CHAPTER 6

INCAPACITANTS

SECTION I-GENERAL

601. Introduction.

a. An incapacitant is a chemical agent which produces a temporary disabling condition that persists for hours to days after exposure to the agent has occurred (unlike that produced by riot control agents). Medical treatment while not essential may in some cases facilitate more rapid recovery. In the narrower sense the term 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 by altering the higher regulatory activity of the central nervous system.
(3) Of a duration of action lasting hours or days, rather than of a momentary or fleeting action.
(4) Not seriously dangerous to life except at doses many times the effective dose.
(5) Not likely to produce permanent injury in concentrations which are militarily effective.

b. These criteria eliminate many drugs that might otherwise be considered as incapacitants. Opiates and strong sedatives are too dangerous on account of their low margin of safety and milder tranquilizers cause little actual loss of performance capability. Many compounds have been considered as incapacitants and medical staffs must be on the alert to detect and report any unusual clinical appearances. All lethal agents in low doses may produce incapacitating effects and it is possible that new agents for incapacitation may be developed. Agents which produce unconsciousness or induce vomiting may well be developed in the future.

c. In this chapter, consideration will be given to two categories which are well known: CNS depressants (anticholinergics) and CNS stimulants (LSD). Although cannabinols and psylocibin, for instance, have been considered in the past, their effective dose is too high for these to be regarded as likely agents for use in the field.

602. CNS Depressants.

CNS depressants produce their effects by interfering with transmission of information across central synapses. An example of this type of agent is 3-quinuclidinyl benzilate (BZ), which blocks the muscarinic action of acetylcholine both peripherally and centrally. In the central nervous system anticholinergic compounds disrupt the high integrative functions of memory, problem solving, attention and comprehension. Relatively high doses produce toxic delirium which destroys the ability to perform any military task.

603. CNS Stimulants.

CNS stimulants cause excessive nervous activity by facilitating transmission of impulses. The effect is to flood the cortex and other higher regulatory centres with too much information, making concentration difficult and causing indecisiveness and inability to act in a sustained purposeful manner. A well known drug which acts in this way is D-lysergic acid diethylamide; similar effects are sometimes produced by large doses of amphetamines.

604. Detection.

a. Field laboratory methods are not yet sufficiently developed to permit isolation and identification of specific agents in the environment and 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 may be available. Following the occurrence of a suspected chemical attack with incapacitating agents, the medical officer should be prepared to take the steps listed below.

b. Instruct field evacuation teams to transport casualties to an uncontaminated area. Resistant or disoriented individuals should be restrained in the triage area after they have been given the necessary first aid.

c. 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 6-I.

d. In a large-scale attack, the diagnosis will be simplified by the epidemiological distribution of the casualties. It is better to look for characteristics common to all or most casualties, then to be overly impressed with atypical features. For example, some anticholinergics are capable of causing marked disorientation, incoherence, hallucinations and confusion (the pathognomonic features of delirium) with very little, if any, evidence of peripheral autonomic effect (such as tachycardia and dilated pupils). This should not dissuade the medical officer from considering the likelihood of a centrally predominant anticholinergic being the causative agent, since very few other pharmaceutical classes can produce delirium in militarily effective doses. The disturbance produced in indoles (such as LSD) or the cannabinols (such as marihuana extracts) is not really delirium, because the casualties remain receptive to their environment and can comprehend quite well, even though they may have great difficulty reacting appropriately.

605. Protection.

It is likely that such agents will be dispersed by smoke-producing munitions or aerosols, using the respiratory tract as a portal of entry. The use of the protective mask, therefore, is essential. With some agents the percutaneous route may be used and full individual protective equipment will be required.

