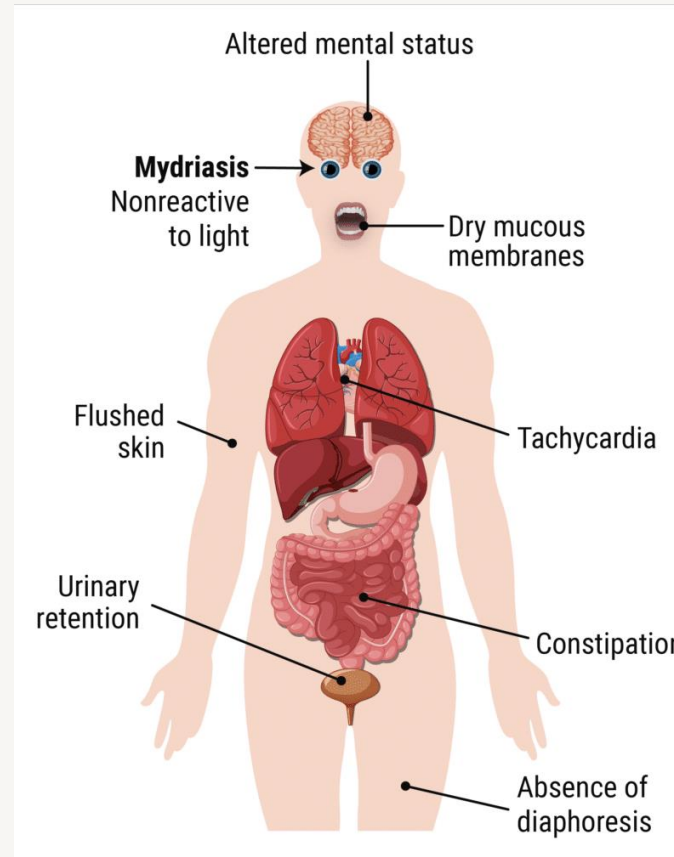
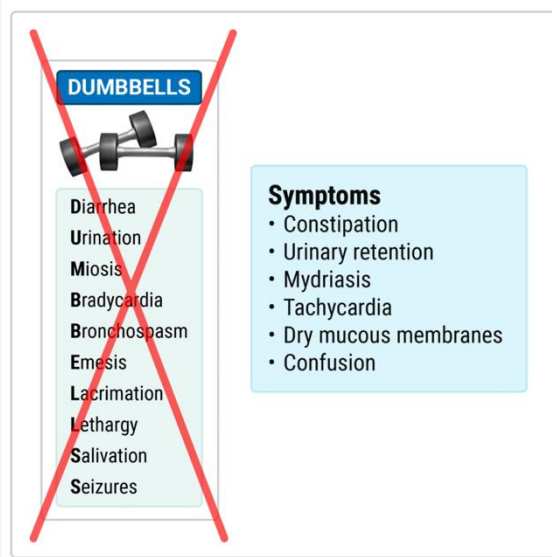
Complication

Respiratory failure can result from secretions, bronchospasm and muscle weakness.

Anticholinergic toxidrome: dry and delirious

Causes

Classes of medications with anticholinergic properties include antihistamines, TCAs, and sleep aids



Treatment

Supportive care and cooling.

Benzodiazepines for severe agitation or seizures.

ECG for QRS widening;

