

Blast Injuries

Explosions inflict injury in a number of ways. *Primary blast injury* is due solely to the direct effect of the pressure wave on the body. *Secondary blast injury* results from penetrating or nonpenetrating damage caused by ordinance projectiles or secondary missiles, which are energized by the explosion and strike the victim. *Tertiary blast injury* results from whole body displacement and subsequent traumatic impact with environmental objects. Tertiary effects generally result from the bulk flow of gases away from an explosion and occur when the individual is in very close proximity to the explosion. Displacement may take place relatively far from the point of detonation if an individual is positioned in the path that gases must take to vent from a structure, such as in a hatch, in a doorway, or by a window. Thermal injury from radiation, hot gases, or fires started by the explosion are considered to be miscellaneous blast effects. Other indirect effects include crush injury from the collapse of structures and toxic effects from the inhalation of combustion gases.

The pressure wave close to the explosion moves outward at supersonic speed. As the wave spherically propagates, it decelerates and loses energy. In water, because of its incompressibility, the speed of wave propagation is much greater and the wave loses energy less quickly with distance. The lethal radius around an explosion in water is about three times the lethal radius of a similar explosion in air.

A typical pressure wave from an explosion in air is shown in Figure 17. Pressure rises almost instantaneously in the ambient environment and then decays exponentially. The peak pressure and duration of the initial positive phase are a function of the size of the explosion and the distance from the detonation. In air, the peak pressure is proportional to the cube root of explosive weight and the inverse of the cube of the distance from detonation. If the pressure wave is in close apposition to a solid barrier, the pressure exerted at the reflecting surface may be many times that of the incident wave.

A blast wave that causes only modest primary injury in the open can be lethal if the casualty was caught near a reflecting surface, such as a solid wall. The bulk flow of gases away from the explosion (blast wind) travels much slower than the shock wave, but may be of importance in causing displacement close to the point of explosion, especially with very large explosions.

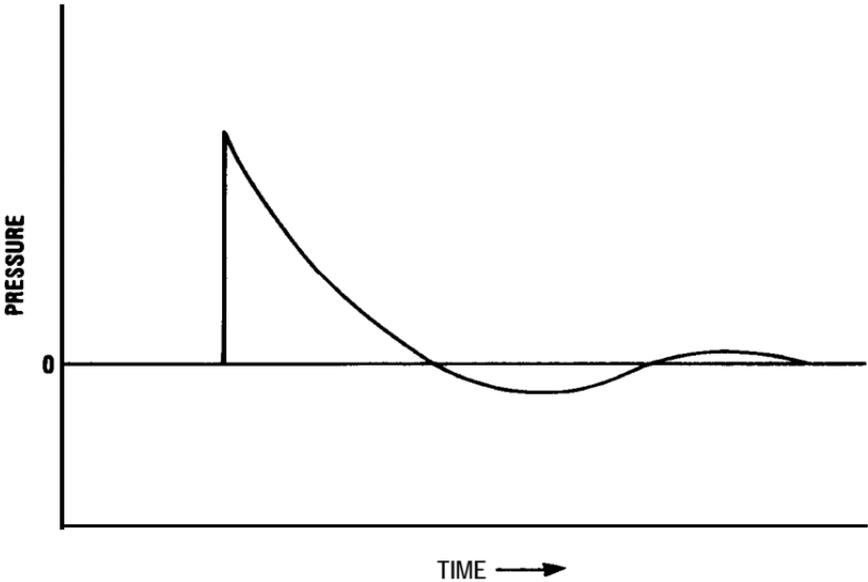


Figure 17.—Idealized representation of pressure - time history of an explosion in air.

For a sharp rising blast wave, damage to both inanimate and biological structures has been shown to be a function of the peak pressure and the duration of the initial positive phase (Figure 18). This figure illustrates the estimated blast levels necessary to cause a range of primary effects in man.

PATHOLOGY OF PRIMARY BLAST INJURY

Primary blast injury is seen almost exclusively in gas containing organs: the ear and the respiratory and gastrointestinal tracts. Of the three organ systems, the ear is the most sensitive. The pinna and the external canal collect and in some cases, amplify pressure signals so that the tympanic membrane, converting acoustic energy to mechanical displacement, is displaced into the middle

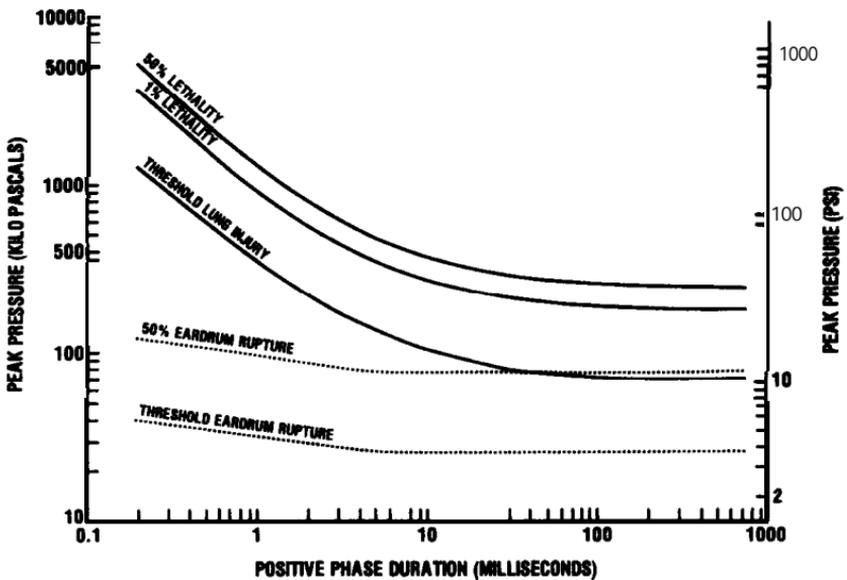


Figure 18.—Estimated human tolerances for single, sharp, rising blast waves.

ear. At pressures of approximately 35 kiloPascals (kPa, with 7 kPa equalling 1 pound per square inch), the human eardrum may rupture (Figure 18). Above 100 kPa, almost all eardrums will be ruptured. The eardrum generally perforates inferiorly in *the pars tensa* but there may be hemorrhage into the membrane without rupture. At higher pressures, the drum may be almost obliterated and the ossicles can be dislocated or fractured. Vestibular function is usually not affected.

Injury to the lung is the cause of the greatest morbidity and mortality. Grossly, one sees diffuse, pleurally-based pulmonary contusions with a stiffened, heavy lung. The *costal* surface may show transverse stripes called "rib markings" which, in fact, are more closely associated with intercostal spaces. Lung weights may be two or three times normal. Pleural rents or blebs may result in pneumothorax, hemothorax, or mediastinal extravasation of

air. Rib fractures or evidence of significant chest wall damage are not seen in the absence of other mechanisms of trauma. Microscopically, the hemorrhage is mainly intra-alveolar with some perivascular or peribronchial disruption and bleeding. Alveolar walls are torn, sometimes producing giant blood-filled alveolar spaces. Alveolar-pulmonary venous communications, the source of air emboli within the arterial circulation, are created. These **fistulae** are responsible for most of the early mortality resulting from primary blast injury. Critical vascular beds in the central nervous or coronary arterial circulations can be occluded by entrained air emboli with subsequent disastrous results.

The gastrointestinal tract may be damaged wherever there are collections of gas. Injury to the gut is particularly severe in underwater blasts. While hollow visceral injury is also present in airblast, it is generally overshadowed by the more dramatic presentation of air emboli or acute respiratory insufficiency. The colon is the hollow **viscus** most commonly disrupted. Gastric injuries are usually less common and less severe. Rarely, one encounters rupture of the spleen or liver in the absence of superimposed blunt abdominal trauma. Pathologically, injuries to the bowel range from subserosal or intramural hemorrhage to frank rupture. The natural history of such bowel wall hematomas is not known, but it is clear that some can progress to perforation during the **post-injury** course.

MECHANISMS OF INJURY

The blast wave exerts a force (pressure times exposed area) on the body surface. That force is transmitted to internal structures by bulk movement of tissue. Inertial effects may play a role in the injuries seen around the relatively massive airways and vessels suspended in the lighter tissue of the lungs. Mass differences, the compressibility of isolated gas pockets, and the material properties of the foam-like lung tissue are probably critical factors in blast injury. Pressure waves propagate in the lung parenchyma as a result of blast exposure. At some point, the lung is unable to pass on the local stresses generated at the pleural surface as fast as the chest wall moves and delivers energy. In such a case, the local compressions, shears, or tensile stresses exceed the physical limits of the lung substance and injury occurs.

For the gut and tympanic membrane, the physical events leading

to injury are probably much simpler. Isolated collections of gas within the bowel lumen are compressed by the pressure wave within the abdomen. At some point, the bowel wall is stressed to the point of failure, manifested as either intramural hemorrhage or frank rupture. The eardrum is a relatively simple membrane which completely closes one end of a tube, the other end of which is open to the air. The middle ear airspace behind the drum is unable to equilibrate pressures rapidly enough through the Eustachian tube. When the stress on the drum exceeds the limits of the tissue, the tympanic membrane ruptures.

CLINICAL PRESENTATION OF PRIMARY BLAST INJURY

Primary blast injury effects may be only a part of the problem in a casualty suffering from multiple trauma. In the setting of injury associated with a large blast, the basic principles of trauma care still apply, and resuscitation and evaluation should proceed by the usual numbers. The key to recognizing that primary blast injury is present is a history or setting suggestive of a powerful explosion. One should then search for corroborative findings with a careful examination of the tympanic membranes, retinal arteries, chest, and abdomen. Specialized military ordinance such as a fuel air explosive or underwater blast may cause a relatively pure form of primary blast injury. Fuel air explosive ordinance is a particularly powerful air blast designed to clear mine fields by detonating the land mines in place.