Table 6-I. Signs and Symptoms Produced by Incapacitating Agents

Signs and symptoms	Possible aetiology
Restlessness, dizziness, or giddiness; failure to obey orders, confusion, erratic behaviour; stumbling or staggering; vomiting.	Anticholinergics (e.g., BZ), indoles (e.g., LSD), cannabinols (e.g., marihuana), anxiety reaction, other intoxications (e.g., alcohol, bromides, barbiturates, lead).
Dryness of mouth, tachycardia at rest, elevated temperature, flushing of face; blurred vision, pupillary dilation; slurred or nonsensical speech, hallucinatory behaviour, disrobing, mumbling and picking behaviour, stupor and coma.	Anticholinergics.
Inappropriate smiling or laughter, irrational fear, distractability, difficulty expressing self, perceptual distortions; labile increase in pupil size, heart rate, blood pressure. Stomach cramps and vomiting may occur.	Indoles. (Schizophrenic psychosis may mimic in some respects.)
Euphoric relaxed, unconcerned daydreaming attitude, easy laughter; hypotension and dizziness on sudden standing.	Cannabinols.
Tremor, clinging or pleading, crying; clear answers, decrease in disturbance with reassurance; history of nervousness or immaturity, phobias.	Anxiety reaction.

606. Decontamination.

Complete cleansing of the skin with soap and water should be accomplished at the earliest opportunity. 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.

SECTION II - CNS DEPRESSANTS - BZ (3-QUINOCLINIDINYL BENZILATE) AND SIMILAR COMPOUNDS

607. Detection.

There is no device available at present for detecting this agent.

608. Protection.

Protection is given by the respirator, NBC suit, overboots and gloves.

609. Properties.

a. BZ and its analogues are glycollic acid esters. Some members of the group are liquid at ambient temperatures but BZ is a stable white crystalline powder that is only slightly soluble in water.
b. These agents are metabolised primarily in the liver and excreted by the kidneys.

610. Mechanism of Action.

a. BZ (3-quinuclidinyl benzilate) is a cholinergic blocking agent that at single doses of less than 1 mg produces delirium lasting several days. In this respect it resembles the well known belladonna alkaloids, atropine and scopolamine, except that it is more potent and its effects last longer. The safety margin (ratio of lethal to incapacitating dose) in people is estimated to be at least 30. No permanent adverse effects have been reported from clinical studies.
b. BZ is effective by all routes of administration, but its effectiveness percutaneously (when mixed with a suitable solvent) is limited, so that route is not likely to be used. However there are other related compounds which are effective percutaneously.
c. It readily crosses the blood-brain barrier and is distributed to all areas of the brain and spinal cord.
d. After administration of an effective dose by inhalation by mouth or by injection mild peripheral effects of BZ occur within 1 hour and maximal central effects occur after about 4 hours lasting 24 to 48 hours, with a peak at 8 to 10 hours. Some other compounds in this group may take longer for their effects to develop and to disappear. Doubling the dose prolongs the duration of severe central effects by about 40 hours and shortens the onset time of severe effects to about 1 hour.
e. BZ and other glycollates produce their effects within the nervous system in the same way as atropine and scopolamine, that is by interfering with cholinergic transmission at muscarinic sites, both in the peripheral autonomic nervous system and in the brain and spinal cord. Because of the wide distribution of these sites measurable effects upon almost every phase of neural regulation may be observed.

611. Signs and Symptoms.

Small doses of BZ cause sleepiness and diminished alertness. Diagnosis can be made by noting increased heart rate, dry skin and lips, drowsiness and a progressive intoxication in the untreated individual as follows:

a. *1-4 hours:* Tachycardia, dizziness, ataxia, vomiting, dry mouth, blurred vision, confusion, sedation progressing to stupor.
b. *4-12 hours:* Inability to respond to the environment effectively or to move about.

c. *12-96 hours:* Increasing activity, random unpredictable behaviour with delusions and hallucination; gradual return to normal 48 to 96 hours after exposure.

