3-1. General

a. An incapacitating agent is a chemical agent which produces temporary disabling conditions. The disabling conditions persist for hours to days after exposure to the agent (unlike that produced by riot control agents, which usually are momentary or fleeting in action). Medical treatment, while not essential, may facilitate more rapid recovery. In the narrower sense, the term “incapacitating agents” has come to mean those agents that are—

(1) Highly potent (an extremely low dose is effective) and logistically feasible.
(2) Able to produce their effects mainly by altering the higher regulatory activity of the CNS.
(3) Temporary in duration of action lasting hours or days, rather than of a momentary or fleeting action.
(4) Not likely to produce permanent injury in concentrations which are militarily effective.

b. Incapacitating agents are not considered to include the following:

(1) Lethal agents, such as nerve agents which are incapacitating at sublethal doses.
(2) Substances which cause permanent or long-lasting injury, such as blister agents, choking agents, and those injuring the eyes.
(3) Common pharmaceutical substances with strong CNS actions, such as the belladonna alkaloids, tranquilizers, and many hallucinogens. These drugs, although effective and relatively safe, are logistically infeasible for large-scale use because of the large amounts required.
(4) Agents which are transiently effective by producing reflex responses interfering with duty performance. These include vomiting and irritant agents.
(5) Agents which disrupt basic life-sustaining systems and prevent physical activity. Examples include agents which lower the blood pressure, paralyzing agents (such as curare), respiratory depressants, and agents that interfere with oxygen transport. Although theoretically effective, such agents almost invariably have a low margin of safety between the effective dose and possible lethal dose. Therefore, these agents defeat the basic purpose of an incapacitating agent: to reduce military effectiveness without endangering life.

c. Despite restrictions imposed by the above definition, a great variety of mechanisms remain by which CNS regulation and maintenance of performance could theoretically be disrupted. In reality, however, only two general types of chemical agents are likely to be encountered in military use.

(1) Central nervous system depressants.

(a) These compounds produce their effects by interfering with information transmission across central synapses. An example of this type of agent is BZ [table 1-1] which blocks the muscarinic action of acetylcholine, both peripherally and centrally. The CNS anticholinergic compounds disrupt the high integrative functions of memory, problem solving, attention, and comprehension. A relatively high dose produces toxic delirium, destroying the individual’s ability to perform any military task.

(b) Cannabinols and phenothiazine-type compounds are potential incapacitating agents which seem to act as CNS depressants. The primary effects of these agents are to sedate and destroy motivation rather than disrupt the ability to think.

(2) Central nervous system stimulants. These agents cause excessive nervous activity by facilitating transmission of impulses. The effect is to flood the cortex and other higher regulatory centers with too much information. This flooding makes concentration difficult and causes indecisiveness and an inability to act in a sustained, purposeful manner. A well-known drug which appears to act in this manner is d-lysergic acid diethylamide (LSD); similar effects are sometimes produced by large doses of amphetamines.

3-2. Diagnosis

Current, field laboratory methods do not permit isolation and identification of specific agents in the environment or in samples of body fluid (for example, blood, urine, cerebrospinal fluid). Therefore, diagnosis rests almost entirely upon chemical acumen, combined with whatever field intelligence or detector system data that may be available. Following a suspected incapacitating agent attack, the medical officer should take the steps listed below.

a. Instruct evacuation teams to transport casualties to an uncontaminated area. After initial treatment, resistant or disoriented individuals should be restrained in the triage area.

b. Once the diagnosis of a nerve agent or other lethal substance has been ruled out, the principal signs and symptoms to consider are those shown in [table 3-1].
3-3. Protection, Decontamination, and First Aid

a. Protection. It is likely that such agents will be dispersed by smoke-producing munitions or aerosols and use the respiratory tract as the portal of entry. The use of the protective mask is essential to prevent inhaling the agent. With some agents, the percutaneous route may be used, thus MOPP 4 will be required.

b. Decontamination. Complete cleansing of the skin with soap and water should be accomplished at the earliest opportunity. The M291 Skin Decontaminating Kit can be used (APP D) if washing is impossible. Symptoms may appear as late as 36 hours after percutaneous exposure, even if the skin is washed within an hour. In fact, a delay in onset of several hours is typical. This time should be used to prepare for the possibility of an epidemic outbreak 6 to 24 hours after the attack.

c. First Aid. The most important considerations are the following:

   (1) If the casualty is stuporous or comatose, be sure that respiration is unobstructed; then turn the casualty onto one side to avoid aspiration in case vomiting should occur.