The ear and the upper respiratory tract are the structures most sensitive to primary blast injury. Rupture of the tympanic membrane may cause tinnitus, pain, and **hearing** loss. Physical examination will reveal blood in the external canal and otoscopic evidence of perforation. In severe injury, there can be vestibular damage with disordered equilibrium. Pressure levels high enough to cause serious injury to the lungs or gut almost invariably rupture the eardrums. This may not be the case when ears were protected by ear muffs or ear plugs. Often the tympanic membranes are not ruptured by high-grade underwater explosions if the head is above water and the tympanic membranes are not exposed to the underwater pressure wave. Petechial hemorrhage in the hypopharynx and larynx is also observed at relatively low pressure levels and, like tympanic rupture, its absence speaks against exposure to high levels of blast. Upper airway petechial hemorrhage such as this is

unlikely to cause airway compromise or other symptoms.

Arterial air emboli represent an immediate threat to life. Clinical evaluation in the presence of air emboli will reveal evidence of cerebral dysfunction such as altered affect, confusion, disorientation, or focal neurologic signs. When such findings are noted after an explosion, one must first consider a skull fracture or other closed head injury. Direct trauma to the skull from secondary or tertiary blast effects is more likely than air emboli in most settings. It may be possible to directly visualize air bubbles in the retinal vessels or to observe patchy blanching of the tongue. Emboli to the coronary arteries will be evidenced by arrhythmias or ischemic electrocardiograph changes. Emboli to other vascular beds might be expected to give a clinical picture similar to the "bends" or decompression illness.

Primary blast injury of the lung presents a clinical picture similar to that of pulmonary contusion from blunt chest trauma, but without rib fractures or chest wall injury. Chest tightness, pain, and hemoptysis are common complaints. One observes tachypnea, the employment of accessory muscles of respiration, and other signs of respiratory distress. Evidence of pulmonary consolidation may indicate either contusion or a hemothorax. A pneumothorax may present as unilateral hyper-resonance with decreased breath sounds and a contralateral shift of the trachea and mediastinum. A precordial systolic crunch on auscultation indicates extravasation of air into the mediastinum. Roentgenographic examination of the chest is mandatory. A simple, frontal view will be diagnostic in most instances of significant barotrauma. Pneumothorax, hemothorax, pneumomediastinum, pleural blebs, subcutaneous emphysema and pulmonary interstitial emphysema can be confirmed by the chest X-ray. The manifestations of contusion may develop over the course of hours and may have the appearance of a local or diffuse infiltrate. The clinical picture of "blast lung" may develop over 24-48 hours. In a complex trauma setting, it is very difficult to differentiate the respiratory insufficiency of the adult respiratory distress syndrome with its varied etiologies from that due solely to primary blast injury to the lungs. Aside from arterial blood gas determinations, laboratory studies have little to offer early on.

Gastrointestinal injury usually presents a less dramatic clinical picture and its diagnosis may be suppressed by the more life-

threatening effects of air emboli or respiratory insufficiency. Signs of peritoneal irritation such as involuntary guarding, rebound tenderness, and absent bowel sounds may indicate visceral rupture. Bright red rectal bleeding has occurred with low sigmoid injury. Contused bowel may necrose and perforate several days after the initial trauma. Abdominal X-rays may reveal free peritoneal air or air within the lumen of the bowel wall. Although multiple organ injury is the usual case in underwater blast injury, visceral injury may predominate and may represent the sole major injury.

TREATMENT OF PRIMARY BLAST INJURY

The individual with primary blast injury usually presents with associated injuries. The basic principles of triage and trauma care management still pertain. Airway establishment, control of hemorrhage, and reversal of shock should proceed without consideration of the presence of blast injury. Since an overly generous administration of fluids during the resuscitation may complicate pulmonary injury, pulmonary artery catheterization and pressure monitoring may be necessary to guide fluid therapy in complex cases. When possible, blast victims should be kept sedentary, as exercise may increase mortality by increasing air emboli or by worsening lung hemorrhage.

Tympanic rupture is treated conservatively. After examination, any debris should be cleared from the external canal; however, no irrigation should be attempted. The majority of tympanic tears will heal spontaneously. About one fourth will require surgical closure which can be delayed for weeks.

Air emboli from a severe blast may be lethal within minutes. The incidence of severe air embolism can be lessened by placing the individual in the prone position with the left side down, the back at a 45° angle to the ground, and the head lower than the feet. This position is thought to distribute emboli to the lower extremities rather than to the head vessels, and is also thought to trap air in the right heart. If seen early enough, prompt use of a compression chamber may be lifesaving. Hyperbaric therapy works both by physically reducing the size of the bubbles and by speeding their absorption. The addition of oxygen to the hyperbaric environment probably adds little to the effect of the increased pressure. In the absence of hyperbaric capability, empiric therapy for CNS injury or cardiac ischemia should be instituted.

Respiratory distress should be immediately treated with supplemental oxygen, and the individual should be evaluated to establish whether the etiology is pneumothorax or pulmonary parenchymal failure from blast or other causes (e.g., inhalation of toxic gases). Progressive respiratory failure poses a particular problem since positive pressure ventilation may increase the incidence and severity of both air emboli and pulmonary barotrauma. If oxygen delivery via conventional binasal prongs or a face mask is insufficient to produce adequate tissue oxygenation, constant positive airway pressure (CPAP), either by face mask or endotracheal tube, should be employed to keep small airways open and to improve oxygenation. Positive pressure ventilation assistance should not be withheld if the clinical situation deteriorates.

Inhalation anesthesia carries a very high morbidity in blast injury. This is probably due to the unmonitored use of positive pressure ventilation intraoperatively and to the difficulty of neurologically assessing the patient. Every effort should be made to perform surgical procedures under regional or spinal anesthesia. Airway pressures during inhalation anesthesia should be kept as low as possible since intraoperative pneumothorax can be produced. Consideration should be given to the prophylactic use of chest tubes. One should anticipate the very possible occurrence of pleural complications by performing frequent physical examinations and chest roentgenograms.

Blast injury of the gastrointestinal tract should be managed in the same way as blunt trauma. Hypovolemic shock in the absence of other obvious etiology should suggest visceral rupture, and warrants diagnostic peritoneal lavage and consideration of laparotomy. Decompression via a nasogastric tube should be undertaken with any peritoneal signs and whenever ventilatory assistance is instituted. The patient should be observed for several days because of the risk of delayed perforation. The role of antibiotics and anti-inflammatory medication is unclear, although both have their advocates.

CONCLUSION

Primary blast injury may present in individuals exposed to powerful explosions in military operations. It is likely to coexist with missile injuries, blunt trauma, burns, and other injuries.

Diagnosis is suspected in the presence of tympanic rupture or hypopharyngeal petechial hemorrhage. Treatment is similar to that for blunt trauma to the chest or abdomen. An important feature is the recognition of pneumothorax and arterial air emboli, both of which may be made worse by positive pressure ventilation.

CHAPTER VI

Chemical Injury

INTRODUCTION

This chapter provides updated guidance for initial care of chemical casualties so as to save life and limb and to aid in the soldier's early return to duty. The recommendations include current research views and should be considered as an adjunct to, not a substitute for, doctrinal management as outlined in Army TM S-285, *Treatment of Chemical Agent Casualties and Conventional Military Chemical Injuries*, dated May 1974 (Change #2, November 1985).

BACKGROUND

Since some of our potential adversaries maintain large stocks of chemical agents, train under realistic conditions, and aim for tactical surprise, the threat from chemical warfare (CW) agents on the battlefield is very real. It should also be borne in mind that CW agents are easily synthesized from readily available chemicals and could therefore be employed to advantage by third-world nations. The operational scenario may well be one of initial chemical attack compelling defending troops to "button up," followed by assault with conventional weapons. Thus, the high likelihood of having to deal with combined chemical and traumatic injuries must always be kept in mind.

CLASSIFICATION

The operational classification of CW agents is shown in Table 3. Riot control agents (eg., tear gas) are not considered CW agents since symptoms are transient and self-limiting. Conversely certain extremely potent chemicals, such as mycotoxins and neurotoxins, are classified with the biological warfare (BW) agents.

TABLE 3.-*Chemical warfare Agent Classification*

Category	US Code	Common Name
Nerve Agents	GA	Tabun
	GB	Sarin
	GD	Soman
	VX	- -
Blister Agents	HD	Distilled Mustard
	L	Lewisite
	c x	Phosgene oxime
Blood Agents	AC	Hydrogen cyanide
	CK	Cyanogen chloride
Choking Agents	CG	Phosgene
	CL	Chlorine
Incapacitation Agents	BZ, QNB	Quinuclidinyl benzilate

IDENTIFICATION AND DIAGNOSIS

Table 4 provides rough initial identification guidance based on the time lag between CW agent exposure and onset of symptoms and signs. Table 5 then aids in diagnostic differentiation once the early signs and symptoms of chemical exposure are established.

TABLE 4.—*Time of Onset*

Precipitous Onset	Rapid Onset	Delayed Onset
Choking Agent: CL	Inhaled Nerve Agent	Absorbed Nerve Agent
Blister Agent: L	Blood Agent	Inhaled Blister Agent
Incap Agent	Liquid in Eye: HD	Choking Agent: CX

TABLE 5.—*Early Signs and Symptoms of Chemical Exposure*

	Signs/Symptoms	Causative Chemical Agent
CNS	convulsions confusion, odd behavior stupor	nerve; blood incap any agent
Respiration	copious ore-nasal secretions chest pain, wheezing frothy sputum hyperpnea, dyspnea apnea cyanosis	nerve nerve; choking; blister blister; choking choking; blister; blood nerve; blood blood; nerve; choking
Circulation	bradycardia tachycardia shock	nerve; blood blood; nerve; incap any agent
Skin	hot, dry, flushed vesication pain on contact muscle tremors erythema	incap blister lewisite nerve unknown liquid
GI/GU	involuntary evacuation vomiting	nerve any agent

DELAYED EFFECTS

While several CW agents produce immediate signs and symptoms, the effects of others may be delayed, depending on the agent concentration and duration of exposure (Table 4). For instance, mustards can seriously damage the skin without immediately producing pain; likewise, pulmonary edema from phosgene may take hours to become manifest. Mixtures of CW agents or newly introduced chemicals further complicate the **diagnostic** picture. Hence, a holding period following unidentified CW agent(s) **exposure**, and careful reexamination prior to discharge, may be prudent, circumstances permitting.