612. Treatment.

a. For most casualties, symptomatic treatment is all that will be necessary. Firm restraint when necessary and a friendly attitude are called for especially in dealing with these subjects who are capable of walking. All dangerous objects must be removed and anything likely to be swallowed should be kept away from the subject as bizarre delusions may occur.

b. The most important single medical consideration is the possibility of heat stroke. Clothing should be removed if the temperature is greater than 25°C. If the body temperature is greater than 39°C vigorous cooling is indicated. The casualty should be placed in the shade and air allowed to circulate. Water may be sprayed on the casualty to aid cooling, ice should *not* be applied to the skin.

c. Physostigmine, which is used as an antidote to BZ, should be reserved for casualties who appear to be in danger. Where this treatment is deemed to be necessary an injection of 2-3 mg 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 injection or infusion. Doses of 2-4 mg every 1 or 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 days. Oral dosing should replace intravenous therapy as soon as possible (2 to 5 mg every 1 to 2 hours).

d. Peripherally acting drugs, which do not cross the blood-brain barrier, such as pyridostigmine, neostigrnine and pilocarpine are ineffective antagonists of the central effects of BZ and should not be used in place of physostigmine.

SECTION III - CNS STIMULANTS-LSD (D-LYSERGIC ACID DIETHYLAMIDE)

613. Properties.

LSD is solid at normal temperatures and is soluble in water. It is a very difficult agent to disseminate and consequently is likely to be used by an enemy only in a clandestine manner.

614. Detection.

There is no device available for detecting this agent in the field.

615. Protection.

No personal protection is available against clandestine attack, but it seems probable that only small quantities of food or water could be contaminated. Good security of the food and water supply are therefore required.

616. Mechanism of Action.

a. Very small doses (for example 50 micrograms per person) are capable of inducing a psychotic state in people, but the precise mechanism of action is not yet known.
b. LSD has been shown to facilitate neural activity in the reticular activating system of the brain stem. It appears to interfere with the normal filtering action of this system, permitting sensory input to reach higher integrative centres without regard to its importance or relevance. The result is a decrease in the ability of the brain to process information selectively and in logical sequence.

617. Pathophysiology.

a. D-lysergic acid diethylamide is the most potent of the biologically active indole compounds, as little as 50 µg being required to produce dramatic psychological changes. Doses of 2 to 5 mg have been taken without permanent sequelae, and animal studies suggest that much higher doses may be tolerated. Convulsions may occur at doses above 2 mg.
b. LSD may be inhaled or ingested. Initial effects appear within a few minutes of inhalation or within 30 to 60 minutes of ingestion. Maximum effects are reached within 2 to 3 hours and gradually subside over the next 4 to 8 hours. The half-life in human plasma is about 3 hours. Tolerance is acquired rapidly on repeated exposures at daily intervals, but is shortlived.
c. LSD appears to interact with endogenous neurotransmitters such as serotonin with which it shares the common feature of an indole nucleus. It is metabolised by the liver and excreted through the kidneys.

618. Signs and Symptoms.

a. The clinical manifestations of LSD intoxication often include an early stage of nausea followed 45-60 minutes after dosage by a confused state in which delusions and hallucinations are common but not always experienced. There is some evidence that the effects may be held off, at least for a time, by determination to continue duty and that the presence of non-intoxicated comrades enables affected subjects to maintain contact with reality.
b. Subjects intoxicated with LSD show evidence of sympathetic stimulation (rapid heart rate, sweating palms, pupillary enlargement, cold extremities) and mental excitation (nervousness, trembling or spasms, anxiety, euphoria and inability to relax or sleep). Hyperthermia has been reported. Subjectively, feelings of tension, heightened awareness, exhilaration, kaleidoscopic imagery, emotions of every type, hilarity and exultation are characteristic. Paranoid ideas and more profound states of terror and ecstasy may also occur, especially in highly suggestible individuals. True hallucinations are rare, as is homicidal or suicidal behaviour.

619. Treatment.

No true antagonist to the indoles is as yet known. The best treatment known at present for LSD intoxication is the administration of diazepam 10-20 mg intravenously or intramuscularly or sodium amytal 200-400 mg intravenously to sedate the patient until spontaneous recovery occurs. Chlorpromazine has also been suggested but does not appear to have any advantage over these drugs.

620. Course and Prognosis.

The question of long term effects is still unresolved, but single exposures to doses in the clinical range (0.1 to 1.0 mg total dose) appear unlikely to cause any permanent biological damage.

621. Other Agents.

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