COUNTY OF SAN DIEGO
HHSA
HEALTH AND HUMAN SERVICES AGENCY

ZEBRA PACKET 2010

Biological, Chemical and Radiological Terrorism Information for Clinicians

County of San Diego
Public Health Services
Emergency Medical Services

COUNTY OF SAN DIEGO
HHSA
HEALTH AND HUMAN SERVICES AGENCY

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PUBLIC HEALTH SERVICES
EMERGENCY MEDICAL SERVICES

BIOLOGICAL, CHEMICAL & RADIOLOGICAL TERRORISM INFORMATION FOR CLINICIANS

ZEBRA PACKET

September 2010

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COUNTY OF SAN DIEGO
HEALTH AND HUMAN SERVICES AGENCY
PUBLIC HEALTH SERVICES
EMERGENCY MEDICAL SERVICES

ZEBRA PACKET

Adapted by the County of San Diego Division of Emergency Medical Services from the Santa Clara County Public Health Department of the same title.

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Project Research

COUNTY OF SAN DIEGO
HEALTH AND HUMAN SERVICES AGENCY
PUBLIC HEALTH SERVICES
EMERGENCY MEDICAL SERVICES

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DISEASE REPORTING

County of San Diego, Office of Public Health

Physicians and health care providers must report the following conditions.
Suspected, lab-confirmed, and/or clinical diagnoses are reportable within specified time intervals.
Reporting enables appropriate public health interventions.

PHONE
619-515-6620
or after 5:00 P.M.
858-565-5255

PHONE
619-515-6620
FAX 619-515-6644

IMMEDIATELY:

Anthrax
Botulism
Brucellosis*
Cholera
Dengue
Diphtheria
E-coli O157 Infection
Hantavirus infections
Hemolytic Uremic Syndrome
Measles
Meningococcal Infections
Plague (any form)
Rabies (any form)
Seafood poisoning
- Domoic Acid
- Ciguatera
- Scrombroid
- Paralytic shellfish

Tularemia*
Viral Hemorrhagic Fevers
Yellow Fever
Outbreaks of any disease
Outbreak of Neonatal diarrhea
Unusual disease occurrence

ONE WORKING DAY:

Amebiasis
Anisakiasis
Babesiosis
Campylobacteriosis
Colorado Tick Fever
Cryptosporidiosis
Encephalitis (infectious)
Ehrlichiosis
Haemophilus influenzae (invasive)
Hepatitis A
Listeriosis
Lymphocytic choriomeningitis
Malaria
Meningitis
Neonatal conjunctivitis
Pertussis
Any food-or-water-borne illness
Psittacosis
Poliomyelitis
Q Fever
Relapsing Fever
RMSF
Salmonellosis
Shigellosis
Streptococcal Infections
- Food Handlers and
- Dairy workers only

Syphilis
Swimmer's itch
Trichinosis
Typhoid
Typus Fever
Tuberculosis
Vibrio infections
Yersiniosis

PHONE, FAX, OR MAIL WITHIN ONE WEEK:

AIDS
Aspergillosis
Chancroid
Chlamydial infections
Coccidioidomycosis
Cysticercosis
Echinococcosis
Giardiasis
Gonococcal infections
Hepatitis B,C, D
Hepatitis, other viral
Invasive Group A Streptococcus
Kawasaki's syndrome
Legionellosis
Leprosy
Leptospirosis
Lyme Disease
MRSA
Mumps
NGU
PID
Reye's syndrome
Rheumatic fever, acute
Rubella
Rubella syndrome, congenital
Tetanus
Toxic shock syndrome
Toxoplasmosis
VRE

Monday - Friday 8 AM to 5 PM, call County of San Diego, Office of Public Health
TEL (619) 515-6620 FAX (619)515-6644
1700 Pacific Highway, Room 107, San Diego, CA 92101

*Not reportable immediately in current regulations, however, because Brucellosis and Tularemia may be used as possible bioterrorism agents, immediate reporting is requested if these conditions are suspected.

CONFIDENTIAL MORBIDITY REPORT

NOTE: For STD, Hepatitis, or TB, complete appropriate section below. Special reporting requirements and reportable diseases on back.

DISEASE BEING REPORTED: _____

Patient's Last Name

Social Security Number ___ — ___ — ___

First Name/Middle Name (or initial)

Birth Date Month Day Year

Age

Address: Number, Street

Apt./Unit Number

City/Town

State

ZIP Code

Area Code **Home Telephone**

Gender M F

Pregnant? Y N Unk

Estimated Delivery Date Month Day Year

Area Code **Work Telephone**

Patient's Occupation/Setting

❒ Food service ❒ Day care ❒ Correctional facility
❒ Health care ❒ School ❒ Other _____

Ethnicity (✓ one)

❒ Hispanic/Latino
❒ Non-Hispanic/Non-Latino

Race (✓ one)

❒ African-American/Black
❒ Asian/Pacific Islander (✓ one):
- ❒ Asian-Indian ❒ Japanese
- ❒ Cambodian ❒ Korean
- ❒ Chinese ❒ Laotian
- ❒ Filipino ❒ Samoan
- ❒ Guamanian ❒ Vietnamese
- ❒ Hawaiian
- ❒ Other: _____

❒ Native American/Alaskan Native
❒ White: _____
❒ Other: _____

DATE OF ONSET Month Day Year

DATE DIAGNOSED Month Day Year

DATE OF DEATH Month Day Year

Reporting Health Care Provider

Reporting Health Care Facility

Address

City **State** **ZIP Code**

Telephone Number ( )

Fax ( )

Submitted by

Date Submitted (Month/Day/Year)

REPORT TO

Epidemiology
Fax: (858) 715-6458 ☐
Phone: (619) 515-6620 ☐
Sexually Transmitted Diseases
Fax: (619) 692-8541
Phone: (619) 692-8520 ☐
Tuberculosis
Fax: (619) 692-5516 ☐
Phone: (619) 692-8610 ☐
(Obtain additional forms from your local health department.)

SEXUALLY TRANSMITTED DISEASES (STD)

Syphilis

❒ Primary (lesion present)
❒ Secondary
❒ Early latent < 1 year
❒ Latent (unknown duration)
❒ Late latent > 1 year
❒ Late (tertiary)
❒ Congenital
❒ **Neurosyphilis**

Syphilis Test Results

❒ RPR Titer:_____
❒ VDRL Titer:_____
❒ FTA/MHA: ❒ Pos ❒ Neg
❒ CSF-VDRL: ❒ Pos ❒ Neg
❒ Other:_____

Gonorrhea

❒ Urethral/Cervical
❒ PID
❒ Other: _____

Chlamydia

❒ Urethral/Cervical
❒ PID
❒ Other: _____

❒ **PID (Unknown Etiology)**
❒ **Chancroid**
❒ **Non-Gonococcal Urethritis**

STD TREATMENT INFORMATION

❒ **Treated (Drugs, Dosage, Route):**

_____
_____

Date Treatment Initiated Month Day Year

❒ **Untreated**
❒ Will treat
❒ Unable to contact patient
❒ Refused treatment
❒ Referred to: _____

VIRAL HEPATITIS

		Pos	Neg	Pend	Not Done
❒ **Hep A**	anti-HAV IgM	❒	❒	❒	❒
❒ **Hep B**	HBsAg	❒	❒	❒	❒
❒ **Acute**	anti-HBc	❒	❒	❒	❒
❒ **Chronic**	anti-HBc IgM	❒	❒	❒	❒
	anti-HBs	❒	❒	❒	❒
❒ **Hep C**	anti-HCV	❒	❒	❒	❒
❒ **Acute** ❒ **Chronic**	PCR-HCV	❒	❒	❒	❒
❒ **Hep D (Delta)**	anti-Delta	❒	❒	❒	❒
❒ **Other:** _____		❒	❒	❒	❒

Suspected Exposure Type

❒ Blood transfusion ❒ Other needle exposure ❒ Sexual contact ❒ Household contact
❒ Child care ❒ Other: _____

TUBERCULOSIS (TB)

Status

❒ **Active Disease**
- ❒ Confirmed
- ❒ Suspected

❒ **Infected, No Disease**
- ❒ Convertor
- ❒ Reactor

Site(s)

❒ Pulmonary
❒ Extra-Pulmonary
❒ Both

Mantoux TB Skin Test

Date Performed Month Day Year

Results:_____ mm ❒ Pending ❒ Not Done

Chest X-Ray Month Day Year

Date Performed

❒ Normal ❒ Pending ❒ Not done
❒ Cavitary ❒ Abnormal/Noncavitary

Bacteriology

Date Specimen Collected Month Day Year

Source _____

Smear: ❒ Pos ❒ Neg ❒ Pending ❒ Not done
Culture: ❒ Pos ❒ Neg ❒ Pending ❒ Not done

BCG Vaccine Given? ❒ Yes ❒ No
If yes, at what age/year? _____

Other test(s) _____

TB TREATMENT INFORMATION

❒ **Current Treatment**
❒ INH ❒ RIF ❒ PZA
❒ EMB ❒ Other: _____

Date Treatment Initiated Month Day Year

❒ **Untreated**
❒ Will treat
❒ Unable to contact patient
❒ Refused treatment
❒ Referred to: _____

REMARKS

Title 17, California Code of Regulations (CCR) §2500, §2593, §2641.5-2643.20, and §2800-2812 Reportable Diseases and Conditions*

§ 2500. REPORTING TO THE LOCAL HEALTH AUTHORITY.

- **§ 2500(b)** It shall be the duty of every health care provider, knowing of or in attendance on a case or suspected case of any of the diseases or condition listed below, to report to the local health officer for the juridiction where the patient resides. Where no health care provider is in attendance, any individual having knowledge of a person who is suspected to be suffering from one of the diseases or conditions listed below may make such a report to the local health officer for the jurisdiction where the patient resides.
- **§ 2500(c)** The administrator of each health facility, clinic, or other setting where more than one health care provider may know of a case, a suspected case or an outbreak of disease within the facility shall establish and be responsible for administrative procedures to assure that reports are made to the local officer.
- **§ 2500(a)(14)** "Health care provider" means a physician and surgeon, a veterinarian, a podiatrist, a nurse practitioner, a physician assistant, a registered nurse, a nurse midwife, a school nurse, an infection control practitioner, a medical examiner, a coroner, or a dentist.

URGENCY REPORTING REQUIREMENTS [17 CCR §2500(h)(i)]

✆ ✆ =Report immediately by telephone (designated by a ♦ in regulations).
† =Report immediately by telephone when two or more cases or suspected cases of foodborne disease from separate households are suspected to have the same source of illness (designated by a ● in regulations.)
FAX ✆ ✉ =Report by electronic transmission (including FAX), telephone, or mail within one working day of identification (designated by a + in regulations).
=All other diseases/conditions should be reported by electronic transmission (including FAX), telephone, or mail within seven calendar days of identification

REPORTABLE COMMUNICABLE DISEASES §2500(j)(1)

Acquired Immune Deficiency Syndrome (AIDS)
(HIV infection only: see "Human Immunodeficiency Virus")
FAX ✆ ✉ Amebiasis
Anaplasmosis/Ehrlichiosis
✆ ✆ Anthrax
✆ ✆ Avian Influenza (human)
FAX ✆ ✉ Babesiosis
✆ ✆ Botulism (Infant, Foodborne, Wound)
✆ ✆ Brucellosis
FAX ✆ ✉ Campylobacteriosis
Chancroid
FAX ✆ ✉ Chickenpox (only hospitalizations and deaths)
Chlamydia trachomatis infections, including Lymphogranuloma Venereum (LGV)
✆ ✆ Cholera
✆ ✆ Ciguatera Fish Poisoning
Coccidioidomycosis
FAX ✆ ✉ Colorado Tick Fever
Creutzfeldt-Jakob Disease (CJD) and other Transmissible Spongiform Encephalopathies (TSE)
FAX ✆ ✉ Cryptosporidiosis
Cysticercosis or Taeniasis
✆ ✆ Dengue
✆ ✆ Diphtheria
✆ ✆ Domoic Acid Poisoning (Amnesic Shellfish Poisoning)
FAX ✆ ✉ Encephalitis, Specify Etiology: Viral, Bacterial, Fungal, Parasitic
✆ ✆ *Escherichia coli*: shiga toxin producing (STEC) including *E. coli* O157
† FAX ✆ ✉ Foodborne Disease
Giardiasis
Gonococcal Infections
FAX ✆ ✉ *Haemophilus influenzae* invasive disease (report an incident less than 15 years of age)
✆ ✆ Hantavirus Infections
✆ ✆ Hemolytic Uremic Syndrome
Hepatitis, Viral
FAX ✆ ✉ Hepatitis A
Hepatitis B (specify acute case or chronic)
Hepatitis C (specify acute case or chronic)
Hepatitis D (Delta)
Hepatitis, other, acute
Influenza deaths (report an incident of less than 18 years of age)
Kawasaki Syndrome (Mucocutaneous Lymph Node Syndrome)
Legionellosis
Leprosy (Hansen Disease)
Leptospirosis
FAX ✆ ✉ Listeriosis
Lyme Disease
FAX ✆ ✉ Malaria
FAX ✆ ✉ Measles (Rubeola)
FAX ✆ ✉ Meningitis, Specify Etiology: Viral, Bacterial, Fungal, Parasitic
✆ ✆ Meningococcal Infections
Mumps
✆ ✆ Paralytic Shellfish Poisoning
Pelvic Inflammatory Disease (PID)
FAX ✆ ✉ Pertussis (Whooping Cough)
✆ ✆ Plague, Human or Animal
FAX ✆ ✉ Poliovirus Infection
FAX ✆ ✉ Psittacosis
FAX ✆ ✉ Q Fever
✆ ✆ Rabies, Human or Animal
FAX ✆ ✉ Relapsing Fever
Rheumatic Fever, Acute
Rocky Mountain Spotted Fever
Rubella (German Measles)
Rubella Syndrome, Congenital
FAX ✆ ✉ Salmonellosis (Other than Typhoid Fever)
✆ ✆ Scombroid Fish Poisoning
✆ ✆ Severe Acute Respiratory Syndrome (SARS)
✆ ✆ Shiga toxin (detected in feces)
FAX ✆ ✉ Shigellosis
✆ ✆ Smallpox (Variola)
FAX ✆ ✉ *Staphylococcus aureus* infection (only a case resulting in death or admission to an intensive care unit of a person who has not been hospitalized or had surgery, dialysis, or residency in a long-term care facility in the past year, and did not have an indwelling catheter or percutaneous medical device at the time of culture)
FAX ✆ ✉ Streptococcal Infections (Outbreaks of Any Type and Individual Cases in Food Handlers and Dairy Workers Only)
FAX ✆ ✉ Syphilis
Tetanus
Toxic Shock Syndrome
FAX ✆ ✉ Trichinosis
FAX ✆ ✉ Tuberculosis
✆ ✆ Tularemia
FAX ✆ ✉ Typhoid Fever, Cases and Carriers
Typhus Fever
FAX ✆ ✉ *Vibrio* Infections
✆ ✆ Viral Hemorrhagic Fevers (e.g., Crimean-Congo, Ebola, Lassa, and Marburg viruses)
FAX ✆ ✉ Water-Associated Disease (e.g., Swimmer's Itch or Hot Tub Rash)
FAX ✆ ✉ West Nile Virus (WNV) Infection
✆ ✆ Yellow Fever
FAX ✆ ✉ Yersiniosis
✆ ✆ **OCCURRENCE of ANY UNUSUAL DISEASE**
✆ ✆ OUTBREAKS of ANY DISEASE (Including diseases not listed in §2500). Specify if institutional and/or open community.

HIV REPORTING BY HEALTH CARE PROVIDERS §2641.5-2643.20

Human Immunodeficiency Virus (HIV) infection is reportable by traceable mail or person-to-person transfer within seven calendar days by completion of the HIV/AIDS Case Report form (CDPH 8641A) available from the local health department. For completing HIV-specific reporting requirements, see Title 17, CCR, §2641.5-2643.20 and http://www.cdph.ca.gov/programs/aids/Pages/OAHIVReporting.aspx

REPORTABLE NONCOMMUNICABLE DISEASES AND CONDITIONS §2800–2812 and §2593(b)

Disorders Characterized by Lapses of Consciousness (§2800-2812)
Pesticide-related illness or injury (known or suspected cases)**
Cancer, including benign and borderline brain tumors (except (1) basal and squamous skin cancer unless occurring on genitalia, and (2) carcinoma in-situ and CIN III of the cervix) (§2593)***

LOCALLY REPORTABLE DISEASES (If Applicable):

NOTE:* For diseases that require "immediate" reporting on weekends/holidays, please call (858) 565-5255.

* This form is designed for health care providers to report those diseases mandated by Title 17, California Code of Regulations (CCR). Failure to report is a misdemeanor (Health and Safety Code §120295) and is a citable offense under the Medical Board of California Citation and Fine Program (Title 16, CCR, §1364.10 and 1364.11).

** Failure to report is a citable offense and subject to civil penalty ($250) (Health and Safety Code §105200).
*** The Confidential Physician Cancer Reporting Form may also be used. See Physician Reporting Requirements for Cancer Reporting in CA at: www.ccrcal.org

DETECTING BIOTERRORISM
The Clinician's Role

Health care providers will be "first responders" in the event of a bioterrorism attack or other public health emergency. **Early detection by astute clinicians and rapid reporting to the local health department will be critical** in minimizing the impact of a bioterrorism event or other disaster.

Bioterrorism attacks are likely to present as acute outbreaks of an unusual syndrome, or outbreak of illnesses in the "wrong" season, or geographic area.

If you see patient(s) with any of the following clinical syndromes:

1. Acute severe pneumonia or respiratory distress
2. Unexplained acute encephalopathy or meningitis
3. Acute onset neuromuscular symptoms
4. Otherwise unexplained rash with fever
5. Fever with mucous membrane bleeding
6. Unexplained acute icteric syndromes
7. Massive diarrhea with dehydration and collapse

In the setting of any of the following:

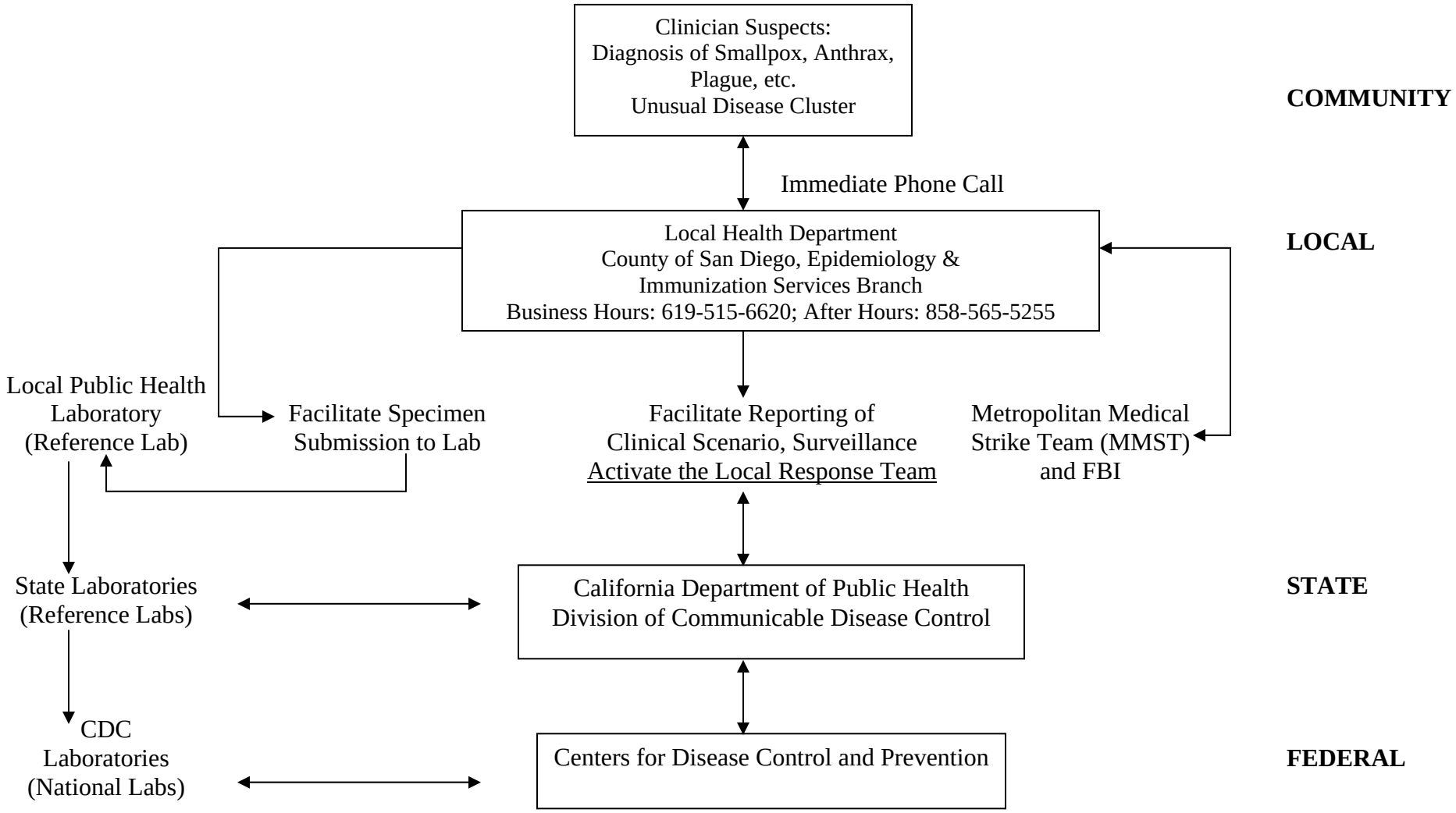
1. Atypical host characteristics:
   - Young (<50 years)
   - Immunologically intact
   - No underlying illness
   - No recent international travel or other exposure to potential source of infection
2. Serious unexpected, acute illness
   - Abrupt onset
   - Prostration
   - Cardiovascular collapse
   - Respiratory distress
   - Obtundation/change in mental status
   - Disseminated intravascular coagulation
3. Multiple similarly presenting cases, especially if
   - Geographically associated, or
   - Closely clustered in time
4. Increases in common syndromes occurring out of season
   - Influenza-like-illness in the summer

Please call the Office of Public Health, Community Epidemiology Division <u>immediately.</u> We would like to hear from you even if you only have <u>some</u> suspicion that something isn't quite right.