General Principles of Management

Personal Hazards. First, do not become a casualty yourself: protect yourself and instruct your personnel to do likewise. Next,

prevent further injury of the casualty: apply his protective mask and cover him, administer treatment, remove clothing, and decontaminate exposed body surfaces. Casualty decontamination may not always be as complete as desired because of the urgency of the situation or resource constraints. Thus, the potential for vapor exposure from an off-gassing residual agent or inadvertent contact with unsuspected, undetected liquid is an ever-present hazard for medical personnel.

Route of Entry. The nerve agents and blood agents are liquids, the vapors of which gain systemic access mainly via the respiratory tract. Their onset of action is precipitous and lethality can be swift. Other nerve agents, VX and thickened GD for instance, are absorbed percutaneously so that the onset of first effect may be delayed. Once in the blood stream, however, they act as quickly as the inhaled nerve agents.

Although agents such as mustard rapidly fix in the skin, the visible dermal injury takes time to develop. One observes both the early irritant effect of a mustard gas on the eyes and respiratory tract, and the delayed systemic effects of leukocytopenia with mustard and hemolysis with lewisite.

Persistent (non-volatile) agents also can contaminate uncovered food or water supplies. Ingestion of blister agent, for instance, may cause necrotic changes in the gastrointestinal tract.

Initial Priorities

Casualties may present with combined injuries on the integrated battlefield—that is, chemical/nuclear exposure combined with trauma or illness. Body heat build-up inside the protective ensemble, with resultant dehydration or hyperthermia, further complicates the picture. The initial issue facing the medical officer, then is determination of treatment priorities for such **combined injuries**.

There is no single “best” way to prioritize emergency treatment for chemical or mixed casualties. In general, respiratory insufficiency and circulatory shock, whatever the cause, present the more immediate life-threatening problems. One possible approach is suggested below:

1. Treat respiratory failure and control massive hemorrhage
 2. Administer chemical agent antidote(s).
 3. Decontaminate the face (and protective mask if donned).
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4. Remove contaminated clothing and decontaminate exposed skin.

5. Render emergency care for shock, wounds, and open fractures.

6. Administer supportive medical care as resources permit.

7. Transport the stabilized patient to a chemically clean area.

The following five sections group CW agents according to the categories of Table 3.

Nerve Agents

Nerve agents inhibit the ability of choline esterase to hydrolyz acetylcholine (ACh) which in turn stimulates muscarinic and nicotinic receptors as well as the central nervous system (CNS) directly. As a result, the casualty will manifest a classic cholinergic syndrome that, depending on exposure and treatment, can span the range from simple miosis and "red eye" to a fulminating cholinergic crisis progressing within minutes to respiratory arrest and death.

Diagnosis: The diagnosis of nerve agent exposure is readily made from physical signs: fasciculation of skeletal muscle (perhaps progressing to depolarization paralysis), smooth muscle contraction of airways, bladder, and bowel; intense miosis and cycloplegia; marked bradycardia (may be masked by excitement or atropine); copious secretions; convulsions; rapidly weakening respiratory effort; pale cyanosis; and terminal apnea.

Treatment: Immediate IM or IV injection with atropine to block muscarinic cholinergic receptors, and with Z-PAM (if given soon after exposure) to reactivate cholinesterase, is effective. Each U.S. soldier has in a pocket of his protective mask carrier three MARK I kits for intramuscular self-injection, each kit delivering 2 mg injections of atropine sulfate and 600 mg pralidoxime chloride (3-PAMCl). Additional 2 mg injections of atropine may need to be given by medical personnel until clear clinical evidence of atropinization is obtained (dry red skin, easier breathing decreased wheezing, dry mouth and, less consistently, dilating pupils).

Airway obstruction requires the clearing of secretions (by suction, if possible, or else by prone turning for postural drainage), the placement of an oropharyngeal or nasopharyngeal airway, and supplementary oxygen, if available. Endotracheal intubation or

trichotomy may be required in conjunction with manual or mechanical ventilation. If the environment is chemically contaminated, a closed system or charcoal-filtered air must be used for ventilation.

Experimental evidence suggests that benzodiazepine **anticonvulsants** reduce the morbidity associated with **organophosphate**-induced convulsions. Suggested doses are 5 mg Valium (IM) repeated as needed, or 2.5 mg increments of (IV) Valium.

Blister **Agents**

The blister (vesicant) agents are cytotoxic alkylating compounds exemplified by the mixture of compounds collectively known as "mustard" or "mustard gas" (H). Other blister agents are sulfur mustard (HD), nitrogen mustard (HN), phosgene oxime (CX), and Lewisite (L), an arsenical vesicant. Mustard vapor injury is a particular threat in hot climates. High humidity in a hot environment further enhances contact damage to the skin.

Diagnosis: The diagnosis of chemical skin injury is straightforward once blisters have appeared, but early and correct recognition of blister agent exposure can be difficult because:

a. Eye inflammation and upper respiratory tract irritation, often the first effects noted, present a picture similar to that produced by choking agents.

b. Although dermal damage occurs within minutes of contact, it cannot always be seen immediately and is commonly painless until subdermal layers become involved and blisters form several hours later.

After a 1-12 hour (or more) latent period, during which burning and itching may occur, erythema appears on exposed skin. In dark-skinned casualties, sulfur mustard lesions may turn **coal-black** in such areas as the face, neck, axilla, groin, and genitalia. Erythema is followed by coalescing, translucent, yellowish blisters on a red base. Healing and resorption of non-infected blisters occur in 1-3 weeks. Broken blisters must be protected to minimize chances for infection and subsequent scarring of denuded skin.

Lewisite is differentiated from the mustards by pain immediately upon skin contact. Nasal irritation, sneezing, and pungent odor provide early warning of the presence of Lewisite vapor. Only those without, or incapable of donning the mask will suffer serious respiratory effects.

Treatment: Forward treatment of vesicant injuries is mainly preventive and supportive. Immediate decontamination of the casualty has top priority. Agent droplets should be removed as expeditiously as possible by blotting or flushing. The M-25841 **decon** kit is extremely effective in inactivating mustard, but it is also quite caustic. A surgical soap and cool water wash suffices, particularly for widely contaminated skin. Neither scrubbing nor hot water is recommended since both accelerate absorption and increased vapor formation. Army TM 8-285 provides further details regarding proper decontamination procedures.

Eye. Immediately flush the contaminated eye with water. Antibiotic ointment, with or without steroid, helps minimize infection. In more severe cases, blepharospasm and pain are extreme, requiring local anesthetic drops or ointment (e.g., tetracaine). Irrigation with sterile saline will remove crusted exudate.

Respiratory Tract. Inhalation of mustard vapor produces severe irritation of the upper respiratory tract, with painful cough, bloody sputum, chest pain, and dyspnea. Treatment is symptomatic at first, since the severity of the broncho-pulmonary lesion may not become evident for some time. Even asymptomatic patients should be observed for at least 4-6 hours, and not released until after re-examination of the chest. Lewisite vapor produces similar effects, except for more pronounced nasal irritation and sneezing.

Tracheitis and bronchitis are prominent; however, fulminant pulmonary edema is much less common with the blister agents than with the choking agents. Bronchopneumonia is a common complication; a change in the appearance of the sputum (culture if possible) is a clear indication for antibiotic therapy.

Prophylactic antibiotic administration is neither necessary nor recommended.

Skin. Doctrinal (TM 8-285) treatment recommends the opening and draining of blisters with removal of the blister fluid. Syringe aspiration of bullous fluid from large blisters might be as effective. Supportive therapy for mustard burns is essentially similar to that for thermal burns: aggressive fluid replacement, pain relief, and vigilance against bacterial infection. From the standpoint of personnel, facilities, and resupply, forward-positioned medical

resources would be severely stressed in the event of widespread utilization of mustard gas by the enemy.

Systemic Bone marrow depression with severe leukopenia and thrombocytopenia follows extensive mustard absorption. Resistance to infection is diminished, with correspondingly high mortality from pneumonitis or other bacterial infections. Mustard ingested with water or food may damage gastrointestinal epithelium, resulting in blood and fluid losses.

Arsenical vesicants, such as Lewisite, increase capillary permeability, causing extensive third-space fluid shifts. Intravascular hemolysis of erythrocytes, and subsequent hemolytic anemia, complicate the clinical picture and may lead to renal failure. Intramuscular BAL (1ml per 50 pounds, not to exceed 4 ml) is given at **4-hour** intervals for a total of 4 doses. In severe cases, follow-up treatment for 3-4 days with the daily deep IM injections of 1 ml per 100 pounds is recommended.

Choking Agents

This group, so called because of the pronounced irritation of the upper as well as the lower respiratory tract, consists of phosgene (CG), diphosgene (DP), and chlorine (CL). Phosgene gas, approximately three times heavier than air, hugs the ground as a white cloud that spills into bunkers and fighting pits.

The choking agents are treacherous in that the onset of fulminating pulmonary edema is delayed several hours following exposure. Thus, a soldier with dyspnea and mild substernal discomfort may have normal auscultatory and radiographic signs and be returned to duty, only to progress to extremis a few hours later.

It is imperative that any casualty with known phosgene exposure, no matter how minor his dyspnea or upper respiratory irritation, be observed for **4-6** hours at the very least, then carefully checked for impending pulmonary edema before being returned to duty (see below).

Diagnosis: Phosgene has the characteristic odor of freshly mown grass. The threshold for detecting phosgene's characteristic odor is so close to the irritant threshold as to be useless as a warning. The individual's sense of dyspnea as the lung stiffens still serves as the best indicator of impending pulmonary edema.

