(A) The neurotransmitter acetylcholine (ACh) is released into the synapse followed by binding to acetylcholine receptors which results in muscle contraction. Immediately after ACh binding, the enzyme acetylcholinesterase (AChE) breaks down ACh, allowing it to be taken up by the presynaptic neuron.

Nerve agents inhibit AChE, which results in an excess of acetylcholine and over-activation of the neuromuscular junction. SLUDGE syndrome followed by paralysis and death results.

Non-aged soman (GD) conjugate of Torpedo californica acetylcholinesterase (Protein Data Bank structure 2WFD)

- Inhalation toxicity
- Dermal toxicity
- Neurological complications

Nerve agent countermeasures
Atropine and Pralidoxime Chloride auto injector

Atropine, blocks the action of acetylcholine at muscarinic receptors and treats SLUDGE syndrome (salivation, lacrimation, urination, diaphoresis, gastrointestinal motility, emesis).

Oximes, Reactivate acetyl cholinesterase before the process of aging (e.g., irreversible inhibition of the enzyme). Oximes can be co-administered with atropine. Commonly used oximes include pralidoxime chloride, HI-6, trimedoxime and obidoxime.

(B) Serine in protein + Sarin → Inhibited Serine + Aged Form Isopropanol

OH

O

N

O

F

P

O

O

P

O

O

N

O

OH