During business hours (M-F, 8 am – 5 pm)	(619) 515-6620
After hours, call county communications and ask to speak with the Epidemiologist on call	(858) 565-5255
Public Health Laboratory (specimen submission, routing info) during business hours (M-F, 8 am – 5 pm)	(619) 692-8500

After hours, see instructions above for contacting Epidemiologist on call.

Reporting Suspected Bioterrorism Related Illness



Evaluation for each level is situation dependent.

WHAT CAN CAHAN DO FOR MY ORGANIZATION?

- **Alerting**

  Send alerts to alpha-numeric pager, email, fax, and phone devices.

- **Custom Groups**

  Enroll partners to fit your public health emergency planning and response needs.

- **Directory**

  Find contact information for CAHAN participants.

- **Document Library**

  Securely store and share emergency preparedness materials (call-down lists, drill and exercise documents, and response plans).

- **High Availability**

  Access the secure, web-based system on a 24x7x365 basis via the internet.

HOW CAN I BECOME A CAHAN SAN DIEGO PARTICIPANT?

A CAHAN San Diego application may be submitted at **http://www.cahansandiego.com**. A CAHAN San Diego application must be submitted and approved prior to CAHAN San Diego participation.

The State CAHAN Team is available to conduct demonstrations, presentations, meetings, trainings, and various workshops. Upcoming CAHAN training sessions schedules and enrollment can be accessed at www.cahanworkshops.com.

California Department of Public Health
Emergency Preparedness Office
1615 Capitol Ave, Sacramento, CA 95814

CONTACT INFORMATION
Monday-Friday 8am-5pm
San Diego County e-mail: cahan@sdcounty.ca.gov
County of San Diego Emergency Medical Services
(619) 285-6429
State e-mail: CAHANInfo@cdph.ca.gov

10/09

California Health Alert Network (CAHAN)

San Diego

BEPREPAREDCALIFORNIA

CAHAN California HEALTH ALERT NETWORK

CDPH California Department of PublicHealth

EXAMPLES OF CAHAN SAN DIEGO PARTICIPANTS

- **Public health and safety officials**
- **Physicians (especially those with specialties in infectious disease, emergency medicine and primary care)**
- **Nurses (especially those with specialties in infectious disease, emergency medicine and primary care)**
- **Infection control practitioners**
- **Laboratory directors and managers**
- **Pharmacy directors and managers**
- **Hospital public information officers**
- **Long Term Care Facilities**
- **Community Clinics**
- **Indian Health Clinics**
- **First Responders**
- **School healthcare providers**
- **Others approved by the County of San Diego Health and Human Services Agency**

WHAT IS CAHAN?

The Centers for Disease Control and Prevention (CDC) requires each state to have a Health Alert Network (HAN). The California Department of Public Health's Emergency Preparedness Office implemented the California Health Alert Network (CAHAN) in 2003 as the State of California's official public health emergency alerting system.

CAHAN is a secure web-based information and communications system available on a 24x7x365 basis for distribution of health alerts, dissemination of prevention guidelines, coordination of disease investigation efforts, preparedness planning, and other initiatives that strengthen state and local preparedness.

The Emergency Preparedness Office administers CAHAN to facilitate alerting and collaboration between federal, state, local health departments, clinics, hospitals, long term care facilities, Indian Health clinics, and other public health emergency partners.

CAHAN also provides a central collaborative work environment for public health emergency partners to securely share and store confidential and/or sensitive information.




You can receive CAHAN San Diego alerts via alpha-numeric pager, e-mail, fax, Personal Digital Assistant (PDA), phone (landline and cellular), or directly on the CAHAN portal.

CAHAN
California HEALTH ALERT NETWORK

CAHAN Team
cahaninfo@cdph.ca.gov

Welcome to the California Health Alert Network (CAHAN)

The California Health Alert Network (CAHAN) is the State of California's web-based information and communications system available on a 24/7/365 basis for distribution of health alerts, dissemination of prevention guidelines, coordination of disease investigation efforts, preparedness planning, and other initiatives that strengthen state and local preparedness. CAHAN participants have the ability to receive alerts and notifications via alphanumeric pager, e-mail, fax, and phone (cellular and landline).

CAHAN links critical health and emergency response partners together to provide:

- Rapid and secure communications system among state and local health agencies, health care providers, emergency management officials, and other emergency response partners
- Dissemination of announcements from local, state or federal public health authorities to inform health and medical service personnel of likely or imminent dangers to the health of their community
- Secure collaborative environment to develop and share information for emergency preparedness planning and response

The California Department of Public Health Emergency Preparedness Office provides CAHAN training, Help Desk support, and statewide administration. To request access, training, or assistance, contact the CAHAN Team at cahaninfo@cdph.ca.gov.

Terms of Use

The California Health Alert Network (CAHAN) is for authorized users for official use only. By logging into CAHAN, you hereby acknowledge you are an authorized participant of this network and are using this system solely for the purposes for which it was designed. You agree that you will not use any information obtained from CAHAN for personal use, nor will you disclose such information to parties other than those to whom the information is vital or who have authorized access to the information. **Log off immediately** if you are not authorized to use CAHAN or do not agree to these terms.

https://login.cahan2.ca.gov/GSS_SSO/Login.aspx

QUICKSHEET: ACCESS TO SAN DIEGO CONTENT ON CAHAN

OPTION 1

1. Click on this link https://cahan2.ca.gov/cahan/Documents/2%20-State%20and%20Local%20Health/San%20Diego%2037/
2. Enter CAHAN username and password
3. Navigate to desired folder in San Diego's Document Library

OPTION 2

1. Go to main CAHAN website http://cahan.ca.gov/
2. Enter CAHAN username and password
3. From the CAHAN Gateway Page, find the column **~State and Local Health~**
4. Scroll down and click on *** San Diego ***
5. Navigate to desired folder in San Diego's Document Library

USER PROFILE UPDATE REQUIRED MONTHLY

If it has been longer than 1 month since you lasted logged on to CAHAN, you will be required to update your user profile ***if*** you log in through OPTION 2.

1. Click on "Change My Profile," then "Save"
2. Go to the CAHAN Gateway Page (from the CAHAN Home Portal)
3. Under **~State and Local Health~**, scroll down and click on *** San Diego ***
4. Navigate to the desired folder in San Diego's Document Library

QUESTIONS ABOUT CAHAN?

Contact the CAHAN San Diego Manager at 619-285-6429 or cahan@sdcounty.ca.gov.

Thank you for your participation.

CAHAN San Diego alerts are generally sent by electronic mail. Please be sure your e-mail address is listed correctly. The information you provide is confidential and will not be distributed further. Completing an application does not ensure inclusion in the network. Most applications will be added within 1-2 weeks of submitting this form. Thank you for your interest in CAHAN San Diego.

PLEASE PRINT CLEARLY.

Applicant Profile

First and Last Name: _____

Organization: _____

Job Title: _____

Degrees: _____

Work Contact Information

Work Address: _____

Work City: _____

Work State: _____ Zip: _____

Work E-mail: _____

Work Phone: _____ (format: 999-999-9999)

Work Fax: _____ (format: 999-999-9999)

Home Contact Information (for High Priority Alerts only)

Home City: _____ Zip: _____

Home Phone: _____ (format: 999-999-9999)

Other Emergency Contact Information

Alternate E-mail: _____

Cell Phone: _____ (format: 999-999-9999)

Alpha Pager E-mail: _____
(Example format: 6195552222@archwireless.net)

Alternate Phone: _____ (format: 999-999-9999)

(Please flip over to continue)

Miscellaneous Emergency Information

Languages Spoken (other than English): _____

CPR Certified: _____ yes _____ no

Preferred Order of Contact for High Priority Alerts (e.g. public health emergency)

Location 1: _____ (Example: Cell phone, alpha pager, work phone, home phone, work e-mail)

Location 2: _____

Location 3: _____

Location 4: _____

Location 5: _____

Preferred Order of contact for Medium Priority Alerts (e.g. outbreak, unusual disease)

Location 1: _____ (Example: Cell phone, alpha pager, work phone, work e-mail)

Location 2: _____

Location 3: _____

Preferred Order of Contact for Low Priority Alerts (e.g. disease clusters, surveillance)

Note: Most CAHAN San Diego alerts are low priority and are issued 1-3 times per month via e-mail only

Location 1: _____ (Example: Work e-mail, alternate e-mail)

Location 2: _____

Applicant Security

CAHAN Password (minimum length of 6 characters)
Note: All CAHAN San Diego alerts, including low-priority e-mail alerts, require a unique username and password. Choose a memorable, but not obvious, preferred password. When you're notified via e-mail, if your application has been accepted, you will be assigned an CAHAN username and password at that time.

Preferred Password: _____

4-digit Phone Alert Access Code (maximum length of 4 digits)
Note: Use last 4 digits of your Social Security # to help remember this code. If you are alerted by phone, you will need this 4-digit code to identify yourself as the intended phone alert recipient. If you do not pick up the phone, a generic voice mail message will be left on your phone, and the 4-digit code will no longer enable you to access the alert.

Phone Alert Access Code: _____

Do you agree to maintain the confidentiality of information received via CAHAN?

Agree? _____ Disagree? _____

Date of Application: _____ Signature: _____

Volunteer Questions and Answers:

Q: Is there protection for liability and workers compensation for volunteer health professionals?

A: Volunteers deployed through Disaster Healthcare Volunteers will be registered in their local county as Disaster Service Workers, a program providing these protections.

Q: Do I need to have prior disaster experience?

A: No! All volunteers are welcome.

Q: I'm retired. Can I still volunteer?

A: Yes! Just be sure to indicate your license status when you register.

Q: What other issues should I consider?

A: Care for your family if you respond. Emergency response can be physically and emotionally difficult; personal medical conditions may need to be evaluated. You may have work or other commitments that would prevent you from responding to an activation. Missions may be up to ten days in duration.

WWW.HEALTHCAREVOLUNTEERS.CA.GOV

Who Should Register?

- Audiologists and Audiology Aides
- Certified Nurse Assistants
- Chiropractors
- Clinical Laboratory Scientists
- Medical Laboratory Technologists
- Clinical Nurse Specialists
- Cytotechnologists
- Dentists
- Diagnostic Radiologic Technologists
- EMT-Is and EMT-Paramedics
- Hemodialysis Technicians
- Home Health Aides
- Licensed Clinical Social Workers
- Licensed Midwives
- Licensed Vocational Nurses
- Marriage and Family Therapists
- Nuclear Medicine Technologists
- Nurse Anesthetists
- Nurse Midwives
- Nurse Midwife Furnishers
- Nurse Practitioner Furnishers
- Nurse Practitioners
- Occupational Therapists
- Occupational Therapy Assistants
- Optometrists
- Osteopathic Physicians and Surgeons
- Pharmacists
- Pharmacy Technicians
- Phlebotomists
- Physical Therapists
- Physical Therapist Assistants
- Physicians and Surgeons
- Physician Assistants
- Podiatrists
- Psychiatric Mental Health Nurses
- Psychiatric Technicians
- Psychologists
- Public Health Microbiologists
- Public Health Nurses
- Registered Associate Social Workers
- Registered Dental Assistants
- Registered Dental Hygienists
- Registered Nurses
- Registered Veterinary Technicians
- Respiratory Care Practitioners
- Speech-Language Pathologists
- Speech-Language Pathology Aides
- Veterinarians

Managed by the California Emergency Medical Services Authority, in partnership with the California Department of Public Health. Funds are provided by the United States Department of Health and Human Services.

California Emergency Medical Services Authority
1930 9th Street • Sacramento, CA 95811
Phone: 916-322-4336 • Fax: 916-323-4898
Email: healthcarevolunteers@emsa.ca.gov
Web: http://www.emsa.ca.gov

WHEN DISASTER STRIKES, YOU CAN MAKE THE DIFFERENCE

REGISTER TODAY

WWW.HEALTHCAREVOLUNTEERS.CA.GOV

EARTHQUAKES | FLOODS | TERRORISM | FIRES | CATASTROPHES

Who are "Disaster Healthcare Volunteers?"

Disaster Healthcare Volunteers are professionals like you who want to volunteer during an emergency or disaster. When you register on our secure web-based registry, you will indicate your volunteer preferences and enter information about your skills. The registry will automatically notify you in the case of a disaster and track your deployment.

What role will I have in a large-scale disaster or emergency?

Your role will be to practice your profession or skill as either an individual called up at the time of a disaster, or as part of an organized response team. Volunteers may participate in several ways, including:

- As an individual called upon during extreme emergencies for your county
- As part of a community-based Medical Reserve Corps;
- As a member of a State of California Medical Assistance Team.

Every attempt will be made to match your skills, competencies and license or registration level with your responsibilities during a disaster. However, there might be situations in which you will be asked to assist with activities that are less challenging than your normal work duties.

How do I register?

Visit the Disaster Healthcare Volunteers' site at: **WWW.HEALTHCAREVOLUNTEERS.CA.GOV,** click the "Register Now" button and you're on your way!

For local information or questions, please contact:

San Diego Medical Reserve Corps
County of San Diego, Emergency Medical Services
Phone: 619-285-6429 | Fax: 619-285-6531
mrcvolcoord@sdcounty.ca.gov • sandiegomrc.org

How does the "Disaster Healthcare Volunteers" program work?

Once you have registered to become a Disaster Healthcare Volunteer, your professional license will be verified electronically with your licensing board by the Emergency Medical Services Authority. This information will become a part of the secure Disaster Healthcare Volunteer Statewide Registry.

During a disaster, state or local (county) officials will determine what kind of health professionals are needed, search the database for available volunteers, and send an alert to selected members via e-mail, telephone and pager.

If you receive an alert in the event of a disaster, you will have the chance to accept or decline the volunteer request. If you accept, you will receive specific instructions on where and when to report, and what is needed for the incident. There is no obligation to participate during an activation.

Why register now, before a disaster?

Registering now allows verification of your license and credentials, promotes training opportunities, and helps disaster managers understand how many volunteers might be available. This will help us match your skills with the needs required in each emergency situation.

Registering now makes it easier to help when disaster strikes!

IN TIMES OF DISASTER, CALIFORNIA NEEDS YOU!

BECOME A DISASTER HEALTHCARE VOLUNTEER

REGISTER TODAY AT WWW.HEALTHCAREVOLUNTEERS.CA.GOV

REGISTERING IS EASY!

Visit the Disaster Healthcare Volunteers' site at: WWW.HEALTHCAREVOLUNTEERS.CA.GOV, click the "Register Now" button and you're on your way!

BECOME A DISASTER HEALTHCARE VOLUNTEER

REGISTER TODAY AT WWW.HEALTHCAREVOLUNTEERS.CA.GOV

Who Should Register?

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- Licensed Clinical Social Workers
- Licensed Midwives
- Licensed Vocational Nurses
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- Nuclear Medicine Technologists
- Nurse Anesthetists
- Nurse Midwives
- Nurse Midwife Furnishers
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- Physical Therapists
- Physical Therapist Assistants
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- Psychologists
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- Public Health Nurses
- Registered Associate Social Workers
- Registered Dental Assistants
- Registered Dental Hygienists
- Registered Nurses
- Registered Veterinary Technicians
- Respiratory Care Practitioners
- Speech-Language Pathologists
- Speech-Language Pathology Aides
- Veterinarians

REGISTER TODAY

WWW.HEALTHCAREVOLUNTEERS.CA.GOV

Your Healthcare Expertise Makes the Difference.

All healthcare professionals are needed to volunteer for public service in the event of a significant disaster or a public health emergency. You can make a difference during times of disaster by helping people, animals and your community.

To become a San Diego Medical Reserve Corps Volunteer...

medical reserve corps
san diego county

1. Go to https://www.healthcarevolunteers.ca.gov to register with Disaster Healthcare Volunteers of California
   a. Select San Diego > MRC

2. After completing your online registration, contact us to set up an appointment for a 30-minute Orientation.

Melissa Dredge
MRC Volunteer Coordinator
Melissa.Dredge@sdcounty.ca.gov
(619)-641-5015

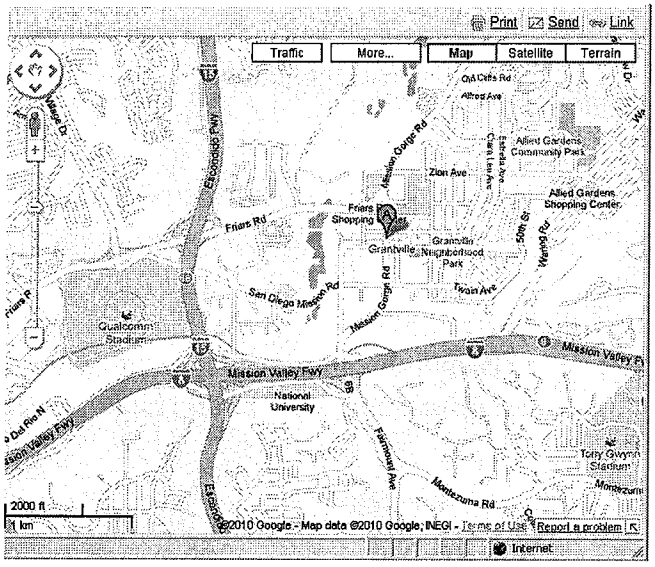
Flexible Scheduling for Orientation is Available

On your scheduled orientation day, please bring with you the following:

1.) Resume including two professional references
2.) Driver's License or Government-issued Identification Card
3.) Your Current Professional License
4.) Any certificates of special training, if applicable.

Orientation will be held at the following address:

County of San Diego: Emergency Medical Services
6255 Mission Gorge Road
San Diego, CA
92120-3599



Orientation and Post-Orientation Check-list…

medical reserve corps
san diego county

1. Orientation: ☐
   a.) Fill out Registration Form for MRC ☐
   b.) Sign Disaster Service Worker Oath ☐
   c.) Take Badge Photo ☐
   d.) Receive MRC Scrubs ☐

2. Go to Ready San Diego: ☐
   http://www.sdcounty.ca.gov/oes/ready/signup.html
   a.) Register your cell phone with **Alert San Diego** ☐
   b.) Organize your personal and family preparedness plan by clicking on the green **"Family"** tab at the top of the page ☐
   c.) Encourage your family, friends, and co-workers to register with Alert San Diego as well! ☐

3. Take FEMA courses: http://training.fema.gov/IS/crslist.asp ☐
   a.) IS-100 Introduction to the Incident Command System ☐
   b.) IS-700 National Management System (NIMS), An Introduction ☐
   c.) Email or fax your certifications of completion to Melissa Dredge ☐
   Email: Melissa.Dredge@sdcounty.ca.gov
   Fax: (619)-285-6560

4. Register with CAHAN San Diego: ☐
   http://www.sdcounty.ca.gov/hhsa/programs/phs/cahan_san_diego/
   CAHAN San Diego is a communications system for the Health and Human Services Agency, Public Health Services, hospitals, clinics, emergency rooms, laboratories, law enforcement, fire service, EMS, volunteer and other health agencies.

5. Keep your contact information and profile current at: ☐
   https://www.healthcarevolunteers.ca.gov.

6. Participate!
   in communication drills, monthly trainings, community disaster exercises, and local public health emergency responses.

Ten Critical Steps for Handling Possible Bioterrorism Events

California Department of Public Health

1. Maintain an Index of Suspicion

In an otherwise healthy population, some associations are very suggestive, especially when seen in clusters, high numbers or unusual presentations.

- Hemoptysis: Plague
- Flaccid Paralysis: Botulism
- Purpura: Viral Hemorrhagic Fevers (VHF)
- Wide Mediastinum: Anthrax
- Centripetal* Rash: Smallpox

*Rash more abundant on face and extremities

2. Protect Yourself and Your Patients

Use appropriate personal protection equipment (PPE). For **smallpox**, triage and evaluate patient in an isolation room; wear an appropriate respirator (N-95 or higher).