Irritant effects on the eyes' and trachea-bronchial tree—accompanied by tearing, cough, or chest discomfort and dyspnea or tachypnea are the first effects noted. These symptoms may appear to be rather minor at first, resembling the symptoms of a common cold or anxiety reaction. From one-half hour to four or six (rarely twelve) hours following exposure, cough, chest pain, cyanosis, and progressive dyspnea herald impending overt pulmonary edema. Painful cough, frothy sputum, cyanosis, rales, dullness to percussion, and radiographic evidence support the diagnosis.

Uncomplicated cases recover without permanent aftereffects. However, the possibility of secondary bacterial pulmonary infection is considerable. Circulatory failure or the patient appearing "mouse-grey cyanotic" are ominous signs. The "plum-blue cyanotic" patient, conversely, has a good chance to survive. These gross observations may be helpful in mass casualty triage

Treatment: Recall the treacherous "silent period" that follows the inhalation of a pulmonary irritant. Observe the symptom-free patient with known exposure to a choking agent for at least 4-6 hours and the casualty with known exposure and minimal symptoms (itching eyes, runny nose, mild cough, vague chest discomfort) for at least 12 hours. In either case, do not release these personnel to duty without a careful re-examination of the chest.

Treatment of non-cardiac pulmonary edema in the field is mostly supportive, with enforced rest to minimize exertion. Oxygen with air admixed to 46% is sufficient and will help conserve limited supplies. Intravenous fluids (as in any case of pulmonary edema) should be administered sparingly. The risk of bronchopneumonia is greatest when pulmonary edema begins to subside. The clinician must be alert for changes in sputum color or a sudden rise in body temperature.

According to current thinking, (a) prophylactic antibiotics are of no proven benefit, (b) steroids, whether given intravenously or by inhalation, appear to offer no advantage, (c) early positive end-expiratory pressure (PEEP) breathing may well reduce the severity of the subsequent pulmonary edema, and (d) patients in severe distress will require endotracheal intubation or tracheostomy and positive pressure mechanical ventilation.

Phosphorus pentoxide, the dense white smoke associated with white phosphorus munitions, presents the same clinical picture

as phosgene exposure. Experience has shown that the chemical burn of the alveolus produced by phosphorus pentoxide is irreversible and fatal in those who progress to pulmonary edema. It is a sobering and frustrating experience to observe a totally asymptomatic soldier, exposed to phosphorus pentoxide inhalation, progress from the asymptomatic state to intractable pulmonary edema and death over the span of eight hours in spite of every supportive effort.

Blood Agents

Hydrogen cyanide (AC) and certain of its congeners form highly stable complexes with metalloporphyrins such as cytochrome oxidase. Aerobic cellular metabolism comes to a virtual halt. Venous blood remains as oxygen rich as arterial blood, accounting for the "cherry red" postmortem appearance of cyanide victims. Death is due to cytotoxic hypoxia.

High volatility and relatively low toxicity (50-100 times less than those of nerve agents) limits its operational utility in open spaces. Potassium cyanide poisoning of water and food supplies is an old terrorist tactic that one should be aware of.

Diagnosis: The diagnosis of hydrogen cyanide inhalation is difficult to make without a history. Cyanogen chloride (CK) is more readily recognized because its irritant properties cause tearing and coughing in sublethal doses.

Treatment: Immediate removal of casualties from the contaminated atmosphere prevents further inhalation. Nitrites are effective antidotes. They form methemoglobin, to which the cyanide ion binds preferentially; however, overly enthusiastic methemoglobin' conversion reduces available hemoglobin, imperiling intravascular oxygen transport. Sodium nitrite for intravenous use (10 ml of 3% solution) is stocked in forward field medical facilities. (The amyl nitrite "pearl" for inhalation has been removed from issue.) Sodium thiosulfate (50 ml of 25% solution, IV) provides free sulfur to convert toxic cyanide to a far less toxic thiocyanate ion.

Manual or mechanical ventilation is central to resuscitation of apneic casualties. Those in respiratory distress will be aided by oxygen inhalation.

Incapacitation (INCAP) Agents

Incapacitation agents (incaps) are a heterogenous group of chemical agents with potent CNS effects that seriously impair normal function but do not endanger life or cause permanent tissue damage in operationally effective doses. Atropine and scopolamine were early forerunners; other cholinolytics such as benactyzine followed. Quinuclidinyl benzilate (BZ or QND) is a potent glycolate representative of this class. The diagnosis of incap exposure may be extremely difficult to make in isolated instances due to the paucity of distinct diagnostic signs and criteria.

An essential precaution with these confused, perhaps disturbed, casualties is immediate removal of firearms and other weapons to insure the safety of themselves, other patients, and nearby personnel. Be aware that interaction between incaps and pharmacologic agents such as analgesics, antidotes, and anesthetics is probable, but little specific information is available. Caution in their use is advisable.

Belladonna-type drugs: These cholinolytics cause widely dilated pupils, tachycardia, dry mouth, hot dry skin, and decreased intestinal motility and bladder tone. The CNS symptoms and signs run the gamut from inattention, confusion, anxiety, restlessness, and hallucinations on up to delirium.

Recommended treatment is physostigmine, given IM in 2-3 mg doses every 45 minutes. Since the CNS effects of BZ may persist for days, close observation and continued treatment with 3-4 mg physostigmine orally every 1-2 hours are essential elements in managing toxic delirium. Titrate therapeutic dosage against clearing of mental status, should heart rate fall below 70, in which case dosage may be decreased, but physostigmine should not be discontinued. The ability of the body to thermoregulate is damaged by cholinolytics. This is of concern, particularly with personnel in protective clothing. Administering fluids, recording body temperature and urine output, and catheterizing the bladder to relieve distention are key supportive measures.

CNS Depressants. In this group are cannabinoids, barbiturates, and morphine-like compounds that destroy motivation and produce tranquillity and sedation. If treatment of severe indolence is required, CNS stimulants such as the amphetamines have been

effective

CNS Excitants. These agents incapacitate by raising the level of neurotransmitters, causing cerebral hyperstimulation. **Indoles** such as lysergic acid diethylamide (LSD) produce inappropriate behavior, restlessness, fear, perceptual aberrations, and a general schizoid psychosis-like syndrome. In hyperexcitable casualties, sedative barbiturate or chlorpromazine administration has been proposed. Benzodiazepines may be useful, with Valium having the advantage of ready oral absorption.

Mass Casualties in Thermonuclear Warfare

GENERAL

Nuclear weapons range in size from very small (not many times larger in total energy yield than the largest conventional bombs), to immensely large (the so-called thermonuclear or hydrogen devices), with yields in the megaton range. Total energy yield of nuclear weapons are rated in terms of equivalent amounts of TNT. Therefore, a weapon with a **20-kiloton** yield has the same total energy output as 20,000 tons of TNT. A **1.0-megaton** weapon has the energy output of 1,000,000 tons of TNT. Energy is released by nuclear detonations in three forms: thermal radiation, blast, and ionizing radiation. The relative-casualty causing potential of each depends primarily upon three factors: the yield of the weapon, the environmental conditions in which the detonation occurs, and the distribution of troops in the target area. The thermal output may be the most significant casualty producer, particularly for the larger weapons; however, blast will produce nearly as many casualties, and blast and thermal injuries together will account for most of the casualties under almost all circumstances. Radiation, either at the time of detonation or later from fallout, will be responsible for significant numbers of delayed casualties.

Radiation-associated injuries pose many new challenges to medical management. Many organ systems are affected by radiation, often compounding problems produced by conventional injuries. These challenges are magnified by the very real potential of nuclear weapons to produce very large numbers of casualties instantaneously. Thus, new concepts of mass-casualty medicine that utilize simplified and standardized regimens will be required

to accomplish what is now done by labor and resource-intensive means.

LOGISTICS OF CASUALTY MANAGEMENT

If nuclear weapons are employed within the theater, the entire medical evacuation and treatment system will be severely overburdened and some system of classification and sorting of casualties must be added to the normal procedures of evacuation and hospitalization. In addition, a system must be established to hold casualties who are too seriously injured to remain with their units, but who do not need to or cannot be hospitalized. These two requirements, the sorting of casualties and the holding of the excess numbers, must be planned for as part of the normal organization and operation of the medical support system in a theater of operations.

In applying the principle of providing the greatest good for the greatest number to the management of mass casualties, a field medical system must face and solve several problems. The location and number of casualties must be determined. This requires intact communications, since isolated units on a dispersed battlefield could suffer severe casualties and be unable to notify higher headquarters. Subsequent delay in initiating treatment and hospitalization will result in greatly increased morbidity and mortality.

The casualties must be evacuated. In frontline areas, follow-up enemy action exploiting the use of nuclear weapons could greatly hinder or prevent evacuation. In rear areas, adequate evacuation means may not be available to handle the massive number of casualties produced by an attack. The availability of helicopters would help since they can be diverted from one area to another much more readily than ground transportation. The use of nonmedical transportation **systems** may be required but cannot be planned on.

TRIAGE

If nuclear weapons casualties are encountered, the basic principles of mass casualty management (triage, evacuation, and the use of standardized care interventions) will have to be followed. Our relative inexperience in dealing with these types of patients

will make matters worse. Life-threatening doses of acute total body radiation are so infrequently encountered that management policies must be derived in part from different but analogous clinical situations and from studies in experimental animals.

Conventional injuries should be treated first and initial triage should be based on these injuries, since no immediate life-threatening hazard exists for radiation casualties who can ultimately survive. The patient with multiple injuries should be resuscitated and stabilized. During this process, standard preoperative preparation for surgery will accomplish much radioactive decontamination. More definitive evaluation of the radiation injury can be initiated postoperatively.

Three groups of conventional injury patients will have to be considered:

1. Those with minimal injuries that do not incapacitate them completely and are not a significant threat to life. These casualties could continue as at least partially effective soldiers and would not qualify for immediate or early evacuation.

2. Those with severe multiple injuries who obviously are going to require extensive, time-consuming care. These also would be delayed.

3. Finally, those with relatively simple injuries which require immediate surgical treatment. These would get first priority for evacuation.

Further classification of patients will not be required prior to evacuation. The presence or absence of radiation injury, in general, will be ignored in this preliminary sorting, since there are no reliable guidelines to aid in the early diagnosis of extent of radiation injury. Eventually, however, all casualties unable to continue as effective soldiers will have to be evacuated.