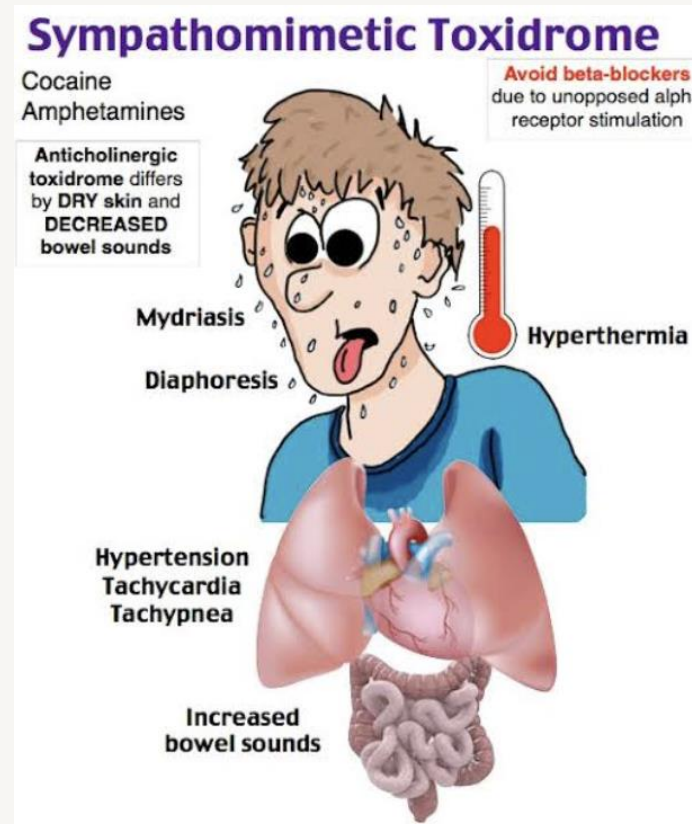
Physostigmine only in selected pure cases with toxicology guidance.

Sodium bicarbonate may be needed for sodium-channel blockade in a mixed or TCA ingestion.

Sympathomimetic toxidrome: sweaty and activated

Causes

Cocaine, amphetamines and related stimulants increase catecholamine effects.



Treatment

Benzodiazepines for agitation.

Active cooling for
hyperthermia

IV fluids

Evaluation for cardiac
ischemia or rhabdomyolysis.

Sweating helps distinguish this pattern from the dry anticholinergic syndrome.

Sedative–hypnotic toxidrome: depressed CNS

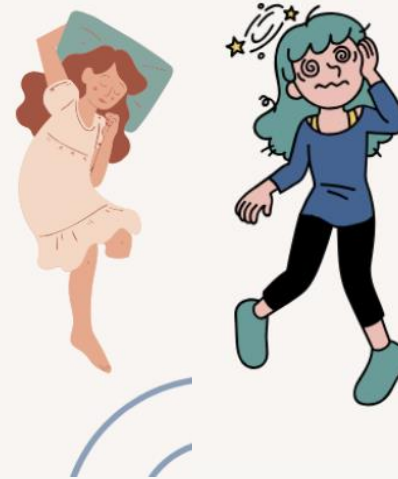


Causes and signs

- Affects GABA system
- Including: ethanol, BZDs, barbiturate, etc.

MNEMONIC

- SAM'S GIN
 - S – Slurred speech
 - A – Attention impairment
 - M – Memory impairment
 - S – Stupor or coma
 - G – Gait unsteady
 - I – Incoordination
 - N – Nystagmus



EXAM FINDINGS

- Bradypnea
- Hypoxia
- Hypotension
- Normo or bradycardia
- Somnolence
- Altered mental status
- Hypothermia

Initial treatment

Protect airway, support breathing

Monitor vitals and check for opioid or other co-ingestions.

Ethanol has no reversal antidote; give glucose when hypoglycemic and thiamine when indicated.

Flumazenil is not routine for unknown overdose. It may precipitate seizures in dependent patients or mixed ingestions, especially with tricyclic antidepressants.