3. Adequately Assess the Patient

Review and assess the patient's history. Also, ask:

- Are others ill?
- Has the patient been traveling?
- Were there any unusual events?
- What is the patient's immunization record?
- Was there a possible contaminated food item?
- What is the patient's occupation?
- Was there vector exposure?

Perform a physical examination with special attention to the respiratory system, nervous system, skin condition and hematologic and vascular status.

4. Decontaminate as Appropriate

Do not use bleach on exposed people. Soap, water and shampoo are perfectly adequate for all biological and most chemical agents. Chemically contaminated clothes should be removed and discarded safely. Biologically contaminated clothes can be laundered with soap, water and, perhaps, bleach.

5. Establish a Diagnosis

Think clinically and epidemiologically; always send specimens for culture.

Symptom (individuals)	**Possible Diagnosis**
Pulmonary (SEB)	Anthrax, tularemia, plague, staph enterotoxin B
Neuromuscular	Botulism, Venezuelan equine encephalitis (VEE)
Bleeding/purpura	VHF, ricin, plague (late)
Rash (various types)	VHF, T2 mycotoxin, smallpox, plague
Flu-like symptoms	Varies

Immediate Symptoms (large numbers)	**Possible Diagnosis**
Pulmonary	SEB, mustard, Lewisite, phosgene, cyanide
Neurologic	Nerve gases, cyanide

Delayed Symptoms (large numbers)	**Possible Diagnosis**
Pulmonary	Biologic agents, mustard, phosgene
Neurologic	Botulism, VEE, other encephalitis

6. Render Prompt Treatment

- Doxycycline can be used to treat virtually everything (except virals or toxins) while awaiting lab results.
- Inhalational anthrax should be treated with two or more antibiotics, including doxycycline or ciprofloxacin plus one or more other antibiotics. Observe pediatric precautions as appropriate.
- Prophylaxis (antibiotics and/or vaccines) should be administered according to public health recommendations.

7. Provide Good Infection Control

Recommended isolation precautions (in addition to standard precautions) for biologic agents include:

- **Anthrax:** Contact precautions for cutaneous anthrax
- **Pneumonic Plague:** Droplet precautions; contact precautions if draining buboes present
- **Smallpox:** Airborne and contact precautions
- **Tularemia:** Contact precautions if lesions present
- **Viral Hemorrhagic Fevers:** Contact precautions; airborne precautions especially in late stages

8. Alert the Proper Authorities

Agency	**Telephone Number**
FBI	858-565-1255 (San Diego)
Municipal Police/County Sheriff	_____
California State Police	_____
County Health Department	_____
California State Health Department	916-445-4171
Local Emergency Medical Services Unit	_____
Local Hospitals	_____

Centers for Disease Control 770-488-7100

9. Assist in Epidemiologic Investigations so as to Determine Who May be at Risk

Steps in an epidemiologic investigation:

- Count cases
- Relate to the at-risk population
- Make comparisons
- Develop hypotheses
- Test hypotheses
- Make inferences
- Conduct studies
- Interpret and evaluate

10. Know and Spread This Information

Information adapted from the New York State Department of Health.

http://bepreparedcalifornia.ca.gov/EPO/Partners/HealthcareProviders/ForHealthcareProviders/PracticeGuidelinesFactSheets/BioterrorismPreparedness/Ten+Critical+Steps+for+Handling+Possible+Bioterrorist+Events.htm

Isolation Guidelines

Patient Management
Negative Pressure Rooms are:

IMPORTANT PHONE NUMBERS:
Infectious Diseases _____-_____
Infection Control _____-_____
ER _____-_____
USAMRIID 301-619-2833,
CDC Emergency Responce Office 770-488-7100

	BACTERIAL AGENTS	Anthrax	Brucellosis	Cholera	Glanders	Bubonic Plague	Pneumonic Plague	Tularemia	Q Fever	VIRUSES	Smallpox	Venez. Equine Encephalitis	Viral Encephalitis	Viral Hemorrhagic Fever	BIOLOGICAL TOXINS	Botulism	Ricin	T-2 Mycotoxins	Staph. Enterotoxin B
Isolation Precautions																			
Standard Precautions for all aspects of patient care		X	X	X	X	X	X	X	X		X	X	X	X		X	X	X	X
Contact Precautions (gown & gloves; wash hands after each pt encounter)				X***	X*	X*					X			X				X*	
Airborne Precautions (negative pressure room & N95 masks for all individuals entering the room)											X			X**					
Droplet Precautions (surgical mask)							X							X**					
Patient Placement																			
No restrictions		X	X	X	X			X	X			X	X			X	X	X	X
Cohort 'like' patients when private room unavailable				X***	X*	X	X				X			X				X*	
Private Room				X***	X*	X*	X				X			X				X*	
Negative Pressure											X			X**					
Door closed at all times											X			X**					
Patient Transport																			
No restrictions		X	X	X	X	X		X	X			X	X			X	X	X	X
Limit movement to essential medical purposes only				X***	X*	X*	X				X			X				X*	
Place mask on patient to minimize dispersal of droplets							X				X			X**					
Cleaning and Disinfection																			
Routine cleaning of room with hospital approved disinfectant		X	X	X	X	X	X	X	X		X	X	X			X	X	X	X
Disinfect surfaces with 10% bleach solution or phenolic disinfectant														X					
Dedicated equipment (disinfect prior to leaving room)				X***	X*	X*					X			X				X*	
Linen management as with all other patients		X	X	X	X	X	X	X	X			X	X	X		X	X	X	X
Linens autoclaved before laundering in hot water with bleach added											X								
Post-mortem Care																			
Follow principles of Standard Precautions		X	X	X	X	X	X	X	X		X	X	X	X		X	X	X	X
Droplet Precautions (surgical mask)							X												
Contact Precautions (gown & gloves)					X*	X*					X			X				X*	
Avoid autopsy or use Airborne Precautions & HEPA filter							X				X			X**					
Routine terminal cleaning of room with hospital approved disinfectant		X	X	X	X	X	X	X	X		X	X	X			X	X	X	X
Disinfect surfaces with 10% bleach solution or phenolic disinfectant														X					
Minimal handling of body; seal body in leaf-proof material														X					
Cremete body whenever possible											X								
Discontinuation of Isolation																			
48 hrs of appropriate antibiotic and clinical improvement							X												
Until all scabs separate											X								
Until skin decontamination completed (1 hr contact time)																		X	
Duration of illness				X***	X*	X*								X					

STANDARD PRECAUTIONS - Standard Precautions prevent direct contact with all body fluids (including blood), secretions, excretions, non-intact skin (including rashes) and mucous membranes. Standard Precautions routinely practiced by healthcare providers include: splash/spray, and gowns to protect skin and clothing during procedures.

*Contact precautions needed only if the patient has skin involvement (bubonic plague: draining bubo) or until decontamination of skin is complete (T2 Mycotoxins).

**A surgical mask and eye protection should be worn if you come within 3 feet of pt. Airborne precautions are needed if patient has cough, vomiting, diarrhea or hemorrhage.

*** Contact precautions needed only if the patient is diapered or incontinent

Designed by LTC Suzanne E. Johnson, RN, MSN, CIC, Walter Reed Army Medical Center; Revised by Center for the Study of Bioterrorism and Emerging Infections
11/01

Bioterrorism Overview

What is Bioterrorism?

A bioterrorism attack is the deliberate release of viruses, bacteria, or other germs (agents) used to cause illness or death in people, animals, or plants. These agents are typically found in nature, but it is possible that they could be changed to increase their ability to cause disease, make them resistant to current medicines, or to increase their ability to be spread into the environment. Biological agents can be spread through the air, through water, or in food. Terrorists may use biological agents because they can be extremely difficult to detect and do not cause illness for several hours to several days. Some bioterrorism agents, like the smallpox virus, can be spread from person to person and some, like anthrax, can not. For information on which bioterrorism agents can be spread from person to person, please see the alphabetical list of bioterrorism agents at www.bt.cdc.gov/agent/agentlist.asp.

Bioterrorism Agent Categories

Bioterrorism agents can be separated into three categories, depending on how easily they can be spread and the severity of illness or death they cause. Category A agents are considered the highest risk and Category C agents are those that are considered emerging threats for disease.

Category A

These high-priority agents include organisms or toxins that pose the highest risk to the public and national security because:

- They can be easily spread or transmitted from person to person
- They result in high death rates and have the potential for major public health impact
- They might cause public panic and social disruption
- They require special action for public health preparedness.

Category B

- These agents are the second highest priority because:
- They are moderately easy to spread
- They result in moderate illness rates and low death rates
- They require specific enhancements of CDC's laboratory capacity and enhanced disease monitoring.

Category C

These third highest priority agents include emerging pathogens that could be engineered for mass spread in the future because:

- They are easily available
- They are easily produced and spread
- They have potential for high morbidity and mortality rates and major health impact.

DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION
SAFER • HEALTHIER • PEOPLE™

Bioterrorism Agents by Name

You can look for the bioterrorism agent by name at www.bt.cdc.gov/agent/agentlist.asp.

What You Can Do to Prepare for Bioterrorism

The CDC and the American Red Cross have teamed up to answer questions and provide advice on steps you can take to prepare yourself and your loved ones in the event of a bioterrorist attack. For preparedness information and guidelines, please see "Preparedness Today: What You Need to Do" at http://www.redcross.org/preparedness.

The Department of Homeland Security has established a website to provide information to the public about emergencies and emergency preparedness. For information on what to do in the event of a bioterrorist attack, please see Ready.gov.

More Resources

- Department of Homeland Security – Bioterrorism Information and Preparedness
  www.ready.gov/america/biological.html
- Department of Homeland Security – National Response Plan
  www.dhs.gov/dhspublic/interapp/editorial/editorial_0566.xml
- American Red Cross – Terrorism Preparedness
  www.redcross.org/services/disaster/0,1082,0_589_,00.html
- The American Medical Association's – Bioterrorism: Frequently Asked Questions
  www.ama-assn.org/ama/pub/category/6667.html
- The Food and Drug Administration – Drug Preparedness and Response to Bioterrorism (information on antibiotics and dosage)
  www.fda.gov/cder/drugprepare
- Environmental Protection Agency – Water Security
  http://cfpub.epa.gov/safewater/watersecurity
- National Library of Medicine/National Institutes of Health Medline Plus – Biodefense and Bioterrorism
  www.nlm.nih.gov/medlineplus/biodefenseandbioterrorism.html

Category A Bioterrorism Agents

Definition

The U.S. public health system and primary healthcare providers must be prepared to address various biological agents, including pathogens that are rarely seen in the United States. High-priority agents include organisms that pose a risk to national security because they

- can be easily disseminated or transmitted from person to person;
- result in high mortality rates and have the potential for major public health impact;
- might cause public panic and social disruption; and
- require special action for public health preparedness.

Agents/Diseases

- Anthrax (*Bacillus anthracis*)
- Botulism (*Clostridium botulinum* toxin)
- Plague (*Yersinia pestis*)
- Smallpox (variola major)
- Tularemia (*Francisella tularensis*)
- Viral hemorrhagic fevers

  (filoviruses [e.g., Ebola, Marburg] and arenaviruses [e.g., Lassa, Machupo])

For the most accurate and up-to-date information, including resources on the specific agents, please refer to the CDC web site at: http://www.cdc.gov/

ANTHRAX

ALL SUSPECT CASES OF ANTHRAX MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY EPIDEMIOLOGY PROGRAM

During Business Hours: 619-515-6620
After Hours (County Communications): 858-565-5255

Epidemiology:

- A spore forming gram-positive rod, which can cause disease by inhalation, inoculation, or ingestion of spores, which, upon reversion to regular bacterial forms, produce potent "edema" and "lethal" toxins (inhalation is the most likely during a bioterrorist attack).
- The spore form of anthrax is highly resistant to physical and chemical agents; spores can persist in the environment for years.
- **Anthrax is <u>not</u> transmitted from person to person.**

Clinical:

- Incubation period is:
  - 2-60 days following pulmonary exposure
  - 1-7 days following either cutaneous exposure or ingestion
- The pneumonic or inhalational form of exposure usually starts with fever, myalgias, cough and fatigue, which after a brief improvement, progresses to an abrupt respiratory distress and shock. There are no specific physical findings, but the chest x-ray may show a widened mediastinum, with or without effusion, but mostly without infiltrates, because the disease is primarily a mediastinitis. Fifty percent have meningitis.
- Biphasic illness, with initial phase characterized by nonspecific flu-like illness followed by acute phase characterized by acute respiratory distress and toxemia (sepsis).
- Chest x-ray findings: **Mediastinal widening in a previously healthy patient in the absence of trauma is pathognomonic for anthrax.**
- Mortality rate for inhalation anthrax approaches 90%, even with treatment. Shock and death occur within 24-36 hours.
- Cutaneous Anthrax may appear in conjunction with inhalation cases. Local tissue destruction results in the formation of a black eschar or ulcer with (+/-severe) surrounding edema. Some develop septicemia.
- Gastrointestinal anthrax occurs when large numbers of spores are ingested. It may present with nausea and vomiting, abdominal pain, bloody diarrhea +/- ascites, which progresses to an acute abdomen. The toxins destroy the mesenteric lymph nodes and the circulation to the small bowel.

Laboratory:

- Laboratory specimens should be handled in a Biosafety Level 2 facility (most clinical microbiology laboratories).
- Gram stain shows gram positive bacilli, occurring singly or in short chains, often with squared off ends (safety pin appearance). In advanced disease, a gram stain of unspun blood may be positive.
- Distinguishing characteristics on culture include: non-hemolytic, non-motile, capsulated bacteria that are susceptible to gamma phage lysis.
- Culture & identification of isolates and PCR testing of clinical samples are available at the San Diego County Public Health Lab.
- ELISA and PCR tests are also available at national reference laboratories.

Patient Isolation:

Standard barrier isolation precautions including routine use of gloves for contact with nonintact skin, including rashes and skin lesions. Patients do <u>not</u> require isolation rooms.

Treatment:

- Prompt initiation of antibiotic therapy is essential. Antibiotic susceptibility testing is key to guiding treatment.
- Ciprofloxin (400 mg IV q 12h) is the antibiotic of choice for penicillin-resistant anthrax or for empiric therapy while awaiting susceptibility results.
- All patients should be treated with anthrax vaccine if available; antibiotic treatment should be continued until 3 doses of vaccine have been administered (day 0, 14 and 28). If vaccine is unavailable, antibiotic treatment should be continued for 8 weeks.

Prophylaxis:

- If vaccine is available, all exposed persons (as determined by local and state health depts.) should be vaccinated with 3 doses of anthrax vaccine (days 0, 14 and 28).
- Start antibiotic prophylaxis immediately after exposure with ciprofloxin (500 mg po q 12 hrs.) or doxycycline (100 mg po q 12 hrs). If strain is penicillin-susceptible, therapy can be modified to penicillin or amoxicillin.
- Antibiotic prophylaxis should be continued until 3 doses of vaccine have been administered; if vaccine is unavailable, antibiotics should be continued for 8 weeks.

FACT SHEET

Facts about Anthrax

Anthrax is a serious disease caused by ***Bacillus anthracis***, a bacterium that forms spores. A bacterium is a very small organism made up of one cell. Many bacteria can cause disease. A spore is a cell that is dormant (asleep) but may come to life with the right conditions.

There are three types of anthrax:

- Skin (cutaneous)
- Lungs (inhalation)
- Digestive (gastrointestinal)

How Do You Get It?

Anthrax is not known to spread from one person to another.

Anthrax from animals. Humans can become infected with anthrax by handling products from infected animals or by breathing in anthrax spores from infected animal products (like wool, for example). People also can become infected with gastrointestinal anthrax by eating undercooked meat from infected animals.

Anthrax as a weapon. Anthrax also can be used as a weapon. This happened in the United States in 2001. Anthrax was deliberately spread through the postal system by sending letters with powder containing anthrax. This caused 22 cases of anthrax infection.

Symptoms

The symptoms (warning signs) of anthrax are different depending on the type of the disease:

- Cutaneous: The first symptom is a small sore that develops into a blister. The blister then develops into a skin ulcer with a black area in the center. The sore, blister and ulcer do not hurt.
- Gastrointestinal: The first symptoms are nausea, loss of appetite, bloody diarrhea, and fever, followed by bad stomach pain.
- Inhalation: The first symptoms of inhalation anthrax are like cold or flu symptoms and can include a sore throat, mild fever and muscle aches. Later symptoms include cough, chest discomfort, shortness of breath, tiredness and muscle aches. (Caution: Do not assume that just because a person has cold or flu symptoms that they have inhalation anthrax.)

How Dangerous Is Anthrax?

The Centers for Disease Control and Prevention classifies agents with recognized bioterrorism potential into three priority areas (A, B and C). Anthrax is classified as a Category A agent. Category A agents are those that:

- pose the greatest possible threat for a bad effect on public health
- may spread across a large area or need public awareness
- need a great deal of planning to protect the public's health

(Continued on back side)

In most cases, early treatment with antibiotics can cure cutaneous anthrax. Even if untreated, 80 percent of people who become infected with cutaneous anthrax do not die. Gastrointestinal anthrax is more serious because between one-fourth and more than half of cases lead to death. Inhalation anthrax is much more severe. In 2001, about half of the cases of inhalation anthrax ended in death.

How Soon Do Infected People Get Sick?

Symptoms can appear within 7 days of coming in contact with the bacterium for all three types of anthrax. For inhalation anthrax, symptoms can appear within a week or can take up to 42 days to appear.

How Is Anthrax Treated?

Antibiotics are used to treat all three types of anthrax. Early identification and treatment are important.

Prevention after exposure. Treatment is different for a person who is exposed to anthrax, but is not yet sick. Health-care providers will use antibiotics (such as ciprofloxacin, levofloxacin, doxycycline, or penicillin) combined with the anthrax vaccine to prevent anthrax infection.

Treatment after infection. Treatment is usually a 60-day course of antibiotics. Success depends on the type of anthrax and how soon treatment begins.

Can Anthrax Be Prevented?

Vaccination. There is a vaccine to prevent anthrax, but it is not yet available for the general public. Anyone who may be exposed to anthrax, including certain members of the U.S. armed forces, laboratory workers, and workers who may enter or re-enter contaminated areas, may get the vaccine. Also, in the event of an attack using anthrax as a weapon, people exposed would get the vaccine.

What Should I Do if I Think I Have Anthrax?

If you are showing symptoms of anthrax infection, call your health-care provider right away.

What Should I Do if I Think I Have Been Exposed to Anthrax?

Contact local law enforcement immediately if you think that you may have been exposed to anthrax. This includes being exposed to a suspicious package or envelope that contains powder.

For more information on anthrax, call 619-515-6620 or visit the Centers for Disease Control and Prevention's (CDC) website at www.bt.cdc.gov or call the CDC public response hotline at (888) 246-2675 (English), (888) 246-2857 (Español), or (866) 874-2646 (TTY). The above information has been adapted from the CDC fact sheet "Anthrax: What You Need to Know."

County of San Diego
Health and Human Services Agency
Public Health Services
P.O. Box 85222
San Diego, CA 92186-5222

FACT SHEET

Anthrax: What You Need To Know

What Is Anthrax?

Anthrax is a serious disease caused by ***Bacillus anthracis***, a bacterium that forms spores. A bacterium is a very small organism made up of one cell. Many bacteria can cause disease. A spore is a cell that is dormant (asleep) but may come to life with the right conditions.

There are three types of anthrax:

- **skin (cutaneous)**
- **lungs (inhalation)**
- **digestive (gastrointestinal)**

How Do You Get It?

Anthrax is not known to spread from one person to another.

Anthrax from animals. Humans can become infected with anthrax by handling products from infected animals or by breathing in anthrax spores from infected animal products (like wool, for example). People also can become infected with gastrointestinal anthrax by eating undercooked meat from infected animals.

Anthrax as a weapon. Anthrax also can be used as a weapon. This happened in the United States in 2001. Anthrax was deliberately spread through the postal system by sending letters with powder containing anthrax. This caused 22 cases of anthrax infection.

How Dangerous Is Anthrax?

The Centers for Disease Control and Prevention classifies agents with recognized bioterrorism potential into three priority areas (A, B and C). Anthrax is classified as a Category A agent. Category A agents are those that:

- pose the greatest possible threat for a bad effect on public health
- may spread across a large area or need public awareness
- need a great deal of planning to protect the public's health

In most cases, early treatment with antibiotics can cure cutaneous anthrax. Even if untreated, 80 percent of people who become infected with cutaneous anthrax do not die. Gastrointestinal anthrax is more serious because between one-fourth and more than half of cases lead to death. Inhalation anthrax is much more severe. In 2001, about half of the cases of inhalation anthrax ended in death.

What Are the Symptoms?

The symptoms (warning signs) of anthrax are different depending on the type of the disease:

- Cutaneous: The first symptom is a small sore that develops into a blister. The blister then develops into a skin ulcer with a black area in the center. The sore, blister and ulcer do not hurt.
- Gastrointestinal: The first symptoms are nausea, loss of appetite, bloody diarrhea, and fever, followed by bad stomach pain.