As noted, there is a requirement for appropriate holding facilities to which patients who cannot be treated immediately or who require only minimal treatment can be evacuated. These facilities should be set up with limited equipment and staffed with small numbers of medical personnel, and should be part of the expansion plans of all field hospitals regardless of size or location. Holding facilities should be as close to hospitals as possible so as to optimize the availability of appropriate additional care and to allow the transfer of patients as the overall situation and balance between medical resources and patient load change. A great variety of patients, including those not fit for field duty but not

requiring full-care-type hospitalization, as well as the very severely injured, should be kept there. These should include patients in the following categories:

1. Minimal burns.
2. Mild trauma cases.
3. Mild chemical injury cases.
4. Severely injured patients who are not expected to survive and for whom treatment is not immediately available, but for whom supportive measures may be enough to keep them alive until treatment does become available.

Radiation injury introduces many complications into the patient's course. Hematologic injuries cause anemia, infection, bleeding, and delayed wound healing. Performance decrements due largely to neuromediator release can also impact the patient. At higher doses of radiation, dehydration due to severe fluid and electrolyte losses through the intestinal wall will be encountered.

After conventional injuries have been managed, the physician is faced with the problem of triaging the patients according to the severity of their radiation injuries so that appropriate treatment can begin. This problem is difficult since the response of any given individual may vary greatly, and a nonhomogenous exposure of radiation (especially if bone marrow and gut are spared) may result in a markedly decreased effect. U.S. forces do not carry individual personal dosimeters that measure neutron and photon exposures. Finally, dose rate effects can be very profound, especially in a fallout environment. In this situation, tactical dosimeters (two per platoon) may be useful to a commander deciding whether to commit exposed troops to battle, but they are less useful to the health care provider. Other problems will also exist. Casualties will be numerous and resources certainly will be strained. Complicating this will be the occurrence of blast and thermal injuries (in addition to radiation injuries). Improved dosimetry is needed for triage since the goal of military medical personnel should be the appropriate allocation of precious resources to salvage the maximum number of casualties. Improved dosimetry is currently unavailable, but its desirability is currently undergoing evaluation by the U.S. Army Academy of Health Sciences.

Based on recent recommendations, the following guidelines apply to medical personnel operating in austere field conditions. The lymphocyte level can be used as a biological dosimeter to confirm the presence of pure radiation injury, but not in combined

injuries. If the physician has the resources of a clinical laboratory, additional information can be obtained to support the original working diagnosis suggested by the presence of prodromal symptoms. An initial blood sample for concentrations of circulating lymphocytes should be obtained as soon as possible from any patient classified as "radiation injury possible" or "radiation injury probable." After the initial assessment, or at least no later than 24 hours after the event in question, additional comparative blood samples should be taken. The samples may be interpreted as follows:

(1) Lymphocyte levels in excess of **1500/mm³**: There is minimal likelihood of significant dose that would require treatment.

(2) Lymphocyte levels between 500 and **1000/mm³**: These indicate treatment for severe radiation injury. These patients should be hospitalized to minimize the complications from hemorrhage and infection that will present within 2-3 weeks postexposure.

(3) Lymphocyte levels of less than **500/mm³**: These patients have received a radiation dose that may prove fatal. All of these patients need to be hospitalized for the inevitable pancytopenic complications.

(4) Lymphocytes not detectable: These patients have received a supralethal radiation dose. Survival is very unlikely. Most have received severe injuries to their gastrointestinal and cardiovascular systems and will not survive for more than two weeks.

A useful rule of thumb is: If lymphocytes have decreased by 50 % or are less than **1000/mm³**, the individual has received a significant radiation exposure. In the event of combined injuries, the diagnostic use of lymphocytes may be unreliable. It should be borne in mind that those with severe burns or **multisystem** trauma often develop lymphopenia.

It is difficult to establish an early definitive diagnosis. Therefore, it is best to utilize a simple, tentative classification system based on three possible categories of patients as discussed below.

1. Radiation Injury Unlikely. If there are no symptoms associated with radiation injury, patients are judged to be at minimal risk for radiation complications. These patients should be triaged according to the severity of their conventional injuries. If the patients are free of conventional injuries or disease states that require treatment, they should be released and returned to duty.

2. Radiation Injury Probable. Anorexia, nausea, and vomiting are the primary prodromal symptoms associated with radiation injury. Priority for further evaluation will be assigned after all **life-threatening** injuries have been stabilized. Casualties in this category will not require any medical treatment within the first few days for their radiation injuries. Evidence to support the diagnosis of significant radiation injury in the absence of burns and trauma may be obtained from serial lymphocyte assays taken over the next two days. If the evidence indicates that a significant radiation injury was received, these casualties should be monitored for pancytopenic complications.

3. Radiation Injury Severe These casualties are judged to have received a potentially fatal radiation dose Nausea and vomiting will be almost universal for persons in this group. The prodromal phase may also include prompt, explosive bloody diarrhea, significant hypotension, and signs of **neurologic** injury. These patients should be sorted according to the availability of resources. Patients should receive symptomatic care Lymphocyte analysis is necessary to support this classification.

Categorization of these patients into one of these three irradiation categories will be facilitated by an appreciation for the characteristic symptoms induced by radiation. These are:

a. Nausea and Vomiting. Nausea and vomiting occur with increasing frequency as the radiation exceeds 100-200 **centigrays (cGy)**. Their onset may be as late as 6-12 hours postexposure. They usually subside within the first day. The occurrence of vomiting within the first two hours is associated with a severe radiation dose Vomiting within the first hour, especially if accompanied by explosive diarrhea, is associated with doses that frequently prove fatal. Due to the transient nature of these symptoms, it is possible that the patient will have already passed through this initial phase of gastrointestinal distress before being seen by a physician. It will be necessary to inquire about these symptoms at the initial examination.

b. Hyperthermia. Casualties who have received a potentially lethal radiation injury show a significant rise in body temperature within the first few hours postexposure Although our experience is limited, this appears to be a consistent finding. The occurrence

of fever and chills within the first day postexposure is associated with a severe and life-threatening radiation dose. Hyperthermia may occur in patients who receive lower (200 cGy or more) but still serious radiation doses. Present evidence indicates that hyperthermia is frequently overlooked. Individuals wearing a chemical ensemble will normally be hyperthermic; consequently, this may not be a useful sign.

c. Erythema. A person who receives whole-body radiation in excess of 1000-2000 cGy will experience erythema within the first day postexposure. This is also true for those who receive a comparable dose to a local body region in which case the erythema will be restricted to the affected area. With lower but still potentially fatal doses (200 cGy or more), erythema is less frequently seen.

d. Hypotension. A noticeable and sometimes clinically significant decline in systemic blood pressure has been recorded in victims who received a supralethal whole-body radiation dose. A severe hypotensive episode has been observed in one person who had received several thousand rads. In persons who received several hundred rads, a drop in systemic blood pressure of more than 10% has been noted. Severe hypotension after irradiation is associated with a poor prognosis.

e. Neurologic Dysfunction. Experience indicates that almost all persons who demonstrate obvious signs of CNS injury within the first hour postexposure have received a supralethal dose. Symptoms include mental confusion, convulsions, and coma. Intracranial hypotension will probably accompany these symptoms. Despite vascular support, these patients succumb within 48 hours.

Casualties receiving a potentially fatal dose of radiation will most likely experience a pattern of prodromal symptoms that is associated with the radiation exposure itself. Unfortunately, these symptoms are nonspecific and may be seen with other forms of illness or injury, thereby seriously complicating the radiation exposure diagnosis. Therefore, the triage officer must determine if the symptoms occurred within the first day postexposure, evaluate the possibility that they are indeed related to radiation exposure, and then assign the patient to one of the three categories:

“Radiation Injury Unlikely,” “Radiation Injury Probable,” or “Radiation Injury Severe? In the last two categories, the observation of changes in circulating lymphocyte counts may either support or rule out the original working diagnosis. All individuals with multiple injuries should be treated initially as if no significant radiation injury is present. Triage and care of any life-threatening injuries should be rendered without regard to the probability of radiation injury. The medical officer should make a preliminary diagnosis of radiation injury only in those patients for whom radiation is the sole source of the problem. This is based on the appearance of nausea, vomiting, diarrhea, erythema, hyperthermia, hypotension, and neurologic dysfunction.

Decontamination of the Patient. Radiation injury per se does not imply that the patient is a health hazard to the medical staff. Studies indicate that the levels of intrinsic radiation present within the patient from activation (after exposure to neutron and high-energy photon sources) are not life-threatening to the medical staff.

Patients entering a medical treatment facility should be routinely decontaminated if monitoring for radiation is not available. Removal of the patient’s clothing will usually reduce most of the contamination. Washing exposed body surfaces will further reduce this problem. Both of these procedures can be performed in the field or on the way to the treatment facility. Once the patient has entered the treatment facility, care should be based on the obvious injuries. Care for life-threatening injuries should not be delayed until the decontamination procedures are completed.

When radiation safety personnel are available, decontamination procedures will be established to assist in rendering care and to minimize the hazard from radioactive contaminants. A more extensive decontamination procedure is to scrub the areas of persistent contamination with a mild detergent or a diluted strong detergent. Caution should be taken to not disrupt the integrity of the skin while scrubbing, because disruption can lead to incorporation of the radioisotopes into deeper layers of the skin. Contaminated wounds should be treated first, since they will rapidly incorporate the contaminant. Washing, gentle scrubbing, or even debridement may be necessary to reduce the level of contaminants.

Wearing surgical attire will reduce the possible contamination of health personnel. If additional precautions are warranted, rotation of the attending personnel will further reduce the possibility of significant contamination or exposure. The prevention of incorporation is of paramount importance. The inhalation or ingestion of radioactive particles is a much more difficult problem, and resources to deal with it will not be available in a field situation.

