CHEMICAL AGENTS AND MASS CASUALTIES

FEW CHEMICALS CAN BE DELIVERED BY TERRORISTS IN THE APPROPRIATE FASHION TO CREATE LARGE NUMBERS OF DEATHS

THE EMERGENCY PHYSICIAN IS MORE LIKELY TO ENCOUNTER THE ACCIDENTAL RELEASE OF A CHEMICAL IN AN INDUSTRIAL OR TRANSPORTATION ACCIDENT

AGENTS RELEASED CAUSE A "PENUMBRA EFFECT" → TRUE EMERGENCIES OCCUR IN THE EPICENTRE AND A LONG SHADOW OF FEAR AND PANIC SURROUND THIS

AT THE EPICENTRE, INDIVIDUALS USUALLY SUCCUMB EARLY

IN THE IMMEDIATE SURROUNDING AREA (THE "WARM ZONE"), INDIVIDUALS MAY REQUIRE DECONTAMINATION AND INTENSIVE CARE

SCENE HAZARDOUS MATERIALS RESPONSE

STEPS ARE OUTLINED BELOW

Table 9-2 Scene Hazardous Materials Planning Guidelines
1. Community risk assessment
2. Recognition of an event
3. Identification of the substances involved
4. Isolation and scene control
5. Decontamination
6. Stabilization and triage

COMMUNITY RISK ASSESSMENT:

- In US → there is an inventory of stored chemicals that has to be reported annually as mandated by law which assists in assessing risk

RECOGNITION OF AN EVENT:

- Most chemical releases are recognised early because many chemicals have early warning properties → noxious/unusual odour, cause eye or upper airway irritation → can cause rapid death at the site of release
- Many industries have monitors for leaks of chemicals at nontoxic threshold levels that have no warning properties

IDENTIFICATION OF THE SUBSTANCES INVOLVED:

- Most common agents released are pulmonary irritants → ammonia and chlorine
- Although it may seem important to identify the substance that has been released, it is more important to RECOGNISE THE CLINICAL SYNDROMIC MANIFESTATIONS OF THE VICTIMS → e.g. the symptom complex of cholinergic crisis is more important than recognising organophosphate release
- If the symptoms of the patient do not fit the initial chemical identification, then the accuracy of the identification should be called into question

ISOLATION AND SCENE CONTROL:

- Once a suspected chemical release occurs → establish an incident command system, designation of hot, warm and cold zones as well as establishing an isolation process
- Only trained personnel in fully encapsulated protective gear should be allowed to enter the hot zone and their main role is rescue of victims by removing them from further exposure
- A surrounding corridor through which each victim is washed off and decontaminated is created outside the hot zone (the warm zone) → appropriate resuscitation can occur here also
- Once appropriately decontaminated, the patient can be transferred to the cold zone where a lower level of protective equipment is needed and very low risk of secondary contamination exists

DECONTAMINATION IN THE WARM ZONE:

- Primary contamination is caused by contact with the release
- Secondary contamination is the inadvertent contamination of rescue personnel through contact with a contaminated patient or environment → avoided by use of proper protective equipment and adequate deconatmination of adherent solids and liquids
- After removal from the hot zone, the main management issue is adequate decontamination → remove clothing, brush off solid particle and wash the face → WATER IS THE UNIVERSAL DECONTAMINANT → in general five minutes of decontamination with warm water is sufficient for most ambulatory patients → pay special attention to hair, skin folds, axilla, groin, toes and eyes

STABILISATION AND TRIAGE IN THE COLD ZONE:

- Triage should occur in the cold zone, at a point upwind and uphill from the hot zone
- Once decontaminated, patients should be stabilised and evaluated for syndromic symptoms and the need for antidotes
- Only a few antidotes may be truly beneficial if given early → HYDROXYCOBALAMIN in cyanide poisoning and ATROPINE for muscarinic effects of organophosphates
- Many people will make their own way to ED and potentially NOT be decontaminated → we cannot rely solely on ambulance notification