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- Inhalation: The first symptoms of inhalation anthrax are like cold or flu symptoms and can include a sore throat, mild fever and muscle aches. Later symptoms include cough, chest discomfort, shortness of breath, tiredness and muscle aches. (Caution: Do not assume that just because a person has cold or flu symptoms that they have inhalation anthrax.)

How Soon Do Infected People Get Sick?

Symptoms can appear within 7 days of coming in contact with the bacterium for all three types of anthrax. For inhalation anthrax, symptoms can appear within a week or can take up to 42 days to appear.

How Is Anthrax Treated?

Antibiotics are used to treat all three types of anthrax. Early identification and treatment are important.

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What Should I Do if I Think I Have Been Exposed to Anthrax?

Contact local law enforcement immediately if you think that you may have been exposed to anthrax. This includes being exposed to a suspicious package or envelope that contains powder.

What Is CDC Doing To Prepare For a Possible Anthrax Attack?

CDC is working with state and local health authorities to prepare for an anthrax attack. Activities include:

- Developing plans and procedures to respond to an attack using anthrax.
- Training and equipping emergency response teams to help state and local governments control infection, gather samples, and perform tests. Educating health-care providers, media, and the general public about what to do in the event of an attack.
- Working closely with health departments, veterinarians, and laboratories to watch for suspected cases of anthrax. Developing a national electronic database to track potential cases of anthrax.
- Ensuring that there are enough safe laboratories for quickly testing of suspected anthrax cases.
- Working with hospitals, laboratories, emergency response teams, and health-care providers to make sure they have the supplies they need in case of an attack.

For more information, visit www.bt.cdc.gov/agent/anthrax,
or call CDC at 800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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Patient Information:
Amoxicillin 500-mg *Oral Capsules (Pills)*
Amoxicillin *Oral Suspension*

Take this medicine as prescribed.

Amoxicillin belongs to a class of drugs called penicillin antibiotics. It has been approved by the Food and Drug Administration (FDA) to treat people with infections caused by certain types of bacteria. Amoxicillin has not been approved by the FDA to use when treating people who have been exposed to anthrax. However, if test results show that the anthrax bacteria can be killed by penicillin antibiotics, the use of amoxicillin is recommended to prevent the development of anthrax disease in people who have been exposed to anthrax, **when other antibiotics are not as safe to use such as with children and pregnant women**.

How to take amoxicillin

ADULTS: Take one pill three times a day.
CHILDREN: A child's dose depends on body weight. Give the medicine to your child as directed by the doctor.

Take amoxicillin with a large glass of water. This medicine can be taken with or without food. Taking with food may decrease the chance that upset stomach will occur.

If you miss a dose, start again taking 1 pill three times a day. Do not take 2 pills to make up for the missed dose. *Finish all your pills, even if you feel okay, unless your doctor tells you to stop. If you stop taking this medicine too soon, you may become ill.*

Side effects

Common side effects of amoxicillin include an upset stomach, vomiting, and diarrhea. If you have problems with any of these symptoms, tell your doctor.

Allergic reactions are rare. Signs of an allergic reaction include rash, itching, swelling of the tongue, hands or feet, fever, or trouble breathing. If any of these symptoms occur, call your doctor right away.

Precautions

- Be sure to tell your doctor if you are allergic to any medicine.
- It is very important to tell the doctor the names of **ALL** medicines that you are currently taking—even pills bought at the store such as vitamins and antacids.
- Tell your doctor if you have asthma, which is a breathing problem, or any other illnesses.
- Birth control pills may not work as well when taking this medication. Be sure to use condoms or another form of birth control until you have finished the entire course of treatment.
- Amoxicillin is safe to take when you are pregnant but be sure your doctor knows if you are pregnant.
- In women, amoxicillin can cause vaginal itching and discharge commonly known as a yeast infection. Tell your doctor if this happens.

For more information, visit www.bt.cdc.gov/agent/anthrax,
or call CDC at 800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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Patient Information:
Ciprofloxacin 500-mg *Oral Tablet*
Ciprofloxacin *Oral Suspension*

Take this medicine as prescribed.

Ciprofloxacin, commonly known as cipro, belongs to a class of drugs called quinolone antibiotics. It has been approved by the Food and Drug Administration (FDA) to treat and protect people who have been exposed to anthrax spores.

How to take cipro

ADULTS: Take 1 tablet every 12 hours as directed.
CHILDREN: A child's dose depends on body weight. Give the medicine to your child as directed by the doctor.

It is best to take cipro 2 hours before or after a meal with at least one large glass of water. However, if an upset stomach occurs, cipro may be taken with food. Avoid dairy products such as milk and yogurt for at least 3 hours before and after taking the medicine. If you take vitamins or antacids such as Tums or Maalox, take them 6 hours before or 2 hours after taking cipro.

If you miss a dose, start again taking one tablet every 12 hours. Do not take 2 pills to make up for the missed dose. *Finish all your pills, even if you feel okay, unless your doctor tells you to stop. If you stop taking this medicine too soon, you may become ill.*

Side effects

Common side effects of cipro include an upset stomach, vomiting, diarrhea, fatigue, dizziness or headache. If you have problems with any of these symptoms, tell your doctor. Less common side effects include pain in arms or legs, changes in vision, restlessness, ringing in the ears, or mental changes. If any of these symptoms occur, call your doctor right away.

Severe allergic reactions are very rare. Signs of an allergic reaction include rash, itching, swelling of the tongue, hands or feet, fever, or trouble breathing. If any of these symptoms occur, call your doctor right away.

SPECIAL NOTE FOR CHILDREN: *This medicine may cause joint problems in infants and children under 18 years of age. If your child has any joint pain while he/she is taking cipro, tell your doctor.*

Precautions

- Be sure to tell the doctor if you are allergic to any medicine
- It is very important to tell your doctor about **ALL** of the medicine you are currently taking even pills that were bought at the store such as vitamins and antacids.
- Tell the doctor if you have ever had a seizure, stroke, or problems with your kidneys, joints or tendons, liver, or vision. Report any history of unusual bleeding or bruising.
- If this drug makes you dizzy, use caution driving or doing tasks that require you to be alert. Avoid alcohol in this case as it will make the dizziness worse.
- Cipro can make skin very sensitive to the sun which increases the chance of getting severe

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sunburn. Avoid the sun as much as possible. When outside, wear a long sleeve shirt and hat and always apply sunscreen (30 SPF)

- In women, cipro can cause vaginal itching and discharge commonly known as a yeast infection. Tell your doctor if this happens.
- If you are pregnant or breastfeeding, tell your doctor.
- Safety of taking cipro during pregnancy is unknown. If you are pregnant or could become pregnant, tell your doctor. Also, if you are breastfeeding, tell your doctor.
- Cipro can increase the effects of caffeine and theophylline (a medicine).

Patient Information:
Doxycycline 100-mg *Oral Tablet*
Doxycycline *Oral Suspension*

Take this medicine as prescribed.

Doxycycline belongs to a class of drugs called tetracycline antibiotics. It is approved by the Food and Drug Administration (FDA) to treat and protect people who have been exposed to anthrax spores.

How to take doxycycline

ADULTS: Take 1 tablet every 12 hours as directed.
CHILDREN: A child's dose depends on body weight. Give the medicine to your child as directed by the doctor.

Take doxycycline with food and least one full glass of water. Avoid taking antacids (like Tums or Maalox), cholestyramine (Questran), colestipol (Colestid), dairy products (like milk or yogurt) or vitamins 3 hours before or after taking doxycycline.

If you miss a dose, start again taking 1 pill every 12 hours. Do not take 2 pills to make up for the missed dose. *Finish all your pills, even if you feel okay, unless your doctor tells you to stop. If you stop this medication too soon, you may become ill.*

Side effects

Common side effects of doxycycline include an upset stomach, vomiting, or diarrhea. If you have problems with any of these symptoms, tell your doctor. Less common side effects include dark urine, yellowing of the eyes or skin, sore throat, fever, unusual bleeding or bruising, fatigue, white patches in the mouth. If any of these symptoms occur, call your doctor right away.

Allergic reactions are rare. Signs of an allergic reaction are rash, itching, swelling of the tongue, hands or feet, fever, and trouble breathing. If any of these symptoms occur, call you doctor right away.

SPECIAL NOTE FOR CHILDREN: *This medicine may cause staining of the teeth in children younger than 8 years old. This means that their teeth can become grayish in color and this color does not go away. This medicine can also cause bone growth delay in premature infants but this side effect goes away after the medicine is finished.*

SPECIAL NOTE FOR PREGNANT WOMEN: *There is little data about side effects from the use of this drug during pregnancy. If the mother of an unborn baby takes doxycycline, staining of baby teeth or poor bone development can result. There is a remote chance of severe liver disease in some pregnant women.*

Precautions

- Be sure to tell the doctor if you are allergic to any medicine.
- It is very important to tell the doctor the names of ALL medicines that you are currently taking even pills bought at the store such as vitamins and antacids.
- Doxycycline can make skin very sensitive to the sun which increases the chance of getting severe

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sunburn. Avoid the sun as much as possible. When outside, wear a long sleeve shirt and hat and always apply sunscreen (30 SPF).

- Birth control pills may not work as well when taking this medication. Be sure to use condoms or another form of birth control until you are finished the entire course of treatment. If you are pregnant or breastfeeding, tell your doctor.
- In women, doxycycline can cause vaginal itching and discharge commonly known as a yeast infection. Tell your doctor if this happens.
- Tell the doctor if you have ever had problems with your liver or kidneys, or if you have frequent heartburn.

Fact Sheet: Anthrax Information for Health Care Providers

Cause	*Bacillus anthracis* • Encapsulated, aerobic, gram-positive, spore-forming, rod-shaped (bacillus) bacterium
Systems Affected	• Skin or cutaneous (most common) • Respiratory tract or inhalation (rare) • Gastrointestinal (GI) tract (rare) • Oropharyngeal form (least common)
Transmission	• Skin: direct skin contact with spores; in nature, contact with infected animals or animal products (usually related to occupational exposure) • Respiratory tract: inhalation of aerosolized spores • GI: consumption of undercooked or raw meat products or dairy products from infected animals • NO person-to-person transmission of inhalation or GI anthrax
Reporting	• Report suspected or confirmed anthrax cases immediately to your local or state department of health.

Cutaneous Anthrax

Incubation Period	• Usually an immediate response up to 1 day
Typical Signs/Symptoms	• Local skin involvement after direct contact with spores or bacilli • Localized itching followed by 1) papular lesion that turns vesicular and 2) subsequent development of black eschar within 7–10 days of initial lesion
Treatment **(See "Cutaneous Anthrax Treatment Protocol" for specific therapy*)**	• Obtain specimens for culture BEFORE initiating antimicrobial therapy. • Do NOT use extended-spectrum cephalosporins or trimethoprim/sulfamethoxazole because anthrax may be resistant to these drugs.
Precautions	• Standard contact precautions. Avoid direct contact with wound or wound drainage.

* http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5042a1.htm

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Inhalation Anthrax

Incubation Period	• Usually <1 week; may be prolonged for weeks (up to 2 months)	
Typical Signs/Symptoms **(often biphasic, but symptoms may progress rapidly)**	**Initial phase** • Non-specific symptoms such as low-grade fever, nonproductive cough, malaise, fatigue, myalgias, profound sweats, chest discomfort (upper respiratory tract symptoms are rare) • Maybe rhonchi on exam, otherwise normal • Chest X-ray: o mediastinal widening o pleural effusion (often) o infiltrates (rare)	**Subsequent phase** • 1–5 days after onset of initial symptoms • May be preceded by 1–3 days of improvement • Abrupt onset of high fever and severe respiratory distress (dyspnea, stridor, cyanosis) • Shock, death within 24–36 hours
Laboratory	• Coordinate all aspects of testing, packaging, and transporting with public health laboratory/Laboratory Response Network (LRN). • Obtain specimens appropriate to system affected: o blood (essential) o pleural fluid o cerebral spinal fluid (CSF) o skin lesion	**Clues to diagnosis** • Gram-positive bacilli on unspun peripheral blood smear or CSF • Aerobic blood culture growth of large, gram-positive bacilli provides preliminary identification of *Bacillus* species.
Treatment **(See "Inhalational Anthrax Treatment Protocol"* for specific therapy)**	• Obtain specimens for culture BEFORE initiating antimicrobial therapy. • Initiate antimicrobial therapy immediately upon suspicion. • Do NOT use extended-spectrum cephalosporins or trimethoprim/sulfamethoxazole because anthrax may be resistant to these drugs. • Supportive care including controlling pleural effusions	
Precautions	• Standard contact precautions	

* http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5042a1.htm

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Gastrointestinal Anthrax

Incubation Period	• Usually 1–7 days	
Typical Signs/Symptoms	**Initial phase** • Nausea, anorexia, vomiting, and fever progressing to severe abdominal pain, hematemesis, and diarrhea that is almost always bloody • Acute abdomen picture with rebound tenderness may develop. • Mesenteric adenopathy on computed tomography (CT) scan likely. Mediastinal widening on chest X-ray has been reported.	**Subsequent phase** • 2–4 days after onset of symptoms, ascites develops as abdominal pain decreases. • Shock, death within 2–5 days of onset
Laboratory	• Coordinate all aspects of testing, packaging, and transporting with public health laboratory/LRN. • Obtain specimens appropriate to system affected: o blood (essential) o ascitic fluid	**Clues to diagnosis** • Gram-positive bacilli on unspun peripheral blood smear or ascitic fluid • Pharyngeal swab for pharyngeal form • Aerobic blood culture growth of large, gram-positive bacilli provides preliminary identification of *Bacillus* species.
Treatment **(See "Inhalational Anthrax Treatment Protocol"* for specific therapy)**	• Obtain specimens for culture BEFORE initiating antimicrobial therapy. • Early (during initial phase) antimicrobial therapy is critical. • Do **NOT** use extended-spectrum cephalosporins or trimethoprim/sulfamethoxazole because anthrax may be resistant to these drugs.	
Precautions	• Standard precautions	

* http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5042a1.htm

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Oropharyngeal Anthrax

Incubation Period	• Usually 1–7 days	
Typical Signs/Symptoms	**Initial phase** • Fever and marked unilateral or bilateral neck swelling caused by regional lymphadenopathy • Severe throat pain and dysphagia • Ulcers at the base of the tongue, initially edematous and hyperemic	**Subsequent phase** • Ulcers may progress to necrosis • Swelling can be severe enough to compromise the airway
Laboratory	• Coordinate all aspects of testing, packaging, and transporting with public health laboratory/LRN. • Obtain specimens appropriate to system affected: o blood (essential) o throat	**Clues to diagnosis** • Aerobic blood culture growth of large, gram-positive bacilli provides preliminary identification of *Bacillus* species.
Treatment **(See "Inhalational Anthrax Treatment Protocol"* for specific therapy)**	• Obtain specimens for culture BEFORE initiating antimicrobial therapy. • Do **NOT** use extended-spectrum cephalosporins or trimethoprim/sulfamethoxazole because anthrax may be resistant to these drugs. • Supportive care including controlling ascites	
Precautions	• Standard contact precautions	

* http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5042a1.htm

Anthrax Bioterrorism: Lessons Learned and Future Directions

James M. Hughes* and Julie Louise Gerberding*

James M. Hughes

Julie L. Gerberding

On September 11, 2001, the United States experienced the worst terrorist attack in its history. As the nation sought to deal with this tragedy, it would face a second wave of terrorism—this time, in the form of a biological attack. The suspicion of anthrax in a patient by an astute infectious disease clinician along with capable clinical and public health laboratory staff in Florida would lead to the discovery that *Bacillus anthracis* spores had been intentionally distributed through the postal system, causing 22 cases of anthrax, including 5 deaths, and forever changing the realm of public health.

In this issue of Emerging Infectious Diseases, numerous individuals involved in the public health aspect of the anthrax investigation document their experiences. Articles describe the epidemiologic and laboratory investigations, applied research findings, environmental assessment and remediation experiences, workplace safety issues, prophylaxis and clinical care information, international aspects, and collaborations between law enforcement and public health officials. The articles also highlight the widespread efforts made to identify the source of exposure and prevent illness among those exposed. While many of the individuals involved in this effort are acknowledged in these articles, many others are not, including the large numbers of medical, public health, law enforcement, and emergency response personnel throughout the country and the world who dealt with the numerous hoaxes perpetrated in the weeks following the attack. We recognize and thank them for their heroic efforts.

This issue also provides an opportunity to review the valuable lessons we have learned from these experiences. Foremost among them is the knowledge that we cannot afford to be complacent. Throughout the Department of Health and Human Services (DHHS) as well as across other federal, state, and local agencies, we remain alert for the first evidence of a disease outbreak. Multiple systems are now in place, both in the United States and internationally, to detect initial cases. On the local level, clinicians and laboratorians play a key role in this process. Activities such as monitoring emergency room visits, pharmacy requests, calls to emergency response and poison control centers, and animal disease registries for unusual occurrences are also expanding.

These lessons have also led us at the Centers for Disease Control and Prevention (CDC) to change the way we operate. Changes have been made within our programs, among our staff and partners, and in our coordination with other federal agencies. Many of these changes have been based on valuable input provided by public and private sector experts during numerous consultations. Terrorism response capacity is being integrated into existing infrastructures, further strengthening the foundation of public health.

The anthrax cases highlighted the importance of the "golden triangle" of response between clinicians and clinical microbiologists, the health-care delivery system, and public health officials. Steps have been taken to strengthen these and other critical linkages, including those between professionals in the human, veterinary, and public health communities and between the public health, law enforcement, and emergency response systems.

DHHS has made available through CDC more than $918 million for state and local health departments to enhance their terrorism preparedness programs. These funds are intended to strengthen capacity to respond to bioterrorism, other infectious disease emergencies, and other urgent public health threats. Existing programs that proved invaluable during the events of last fall, such as the Laboratory Response Network for Bioterrorism (LRN) and the National Pharmaceutical Stockpile (NPS), both described in this issue in the article by Perkins et al., have also been strengthened. During the anthrax attacks, laboratories within the LRN tested more than 125,000 clinical specimens and approximately 1 million environmental specimens. The number of these specialty laboratories participating in this network has now increased to more than 100, with at least one in each state, enabling widespread testing for microbes that might be used in a terrorist attack to cause illnesses such as anthrax, tularemia, plague, and botulism. New facilities have been opened, and improvements in others are in progress or planned for the near future. The NPS has also been

*Centers for Disease Control and Prevention

expanded to include additional medical supplies and personnel. State and local agencies are implementing measures to ensure the successful transport and delivery of these critical components of effective response.

CDC has established rapid response teams composed of individuals with expertise in field operations, epidemiology, microbiology, data management, and communications. These individuals have received training to enable immediate deployment to affected areas to assist state and local efforts. The Epidemic Intelligence Service (EIS), CDC's long-standing disease investigation training program for epidemiologists, is also undergoing changes. In addition to traditional training for rapid response to disease outbreaks, this year's class of officers, the largest in the program's 51-year history, is receiving specialized field training to respond to terrorist attacks that might involve the intentional release of toxic chemicals or spread of infectious agents.

While the terrorist attacks experienced by the United States have enabled us to better prepare for, recognize, and respond to future attacks, more work needs to be done. The anthrax attack was relatively small and did not involve the use of multiple agents, multiple modes of transmission, a drug-resistant organism, transmission to animals, or global spread. The surge capacity of the health-care delivery system was not challenged. In addition, unlike some of the other threat agents, the causative organism was easily isolated in clinical laboratories; there was no risk of person-to-person transmission and no risk of vector-borne transmission.

Planning and practice are essential to ensure an effective response to urgent public health threats. CDC has activated its emergency operations center in response to the recent outbreak of West Nile virus. During 2002, through mid-September, West Nile virus has been identified in more than 40 states and the District of Columbia and has caused more than 1,700 human cases, including more than 80 deaths. Although West Nile virus is a naturally occurring disease, because of its recent arrival in the United States many physicians are unfamiliar with the signs and symptoms suggestive of infection. As part of this response, we have provided professional education to health-care workers, evaluated the quality of laboratory processing of suspected samples, and streamlined communication—all critical components for responding to this outbreak and for identifying ways to improve our capabilities for addressing future emergencies.

Integral to planning is education. Health-care workers, particularly physicians and nurses, need training about the clinical aspects of diseases that may result from the use of biological agents. As has been evident in many recent investigations (e.g., hantavirus pulmonary syndrome, West Nile virus meningitis/encephalitis, anthrax), alert and knowledgeable clinicians and laboratorians are vital to disease surveillance efforts and recognition of new diseases and syndromes. Education of the public regarding the signs and symptoms of diseases associated with infectious agents is also essential. CDC will continue to work with partners in clinical medicine and public health to provide training for health-care providers and microbiologists and to seek innovative ways to disseminate information to the public.