SPECIFIC MEDICAL EFFECTS OF NUCLEAR WEAPONS

Proper management of radiation casualties of nuclear war requires an understanding of the medical problems to be expected. Nuclear weapons are sufficiently different in their casualty producing potential from conventional weapons that the types of injuries will be different. It is important to understand these differences if triage and medical treatment are to be accomplished effectively and quickly. As has been said, the biologic effects of nuclear weapons are due to thermal burns, blast, and radiation injuries.

Thermal Burns. The extremely high temperatures produced by a nuclear explosion cause release of a large part of the energy in the form of thermal radiation. This radiation travels at the speed of light and is capable of producing severe burns at great distances. In nuclear warfare, burn casualties will constitute a large fraction of the patient load. All echelons of medical care must plan for this increased burden of thousands of burn cases.

A major problem will occur during the initial evacuation of burn patients if massive numbers of casualties must be handled. Sorting will be essential to conserve medical resources and should be done in accordance with the following criteria:

1. Cases involving 20% or less of the body surface should be treated on an outpatient basis or at minimal care facilities. These patients can care for themselves with minimal supervision. They will not be fit for duty and should not remain with their units if those units are actively engaged in combat.

2. Patients whose burns involve certain critical areas, such as the head and neck, hands, or feet will require hospitalization even if the total body surface involvement is less than 20%.

3. Patients with more than 20% body surface involvement, or

with associated blast injuries, 'will require hospitalization for resuscitative treatment and surgical care.

4. Cases with more than 50% involvement have a decreasing chance of survival with increasing degree of involvement and should be given a low priority for needed surgical care. They should be retained in a delayed status in the minimal care section of a medical facility where they will be available for more extensive treatment if resources and time allow. It must be realized that young healthy adults, without other injury or disease, may be more likely to survive such burns with adequate treatment; thus, a rigid classification system denying them available treatment is not desirable.

All patients should receive as much treatment as possible and the above criteria must be flexible. However, any treatment must be accomplished as efficiently and quickly as possible, and long, time-consuming procedures may have to be delayed, or not performed. The greatest good for the greatest number is best achieved by treating each patient as quickly and simply as possible by doing first what is essential to save his life, then what may be possible to save limbs, and last what might be required to save and restore function.

Depending on the weapon yield, some burn patients will have associated radiation injury and will develop bone marrow depression during the course of their illness. These patients cannot be recognized upon admission, since the bone marrow depression does not become clinically evident until after a latent period of 2-6 weeks after the radiation exposure. A blood count in such cases during the first few days after exposure will show a variable leukopenia, particularly of lymphocytes. These patients will have high morbidity and mortality rates due primarily to infection. Unless a procedure is required to save life, these patients should not be subjected to surgery during the phase of bone marrow depression. If there is no evidence of bone marrow recovery, the patient will not survive with the treatment modalities presently available in the field.

Blast Injuries. Blast injuries caused by nuclear detonations are of two types: direct (due to overpressure effects) or indirect (due to drag forces of the winds accompanying the blast wave). This latter category includes a wide variety of missile and translational injuries.

Direct blast injuries will be rare, since persons close enough to the point of detonation to sustain significant direct overpressures will almost invariably sustain lethal thermal and indirect-blast injuries. However, those few patients who survive the direct blast should be managed the same as any other direct blast injury. Their injuries will be complicated by other trauma and they will suffer a high incidence of radiation-induced bone marrow depression during their post-injury phase, resulting in increased morbidity and mortality. Direct blast-induced internal injuries can easily be overlooked in a mass casualty situation.

The blast wave of a nuclear detonation is unlike conventional blast waves in that its formation is associated with the production of severe, transient winds from the violent movement of large masses of air to form the wave itself. These blast winds, perpendicular to the plane of the wave, have velocities reaching several hundred kilometers per hour. They last only a few seconds but can produce considerable damage through drag forces and by the production of large numbers of low-velocity secondary missiles, the size and nature of which depend on the environment. A high percentage of blast trauma will be caused by such missiles, and a large number of patients will have multiple missile injuries. Many Japanese at Hiroshima and Nagasaki had dozens of superficial wounds caused by flying glass and debris. These types of injuries will vary greatly in severity, but in general, there will be a relatively low incidence of deeply penetrating injuries. However, when massive numbers of casualties must be quickly sorted and prepared for evacuation and treatment, a significant number will have penetrating wounds which might be overlooked until clinical signs become obvious. Otherwise, the nonpenetrating missile injuries will not be severely disabling unless critical parts of the body are involved, such as the head, face, neck, or hands.

Radiation Injuries. The detonation of a nuclear weapon produces large amounts of ionizing radiation in two basic forms: electromagnetic (gamma) radiation, which travels at the speed of light and is highly penetrating, and particulate (alpha, beta, and neutron) radiation. Of the particulate radiations, only the neutron is highly penetrating, whereas the alpha and beta are not. All four types are present at the time of the detonation, but the gamma and neutron are by far the most important clinically. All but the neutron radiation are present in fallout and, in this instance,

the gamma is the most important.

Ionizing radiation is emitted both at the time of the nuclear detonation and for a considerable time afterward. That which is emitted at the time of the detonation is termed "prompt radiation", and is produced by the nuclear reactions of fission and fusion. The significant part of prompt radiation consists of a mixture of gamma and neutron radiation, most of which is emitted within a few seconds of the onset of the detonation. However, the duration of significant emission may be longer, particularly with larger weapons. One minute has been established as a reasonable time parameter, after which there is no significant amount of prompt radiation, regardless of the type of weapon or circumstances of the detonation.

Residual radiation is that which persists beyond the first minute after detonation. Its source is the variable amount of residual radioactive material produced by a nuclear detonation. A nuclear fission reaction transforms uranium or plutonium into a large number (about 150) of radioactive isotopes, termed fission products, which constitute by far the most important source of residual radiation. In addition, small amounts of **unfissioned** bomb material, and material in which neutron radiation has induced radioactivity, are present. All of these residually radioactive materials will be found in fallout.

Fission products are the major radiation hazard in fallout, since a large number of them emit penetrating gamma radiation and, as a result, can be hazardous even at great distances. They have half-lives varying from fractions of seconds to several years, but most have half-lives in the range of days to weeks. As a result, the total amount of radiation emitted by a typical mixture of fission products is quite intense early and remains hazardous until the activity decays to negligible levels. This takes several days to several weeks, depending on the original level of activity; however, some isotopes with very long half-lives will be present and detectable for many years.

Figure 19 shows that fallout activity decays down to 1/110 of its initial level within seven hours post detonation. At H plus one hour a significant part of the early fallout will have deposited itself close to the point of detonation. Deviation from this decay curve will be common, depending on the interplay between the various factors controlling- the rate of deposition of fallout and the distance involved. At greater distances from the point of

detonation, it may take several hours before fallout will be deposited and become detectable. A significant amount of radioactive decay will have already occurred while the radioactive material has been airborne and, as a result, the rate of decay, once all the fallout is on the ground, will be similar to the later part of the curve shown in Figure 19. If fallout in a given area is a mixture from several detonations at different times, the observed rate of decay may be quite different from this ideal example. Under these circumstances, the rule of thumb that fallout will have decayed to negligible values by two weeks may not be applicable. It should be obvious that instruments designed to measure fallout activity must be available and used to evaluate the true hazard.

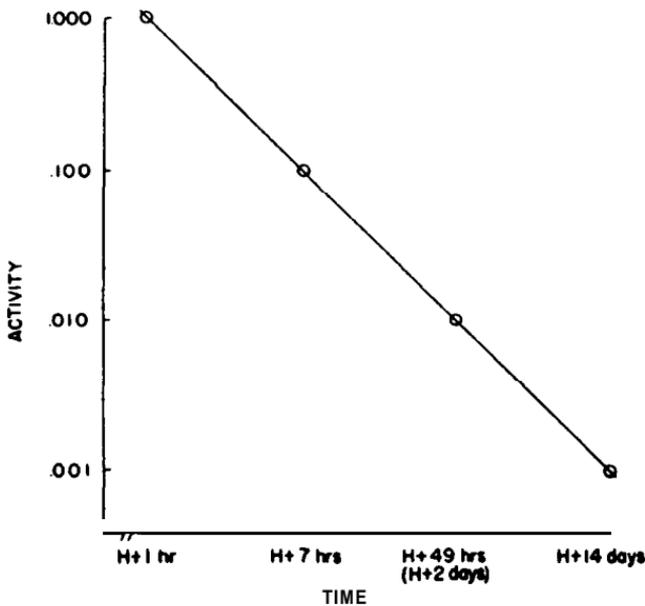


FIGURE 19.—Fallout decay with time after detonation. Fallout activity decreases by a factor of 10 for every sevenfold increase in time after burst. Fallout activity is referenced to 1 hour after burst (H + 1 hour), which is used as the base value in fallout calculations.

Not all the uranium or plutonium in a weapon is fissioned, and fallout, which contains residual weapon material, will contain small amounts of these elements. They add little to the hazards of fallout since they are alpha emitters and are not an external

hazard unless ingested or inhaled. They must be incorporated into body tissue to do damage and their relative insolubility greatly minimizes this hazard. Obviously, ingestion or inhalation of contaminated material should be avoided.

Because of the exceedingly high temperatures generated in a nuclear detonation, all the fission products and the weapon residues are vaporized. As they cool and recondense, they solidify as extremely small particles. In an airburst, these particles will remain suspended in the upper atmosphere (stratosphere) for long periods of time, descending slowly over a period of years and over large parts of the earth's surface. This occurred during the atmospheric testing of weapons. Under such circumstances, there is no significant early or local fallout. When a detonation occurs within a certain critical distance of the surface, however, severe up drafts cause large amounts of terrain debris to be sucked up into the fireball. As a result, as the radioactive materials cool and condense, they become affixed to relatively large particles of dirt and debris. These large particles, with their radioactive contamination attached, tend to fall back to earth rapidly and locally, resulting in high levels of radioactivity downwind from the point of detonation. On occasion, this type of fallout is visible while it descends.

