of a chemical agent is essential. Scrubbing of exposed skin with a stiff brush or bristles is discouraged, because skin damage may occur and may increase absorption of agent. "Gentle" liquid dish soap and copious amounts of water should be used, with mild to moderate friction applied with a single-use sponge or washcloth in the first and second washes. The third wash should be a rinse with copious amounts of warm or tepid water. Shampoo can be used to wash the hair. If only cold water is available, it should be used; decontamination should not be delayed while warm water is sought. Spot (local) decontamination with reactive skin decontamination lotion (RSDL), followed by a soap and water wash/shower, is the method preferred by the Department of Defense. RSDL is available for purchase by civilians and has been shown to be superior across a broad spectrum of nerve agents as well as sulfur mustard. RSDL is the only product approved by the U.S. Food and Drug Administration (FDA) for initial spot decontamination. An important caveat is that RSDL and 0.5% sodium hypochlorite (dilute bleach military field expedient) should *not* be used concurrently because of a potential exothermic reaction. In any event, decontamination must be accomplished before the patient enters the medical facility to avoid contaminating the facility and its staff. In patients with contaminated wounds, potentially contaminated clothing and other foreign material that may serve as a depot for the liquid agent should be extracted.

RESPIRATORY SUPPORT

Death from nerve agent poisoning is almost always attributable to respiratory causes. Ventilation will be complicated by increased resistance and secretions. Atropine should be given before ventilation or as it begins, since it will make ventilation far easier.