The efforts of this past year to improve terrorism response capacities have been widespread, crossing multiple levels and types of organizations and professions as well as international borders. Within the public health system, we intend to continue these efforts, strengthening existing and establishing new partnerships with diverse agencies, specialties, and disciplines. While we believe that these efforts will enable us to respond aggressively and effectively in the event of a future bioterrorist attack, we acknowledge that inherent to terrorism is the unknown. As was evident in the anthrax investigation, we must learn as we go, adapting our responses as new information becomes available and continuing to strive for excellence in our science, service, systems, and strategies. Investments made in the public health system to increase preparedness to address the threat of bioterrorism will also pay dividends in preparedness to confront the next influenza pandemic, other emerging infectious diseases, and other threats to public health.

Dr. Hughes is Director, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, GA, USA. Dr. Gerberding is Director, Centers for Disease Control and Prevention, Atlanta, GA, USA.

SMALLPOX

ALL SUSPECT CASES OF SMALLPOX MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY EPIDEMIOLOGY PROGRAM

During Business Hours:	619-515-6620
After Hours (County Communications):	858-565-5255

Epidemiology:

- Declared eradicated in 1980 by the World Health Organization (WHO), known stockpiles of virus still remain at the CDC in Atlanta and at the Institute for Viral preparations in Moscow and possibly other sites in the world. In the USA, civilian vaccination programs ended in the early 1980's while the military stopped in 1989.
- Highly infectious after aerosolization.
- Person-to-person transmission can occur via droplet nuclei or aerosols expelled from the oropharynx and by direct contact.
- Contaminated clothing or bed linens can also spread the virus.
- About 30% of susceptible contacts will become infected.

Clinical:

- Incubation period averages ~ 12 days (ranges 7-19 days).
- Abrupt onset of malaise, fevers, rigors, headache, emesis, backache, and delirium (15%) followed 2-3 days later by onset of rash on face, hands, forearms, and legs then spreading centrally.
- Lesions typically progress synchronously on any given part of the body from macules to papules to pustular vesicles to crusty scabs.

Laboratory Diagnosis:

- Mask and gloves should be worn by person obtaining specimen, preferably a person who has been recently vaccinated.
- Vesicular fluid is obtained by opening lesions with the blunt edge of a scalpel, harvesting fluid with a cotton swab; scabs can be removed by forceps. Swabs and scabs should be placed in a vacutainer, sealed with tape, and placed in a second, durable, watertight container.
- Laboratory specimens must be handled in a Biosafety Level 4 facility, e.g. Centers for Disease Control (CDC), and will be evaluated with electron microscopy and cell culture.

Patient Isolation:

- **Strict isolation in negative pressure room (high efficiency particulate are filtration ideal) from onset of rash until all scabs separate.**
- Laundry and waste should be autoclaved before being laundered or incinerated.

Treatment:

- Supportive care is the mainstay of therapy.
- In-vitro antiviral activity against poxviruses has been shown with cidofovir and other antivirals, but none are proven effective.

Prophylaxis:

- Vaccination within 3 days of exposure will prevent disease and within 5 days, life-saving. CDC has 12-14 million doses of vaccine; oral vaccine in development.
- Ideally, all exposed persons should be placed in strict quarantine for 17 days after last contact with a smallpox case.

FACT SHEET

Facts about Smallpox

Smallpox is a serious, contagious, and sometimes fatal infectious disease. There is no specific treatment for smallpox disease, and the only prevention is vaccination. The name *smallpox* is derived from the Latin word for "spotted" and refers to the raised bumps that appear on the face and body of an infected person.

There are two clinical forms of smallpox. Variola major is the severe and most common form of smallpox, with a more extensive rash and higher fever. **There are four types of variola major smallpox: ordinary** (the most frequent type, accounting for 90% or more of cases); **modified** (mild and occurring in previously vaccinated persons); **flat**; and **hemorrhagic** (both rare and very severe). Historically, variola major has an overall fatality rate of about 30%; however, flat and hemorrhagic smallpox usually are fatal. Variola minor is a less common presentation of smallpox, and a much less severe disease, with death rates historically of 1% or less.

Smallpox outbreaks have occurred from time to time for thousands of years, but the disease is now eradicated after a successful worldwide vaccination program. The last case of smallpox in the United States was in 1949. The last naturally occurring case in the world was in Somalia in 1977. After the disease was eliminated from the world, routine vaccination against smallpox among the general public was stopped because it was no longer necessary for prevention.

Where Smallpox Comes From

Smallpox is caused by the variola virus that emerged in human populations thousands of years ago. Except for laboratory stockpiles, the variola virus has been eliminated. However, in the aftermath of the events of September and October, 2001, there is heightened concern that the variola virus might be used as an agent of bioterrorism. For this reason, the U.S. government is taking precautions for dealing with a smallpox outbreak.

Transmission

Generally, direct and fairly prolonged face-to-face contact is required to spread smallpox from one person to another. Smallpox also can be spread through direct contact with infected bodily fluids or contaminated objects such as bedding or clothing. Rarely, smallpox has been spread by virus carried in the air in enclosed settings such as buildings, buses, and trains. Humans are the only natural hosts of variola. Smallpox is not known to be transmitted by insects or animals.

A person with smallpox is sometimes contagious with onset of fever (prodrome phase), but the person becomes most contagious with the onset of rash. At this stage the infected person is usually very sick and not able to move around in the community. The infected person is contagious until the last smallpox scab falls off.

(continued)

Smallpox Disease	
Incubation Period (Duration: 7 to 17 days) ***Not contagious***	**Exposure to the virus** is followed by an incubation period during which people do not have any symptoms and may feel fine. This incubation period averages about 12 to 14 days but can range from 7 to 17 days. During this time, people are not contagious.
Initial Symptoms (Duration: 2 to 4 days) ***Sometimes contagious****	The **first symptoms** of smallpox include fever, malaise, head and body aches, and sometimes vomiting. The fever is usually high, in the range of 101 to 104 degrees Fahrenheit. At this time, people are usually too sick to carry on their normal activities. This is called the *prodrome* phase and may last for 2 to 4 days.
Early Rash (Duration: about 4 days) ***Most contagious*** **Rash distribution:**	A **rash emerges** first as small red spots on the tongue and in the mouth. These spots develop into sores that break open and spread large amounts of the virus into the mouth and throat. At this time, the person becomes **most contagious**. Around the time the sores in the mouth break down, a rash appears on the skin, starting on the face and spreading to the arms and legs and then to the hands and feet. Usually the rash spreads to all parts of the body within 24 hours. As the rash appears, the fever usually falls and the person may start to feel better. By the third day of the rash, the rash becomes raised bumps. By the fourth day, the bumps fill with a thick, opaque fluid and often have a depression in the center that looks like a bellybutton. (This is a major distinguishing characteristic of smallpox.) Fever often will rise again at this time and remain high until scabs form over the bumps.
Pustular Rash (Duration: about 5 days) ***Contagious***	The bumps become **pustules**—sharply raised, usually round and firm to the touch as if there's a small round object under the skin. People often say the bumps feel like BB pellets embedded in the skin.
Pustules and Scabs (Duration: about 5 days) ***Contagious***	The pustules begin to form a crust and then **scab**. By the end of the second week after the rash appears, most of the sores have scabbed over.
Resolving Scabs (Duration: about 6 days) ***Contagious***	The scabs begin to fall off, leaving marks on the skin that eventually become pitted **scars**. Most scabs will have fallen off three weeks after the rash appears. The person is contagious to others until all of the scabs have fallen off.
Scabs resolved ***Not contagious***	Scabs have fallen off. Person is no longer contagious.

*Smallpox may be contagious during the *prodrome* phase, but is most infectious during the first 7 to 10 days following rash onset.

For more information on smallpox, call 619-515-6620 or visit the Centers for Disease Control and Prevention's (CDC) website at www.cdc.gov/smallpox or call the CDC at 1-800-CDC-INFO (English and Spanish) or 1-888-232-6348 (TTY). The above information has been adapted from the CDC fact sheet "Smallpox Overview".

County of San Diego
Health and Human Services Agency
Public Health Services
P.O. Box 85222
San Diego, CA 92186-5222

3/04/2002 5:30pm

WORKSHEET: EVALUATING PATIENTS FOR SMALLPOX

Identification Number _____
Person Completing Form _____
Date of Contact with Case _____
Today's Date (mo/da/yr) _____

PATIENT INFORMATION

Name:

_____
LAST FIRST MIDDLE INITIAL

Date of Birth: ___/___/___ **Age:** ___ **Sex:** ☐ Male ☐ Female

Telephone:
Home _____ Other _____

Address: _____

_____
CITY STATE ZIP

Where is the patient now? ☐ Home ☐ Doctor's Office
☐ Emergency Room *(if checked, continue below)*
☐ Hospital *(if checked, continue below)*
☐ Other *(specify)* _____

Hospital Name _____
City/State _____
Admission Date ___/___/___ Discharge Date ___/___/___
Hospital Telephone Number ___( )_____

Race: ☐ White ☐ Black ☐ Asian ☐ Other **Ethnicity:** ☐ Hispanic ☐ Non-Hispanic **Country of Birth:** _____

PROVIDER INFORMATION

Name: _____
Patient Population: ☐ Adult ☐ Peds ☐ Both
Specialty: _____
Telephone:
Type _____ ( ) _____
Type _____ ( ) _____
E-mail Address: _____

Name: _____
Patient Population: ☐ Adult ☐ Peds ☐ Both
Specialty: _____
Telephone:
Type _____ ( ) _____
Type _____ ( ) _____
E-mail Address: _____

CLINICAL INFORMATION

PRODROME / SYMPTOMS 1-4 DAYS BEFORE RASH ONSET

Did the patient have a fever and other illness 1-4 days before rash onset? ☐ Yes ☐ No ☐ Unknown

Date of prodrome onset ___/___/ 200__

Date of first fever ≥101° F: ___/___/___

What was the highest temperature? _____° F or _____° C

On what date? ___/___/___

Check all features of the prodrome that apply:
☐ No/Mild prodrome (<1 day)
☐ Headache
☐ Backache
☐ Chills
☐ Vomiting
☐ Abdominal pain
☐ Sore throat*
☐ Other *(specify)* _____

*In infants, this may manifest as drooling or refusing to eat or drink.

Was the patient toxic or seriously ill? ☐ Yes ☐ No ☐ Unknown

Was the patient able to do most normal activities? ☐ Yes ☐ No ☐ Unknown

RASH

Date of rash onset ___/___/ 200__

Was the rash acute (sudden) in onset? ☐ Yes ☐ No ☐ Unknown

Was a black scar (eschar) present before or at the time of appearance of the rash? ☐ Yes ☐ No ☐ Unknown

Is the rash *generalized* (i.e., multiple parts of the body) or *focal* (i.e., only one part of the body)? ☐ Generalized ☐ Focal

Where on the body were the first lesions noted?
☐ Face
☐ Trunk
☐ Inside the mouth
☐ Other *(specify)* _____
☐ Arms
☐ Legs
☐ Unknown

Since rash onset, where on the body was the rash most dense?
☐ Trunk
☐ Face or scalp
☐ Distal extremities (arms, legs)
☐ Equally distributed everywhere
☐ Other *(describe)* _____

Are there any lesions on the palms or soles? ☐ Yes ☐ No ☐ Unknown

What kind of lesions does the patient have now? *(check all that apply)*
☐ Macules (flat spots)
☐ Papules (solid bumps)
☐ Vesicles (fluid-filled blisters)
☐ Pustules (blisters filled with pus)
☐ Crusts
☐ Other _____

If more than one kind of lesion, which kind of lesion is now the most common? _____

Are the lesions now:
☐ Superficial (on top of the skin)
☐ Deep (feel embedded deeply in the skin)
☐ Neither *(describe)* _____

How many lesions are present? (in total) _____

If no precise count is available, please estimate:
☐ <20
☐ 20-50 (able to count in less than a minute)
☐ 51-499 (typically an average case of varicella has 200-400 lesions)
☐ >500 (lesions confluent in some places, can't see normal skin between)

On any one part of the body (e.g., face or arm), are all the lesions in the same state of development? ☐ Yes ☐ No ☐ Unknown

How big are most of the lesions? *(Do not measure superinfected lesions.)*
☐ Small (1-5 mm)
☐ Large (5-10 mm)
☐ Neither *(describe)* _____

Have any lesions crusted? ☐ Yes ☐ No ☐ Unknown

If *Yes*, how many days did it take for the first lesions to crust? _____

How itchy is the rash? ☐ Not at all ☐ Somewhat ☐ Very ☐ Unknown

Does the patient have lymphadenopathy? ☐ Yes ☐ No ☐ Unknown

If *Yes*, describe: _____

Is the patient toxic or moribund now? ☐ Yes ☐ No ☐ Unknown

If *Yes*, describe: _____

Continues

CLINICAL NOTES

SOURCE / EXPOSURE INFORMATION

Is chickenpox (varicella) occurring in the community? ☐ Yes ☐ No ☐ Unknown

Has the patient had contact with a person with chickenpox or shingles 10-21 days before rash onset? ☐ Yes ☐ No ☐ Unknown

If *Yes*, give date(s) and type of contact: _____

In the 3 weeks before onset of illness: *(applies to remainder of section)*

Has the patient been in contact with a person with any other rash illness? ☐ Yes ☐ No ☐ Unknown

If *Yes*, please specify, with date: _____

Has the patient traveled? ☐ Yes ☐ No ☐ Unknown

If *Yes*, please provide locations and dates of travel:

Place: _____ Dates: _____

Place: _____ Dates: _____

Has the patient had contact with mice? ☐ Yes ☐ No ☐ Unknown

Has the patient been camping, hiking, or exposed to woods before onset of illness? ☐ Yes ☐ No ☐ Unknown

If *Yes*, please provide details and dates:

_____ Dates: _____

_____ Dates: _____

Has the patient received insect bites? ☐ Yes ☐ No ☐ Unknown

Has the patient been exposed to ticks? ☐ Yes ☐ No ☐ Unknown

VACCINATION HISTORY

Has the patient received chickenpox (varicella) vaccine? ☐ Yes ☐ No ☐ Unknown
(Chickenpox vaccine was licensed in the United States in 1995.)

If *Yes*, dose #1 date ____/____/____ or age _____

dose #2 date ____/____/____ or age _____
(only persons >13 years receive a second dose)

Has the patient ever received smallpox vaccine? ☐ Yes ☐ No ☐ Unknown
(The smallpox vaccine was routinely given in the U.S. until 1972, was recommended for health care providers until 1976, was administered in the military until 1990.)

If *Yes*, when was the most recent vaccination? ____/____/____
or at what age? _____

MEDICAL HISTORY

Has the patient ever had chickenpox or shingles? ☐ Yes ☐ No ☐ Unknown

If *Yes*, when? ____/____/____ or at what age? _____

Is the patient immunocompromised? ☐ Yes ☐ No ☐ Unknown

If *Yes*, specify type of illness *(e.g., cancer, HIV/AIDS)* _____

Does the patient have any other serious underlying medical illnesses? *(e.g., asthma)* ☐ Yes ☐ No ☐ Unknown

If *Yes*, please list: _____

Is the patient sexually active? ☐ Yes ☐ No ☐ Unknown

Is the patient pregnant? ☐ Yes ☐ No ☐ Unknown

MEDICATIONS

Is the patient on medications that suppress the immune system? *(e.g., steroids, chemotherapy, radiation)* ☐ Yes ☐ No ☐ Unknown

If *Yes*, name of medication: _____
Dosage: _____
Method of administration: _____

Is the patient taking antiviral medications? ☐ Yes ☐ No ☐ Unknown

If *Yes*, name of medication: _____
Dosage: _____
Method of administration: _____

Please list all prescription and non-prescription medications that the patient has taken in the past three weeks. *(List drug, dosage, route, dates)*

Is there a history of illicit drug use? ☐ Yes ☐ No ☐ Unknown

If *Yes*, please specify drug, amount (if known), route, and dates:

DIFFERENTIAL DIAGNOSIS

LABORATORY

Other lab testing — Please complete last page

Have you tested the patient for chickenpox? ☐ Yes ☐ No ☐ Unknown

If *Yes*, what type of test? _____

Results of tests: _____

Date: ____/____/____

Other comments: _____

DISPOSITION

Risk of smallpox
using CDC criteria (available at www.cdc.gov/nip/smallpox):

☐ **Low** ☐ **Moderate** ☐ **High*** ☐ **Unknown**
**If checked, see contact checklist below in Immediate Response Information*

IMMEDIATE RESPONSE INFORMATION

☐ Institute airborne and contact precautions
☐ Alert infection control
☐ Take digital photographs of rash
☐ Consult ID and/or dermatology

IF THE PATIENT IS AT HIGH RISK:

☐ Contact local health department

Name: _____ Phone: _____

E-mail: _____ Phone: _____

☐ Contact state epidemiologist

Name: _____ Phone: _____

E-mail: _____ Phone: _____

☐ Contact state BT coordinator

Name: _____ Phone: _____

E-mail: _____ Phone: _____

☐ Contact CDC BT coordinator

Name: _____ Phone: _____

E-mail: _____ Phone: _____

24-HOUR FOLLOW-UP INFORMATION

Date of follow -up: ____/____/____

Person making follow-up: _____

Condition of patient: _____

Risk of smallpox 24 hours later: ☐ Low ☐ Moderate ☐ High ☐ Unkn

Action taken: _____

Diagnosis: _____

Was diagnosis confirmed? ☐ Yes ☐ No ☐ Unknown

How was diagnosis confirmed? _____

48-HOUR FOLLOW-UP INFORMATION

Date of follow -up: ____/____/____

Person making follow-up: _____

Condition of patient: _____

Risk of smallpox 48 hours later: ☐ Low ☐ Moderate ☐ High ☐ Unkn

Action taken: _____

Diagnosis: _____

Was diagnosis confirmed? ☐ Yes ☐ No ☐ Unknown

How was diagnosis confirmed? _____

72-HOUR FOLLOW-UP INFORMATION

Date of follow -up: ____/____/____

Person making follow-up: _____

Condition of patient: _____

Risk of smallpox 72 hours later: ☐ Low ☐ Moderate ☐ High ☐ Unkn

Action taken:

Diagnosis: _____

Was diagnosis confirmed? ☐ Yes ☐ No ☐ Unknown

How was diagnosis confirmed? _____

www.cdc.gov/smallpox

CLINICAL NOTES

PLEASE LIST ALL LABORATORY TESTS ORDERED OR PERFORMED REGARDING THIS ILLNESS

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Date: ____/____/____ Results: ____
Disease: ____
Test: ____
Laboratory: ☐ State
☐ Other ____

Review Article

Current Concepts

Diagnosis and Management of Smallpox

Joel G. Breman, M.D., D.T.P.H., and D.A. Henderson, M.D., M.P.H.

THE last case of endemic smallpox occurred in Somalia in 1977, and eradication of the disease was declared in 1980. With no natural reservoir, variola virus, which causes smallpox, has existed only in laboratories; indeed, the last case of smallpox was due to infection acquired in a laboratory in the United Kingdom in 1978. During the global program of smallpox eradication, the World Health Organization (WHO) made concerted efforts to decrease the number of laboratories retaining variola virus. On the basis of contacts with all countries and a total of 823 microbiology institutions, 76 such laboratories had been identified by 1978.[1,2] By 1984, only the Centers for Disease Control and Prevention (CDC), in Atlanta, and the Research Institute of Viral Preparations, in Moscow, retained variola virus isolates. In 1994, the Russian isolates were moved to the State Research Center of Virology and Biotechnology (the Vektor Institute), in Novosibirsk, Russia.

There is concern that variola virus resides outside these laboratories and could be used as a weapon by terrorists. Possible sources are virus in countries that claim that they destroyed their stocks but did not and virus acquired from laboratories in the former Soviet Union.[3,4] An accidental or deliberate release of smallpox could cause a major epidemic.[5-7] Because vaccination against smallpox has not been performed routinely in the United States since 1972 and in the rest of the world since about 1982, there is now a large population of susceptible persons.[1] Thus, if an outbreak occurred, prompt recognition and institution of control measures would be important.

VIROLOGY

Variola virus belongs to the family Poxviridae, subfamily Chordopoxvirinae, and genus orthopoxvirus, which includes vaccinia (smallpox vaccine), monkeypox virus, and several other animal poxviruses that cross-react serologically.[8,9] The poxviruses contain single, linear, double-stranded DNA molecules of 130- to-375-kb pairs and replicate in cell cytoplasm. They are shaped like bricks on electron micrographs and measure about 300 by 250 by 200 nm.

PATHOGENESIS

Studies of mousepox, rabbitpox, and monkeypox have provided information about the pathogenesis of poxviruses.[9-13] The virus enters the respiratory tract, seeding the mucous membranes and passing rapidly into local lymph nodes. After a brief period of viremia, there is a latent period of 4 to 14 days, during which the virus multiplies in the reticuloendothelial system. Another brief period of viremia precedes the prodromal phase. During the prodromal phase, mucous membranes in the mouth and pharynx are infected. The virus invades the capillary epithelium of the dermal layer in skin, leading to the development of lesions (Fig. 1).[14] Oropharyngeal and skin lesions contain abundant viral particles, particularly early in the illness. Virus is also present in urine and conjunctival secretions, with the levels decreasing during convalescence.[15,16] The spleen, lymph nodes, liver, bone marrow, kidneys, and other viscera may contain large quantities of virus.