   (2) If the body temperature is elevated above 102°F (39°C) and mucous membranes are dry, immediate and vigorous cooling (as for heatstroke) is indicated. (Methods that can be used to cool the skin are spraying with 72 to 75°F (22 to 24°C) water and air circulation (fanning); applying alcohol-soaked cloths and air circulation; and providing maximum exposure to air in a shaded area, along with maximum air circulation. DO NOT USE ICE FOR SKIN COOLING.) Such cases are usually the result of anticholinergic intoxication. Rapid evacuation should be accomplished since treatment with appropriate medication may be lifesaving.
(3) Reassurance and a firm, but friendly, attitude by personnel administering first aid will be beneficial if the casualty appears to comprehend what is being said. Conversation is a waste of time if the individual is incoherent or cannot understand what is being said. In such cases, the less said, the better it is—these patients will benefit more from prompt and vigorous restraint and evacuation to an MTF.

(4) Although anticholinergic poisoning may produce alarming dryness and coating of the lips and tongue, there is usually no danger of immediate dehydration. In such cases, fluids should be given sparingly—if at all—because of the danger of vomiting and the likelihood of temporary urinary retention. (Temporary urinary retention is due to paralysis of the bladder smooth muscle.) Cleansing the mouth with an astringent swab may be comforting and will reduce the foul breath associated with membrane parching.

(5) Weapons and other potentially harmful items should be removed from the possession of individuals who are suspected of being casualties. This includes cigarettes, matches, medications, and small items which might be accidentally ingested. Delirious casualties have been known to attempt to eat items bearing only a superficial resemblance to food.

3-4. Treatment

General treatment consists of close observation, restraint and confinement (as required), supportive care with fluids, and appropriate clothing. Underlying medical problems should be identified and treated as they would be ordinarily.

a. Anticholinergics. Certain cholinesterase inhibitors (such as physostigmine) are highly active antagonists of the centrally active anticholinergics. Neostigmine and pyridostigmine are ineffective because they lack the tertiary nitrogen required to enable them to pass the blood-brain barrier. Treatment with 2 to 3 mg of physostigmine salicylate IM will be required to alleviate the condition. Repeated injections at intervals of approximately 15 minutes to 1 hour may be required to build up a sufficient level. Once a desirable effect is achieved, it should be maintained by slow intravenous (IV) injection or infusion. Doses of 2 to 4 mg every 1 to 2 hours may be required. The dose should be titrated against symptoms with gradual tapering of the dose as the effect of the poisoning runs its course. This may vary from a few hours to several weeks. Oral dosing should replace IV therapy as soon as possible (2 to 5 mg every 1 to 2 hours).

NOTE

1. Phenothiazines and other sedatives (such as chloral hydrate) will potentate the effects of these depressant compounds and are specifically contraindicated.

2. An overdose of physostigmine can result in cholinergic toxicity up to and including temporary apnea. If apnea occurs, assisted ventilation is indicated. Small doses (0.5 mg) of atropine given intravenously may be used to control less severe symptoms of overdose. Since the half-life of physostigmine is only about 30 minutes, overtreatment usually does not require any additional therapy for spontaneous recovery to occur. Then treatment can be resumed, using a slightly smaller and less frequent dosage.

b. Indoles. No true antagonist to the indoles is as yet known. The best treatment known at present for LSD intoxication is the administration of diazepam 10 to 20 mg IV or IM to sedate the patient until spontaneous recovery occurs. Chlorpromazine 50 to 100 mg IM injection has been suggested but does not appear to have any advantage over these drugs.

c. Cannabinols. Stimulants such as d-amphetamine (15 mg) can antagonize the sedation and indifference induced by marihuana-like substances. Although amphetamine may slightly potentate the effects of LSD (if given to such individuals in error), this is not a contraindication to its use if cannabinol intoxication is suspected.

d. Other Agents. Unfamiliar agents or mixtures of agents may be encountered on future battlefields. In such instances, the general principles of restraint, close observation, and supportive medical care apply. No medication should be given until an etiological diagnosis can be made with reasonable certainty—unless circumstances require it (for example, concomitant wounds, burns, or fractures requiring major surgical intervention). The judgment of the medical officer remains the only useful guide to action in these complex and unforeseeable circumstances.