The major hazard in this type of fallout will be external **whole-body** irradiation from gamma-emitting isotopes, since they do not actually have to be on a person's skin to cause damage. Gamma radiation has a very long range in air, and large amounts of gamma-emitting material scattered uniformly over many square kilometers can produce a high level of penetrating radiation, which is a hazard to anyone occupying or passing through the area, even though he avoids direct contact with the fallout material. Even personnel traveling through in vehicles will be exposed, although vehicles can provide significant reduction of exposure because of the ability of most metals at least partially to scatter or absorb gamma radiation. The dose rate inside a tank, for example, may be only **4-10%** of that outside.

The potential injury incurred from gamma radiation is a function of the amount of time spent in the fallout field as well as the dose rate present, since these factors together determine the total dose absorbed.

The beta-emitting isotopes in fallout are not a significant hazard, unless a person is directly contaminated with or ingests them. External contamination can result in a moderate degree of

skin damage somewhat similar to a thermal burn, and incorporation into body tissues can result in organ damage of long-term significance. These later effects—that is, interferences with specific organ functions, carcinogenesis, and accelerated aging changes—are not manifested for months or years, and acute whole body irradiation, with resulting radiation sickness, will not occur. Therefore in combat situations, the beta-emitting components of fallout are not considered to be a serious hazard.

RADIATION SYNDROMES

Radiation sickness caused by whole body irradiation may be lethal within a few days to several weeks, depending upon the dose sustained. Clinically, radiation sickness occurs in a **dose**-dependent pattern of three syndromes, determined by the organ system most seriously involved. These are (1) the neurovascular syndrome, caused by very high doses and uniformly fatal within 2-4 days; (2) **the** gastrointestinal syndrome, due to somewhat lower doses but also uniformly fatal; and (3) the hematopoietic syndrome, caused by still lower doses and associated with the possibility of recovery and survival.

The neurovascular syndrome will be extremely rare in combat. The gastrointestinal syndrome will be relatively uncommon but may be seen. The hematopoietic syndrome will be the most commonly seen.

All three syndromes have certain characteristics in common. These include:

1. An initial nonspecific response.
2. A latent period.
- 3.** A clinical phase

1. Initial Response. Within a few hours after a prompt exposure, all patients, regardless of which syndrome later develops, pass through a nonspecific, transient period of malaise, weakness, anorexia, vomiting, and diarrhea. This response is probably toxic in nature due to tissue, breakdown products associated with radiation-induced cellular damage. The exact mechanism responsible or the cell mass involved is not known. The initial response to irradiation lasts up to a few hours and then subsides. It is followed by a latent period during which there are no significant symptoms or obvious physical signs of radiation injury. At present,

no diagnostic clues are available to establish firmly the presence or extent of radiation injury during the initial response phase. Its severity and duration are not reliable indexes of the degree of radiation exposure and it may be absent following low dose-rate fallout exposures, despite their magnitude.

2. Latent Phase. All three syndromes have latent periods between the initial response and the onset of the clinical phase. This latent period is shortest for the neurovascular syndrome, from an almost negligible period to three days, and longest for the hematopoietic syndrome, lasting 2-6 weeks, with an occasional patient demonstrating an even longer latent period. The gastrointestinal syndrome has an intermediate latent period of a few days. This phase is characterized by a feeling of relative well-being.

3. Clinical Phase. The clinical phase follows the latent period and many patients will not be hospitalized until this time, unless they have had other injuries for which they require treatment. As noted previously, there are three distinct syndromes (Figure 20), depending upon the dose of radiation sustained, as follows:

(a) Neurovascular Syndrome. The dose of radiation required to cause each type of clinical response varies considerably. The neurovascular syndrome requires very high doses (3,000 cGy or more). Such doses are rare in a battlefield situation, except for unprotected personnel exposed to extremely intense fallout very close to the point of surface detonation of a large weapon or in an armored vehicle near the detonation of a small device. Therefore, these patients will be rare and in most cases will usually not survive to be seen in medical facilities because of other lethal injuries.

The clinical course of the neurovascular syndrome is one of progressive depression leading to coma and finally death. In its early stage, patients will be ataxic; convulsions are frequent as the clinical condition deteriorates. This syndrome progresses too rapidly for significant hematologic changes to occur; therefore, diagnosis will not be easy, particularly if patients have sustained head injuries.

(b) Gastrointestinal syndrome. The gastrointestinal syndrome is caused by doses in the range of about 1,000 cGy and higher.

These doses will not be common', but exposure to prompt radiation from small weapons or to intense levels of fallout will result in a small number of such patients. Small numbers of patients with this type of radiation sickness were seen among the victims at Hiroshima and Nagasaki.

A typical patient with this syndrome will have to be hospitalized for other injuries and will, within four to 4-5 days of injury, develop severe, bloody diarrhea. A peripheral blood count will show a depression of lymphocytes and beginning depressions of other leukocytes and platelets. Differentiating between this syndrome and an infectious, nonradiation-induced diarrhea, superimposed upon radiation-induced bone marrow depression, could well be difficult because of the widespread occurrence of various dysenteries in combat. As the bone marrow depression becomes more severe, a point will be reached from which recovery will be impossible. Such patients eventually will succumb to the effects of overwhelming infection and hemorrhage, despite antibiotic therapy and massive fluid, electrolyte, and blood replacement. If patients with gastrointestinal damage are not treated, they will die early due to their massive fluid and blood losses. Replacement therapy can prevent this type of death, but then such patients will progress to the clinical phase of irreparable bone marrow injury. The survival time of such patients will vary, but may be a few weeks. They could constitute a severe burden on all echelons of medical care.

(c) Hematopoietic syndrome. Patients with exposures below levels causing the gastrointestinal syndrome will have longer latent periods before the clinical picture of bone marrow depression becomes evident. This may take from less than two weeks to more than six weeks to develop, but, in most cases, the latent period will be from 2-3 weeks.

The degree of bone marrow depression will vary with the dose of radiation sustained, and the probability of survival is directly related to the probability of recovery of the bone marrow.

The clinical picture presented by patients with bone marrow depression will vary, depending upon the presence and nature of other injuries. In uncomplicated radiation sickness, the clinical picture will reflect the increased bleeding tendencies which develop. These patients will develop extensive hemorrhages throughout their bodies. Subcutaneous petechiae and ecchymoses and extensive gastrointestinal bleeding will be common. De-

creased resistance to infection will accompany the hemorrhagic diathesis, and infection will be the primary cause of death. Treatment will be limited to supportive measures, such as fluids and antibiotics. Bone marrow transplantation is obviously not practical therapy in the field. Transfusion of blood or blood components will become impractical if the number of casualties is too high.

This syndrome is associated with a chance for survival, depending upon the ability of the bone marrow to recover. Bone marrow recovery and an associated favorable prognosis can be determined by serial peripheral blood counts.

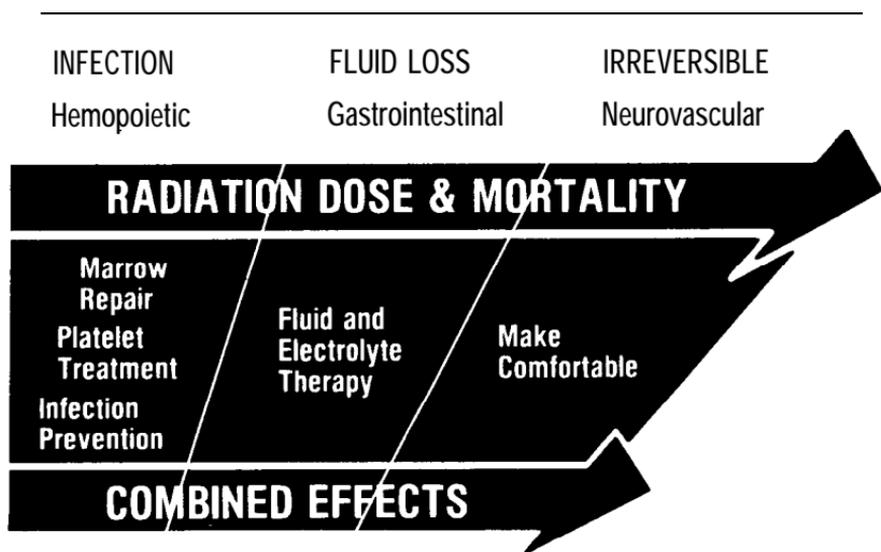


FIGURE 20.—Postradiation syndrome dose and time relationships.

INITIAL TREATMENT FOR PATIENTS WITH WHOLE-BODY RADIATION INJURY

The primary determinants of survival among most patients receiving intermediate (serious but not uniformly fatal if treated) radiation doses are the management of microbial infections and the arrest of bleeding. If high intermediate doses have been received, fluid and electrolyte loss may cause early deaths. If properly resuscitated, however, these patients can survive until the consequences of hematologic failure become apparent.

For those casualties who have received sublethal whole-body

radiation doses, gastrointestinal distress will predominate in the first two days. Antiemetics (metoclopramide, dazopride) may be effective in reducing the symptoms, but currently available drugs have significant side effects. Unless severe radiation injury has occurred, these symptoms will usually subside within the first day. For those patients who continue to experience gastrointestinal distress, parenteral fluids should be considered. If explosive diarrhea occurs within the first hour postexposure, fluids and electrolytes should be administered, if available. For triage purposes, the presence of explosive diarrhea (especially bloody) is likely to be related to a fatal radiation dose.

Cardiovascular support for patients with clinically significant hypotension and **neurologic** dysfunction should be undertaken only when medical resources permit. These patients are not likely to survive injury to the vascular and gastrointestinal systems combined with bone marrow aplasia.

New means of radioprotection and repair of radiation damage are presently on the horizon. Furthermore, immunomodulators are now under study which may not only facilitate marrow regeneration, but also help reduce the profound **immunosuppression** responsible for infections associated with severe injury. These agents may be used in combination with radioprotectors and antibiotics to further enhance survival. Leukopenia is a significant problem in irradiated casualties, but hazards exist with leukocyte transfusion into patients. Induction of stem cell regeneration agents into selected populations probably offers the best opportunity to correct this deficiency. Although platelet transfusions are certainly desirable for radiation victims, they are presently not practical for mass casualty scenarios. Enormous progress is **being** made in autologous bone marrow transplants, but this procedure is not practical in forward facilities. Again, repair by stimulation of surviving stem cells is probably the best near-term hope of solving this problem. Problems of effective wound management and fluid and electrolyte replacement remain to be overcome in the neutropenic patient. Pharmacologic means to regulate performance decrements, such as emesis and early transient incapacitation are still not available for use by military personnel.