The migration of infected macrophages to lymph nodes after the initial infection elicits the production of cytotoxic T cells and B cells; these responses limit the spread of infection. Neutralizing antibodies appear during the first week of illness but are delayed if the infection is severe; hemagglutination-inhibition antibodies are detectable by day 16 of the infection, and complement-fixation antibodies by day 18. Neutralizing antibodies remain present for many years, whereas levels of hemagglutination-inhibition and complement-fixation antibodies begin to decrease after one year.[1] The correlation between humoral antibodies and protection from smallpox is not entirely clear.

CLINICAL MANIFESTATIONS

The incubation period for smallpox is 7 to 17 days (mean, 10 to 12). The prodromal phase, which lasts for two or three days, is characterized by severe headache, backache, and fever, all beginning abruptly.[17] The temperature often rises to more than 40°C and then subsides over a period of two to three days. Enanthema over the tongue, mouth, and oropharynx precedes

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the rash by a day. The rash begins as small, reddish macules, which become papules with a diameter of 2 to 3 mm over a period of one or two days; after an additional one or two days, the papules become vesicles with a diameter of 2 to 5 mm. The lesions occur first on the face and extremities but gradually cover the body. Pustules that are 4 to 6 mm in diameter develop about four to seven days after the onset of the rash and remain for five to eight days, followed by umbilication and crusting. There may be a second, less pronounced temperature spike five to eight days after the onset of the rash, especially if the patient has a secondary bacterial infection. The crusts begin separating by the second week of the eruption. Smallpox lesions have a peripheral or centrifugal distribution and are generally all at the same stage of development. Lesions on the palms and soles persist the longest. Death from smallpox is ascribed to toxemia, associated with immune complexes, and to hypotension.

After severe smallpox, pockmarks, or pitted lesions, are seen in 65 to 80 percent of survivors.[1] These lesions are common on the face because the large sebaceous glands tend to become infected. Panophthalmitis and blindness from viral keratitis or secondary infection of the eye occur in about 1 percent of patients. Arthritis develops in up to 2 percent of children who have smallpox; viral infection of the metaphysis of growing bones is thought to be the cause. Encephalitis occurs in less than 1 percent of patients with smallpox. Although cough is not a prominent symptom, the more severe the disease, the greater the likelihood of respiratory complications; pneumonia or bacteremia may result in high mortality.

A useful classification proposed by WHO encompasses five types of smallpox.[1] The classification is based on a study of 3544 patients in India. In that study, the "ordinary" type of smallpox, variola major (described above), accounted for nearly 90 percent of cases, with a case fatality rate of 30 percent.[15,17] The milder, "modified" type accounted for 2 percent of cases in unvaccinated persons and for 25 percent in previously vaccinated persons. The modified cases were rarely fatal; the lesions were fewer, smaller, and more superficial than those in patients with the first type, and they evolved more rapidly. Seven percent of cases were characterized by flat lesions that evolved more slowly than those of variola major and that coalesced; the case fatality rate for the flat type was 97 percent among unvaccinated patients. Hemorrhagic smallpox, which is difficult to diagnose, accounted for less than 3 percent of cases; almost all patients with this type of smallpox died within the first seven days of illness. In the Yugoslav outbreak of 1972, a fatal case of hemorrhagic smallpox was misdiagnosed as a penicillin-associated drug eruption. The patient was treated in four medical institutions and infected 38 persons, 8 of whom died.[1]

The last type of smallpox, variola sine eruptione, occurs in previously vaccinated contacts or in infants with maternal antibodies. Affected persons are asymptomatic or have a brief rise in temperature, headache, and influenza-like symptoms[18]; the transmission of clinical smallpox has not been documented with variola sine eruptione.[19] In cases of variola minor, which occurs mainly in the Americas and parts of Africa, the disease is mild, causing death in less than 1 percent of patients.[20] Infection with smallpox confers lifelong immunity.

DIAGNOSIS

Many eruptive illnesses can be misdiagnosed as smallpox (Table 1). Severe chickenpox is most frequently misdiagnosed as smallpox, especially in adults who have an extensive rash (Table 2). The prodromal phase of chickenpox lasts for one or two days, fever occurs with the onset of the rash, and the eruption is concentrated over the torso; individual lesions are present at different stages and progress from vesicles, crusting within 24 hours. The interval from the initial appearance of lesions to the crusting of all lesions is about four to six days. Although about 75 percent of children in the United States are immunized against chickenpox, more than 1 million cases occur yearly. Human monkeypox, a zoonotic disease, has never occurred outside west and central Africa. The rash of human monkeypox resembles that of smallpox clinically, but patients with monkeypox often have lymphadenopathy, unlike those with smallpox, and monkeypox is not spread easily between humans, although sequential passage through four persons has been reported in rare cases.[21-23]

Drug-induced rashes, including erythema multiforme exudativum (the Stevens–Johnson syndrome), can be diagnosed by a careful history taking and examination; sulfonamides cause severe vesicular and bullous rashes. A morbilliform rash on the face due to measles virus (rare in the United States) or coxsackievirus may be confused with early smallpox. Insect bites are often linear, and allergic responses can occur. Patients with the acquired immunodeficiency syndrome may have widespread molluscum contagiosum lesions. Lesions associated with secondary syphilis vary in size and distribution, and the papules do not evolve.

EMERGENCY REPORTING

A possible case of smallpox is a public health emergency and of utmost international concern.[5,24,25] State health officials should be contacted immediately, and the diagnosis confirmed in a Biological Safety Level 4 laboratory where staff members have been vacci-

nated. The state officials should contact the CDC at any time of the day or night (telephone number, 770-488-7100). The CDC, in turn, will inform the WHO Department of Communicable Diseases Surveillance and Response Unit in Geneva, Switzerland.

After the patient has been isolated, interviews should be conducted to identify contacts. The contacts should be vaccinated as soon as possible and not more than two or three days after exposure. Smallpox vaccination within this period offers substantial protection, which is the rationale, in part, for the current policy of not launching a program of widespread vaccination of health care personnel before an outbreak has occurred.

All health care providers, regardless of their immunization status, should use airborne and contact precautions.[25,26] Scrapings of skin lesions, papular, vesicular, or pustular fluid, crusts, blood samples, and tonsillar swabbings must be sent to the CDC (or a designated laboratory) after public health officials have been notified.[25]

There are several methods for confirming the diagnosis; some are specific for variola virus, and others are for orthopoxviruses in general.[27,28] Specimens can be examined directly for the presence of virions by electron microscopy, and viral antigen can be identified by immunohistochemical studies; the brick shape of the variola virus distinguishes it from varicella–zoster virus (Fig. 2). A polymerase-chain-reaction assay for orthopoxvirus genes can be used to identify variola virus.[29-32] Isolation of the virus on live-cell cultures, followed by nucleic acid identification of orthopoxvirus species, or growth on chorioallantois, is confirmatory. The results of serologic testing do not differentiate among orthopoxvirus species, and paired serum samples are required to distinguish recent infection from vaccination in the remote past. Newer methods, which detect IgM responses, may enhance the sensitivity and specificity of serologic tests.

EPIDEMIOLOGY

Smallpox spreads primarily through respiratory-droplet nuclei, but infected clothing or bedding can also spread infection.[1,17] Although smallpox is less transmissible than measles, chickenpox, or influenza,[33] secondary attack rates among unvaccinated contacts range from 37 to 88 percent.[1,34] Patients are most infectious from the onset of the enanthema through the first 7 to 10 days of rash. Secondary cases are often limited to family members or health care personnel. Patients who have severe disease or who are coughing can transmit large quantities of virus. In Meschede, Germany, 17 persons on three floors of a hospital contracted smallpox from 1 patient during the incubation period[35]; this extensive outbreak was ascribed to the patient's cough and the low relative humidity and the air currents in the hospital.

The incidence of smallpox is highest during winter and early spring, because aerosolized orthopoxviruses survive longer at lower temperatures and low levels of humidity.[36,37] Virtually no persons in the United States under the age of 30 years have been vaccinated, and therefore, all such persons are susceptible to smallpox. Some persons who were born before 1972 and were vaccinated may still be partially protected; if exposed, they may have milder disease and may be less likely to transmit it to others.

TREATMENT

A suspect case of smallpox should be managed in a negative-pressure room, if possible, and the patient should be vaccinated, particularly if the illness is in an early stage. Strict respiratory and contact isolation is imperative. When there are many patients, an isolation hospital or other facility should be designated.[25] There is no treatment approved by the Food and Drug Administration for orthopoxviruses. Penicillinase-resistant antimicrobial agents should be used if smallpox lesions are secondarily infected, if bacterial infection endangers the eyes, or if the eruption is very dense and widespread. Daily eye rinsing is required in severe cases. Patients need adequate hydration and nutrition, because substantial amounts of fluid and protein can be lost by febrile persons with dense, often weeping lesions. Topical idoxuridine (Dendrid, Herplex, or Stoxil) should be considered for the treatment of corneal lesions, although its efficacy is unproved for smallpox. Cidofovir has been licensed for the treatment of cytomegalovirus.[38] Recent studies in animals suggest that cidofovir and its cyclic analogues, given at the time of or immediately after exposure, have promise for the prevention of cowpox, vaccinia, and monkeypox.[39,40] The drug decreases pulmonary viral levels and pneumonitis in animals with vaccinia or

Figure 1 (facing page). Clinical Manifestations and Pathogenesis of Smallpox and the Immune Response.

Panel A shows the initial phases of infection and the clinical manifestations, which include temperature spikes and progressive skin lesions (photographs of lesions courtesy of Dr. David Heymann, World Health Organization). Panel B shows the pathogenesis of the infection. The photographs at the right-hand side of the panel show the characteristic features of the vesicles caused by smallpox (hematoxylin and eosin, ×90; reprinted from Strano[14]). Panel C shows the immune response to smallpox and the period of infectiousness. HI denotes hemagglutination inhibition, and CF complement fixation.

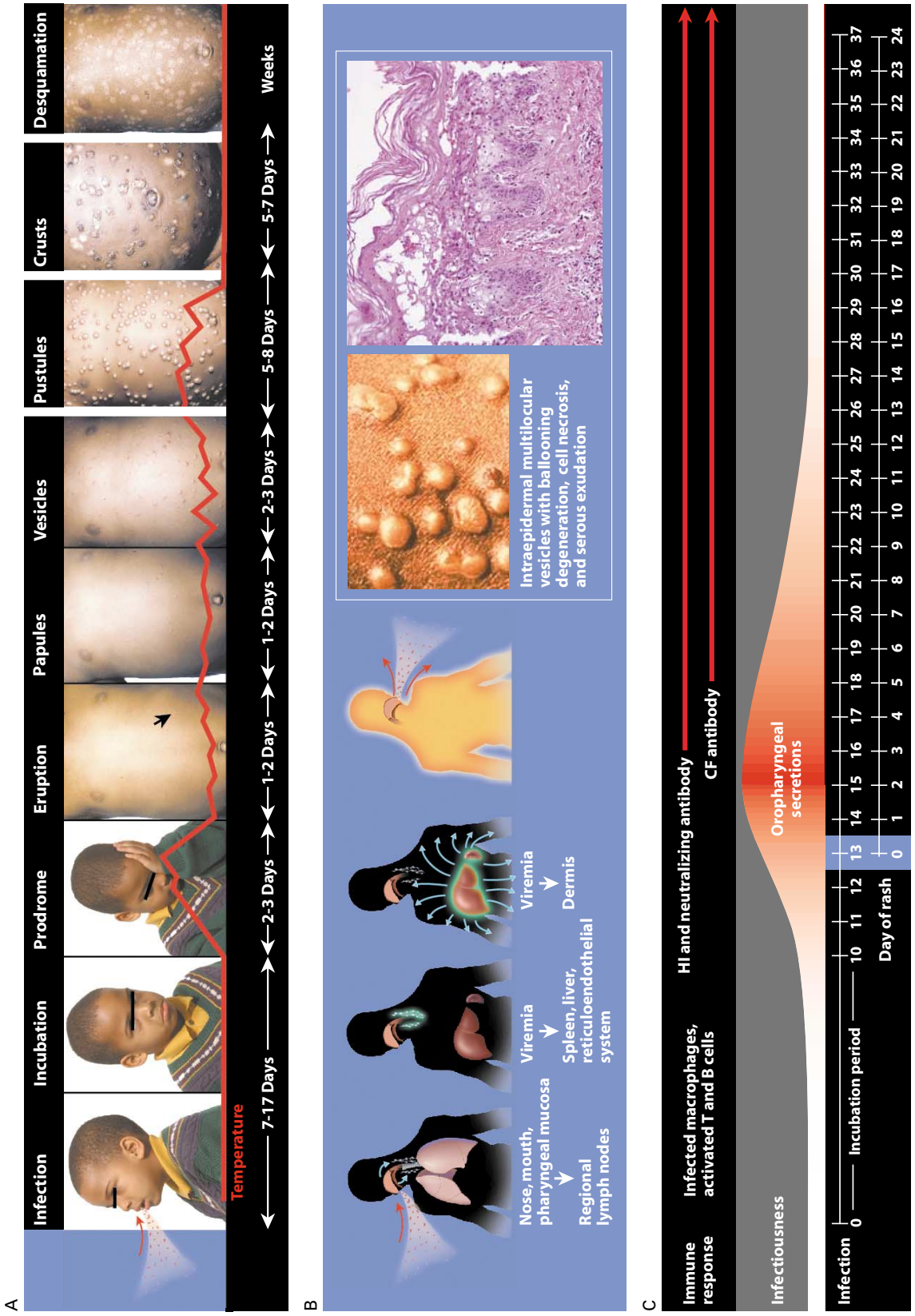

TABLE 1. CAUSES OF PAPULOVESICULAR AND MACULOPAPULAR ERUPTIONS.*

CAUSES OF PAPULOVESICULAR ERUPTIONS	CAUSES OF MACULOPAPULAR ERUPTIONS
Atypical measles (rubeola)	AIDS (HIV)
Acne†	Adenoviruses
Chickenpox (varicella)†	Arboviruses (dengue, chikungunya, and o'nyong-nyong)
Coxsackievirus (hand-foot-and-mouth disease and coxsackievirus A16)	Atypical measles (rubeola)
Dermatitis herpetiformis	Cytomegalovirus
Drug eruptions†	Drug eruptions†
Eczema herpeticum (herpes simplex virus)	Epstein–Barr virus
Generalized vaccinia and eczema vaccinatum (vaccinia)†	Enteroviral infections (echoviruses 1–7, 9, 11, 12, 14, 16, 18–20, 25, 30 and coxsackieviruses A4, A6, A10, A16, B2, B3, B5)
Impetigo	Erythema infectiosum (parvovirus B19)
Insect bites†	Exanthem infectiosum (herpesvirus 6)
Molluscum contagiosum	German measles (rubella)
Monkeypox†	Infectious mononucleosis
Papular urticaria	Measles (rubeola)
Pemphigus	Meningococcemia
Rickettsialpox (*Rickettsia akari*)	Mucocutaneous lymph-node syndrome (Kawasaki's disease)
Shingles (varicella–zoster virus)	*Mycoplasma pneumoniae*
Yaws (*Treponema pallidum*, subspecies *pertenue*)	Roseola infantum
Smallpox (*Variola major* and *V. minor*)	Scalded skin syndrome (*Staphylococcus aureus*)
	Scarlet fever (*Streptococcus pyogenes*)
	Sunburn
	Secondary syphilis (*T. pallidum* subspecies *pallidum*)†
	Rat-bite fever (*Streptobacillus moniliformis*)
	Reoviruses
	Rocky Mountain spotted fever (*R. rickettsii*)
	Toxic erythemas
	Toxic shock syndrome (*S. aureus*, phage group I)
	Toxoplasmosis
	Typhus and tick fevers (*R. prowazekii, R. typhi, Coxiella burnetii*)
	Typhoid
	Vaccine reactions (live virus)†

*AIDS denotes the acquired immunodeficiency syndrome, and HIV human immunodeficiency virus.

†This condition has frequently been confused with smallpox.

cowpox. In the event of a smallpox outbreak, the drug could be made available under an investigational-new-drug protocol for smallpox or adverse effects of vaccine. There is no evidence that prophylaxis with the use of vaccinia immune globulin, given early in the incubation period along with vaccination, has a greater survival benefit than vaccination alone[1]; vaccinia immune globulin has no benefit in patients with clinical smallpox.

PREVENTION

If performed very early in the incubation period, vaccination can markedly attenuate or even prevent clinical manifestations of smallpox. Full protection occurs after a successful vaccination. Vaccinia multiplies in the basilar epithelium after vaccination, causing a local cellular reaction. At six to eight days, the lesion is a grayish-white, loculated pustule 1 to 2 cm in diameter, with central umbilication; it is called a Jennerian pustule. Central crusting begins and spreads peripherally over a period of three to five days. Local edema and a dark crust remain until the third week. A Jennerian pustule is classified as a major reaction, indicating a successful primary vaccination; successful revaccination is indicated by palpable inflammation at six to eight days. Other reactions are classified as equivocal, and another vaccination is required in such cases. A successful primary vaccination confers full immunity to smallpox in more than 95 percent of persons for perhaps 5 to 10 years, and successful revaccination probably provides protection for 10 to 20 years or more.[1]

Guidelines from the CDC address the release of vaccine in the event of bioterrorism.[25,26] Because the risk of a deliberate release of variola virus is considered low, preexposure vaccination is not advised, except for clinical or laboratory personnel working with non–highly attenuated orthopoxviruses.[26] If the risk increased, expanded preexposure vaccination would be considered. According to the ring vaccination and

TABLE 2. DIFFERENTIAL DIAGNOSIS OF SMALLPOX AND CHICKENPOX.

DIAGNOSTIC CRITERIA	SMALLPOX	CHICKENPOX
History		
Recent contact with smallpox	Yes	No
Recent contact with chickenpox	No	Yes
Prior vaccination against smallpox*	In some cases	In some cases
Prior vaccination against chickenpox	In some cases	No
Incubation period (days)	10–12 (range, 7–17)	14–16
Prodromal phase†		
Duration (days)	2–4	0–2
Fever	Yes	In some cases
Headache, backache	Yes	In many cases
Muscle pain, malaise	Yes	In some cases
Pallor, transient rash	In some cases	No
Physical examination		
Scar from smallpox vaccination*	In some cases	In some cases
Skin lesions†		
Distribution	Centrifugal	Central
Peak (days after onset of eruption)	7–10	3–5
Evolution	Same stage	Different stages
Diameter (mm)	4–6	2–4
Shape	Round	Oval
Depth	Deep	Superficial
Desquamation (days after onset of eruption)	14–21	6–14
Lesions on palms and soles	Common	Uncommon
Complications		
Skin infection	In some cases	In some cases
Facial scarring	In most cases	In some cases (superficial)
Pneumonia	In some cases	Rare
Blindness	In some cases	No
Encephalitis	In some cases	Rare
Case-fatality rate (%)		
Chickenpox	—	<1 (2–3/100,000)
Variola major	30	—
V. minor	<1	—
Laboratory diagnosis		
Antigen or nucleic acid detection	Variola virus	Varicella–zoster virus
Electron-microscopical findings	Poxvirus particles	Herpesvirus
Results of culture on chorioallantois	Characteristic pocks	No growth
Serologic findings	Increase in antibody to orthopoxvirus	Increase in antibody to varicella virus

*Routine vaccination against smallpox stopped in 1972 in the United States and in the early 1980s in other countries, except in the case of laboratory personnel working with orthopoxviruses. The vaccination scar may fade with time.