Opioid toxidrome: breathing is the priority

Causes and signs

Fentanyl, heroin, morphine, oxycodone or methadone

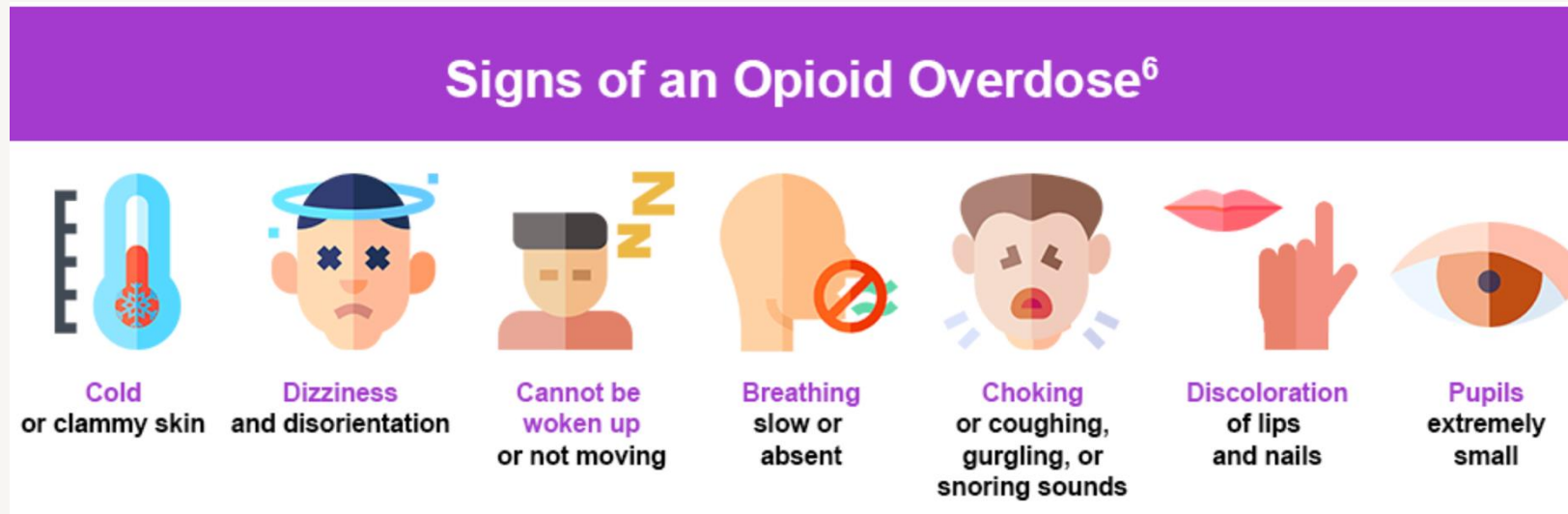
Treatment

Open the airway and assist ventilation.

Give naloxone by available route; repeat if breathing remains inadequate or worsens again.

Monitor

Watch for aspiration and recurrent respiratory depression after naloxone, particularly with long-acting agents or mixed ingestion.



Common mimics and overlap

Depressed consciousness

Hypoglycemia

Seizure

Head injury, stroke

Infection

Ethanol or benzodiazepines
can resemble opioid or
sedative toxicity.

Agitation and hyperthermia

Sepsis

Anticholinergic drugs,
stimulants, serotonin toxicity
and withdrawal can resemble
one another.

Small pupils

Opioids

Cholinergic agents

*Airway secretions and
fasciculations favor
cholinergic poisoning.

Recheck the pattern after treatment; co-ingestions may reveal themselves over time.

Risk and disposition: tailor monitoring

High risk

Respiratory failure

Persistent coma

Seizures

Severe hyperthermia

Shock

Severe acidosis.

Treatment dependence

Repeated naloxone

Antidote infusion

Ongoing sedation

Airway intervention warrants
monitored care.

Before discharge

Confirm stable breathing and
mentation

Evaluate injury and intent

Arrange safety planning or
follow-up when appropriate.

Practice: identify the pattern and first action

A Confused, dry skin, large pupils, urinary retention.

Anticholinergic. Stabilize, check ECG, cool and treat agitation.

B Agitated, sweating, 40°C, tachycardic.

Sympathomimetic is likely. Sedate and cool promptly.

C Wheezing, profuse secretions, diarrhea, pinpoint pupils.

Cholinergic. Protect staff, clear the airway, give atropine; consider pralidoxime.

D Unresponsive with 6 breaths/min and small pupils.

Opioid. Ventilate and administer naloxone.

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Article concepts are paraphrased. Medication decisions follow patient presentation and local toxicology guidance.

Comparison: the five major toxidromes

Pattern	Bedside findings	Primary treatment
Cholinergic	Wet; miosis, secretions, diarrhea, fasciculations	Atropine; pralidoxime for organophosphates
Anticholinergic	Dry, hot; mydriasis, delirium, urinary retention	Supportive care; select physostigmine cases
Sympathomimetic	Sweaty, hot; mydriasis, agitation, tachycardia	Benzodiazepines and cooling
Sedative / hypnotic	Sedation, ataxia; often normal pupils	Airway support; rare flumazenil use
Opioid	Slow breathing, sedation, often miosis	Ventilation and naloxone