HIGH RISK CHEMICALS:

TOXIC GASES:

- Tend to interfere with one or more of the four phases of oxygen delivery
- DISPLACEMENT OF OXYGEN FROM AIR → SIMPLE ASPHYXIANTS:
    - These create an oxygen-poor environment by displacing oxygen from air → inert gases such as CO2, hydrogen, nitrogen and the noble gases
    - Occupants in closed, unventilated space may die as concentration of asphyxiant gas builds up silently
    - When the concentration of any of these nonreactive gases increases, the fraction of inspired oxygen decreases
    - Treatment is restoration of a higher FIO2 with supplemental oxygen and correction of the problem that led to exposure
- IRRITANT GASES THAT INTERRUPT PULMONARY DIFFUSION:
    - Irritant gases are classified according to their water solubility
    - Highly water soluble agents react with water in the URT and produce immediate irritation and discomfort → e.g. ammonia
        - Hydrogen fluoride is also highly water-soluble but it ALSO REACTS WITH INTRACELLULAR CALCIUM, causing life-threatening calcium depletion and cardiac arrhythmia
        - BOTH AGENTS HAVE IMMEDIATE WARNING PROPERTIES DUE TO INTENSE DISCOMFORT
        - Treatment is removal from the source and in the case of HF → treat with IV calcium boluses
    - The only gas classified as intermediate solubility is CHLORINE → used in chemical warfare in WWI → reacts with water to produce hydrochloric and hydrochlorous acids → burning of the conjunctiva, throat and bronchial tree
        - Higher concentrations can produce bronchospasm, lower pulmonary injuries and delayed pulmonary oedema
    - Low solubility → PHOSGENE → can be fatal after even brief exposure. Slowly hydrolyses to hydrochloric acid and reacts IN THE ALVEOLI → delayed onset pulmonary oedema → intubation often needed
- AGENTS THAT INTERRUPT OXYGEN TRANSPORT:
    - Interrupt delivery of oxygen by altering haemoglobin so that it is no longer capable of binding and transporting oxygen (CARBON MONOXIDE AND METHYLENE CHLORIDE)
    - Symptoms of either include those consistent declining oxygenation with headache nausea and fatigue
- AGENTS THAT INTERFEE WITH CELLULAR OXYGEN UTILISATION (CHEMICAL ASPHYXIATION):
    - Those that interfere with electron transport in the mitochondria
    - THESE AGENTS INCLUDE CYANIDE, HYDROGEN SULPHIDE, PHOSPHINE, SODIUM AZIDE
    - By binding to CYTOCHROME OXIDASE –A, these agents disrupt aerobic metabolism and create intracellular acidosis

- Symptoms include headache, alteration of consciousness, seizures and severe lactic acidosis
- Cyanide antidote kit contains → AMYL NITRITE, SODIUM NITRITE AND SODIUM THIOSULFATE
    - The nitrite components may be used with any of above exposure and thiosulfate only in suspected cyanide poisoning (converts cyanide to sodium thiocyanate)
    - HYDROXYCOBALAMIN can also be used

NERVE GASES:

- TABUN, SARIN, SOMAN → all are designated as ORGANOPHOSPHATES → chosen as chemical weapons as they had greater CNS activity and higher lethality than organophosphates used for insecticides
- ORGANOPHOSPHATES:
    - Inhibit several key enzymes by binding to them
        - Bind to ACETYLCHOLINESTERASE in two stages → first is REVERSIBLE WITH PRALIDOXIME. The second step (AGING PROCESS) is irreversible
        - Acetylcholinesterase breaks down acetylcholine, and thus when it is inhibitied, acetylcholine remains at its postsynaptic receptor sites, CAUSING EXCESS CHOLINERGIC STIMULATION
        - Within the PARASYMPATHETIC NERVOUS SYSTEM → cholinergic receptors at MUSCARINIC SITES (tear glands, sweat glands, bronchial secretion glands and SA/AV nodes) → leads to "SLUD" symptoms:
            - Salivation
            - Lacrimation
            - Urination
            - Defecation
                - Also causes bronchospasm, miosis and bradycardia
        - NICOTINIC RECEPTORS AT NEUROMUSCULAR JUNCTION are also affected:
            - PROGRESSION OF MUSCULLAR FASCICULATION TO PROFOUND MUSCULAR WEAKNESS TO COMPLETE PARALYSIS IN A DOSE-DEPENDENT FASHION
        - Last location for cholinergic stimulation is the BRAIN → produces COMA AND SEIZURES
    - The treatment strategy for nerve agents must counteract cholinergic excess at ALL THREE RECEPTOR SITES:
        - ATROPINE ONLY COUNTERACTS MUSCARINIC EFFECTS:
            - IV atropine should be given to anyone experience hypersalivation, bronchial secretions or bradycardia → anticipate large doses
        - PRALIDOXIME counteracts nicotinic effects:

- Reactivates acetylcholinesterase at the NMJ but can only reactivate the enzyme if AGING HAS NOT OCCURRED
- How rapidly an agent "ages" varies
- Recommended dose of pralidoxime is 1-2g IV over 20-30 minutes
- Many patients with neuromuscular weakness will require intubation and ventilation → DO NOT USE SUCCINYLCHOLINE as the duration of action will be greatly increased

▪ Benzodiazepines enhance atropine and stops seizures → phenytoin not effective

INCAPACITATING AGENTS:

- A diverse group of chemicals that immobilise the victim
  - Narcotic gases → fentanyl derivative used in storming of theatre in Russia was CARFENTANYL mixed with halothane gas → killed hundreds

VESICANTS:

- Sulfur mustard first used by Germany in 1917
- Vesicants CAUSE DAMAGE TO SKIN, MUCOUS MEMBRANES AND POTENTIALLY THE LUNGS IF EXPOSED TO HIGH CONCENTRATIONS
- Sulfur mustard skin symptoms are delayed in onset for 4-8 hours, leading to blistering similar to second degree burns
  - Primary goal of therapy is copious irrigation with water to dilute and remove the chemical and then monitoring the patient with serial eye exams and pulmonary monitoring
  - Can also caused delayed marrow suppression

BIOTOXINS:

- Differ from biologic agents, in that the toxins do not replicate in the body and a sufficient dose to cause disease must be delivered
- In many cases the LD50 → i.e. the lethal dose in 50% cases → is quite low

Table 9-7 Lethality of Biotoxins

Agent	LD_{50} (micrograms/kg)
Botulinum toxin	0.001
Tetanus toxin	0.002
Staphylococcal enterotoxin B	0.02
Diphtheria toxin	0.10
Ciguatoxin	0.4
Ricin	3.0
Tetrodotoxin	8.0
Saxitoxin	10.0
Trichothecene toxin	1200

BOTULINUM TOXIN:

- Inhalational botulism has been described in hums after accidental lab exposure and the LD50 is 1-3nanograms/kg (i.e. very low) → case report of those who performed an autopsy on an animal who had died of botulism developed symptoms → all recovered with appropriate antitoxin treatment
- Treatment with antitoxin will PREVENT PROGRESSION, but will not reverse paralysis once it occurs

RICIN:

- Gained notoriety when used to assassinate Bulgarian activist Georgi Markov → pellet containing ricin was removed from a small wound in the back of his leg
- Ricin is a toxin derived from the castor bean
- Not toxic by ingestion and must be inhaled or injected
- Causes destruction of RNA leading to cell death
    - Inflammation, exudates and pulmonary oedema occur causing a necrotising pneumonitis
    - After parenteral administration, local pain occurs, followed in a few hours by weakness and flu-like symptoms
    - Shortly thereafter (15-24 hours) → fever, N+V, localised lymphadenopathy proximal to injection may occur
    - At 48 hours, a sepsis-like syndrome occurs with hypotension, leukocytosis, IDC and MOF
    - Aggressive supportive care is warranted in ICU setting