†Patients who have undergone smallpox vaccination may have attenuated disease.

containment strategy, in the case of an international release of variola virus, the following groups would be vaccinated initially, depending on the supply of vaccine: persons directly exposed to the release; persons with face-to-face or household contact with an infected patient or in close proximity (within 2 m); personnel directly involved in the evaluation, care, or transport of infected patients; laboratory personnel involved in processing specimens; and others likely to have contact with infectious materials.[25,26] Healthy persons with no contraindication to vaccination, who have been vaccinated immediately before or shortly after contact with infected patients, should provide care for patients or work with potentially infectious materials. Those vaccinated before 1972 might have an accelerated immune response after revaccination or exposure.[1] A careful history of all persons and places in contact with patients within a period starting three weeks before the onset of the illness should be obtained for application of the ring vaccination and containment strategy.[25]

The 15 million doses of smallpox vaccine in the United States were derived from the New York Board of Health vaccinia strain. (In addition, 70 to 90 million doses have recently been identified in long-term storage by Aventis, and the U.S. government is reportedly negotiating to acquire this vaccine.) Vaccine is administered with the use of a bifurcated needle, which is dipped into reconstituted vaccine. Fifteen assertive jabs into the dermis of the upper deltoid are

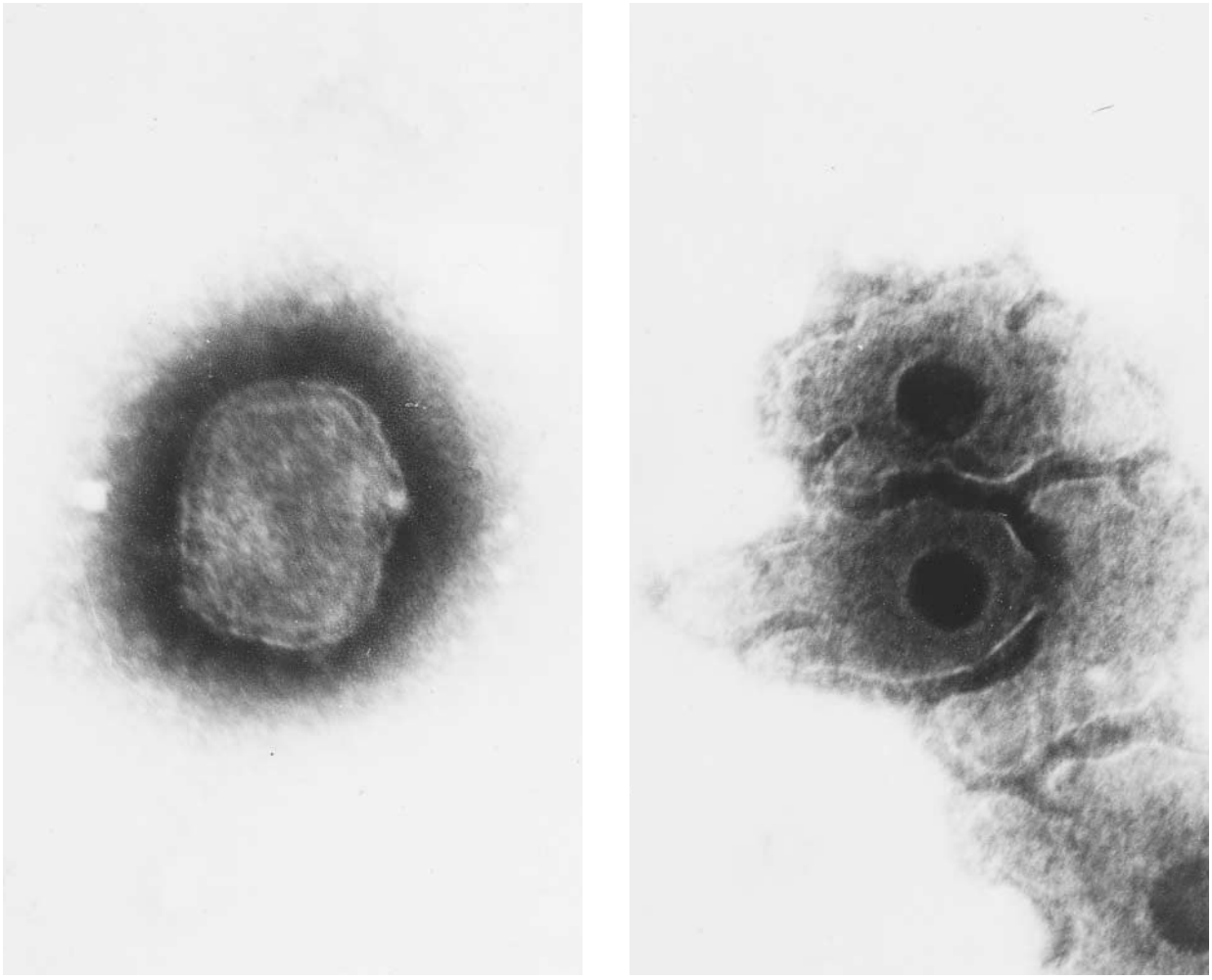

Figure 2. Electron Micrographs of Variola Virus (Panel A, ×200,000) and Varicella–Zoster Virus (Panel B, ×200,000). Photographs courtesy of Dr. Inger Damon, Centers for Disease Control and Prevention.

given in an area with a diameter of about 0.5 cm; a small amount of blood should appear at the vaccination site within 20 to 25 seconds. Studies by the National Institutes of Health indicate that vaccine diluted as much as 5 to 10 times can result in high rates of successful reactions.[41,42] In this issue of the *Journal*, Frey et al. report a 97 percent success rate with a 1:10 dilution of the vaccinia vaccine.[41] These data show that current supplies can be extended. Contracts with vaccine producers call for 280 million doses of vaccine to be available in the United States by late 2002. The newer vaccine will be produced on cell culture, in contrast to the previously used method of production in calves. Because of the different method of production, studies of the vaccine's reactogenicity and immunogenicity are required.

Complications from smallpox vaccination in the United States were closely scrutinized in the 1960s.[43,44] Because of adverse reactions, termination of the vaccination program was advised, because the risk of complications outweighed the threat of endemic smallpox.[1] The most accurate data, from a 10-state study, indicated that there were 1254 complications per 1 million primary vaccinations (Table 3).[44] Children under the age of five years who were undergoing primary vaccination had the highest rates of complications, particularly for the complications that were most severe. A nationwide study showed that the case fa-

TABLE 3. RATES OF COMPLICATIONS FROM VACCINIA, ACCORDING TO VACCINATION STATUS AND AGE.*

COMPLICATION	PRIMARY VACCINATION (N=650,000)				REVACCINATION (N=998,000)			
	0–4 YR	5–19 YR	≥20 YR	ALL AGES	1–4 YR†	5–19 YR	≥20 YR	ALL AGES
	no. of events/1 million vaccinations							
Accidental infection	564	371	606	529	198	48	25	42
Generalized vaccinia	263	140	212	242	0	10	9	9
Erythema multiforme	209	87	30	165	73	2	9	10
Eczema vaccinatum	39	35	30	39	0	2	5	3
Postvaccinal encephalitis	15	9	0	12	0	0	5	2
Progressive vaccinia	3	0	0	2	0	0	7	3
Other	222	214	636	266	18	24	55	39

*Data are from a 1968 survey of 10 states.[44] No deaths occurred.

†No children under the age of one year were revaccinated.

tality rate was 1 per 1 million primary vaccinations[43]; in 1968, there were 9 vaccine-associated deaths.

Persons who have immunologic disorders or severe eczema and pregnant women should not receive vaccinia or be in close contact with recent recipients. There are several million immunosuppressed persons in the United States, including those with human immunodeficiency virus infection and those with organ transplants, who may have vaccinia necrosum or other severe complications.[45] A limited supply of vaccinia immune globulin is available from the CDC through state health departments for the treatment of severe complications.[25] Two attenuated vaccine strains have been developed and tested: modified vaccinia Ankara (MVA) and a Japanese strain (LC16m8).[46,47] Neither has been used in areas where smallpox is endemic, so their efficacy is unknown; MVA is of special interest as a vector for immunization against other infectious diseases.

RESEARCH ISSUES

Studies that might be undertaken with the use of variola virus have been described by the Institute of Medicine, the National Academy of Sciences, and the WHO Advisory Committee on Variola Virus Research.[48] These studies address DNA-sequence information,[49] the development of antiviral drugs,[39,40] the development of an animal model for the evaluation of novel antiviral drugs and vaccines, validation of tests and equipment for early diagnosis,[27-30] establishment of a program for the production of monoclonal antibody, and the development of new vaccines with few adverse events, especially for use in immunosuppressed persons.[46]

The views expressed in this article are those of the authors and do not necessarily reflect those of the institutions with which they are affiliated or the U.S. government.

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PLAGUE

ALL SUSPECT CASES OF PLAGUE MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY EPIDEMIOLOGY PROGRAM

During Business Hours: 619-515-6620
After Hours (County Communications): 858-565-5255

Epidemiology:

- Highly infectious after aerosolization.
- Person-to-person and animal transmission can occur with pneumonic plague via respiratory droplet.

Clinical:

- Incubation period for bubonic plague is 2-8 days.
- Incubation period for primary pneumonic plague is 1-6 days; most often 2-4 days.
- Aerosolization would most likely result in pneumonic plague.
- Pneumonic plague presents with acute onset of high fever, chills, headache, malaise and a productive cough, that is initially watery before becoming bloody. Within 1 day progresses to fulminant pneumonia with dyspnea, stridor, cyanosis, septic shock with DIC and hepatocellular damage.
- Chest x-ray findings: consolidation/infiltrates.

Laboratory Diagnosis:

- Bacterial cultures (blood, sputum, or lymph node aspirate specimens) should be handled in a Biosafety Level 2 facility.
- Wright, Giemsa, or Wayson stain shows gram negative coccobacilli with bipolar "safety-pin" appearance.
- Organism grows slowly (48 hours for observable growth) on standard blood and Mac-Conkey agar.
- Immunofluorescent staining for capsule (F1 antigen) is diagnostic.

Patient Isolation:

- Strict respiratory isolation with droplet precautions (gown, gloves, and eye protection) until the patient has received at least 48 hours of antibiotic therapy and shows clinical improvement.

Treatment:

- Streptomycin (15mg/kg IM bid) or gentamicin (5 mg/kg IM or IV in 3 divided doses) are the preferred antibiotics.
- Tetracyclines or fluoroquinolones are alternative choices.
- Trimethoprim/sulfa is recommended treatment for pregnant woman and children between the ages of 2 months and 8 years.
- Chloramphenicol should be used for plague meningitis.

Prophylaxis:

- Antibiotic prophylaxis is recommended for all persons exposed to the aerosol or persons in close physical contact with a confirmed case.
- Doxycycline or trimethoprim/sulfa for pediatric prophylaxis.
- Tetracyclines or fluoroquinolones are recommended for 7 days from last exposure to a case.

FACT SHEET

Facts about Pneumonic Plague

Plague is an infectious disease that affects animals and humans. It is caused by the bacterium Yersinia pestis. This bacterium is found in rodents and their fleas and occurs in many areas of the world, including the United States. *Y. pestis* is easily destroyed by sunlight and drying. Even so, when released into air, the bacterium will survive for up to one hour, although this could vary depending on conditions.

Forms of Plague

Pneumonic plague is one of several forms of plague. Depending on circumstances, these forms may occur separately or in combination:

- **Pneumonic plague** occurs when *Y. pestis* infects the lungs. This type of plague can spread from person to person through the air. Transmission can take place if someone breathes in aerosolized bacteria, which could happen in a bioterrorist attack. Pneumonic plague is also spread by breathing in *Y. pestis* suspended in respiratory droplets from a person (or animal) with pneumonic plague. Becoming infected in this way usually requires direct and close contact with the ill person or animal. Pneumonic plague may also occur if a person with bubonic or septicemic plague is untreated and the bacteria spread to the lungs.
- **Bubonic plague** is the most common form of plague. This occurs when an infected flea bites a person or when materials contaminated with Y. pestis enter through a break in a person's skin. Patients develop swollen, tender lymph glands (called buboes) and fever, headache, chills, and weakness. Bubonic plague does not spread from person to person.
- **Septicemic plague** occurs when plague bacteria multiply in the blood. It can be a complication of pneumonic or bubonic plague or it can occur by itself. When it occurs alone, it is caused in the same ways as bubonic plague; however, buboes do not develop. Patients have fever, chills, prostration, abdominal pain, shock, and bleeding into skin and other organs. Septicemic plague does not spread from person to person.

Symptoms and Treatment

With pneumonic plague, the first signs of illness are:

- Fever
- Headache
- Weakness
- Rapidly developing pneumonia with shortness of breath, chest pain, cough, and sometimes bloody or watery sputum.

The pneumonia progresses for 2 to 4 days and may cause respiratory failure and shock. Without early treatment, patients may die.

(Continued on reverse side)

Early treatment of pneumonic plague is essential. To reduce the chance of death, antibiotics must be given within 24 hours of first symptoms. Streptomycin, gentamicin, the tetracyclines, and chloramphenicol are all effective against pneumonic plague.

Antibiotic treatment for 7 days will protect people who have had direct, close contact with infected patients. Wearing a close-fitting surgical mask also protects against infection.

A plague vaccine is not currently available for use in the United States.

For more information on pneumonic plague, call 619-515-6620 or visit the Centers for Disease Control and Prevention's website at www.bt.cdc.gov or call the CDC public response hotline at (888) 246-2675 (English), (888) 246-2857 (Español), or (866) 874-2646 (TTY). The above information has been adapted from the CDC fact sheet "Facts about Pneumonic Plague."

County of San Diego
Health and Human Services Agency
Public Health Services
P.O. Box 85222
San Diego, CA 92186-5222

9/08

BOTULISM

ALL SUSPECT CASES OF BOTULISM MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY DIVISION OF EPIDEMIOLOGY

During Business Hours:	619-515-6620
After Hours (County Communications):	858-565-5255

Epidemiology:

- Botulism neurotoxins (A-F) could be transmitted by aerosol or contamination of food and water supplies.
- **Botulism is not transmitted from person to person.**

Clinical:

- Incubation period is 12-72 hours (can be several days).
- Early symptoms include blurred vision, diplopia, and dry mouth.
- Later symptoms include dysarthria, dysphagia, dysphonia, ptosis and the development of a symmetrical, descending progressive paralysis, prominent bulbar palsies and respiratory failure.

Laboratory Diagnosis:

- Diagnosis is primarily based on a compatible clinical presentation.
- Spinal protein is normal and characteristic findings are seen on EMG (facilitation of the compound muscle action potential on repetitive nerve stimulation).
- Contact the SD Public Health Laboratory 619-692-8500 before collecting and submitting specimens.
- Toxin can be detected in serum (collect 30 cc in red top) and stool (foodborne botulism) by mouse neutralization bioassay performed at California Microbial Diseases Laboratory.
- Gastric secretions or vomitus analysis.

Patient Isolation:

- Standard precautions. Patients do not require isolation rooms.

Treatment:

- Supportive care is the mainstay of therapy. Prolonged ventilatory support is often required in severe cases with nutritional and fluid management and treatment of complications.
- Botulism anti-toxin (for A, B and E toxins) is in limited supply and is available only from the Division of Communicable Disease Control, California Dept. of Health Services. It will minimize severity, but not reverse existent paralysis. Paralysis can persist for weeks to months.

Prophylaxis:

- Currently, there is no available post-exposure prophylaxis. Botulism toxin vaccine is an investigational agent for individuals at high risk and is not effective in post-exposure prophylaxis.

County of San Diego

FACT SHEET

Facts about Botulism

Botulism is a muscle-paralyzing disease caused by a toxin made by a bacterium called *Clostridium botulinum*.

There are three main kinds of botulism:

- Foodborne botulism occurs when a person ingests pre-formed toxin that leads to illness within a few hours to days. Foodborne botulism is a public health emergency because the contaminated food may still be available to other persons besides the patient.

- Infant botulism occurs in a small number of susceptible infants each year who harbor C. *botulinum* in their intestinal tract.

- Wound botulism occurs when wounds are infected with C. *botulinum* that secretes the toxin.

Symptoms

With foodborne botulism, symptoms begin within 6 hours to 2 weeks (most commonly between 12 and 36 hours) after eating toxin-containing food. Symptoms of botulism include:

- Double or blurred vision
- Drooping eyelids
- Slurred speech
- Difficulty swallowing
- Dry mouth
- Muscle weakness that always descends through the body: first shoulders are affected, then upper arms, lower arms, thighs, calves, etc. Paralysis of breathing muscles can cause a person to stop breathing and die, unless assistance with breathing (mechanical ventilation) is provided.

Botulism is not spread from one person to another. Foodborne botulism can occur in all age groups. A supply of antitoxin against botulism is maintained by Centers for Disease Control and Prevention (CDC). The antitoxin is effective in reducing the severity of symptoms if administered early in the course of the disease. Most patients eventually recover after weeks to months of supportive care.

For more information on botulism, call 619-515-6620 or visit the Centers for Disease Control and Prevention's (CDC) website at www.bt.cdc.gov or call the CDC public response hotline at (888) 246-2675 (English), (888) 246-2857 (Español), or (866) 874-2646 (TTY). The above information has been adapted from the CDC fact sheet "Facts about Botulism."

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TULAREMIA

ALL SUSPECT CASES OF TULAREMIA MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY DIVISION OF EPIDEMIOLOGY

During Business Hours:	619-515-6620
After Hours (County Communications):	858-565-5255

Epidemiology:

- Caused by Francisella tularensis, a small fastidious Gram-negative bacillus.
- Highly infectious after aerosolization, through contaminated food or water, from arthropod, or through skin exposure.
- Infectious dose can be as low as 10-50 organisms.
- Person-to-person transmission does not occur.
- Endemic to small animals in California.

Clinical:

- Incubation period is 3-6 days (ranges 1-21 days).
- Aerosolization would most likely result in typhoidal tularemia, with pneumonic involvement.
- Ingestion is most likely to cause exudative tonsillitis and supporative cervical adenitis.
- Typhoidal tularemia is a nonspecific illness, with fever, headache, malaise and nonproductive cough, chills and sore throat. Mortality rates can be as high as 30-60%.
- Diagnosis requires high index of suspicion given nonspecific presentation. Each route of infection produces a different clinical picture.

Laboratory Diagnosis:

- Bacterial cultures should be handled in a Biosafety Level 2 facility. Isolation of the organism can otherwise put laboratory workers at risk.
- Blood/pleural fluid cultures may be positive.
- Organism is difficult to culture and grows poorly on standard media; cysteine-enriched media or chocolate agar is recommended.
- Serology is most commonly used for retrospective diagnosis.
- Aspirates of enlarged lymph nodes will also yield the pathogens.
- Four-fold titer above 1:160 is diagnostic, however this usually takes 10-14 days to develop.

Patient Isolation:

- Standard precautions. Respiratory isolation is not required.

Treatment:

- Doxycycline 100mg q 12h; chloramphenicol 15mg/kg q 6h; erythromycin 500 mg q 8h or gentamicin (5mg/kg/day IV or IM in 3 divided doses x 10-14 days) are the preferred antibiotics.
- Tetracyclines are alternatives choices, although they are bacteriostatic, associated with higher relapse rates and must be continued for at least 14 days.

Prophylaxis:

- Antibiotic prophylaxis is most effective if begun within 24 hours after exposure to aerosol.
- Tetracyclines are recommended for 14 days.
- Doxycycline or fluoroquinolones can be used for likely exposure in the absence of symptoms.
- Contaminated surfaces can be cleansed with 10% bleach, then wiped with 70% alcohol.
- Clothes and skin can be washed with soap and water.

County of San Diego

FACT SHEET

Facts about Tularemia

Tularemia is a potentially serious illness that occurs naturally in the United States. It is caused by the bacterium *Francisella tularensis* found in animals (especially rodents, rabbits, and hares).

Symptoms

Symptoms of tularemia could include:

- Sudden fever
- Chills
- Headaches
- Diarrhea
- Muscle aches
- Joint pain
- Dry cough
- Progressive weakness

People can also catch pneumonia and develop chest pain, bloody sputum and can have trouble breathing and even sometimes stop breathing. Other symptoms of tularemia depend on how a person was exposed to the tularemia bacteria. These symptoms can include ulcers on the skin or mouth, swollen and painful lymph glands, swollen and painful eyes, and a sore throat.

Symptoms usually appear 3 to 5 days after exposure to the bacteria, but can take as long as 14 days.

How You Become Infected

People can get tularemia many different ways:

- Being bitten by an infected tick, deerfly or other insect
- Handling infected animal carcasses
- Eating or drinking contaminated food or water
- Breathing in the bacteria, *F. tularensis*

Tularemia is not known to be spread from person to person. People who have tularemia do not need to be isolated. People who have been exposed to the tularemia bacteria should be treated as soon as possible. The disease can be fatal if it is not treated with the right antibiotics.

If You Think You Have Tularemia…

Consult your doctor at the first sign of illness. Be sure to let the doctor know if you are pregnant or have a weakened immune system.

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Treatment

Your doctor will most likely prescribe antibiotics, which must be taken according to the directions supplied with your prescription to ensure the best possible result. Let your doctor know if you have any allergy to antibiotics.

A vaccine for tularemia is under review by the Food and Drug Administration and is not currently available in the United States.

How to Prevent Infection

Tularemia occurs naturally in many parts of the United States. Use insect repellent containing DEET on your skin, or treat clothing with repellent containing permethrin, to prevent insect bites. Wash your hands often, using soap and warm water, especially after handling animal carcasses. Be sure to cook your food thoroughly and that your water is from a safe source.

Note any change in the behavior of your pets (especially rodents, rabbits, and hares) or livestock, and consult a veterinarian if they develop unusual symptoms.

Tularemia As a Weapon

Francisella tularensis is very infectious. A small number (10-50 or so organisms) can cause disease. If F. tularensis were used as a weapon, the bacteria would likely be made airborne for exposure by inhalation. People who inhale an infectious aerosol would generally experience severe respiratory illness, including life-threatening pneumonia and systemic infection, if they are not treated. The bacteria that cause tularemia occur widely in nature and could be isolated and grown in quantity in a laboratory, although manufacturing an effective aerosol weapon would require considerable sophistication.

For more information on tularemia, call 619-515-6620 or visit the Centers for Disease Control and Prevention's (CDC) website at www.bt.cdc.gov/agent/tularemia or call the CDC public response hotline at (888) 246-2675 (English), (888) 246-2857 (Español), or (866) 874-2646 (TTY). The above information was adapted from the CDC fact sheet "Key Facts About Tularemia."