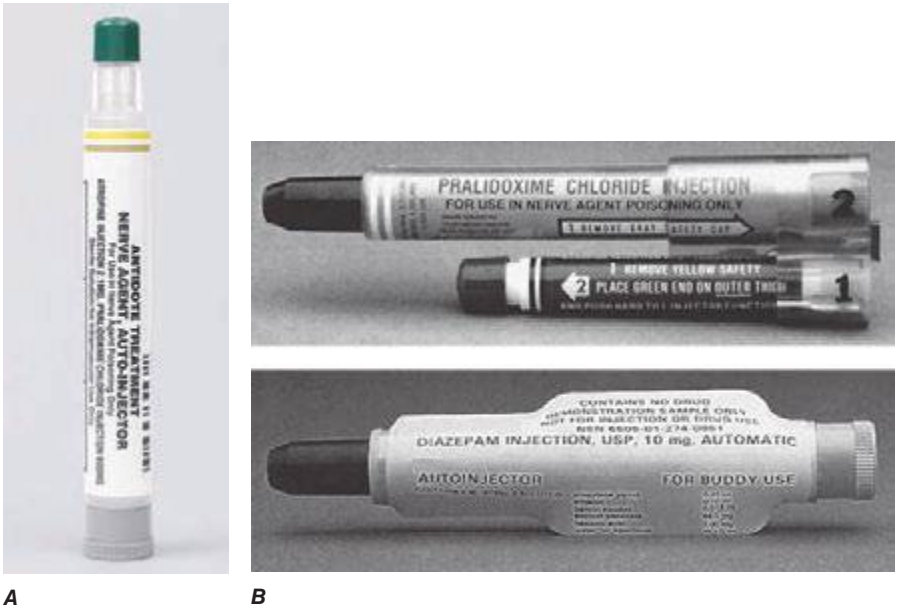
ANTIDOTAL THERAPY

Atropine In theory, any anticholinergic agent could be used to treat nerve agent poisoning. Worldwide, however, the choice is invariably atropine because of its wide temperature stability and rapid effectiveness when administered either IM or IV and because inadvertent administration of this drug usually causes little CNS dysfunction (Table 262e-3). Atropine rapidly reverses cholinergic overload at muscarinic synapses but has little effect at nicotinic synapses. The practical implication is that atropine can quickly treat the life-threatening respiratory effects of nerve agents but probably will not help with neuromuscular (and possibly sympathetic) effects. In the field, military personnel are given a combined autoinjector containing both 2.1 mg of atropine and oxime (2-pralidoxime chloride [2-PAM Cl])—a product licensed by the FDA under the trade name Duodote®. Its miltary designation is the Antidote Treatment Nerve Agent Autoinjector (ATNAA) **(Fig. 262e-5)**. Only full—and not divided—autoinjector doses can be administered. The field loading dose is 2, 4, or 6 mg, with re-treatment every 5–10 min until the patient's breathing improves and secretions diminish. The Iranian military initially used larger doses during the Iran–Iraq war, in which oximes were in short supply. When the patient reaches a level of medical care at which drugs can be given IV, this is the preferred route. In small children, the IV route may be the initial avenue for atropine therapy; however, pediatric autoinjectors of 0.5 mg and 1 mg are manufactured. There is no upper limit to atropine therapy (whether IM or IV), but the total average dose for a severely afflicted adult is usually 20–30 mg.

In a mildly afflicted patient with miosis and no other systemic symptoms, atropine or homatropine eyedrops may suffice for therapy. This treatment will result in ~24 h of mydriasis. Frank miosis or imperfect accommodation may persist for weeks or even months after all other signs and symptoms have resolved.

Oximes Oximes are nucleophiles that reactivate the cholinesterase whose active site has been occupied and bound to nerve agent (Table 262e-3). Therapy with oximes therefore restores normal enzyme function. Oxime therapy is limited by a second side reaction, called "aging," in which a side chain on nerve agents falls off the complex at a characteristic rate. "Aged" complexes are negatively charged, and oximes cannot reactivate negatively charged complexes. The practical effect of this limitation differs from one nerve agent to another since each ages at a characteristic rate. For example, sarin ages in 3–5 h, tabun ages over a longer period (12–13 h), and VX ages much less rapidly (>48 h). All these intervals are so much longer than the patient's expected life span and expected treatment time after acute nerve-agent toxicity that they are irrelevant. Soman, in contrast, ages in 2 min; thus, only a few minutes after exposure, oximes are useless in treating soman poisoning. The oxime used varies by country; the United States has approved and fielded 2-PAM Cl. MARK 1 kits and Duodotes® (Fig. 262e-5*A*) both contain autoinjectors holding 600 mg of 2-PAM Cl. Initial field loading doses are 600, 1200, and 1800 mg. Since blood pressure may become elevated after administration of 45 mg/kg in adults, field use of 2-PAM Cl is restricted to 1800 mg/h IM. During the time when more oxime cannot be given, atropine alone is recommended. In the hospital setting, 2.5–25 mg/kg of 2-PAM Cl by the IV route has been found to reactivate 50% of inhibited cholinesterase. The usual recommendation is 1000 mg by slow IV drip over 20–30 min, with ≤2500 mg over a period of 1–1.5 h. Active research aims to field a more effective and broader-spectrum oxime than 2-PAM Cl.

Anticonvulsants Nerve agent–induced seizures do not respond to the usual anticonvulsants used for status epilepticus, including phenytoin, phenobarbital, carbamazepine, valproic acid, and lamotrigine **(Chap. 445)**. The only anticonvulsants that have been shown to stop this form of status are the benzodiazepines. Diazepam is the only benzodiazepine approved for seizures in humans, although other FDA-approved benzodiazepines (notably midazolam) work well against nerve agent–induced seizures in animal models. Diazepam



A *B*

FIGURE 262e-5 **Antidotes to nerve agents.** ***A.*** The Antidote Treatment Nerve Agent Autoinjector (ATNAA) replaces the MARK I Kit. It is easier to self-administer and allows prompt distribution of the antidotes atropine and 2-pralidoxime chloride (2-PAM Cl). ***B.*** Diazepam 10-mg autoinjectors are carried by all U.S. military forces in a potential chemical battlefield and are being stockpiled by civilian first responders.