DIAGNOSIS AND TREATMENT OF THE PATIENT WITH COMBINED INJURIES

As already noted, radiation injury will be associated with other injuries in a large number of patients, in which event the clinical phase of radiation sickness will come sometime during the course of recovery from the other injuries. With supralethal doses of radiation resulting in the gastrointestinal syndrome, the primary clinical picture of the syndrome will predominate and any lesser effects from radiation on other injuries will be secondary. Following lower doses, however, the bone marrow depression will have significant effects upon the clinical course of certain types of wounds and injuries.

In the event of a nuclear detonation, those patients with burns and traumatic injuries in addition to radiation should be managed on the basis of their conventional injuries. Further reclassification may be warranted on the basis of prodromal symptoms associated with radiation injury. The prognosis for all combined injuries is worse than for radiation injury alone. Animal studies indicate that, when other injuries are accompanied by sublethal doses of radiation, their effects are synergistic: infections are much more difficult to control, and wounds and fractures heal more slowly. Thus, potentially survivable burns and trauma will be fatal in a large percentage of persons who have also received significant injury from sublethal doses of radiation. Because of the delays in wound healing and the subsequent granulocytopenia and thrombocytopenia with injuries from nuclear weapons, most life-saving and **reconstructive** surgery must be performed within 36 hours after the exposure. Then, if possible, no surgery should be performed for the next 6-8 weeks postexposure.

Closed wounds will not be affected greatly, but open wounds, particularly burns, will demonstrate delays in healing. Granulation tissue will disappear and the wounds will become pale. In addition, they will bleed quite easily. Wound infection, caused by both exogenous and endogenous organisms, can become a severe problem. Closed simple fractures will not be markedly affected, although some delay in union may occur. Open fractures, or severe fractures in which infection is a probable complication, are dangerous.

MANAGEMENT OF INFECTION

In spite of antibiotics, infections with opportunistic pathogens are still a major problem. The majority of these organisms today are gram-negative bacteria such as *Escherichia coli* and *Pseudomonas aeruginosa*. These infections occur as a consequence of profound immunosuppression, abnormal colonization of body surfaces, and invasive medical devices. Susceptible body surfaces include the oropharyngeal-respiratory tree and the intestine. Wound sites and artificial invasive devices, such as catheters, are also important sources of infection. Infections may be more prevalent and severe if patients are maintained for long periods in environments containing antibiotic-resistant pathogens.

Wound debridement, appropriate wound dressings, and antibiotics are key elements in infection control. Since infections will be extremely difficult to control in neutropenic subjects, every effort at preventive measures should be made. Antibiotics, preferably in combination therapy, should be used promptly to treat any new fever. When signs or symptoms of infection do appear in the granulocytopenic patient, treatment should be started without waiting for culture and sensitivity studies. Initial coverage should be directed against gram-negative organisms and *Staphylococcus aureus*. Prevalent organisms and antimicrobial susceptibility patterns in the particular medical facility should also be considered. The drugs most often used now for the initial treatment are the synthetic penicillins, such as ticarcillin, combined with an aminoglycoside such as tobramycin. It is recommended either that the treatment continue until the granulocytes return to more than 500, or that the treatment continue for only two weeks and then stop, even if the white cell count is still depressed, as long as all signs of infection have cleared.

Decontamination and Decorporation After External Contamination by Radioactive Materials

The tremendously increased use of radionuclides in medicine, research, Navy nuclear power, and space, in addition to the increased transport of these materials, has increased the likelihood of exposure to military personnel. A nuclear weapon may have its high-explosive detonate, scattering plutonium debris. In addition, a

weapon may be detonated by a 'terrorist, a third-world country, or by a major power in a single strike All of these scenarios may result in radionuclide contamination and traumatic injury one or many casualties. Because of the increased probability of exposure, it is important for military physicians to be trained in decontamination procedures and in the decorporation of radionuclides.

SKIN DECONTAMINATION

Skin contamination with radionuclides is almost never immediately life threatening. As in every other aspect of radiation accident management, the serious medical problems have priority over decontamination. The primary objectives of skin decontamination should be to remove as much radionuclide as possible to reduce the surface dose rate and minimize entry into the body. Decontamination also increases the accuracy of determining incorporated radionuclide burdens by whole-body counting. Zealous decontamination to decrease the percutaneous absorption is to be discouraged. Simple removal of the victim's clothing can remove as much as 70-80% of the contamination. No human exposure to date has represented a significant risk to the personnel giving assistance. Additionally, the principles of time, distance, and shielding can reduce any potential radiation exposure to the attending personnel. Personnel participating in decontamination should wear protective clothing, including surgical gowns, gloves, shoe and head covers, and aprons. Health physics monitoring may suggest the need for additional protective gear. Clothing, personal effects, and biological samples from swabs of the nares, aural canal, and mouth should be placed in plastic bags and glass-stopped tubes with proper identification for later analysis.

The first priority of surface decontamination should be open wounds. Since these areas may allow the rapid incorporation of radionuclides, they should be copiously irrigated with physiological saline for several minutes. If contamination persists, gentle surgical debridement may be necessary. Experiments with plutonium oxides have shown translocation to regional lymph nodes within a few minutes to several hours. After one month, the concentration absorbed is 60% of the implanted dose. For this reason, contaminated wounds must receive first decontamination priority. If the radionuclide is plutonium or other alpha emitters for which DTPA is an effective chelating agent, treatment should

begin immediately. An effective irrigating solution for americium or plutonium contamination is 1 gram calcium DTPA and 10 ml of 2% lidocaine in 100 cc of normal saline. If an extremity is so severely contaminated that it is not possible to decontaminate it adequately, a decision may be required of whether or not to amputate. Amputation should be seriously contemplated only when the extremity injury is so severe that it precludes functional recovery or when the contamination burden is so great that severe radionecrosis will occur. The best conservative advice is still "decontaminate, but do not mutilate."

After contaminated wounds have been treated, other areas can be decontaminated. The eyes, ears, nose, mouth, areas adjacent to uncontaminated wounds, and remaining skin surface should be decontaminated. Gentle, frequent irrigation and suction of the eyes and ears should be sufficient to decontaminate them. Decontamination of the mouth is important because of possible incorporation. The mouth should be irrigated. A nasogastric tube should be inserted and aspirated for analysis. If radionuclides have been ingested, lavage and decorporation therapy should be begun. Decontamination of the skin usually requires only soap and warm water with gentle scrubbing. The use of hot water is contraindicated because of the subsequent vasodilation. If more aggressive decontamination is necessary, a mixture of half cornmeal and Tide (detergent) has been shown to be very effective. Hair can usually be decontaminated with soap and water. If this is inadequate, the scalp should be clipped rather than shaved, to avoid disruption of the skin barrier.

MANAGEMENT OF INTERNAL CONTAMINATION WITH RADIOACTIVE MATERIAL

Radionuclides within the body represent a state of either internal contamination or incorporation. In internal contamination, radionuclides reside in the respiratory and gastrointestinal tracts, and have not crossed the mucous membranes. In incorporation the radionuclide has been transported across mucous membranes, or the radionuclide has been injected or absorbed through the skin or a wound. Once radionuclides are incorporated, they are significantly more difficult to remove; consequently internal contamination must be removed before it is incorporated. The treatment involves reducing the absorption and internal deposition,

and enhancing the excretion of the absorbed radionuclides. A number of important factors must be understood in assessing the hazards and therapy of incorporated radionuclides. These include absorption, excretion, concentration, biologic half-life, and effective half-life. A definitive review of these factors and incorporated radionuclides is provided in Report No. 65 of the National Council on Radiation Protection and Measurements (NCRP). Blocking agents can enhance elimination of the radionuclide or decrease the quantity incorporated. After incorporation, chelating agents or agents that mobilize the radionuclides are much less effective. It should be obvious that the least incorporation will occur with early administration of the proper drug. Chelating agents bind metals into-complexes, thus preventing tissue uptake and allowing urinary excretion. These agents were previously referred to with regard to **CaDTPA** and transuranic incorporation through a skin wound. A handy checklist is provided in the front of NCRP Report No. 65 for guidance in rapidly treating and preventing transuranic incorporation.

Blocking agents are chemicals that saturate a tissue with a nonradioactive element, thereby reducing the uptake of the radionuclide. Dilution of an isotope involves administering large amounts of a stable isotope so that the hazardous radioisotope is diluted. Incorporated radioiodine can be treated by either approach. Radioiodine is an especially important radionuclide because of the increasing number of potential sources of exposure in medicine, nuclear weapons, and nuclear reactors. A power reactor may contain **10-100** million curies of iodine-131. A **loss-of-coolant** accident releasing 1% of the radionuclide under the most adverse weather conditions could give an iodine-131 exposure of **500 cGy (R)** to a child's thyroid at 75 km. Since most of a dose of radioiodine is taken up by the thyroid within several hours, rapid administration is necessary. Early administration is not the only requirement, however, since exposure from a reactor accident will continue for a period of time. Many recommendations have recently been made to prevent the uptake of radioiodine. A recommended protective dose of stable potassium iodide (KI) for a person over 1 year of age is 130 mg per day, while a dose of 65 mg per day is recommended for children under 1 year of age.

A multitude of other radionuclides of potential importance should be encountered only rarely. Extensive guidance for these

can be obtained from NCRP **Reports** No. 65 and 55 and the ***Manual on Early Medical Treatment of Possible Radiation Injury.***

To reiterate, the first several hours after exposure to radionuclides is the best time to prevent uptake, whether by local removal, chelation, physiologic treatment, or limitation of absorption.