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VIRAL HEMORRHAGIC FEVERS

ALL SUSPECT CASES OF VIRAL HEMORRHAGIC FEVERS MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY DIVISION OF EPIDEMIOLOGY

During Business Hours: 619-515-6620
After Hours (County Communications): 858-565-5255

Etiologic Agents: Arenaviridae (Lassa, Argentine, Bolivian, Venezuelan, Brazilian), Bunyaridae (Rift Valley Fever, Congo-Crimean, Hantaviruses), Filoviradae (Ebola, Marburg), and Flaviviridae (Yellow Fever) can all cause viral hemorrhagic fever (VHF).

Epidemiology:

- Caused by various RNA viruses, usually with an animal reservoir.
- All are potentially infectious by aerosol with high morbidity and mortality.
- Risk of person to person transmission depends on virus.
- All hemorrhagic fever viruses can cause capillary leak syndromes.

Clinical:

- Incubation period is 4-21 days depending on virus (5-10 days for Filoviradae).
- Clinical presentation would vary by viral agent; however, dominant clinical features of all are a consequence of microvascular damage and changes in vascular permeability.
- Malaise, fever, myalgias, prostration, conjunctival injection, petechiae, ecchymoses, shock diffuse hemorrhage, neurologic dysfunction and pulmonary collapse.
- Increased LFT's and renal dysfunction indicate a potentially poor prognosis.

Laboratory Diagnosis:

- Viral isolation should be handled in a Biosafety Level 3 or 4 facility and may take 3-10 days
- ELISA or reverse transcriptase PCR available for most VHF viruses.

Patient Isolation:

- Isolation room with contact/respiratory precautions.

Treatment:

- Largely supportive, avoiding ASA/antiplatelet drugs.
- Ribavirin (30mg/kg IV x 1, then 15 mg/kg IV q 6 h x 4 days, then 7.5 mg/kg IV q 8 x 6 days) for Lassa, CCHF, HFRS and RVF.

Prophylaxis:

- Vaccine available for Yellow Fever, Argentine, Bolivian and Rift Valley Fever.

FACT SHEET

Facts about Viral Hemorrhagic Fevers

Viral hemorrhagic fevers (VHFs) refer to a group of illnesses that are caused by several distinct families of viruses. In general, the term "viral hemorrhagic fever" is used to describe a severe multisystem syndrome (multisystem in that multiple organ systems in the body are affected). Characteristically, the overall vascular system is damaged, and the body's ability to regulate itself is impaired. These symptoms are often accompanied by hemorrhage (bleeding); however, the bleeding is itself rarely life-threatening. While some types of hemorrhagic fever viruses can cause relatively mild illnesses, many of these viruses cause severe, life-threatening disease.

Characteristics of Viruses that Cause Hemorrhagic Fever

VHFs are caused by viruses of four distinct families: arenaviruses, filoviruses, bunyaviruses, and flaviviruses. Each of these families share a number of features:

- They are all RNA viruses, and all are covered, or enveloped, in a fatty (lipid) coating.
- Their survival is dependent on an animal or insect host, called the natural reservoir.
- The viruses are geographically restricted to the areas where their host species live.
- Humans are not the natural reservoir for any of these viruses. Humans are infected when they come into contact with infected hosts. However, with some viruses, after the accidental transmission from the host, humans can transmit the virus to one another.
- Human cases or outbreaks of hemorrhagic fevers caused by these viruses occur sporadically and irregularly. The occurrence of outbreaks cannot be easily predicted.
- With a few noteworthy exceptions, there is no cure or established drug treatment for VHFs.
- In rare cases, other viral and bacterial infections can cause a hemorrhagic fever; scrub typhus is a good example.

Transmission

Viruses causing hemorrhagic fever are initially transmitted to humans when the activities of infected reservoir hosts or vectors and humans overlap. The viruses carried in rodent reservoirs are transmitted when humans have contact with urine, fecal matter, saliva, or other body excretions from infected rodents. The viruses associated with arthropod vectors are spread most often when the vector mosquito or tick bites a human, or when a human crushes a tick. However, some of these vectors may spread virus to animals, livestock, for example. Humans then become infected when they care for or slaughter the animals.

Some viruses that cause hemorrhagic fever can spread from one person to another, once an initial person has become infected. Ebola, Marburg, Lassa and Crimean-Congo hemorrhagic fever viruses are examples. This type of secondary transmission of the virus can occur directly, through close contact with infected people or their body fluids. It can also occur indirectly, through contact with objects contaminated with infected body fluids. For example, contaminated syringes and needles have played an important role in spreading infection in outbreaks of Ebola hemorrhagic fever and Lassa fever.

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Symptoms

Specific signs and symptoms vary by the type of VHF, but initial signs and symptoms often include:

- marked fever
- fatigue
- dizziness
- muscle aches
- loss of strength
- exhaustion

Patients with severe cases of VHF often show signs of bleeding under the skin, in internal organs, or from body orifices like the mouth, eyes, or ears. However, although they may bleed from many sites around the body, patients rarely die because of blood loss. Severely ill patient cases may also show shock, nervous system malfunction, coma, delirium, and seizures. Some types of VHF are associated with renal (kidney) failure.

Treatment

Patients receive supportive therapy, but generally speaking, there is no other treatment or established cure for VHFs. Ribavirin, an anti-viral drug, has been effective in treating some individuals with Lassa fever or HFRS. Treatment with convalescent-phase plasma has been used with success in some patients with Argentine hemorrhagic fever.

VHF Prevention and Control

With the exception of yellow fever and Argentine hemorrhagic fever, for which vaccines have been developed, no vaccines exist that can protect against these diseases. Therefore, prevention efforts must concentrate on avoiding contact with host species. If prevention methods fail and a case of VHF does occur, efforts should focus on preventing further transmission from person to person, if the virus can be transmitted in this way.

Because many of the hosts that carry hemorrhagic fever viruses are rodents, disease prevention efforts include:

- Controlling rodent populations
- Discouraging rodents from entering or living in homes or workplaces
- Encouraging safe cleanup of rodent nests and droppings
- For hemorrhagic fever viruses spread by arthropod vectors, prevention efforts often focus on community-wide insect and arthropod control
- Use insect repellant, proper clothing, bednets, window screens, and other insect barriers to avoid being bitten
- For those hemorrhagic fever viruses that can be transmitted from one person to another, avoiding close physical contact with infected people and their body fluids is the most important way of controlling the spread of disease.

For more information on viral hemorrhagic fevers, call 619-515-6620 or visit the Centers for Disease Control and Prevention's (CDC) website at www.bt.cdc.gov or call the CDC public response hotline at (888) 246-2675 (English), (888) 246-2857 (Español), or (866) 874-2646 (TTY). The above information has been adapted from the CDC fact sheet "Viral Hemorrhagic Fevers."

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CHEMICAL TERRORISM

The following section, “Section II: Chemical Terrorism Information and Treatment Guidelines for Hospitals and Clinicians” is an excerpt from the July *2006 Terrorism Agent Information and Treatment Guidelines for Clinicians and Hospitals* published, and approved for reprinting, by the County of Los Angeles Public Health, Emergency Medical Services Agency.

Please refer to local, state, and federal resources for updates and event-specific information.

SECTION II: CHEMICAL TERRORISM INFORMATION AND TREATMENT GUIDELINES FOR HOSPITALS AND CLINICIANS

INTRODUCTION

Background

Hospitals represent a vital disaster resource to local communities. After a terrorist attack, victims, either on their own or by emergency vehicles, will go to emergency departments regardless of the level of preparedness of the medical facility. It is essential that hospitals develop an awareness and operational level of understanding regarding the consequences of a chemical agent terrorist attack.

Historical Perspective

The first successful use of a chemical warfare weapon of mass destruction (WMD) occurred during World War I (WWI) at Ypres, Belgium, in April 1915. In that attack, the Germans released 168 tons of chlorine. The allies claimed that 5,000 troops were killed, but this was probably an inflated number for propaganda purposes.

In July 1917, the Germans first used sulfur mustard, again in Ypres, Belgium. This persistent agent (it does not evaporate readily and stays on terrain for a long time) caused many casualties as it damaged eyes, airways, and skin, but most survived. Sulfur mustard was successful as a weapon because of its persistency in the battlefield, its delayed clinical effects, and its ability to cause casualties.

Chemical agents caused over one million casualties in WW I, but killed fewer than 5 percent of these casualties, excluding those from Russia. After World War II (WWII), Egypt allegedly used chemicals in Yemen, and Iraq used them against Iran and the Iraqi Kurds.

On June 27, 1994, the Aum Shinriky , a well-funded Japanese religious cult, initiated the use of chemical warfare terrorism in Japan. The nerve agent GB, or sarin, was manufactured in a secret facility in Japan and was first released in Matsumoto, Japan with about 280 casualties and 7 deaths. Nine months later, on March 20, 1995, sarin was released in five separate subway cars in downtown Tokyo. There were 12 deaths, hundreds injured (a few dozen seriously), and 5,500 who sought medical care. Over 80 percent of those found their own transportation to the medical facilities. One hundred thirty-five of the first responders were injured. Hence, knowledge about the effects of chemical agents, how to protect oneself, and how to decontaminate and treat victims is essential.

Terrorist Threat

Chemicals make an excellent weapon for the terrorist. The effects can be immediate or delayed, the chemicals can be delivered by a variety of routes, the cost is low, the chemicals are available, and they are easily transported. In addition, most countries are poorly prepared to deal with a terrorist chemical attack.

Transport of phosgene, cyanide, anhydrous ammonia, and chlorine is a daily event in most large cities. Rail cars, which can contain up to 30,000 gallons of chemical, are susceptible to a terrorist pipe bomb. A terrorist wanting to cause panic and fear could use teargas, which is available in many stores. Dual use of industrial chemicals as agents for a terrorist attack is a concern.

Disaster Somatization Reaction

There will likely be a large number of anxious patients who have been determined not to have been exposed. This population of patients should still be considered victims. Most will be exhibiting anxiety, some will exhibit somatic symptoms that they will attribute to exposure and/or infection, referred to here as disaster somatization reaction (DSR). These symptoms can range from symptoms of general anxiety, to mimicking symptoms of true exposure. Mental health referral should be made AFTER appropriate medical triage. Mental health can elicit additional pertainent history which may result in rapid identification of patients in need of medical re-evaluation. Mental health treatment should include reassurance and possible treatment with anxiolytic medications.

Current Preparedness

Not all first responders are universally trained to recognize a chemical incident or that injuries may be due to a chemical release. Thus, first responders (police, fire, EMS) may become secondary victims. Chemical cross-contamination of ambulances and hospitals due to this lack of preparedness could cripple the capacity of the local pre-hospital and hospital system. One patient exposed to a hazardous chemical can contaminate a transport vehicle and temporarily close a hospital emergency department (ED). Convergent casualties, those who leave the incident site without pre hospital care and then seek hospital care, who are chemically contaminated pose a serious threat to the hospital and the health care provider.

Few hospital providers have access to or have been trained to use personal protective equipment (PPE). For communities to be prepared, law enforcement, fire, EMS, and hospital personnel must develop policies and procedures to address:

- The safe identification, decontamination, treatment, and transport of the chemically contaminated victim.
- The procurement of adequate caches of antidotes that are readily available to quickly and accurately treat the victims of a chemical agent attack.
- The purchase of appropriate PPE and decontamination equipment, with frequent training in the use of this gear to protect the safety of the first responder and hospital personnel.

CHEMICAL WARFARE AGENTS

Chemical warfare agents are hazardous chemicals that have been designed for use by the military to irritate, incapacitate, injure, or kill. Some have local effects on the eyes, skin, or airways (riot control agents, chlorine), some have only systemic effects (hydrogen cyanide), and some have both (nerve agents and vesicants).

The types of chemical agents to be discussed are listed below.

Agent	Name
Nerve ~Agents	**Tabun, Sarin, Soman, VX**
Vesicants	**Mustard, Lewisite**
Blood Agents	**Cyanide, Hydrogen Cyanide, Cyanogen**
Pulmonary Intoxicants	**Phosgene, Chlorine, Ammonia**
Riot Control Agents	**Mace7, Pepper Spray**
Incapacitating Agents	**BZ (Incapacitating agents, chemical agents that might cause psychological effects, might be used, but these will not be discussed)**

Some of the chemical warfare agents are said to have characteristic odors. However, these are not adequate warning signs for the purpose of protecting oneself against adverse health effects associated with exposure.

Nerve Agents

The nerve agents are tabun (GA), sarin (GB), soman (GD), and VX. Nerve agents are the most toxic of all the weaponized military agents. These agents can cause sudden loss of consciousness, seizures, apnea, and death. Sarin (GB), one of the more commonly stockpiled nerve agents, may be inhaled as a vapor, or cause toxic effects by contact with the skin in the liquid form. VX is mainly a liquid skin hazard at normal ambient temperatures. These chemicals are easily absorbed through the skin, eyes, and lungs.

The diagnosis of a nerve agent poisoned casualty must be made clinically on the basis of the presenting signs and symptoms **sudden loss of consciousness, seizures, apnea, and death**. There usually is not time for laboratory confirmation. Nerve agents inhibit cholinesterase, an enzyme present in tissues and blood. There is a laboratory blood test to determine cholinesterase activity.

Characteristics

The nerve agents belong to a class of chemicals called organophosphates and have a physiological effect similar to that of many insecticides commonly found in the community, such as malathion, diazinon, and chlorpyrifos. If organophosphate poisoning is not treated with appropriate antidote, the effect on cholinesterase is permanent.

Included among the nerve agents are chemicals called carbamates, which include some drugs (such as physostigmine and pyridostigmine) and some insecticides (Sevin7, Raid, etc.). These compounds cause the same clinical effects as the nerve agents developed for military use, but the latter are more than a hundred-fold more potent. Also, with carbamates, the effect on cholinesterase is only temporary.

Nerve agents are stored and transported in the liquid state. The G-agents such as sarin (GB), soman (GD), and tabun (GA) are volatile liquids at normal temperatures although, the most volatile, sarin, evaporates at about the same rate as water. In liquid form, the G-agents can be absorbed through the skin and eyes; vapor is absorbed by inhalation and through the eyes, but not through the skin unless the concentration of vapors is extremely high. The G-agent liquids are more effective in penetrating skin when the chemical is trapped between the skin and clothes. GB rapidly evaporates and is considered to be a "non-persistent agent," meaning that it does not remain on terrain or equipment very long. VX is a persistent agent due to its low volatility. Though liquid at normal temperatures, VX has the consistency of motor oil, and seldom presents a vapor hazard, unless exploded or subjected to high temperature. VX is much more toxic (100 to 150 times) than sarin when on the skin because sarin evaporates from the skin surface while VX does not.

Mechanism of Action

Nerves communicate with muscles, glands, and other nerves by releasing chemicals (neurotransmitters) at their connection sites (synapses). One of the most common neurotransmitters is acetylcholine (ACh), which is released and collects at the receptor site stimulating the end organ to respond and produce a variety of effects: muscle contractions, gland secretions, and nerve-to-nerve conduction. These are known as cholinergic nerves and synapses.

When a nerve impulse reaches the synapse, ACh is released from the nerve ending and diffuses across the synaptic cleft to combine with receptor sites on the next nerve, muscle, or gland and stimulate a response.

To stop further stimulation of the nerve, muscle, or gland, ACh is rapidly broken down by the enzyme acetylcholinesterase (AChE) located in the postsynaptic receptor region, producing choline, acetic acid, and the regenerated enzyme. Thus, a check and balance system prevents the accumulation of ACh and the resultant over-stimulation of nerves, muscles, and glands.

The term "nerve agent" refers to chemical that produces biological effects by inhibiting the enzyme AChE, thus allowing the neurotransmitter ACh to accumulate. As a result of inhibition of AChE, the neurotransmitter ACh accumulates and over-stimulates the receptors of the

cholinergic nerves and causes hyperactivity of the cholinergic nerves, muscles, and glands.

Cholinergic synapses have two types of receptors: muscarinic receptors, nicotinic receptors, or a combination (central nervous system and cardiovascular system). Organs with muscarinic receptors include smooth muscles and exocrine glands; those with nicotinic sites are skeletal muscles and pre-ganglionic fibers.

Muscarinic receptors

Over-stimulation at muscarinic sites will increase glandular secretions. The victim may experience increased saliva, tearing, runny nose, thick secretions in the airways, and sweating; remembered by the acronym SLUDGE: salivation, lacrimation, urination, defecation and gastrointestinal emesis.

Smooth muscle over-stimulation

Over-stimulation of smooth muscles causes pinpoint pupils (miosis), bronchoconstriction of airways (shortness of breath), and hyperactivity of the gastrointestinal tract (nausea, vomiting, and diarrhea).

Nicotinic receptors

Over-stimulation of nicotinic receptors causes skeletal muscle fasciculations, twitching, cramping, weakness, and finally paralysis. There is also stimulation of the pre-ganglionic fibers, which may contribute to hypertension and tachycardia. **The combination of pinpoint pupils, muscle fasciculations and respiratory distress is reliable clinical evidence of organophosphate (nerve agent) poisoning.**

Cardiovascular

Cardiovascular effects that may occur are bradyarrhythmias and hypotension. Tachyarrhythmias (sinus tachycardia, ventricular tachycardia, and ventricular fibrillation), hypertension, and heart blocks may also occur. Most of these cardiovascular effects disappear once the antidote is given.

Central Nervous System

Acute severe effects include: loss of consciousness, seizures, and apnea. Effects from a mild exposure include: nervousness, fatigue, minor memory disturbances, irritability, and other minor psychological symptoms. The latter, whether caused by a severe or mild exposure, might linger for 4 to 6 weeks after exposure before resolving.

Cause of Death

The cause of death in nerve agent exposure is respiratory failure due to: bronchospasm and thick secretions in the airways; weakness of respiratory muscles to flaccid paralysis; and inhibition of the respiratory center in the CNS.

Clinical Effects

Vapor

After exposure to a small amount of vapor from a volatile nerve agent like GB, the most common effects are miosis - often with pain in the eye or head, complaints of dim or blurred vision or conjunctival injection, rhinorrhea, and some degree of bronchoconstriction and bronchosecretions with associated complaints of a tight chest and/ or shortness of breath.

After exposure to a moderate amount of vapor, besides the signs and symptoms noted above, the victim will show signs of multiple system involvement - especially increasing respiratory distress and nausea, vomiting and diarrhea

After exposure to a large amount of vapor, the victim will almost immediately lose consciousness, and seizures will begin within 1 to 2 minutes. After several minutes of seizing, apnea and flaccid paralysis will occur.

Effects begin within a minute or so after vapor exposure and generally do not worsen significantly once the contamination is removed. Peak effects usually occur within the first 5 minutes following exposure.

If the exposure has been small and a victim is removed from the area of the exposure, shortness of breath may improve. In this situation, the removal of clothing is often adequate decontamination.

Liquid

Persistent agents like VX present more of a liquid contact hazard. The onset of effects following exposure can be delayed from 10 minutes to 18 hours after contact with the agent, depending on the dose. With military grade purity the LD 50 for VX is 10 mg, a droplet the size of the head of a pin. Fortunately, terrorists are unlikely to achieve such purity (the sarin at the Tokyo incident was a 20-40 % solution).

- small dose - A very fine droplet on the skin will cause fasciculations and diaphoresis under the droplet site. There will be no pinpoint pupils.
- moderate dose - With a larger droplet multiple system effects will occur including gastrointestinal (GI), nausea, vomiting, and diarrhea. Generally, there will be no pinpoint pupils.
- large dose - A droplet the size of the LD50 on the skin will cause sudden loss of consciousness, seizures, flaccid paralysis, and apnea within minutes.

Medical Management

Self-protection

The process of treating nerve agent casualties may be divided into several components. The first and most important concept is to protect oneself. Although liquid contaminated casualties

are unlikely to present directly to the hospital ED prior to decontamination by emergency responders, medical personnel should always protect themselves by assuming the presence of liquid contamination, unless a clear vapor-only exposure history is obtained. Whenever possible, areas of liquid contamination should be decontaminated prior to patient handling to minimize spread of contamination and cross-contamination of other providers.

Decontamination

In the immediate aftermath of the sarin nerve agent attack in Tokyo, over 650 patients presented to St. Luke's Hospital within several hours after the release of sarin. With high numbers of vapor-exposed patients presenting to a medical facility under these conditions, minimum decontamination should include removal of patients' clothing and jewelry. This will hopefully prevent secondary chemical exposure of hospital personnel due to vapor off-gassing. If the patient has been exposed to liquid nerve agent (such as spraying or an explosion), survivors will require complete decontamination of skin and hair with water, soap and water, and water rinse at the scene prior to evacuation.

Patients arriving at the ED with an unclear exposure history who are symptomatic from nerve agent exposure should be fully decontaminated as above before entering treatment areas.

Airway and ventilation

Establishment of a patent airway is essential for the survival of the severely exposed patient. Severely intoxicated patients will die if aggressive airway management is not quickly available. With large numbers of victims, rapid scene and resource assessment will influence triage decisions regarding interventional therapy. Because of the intense bronchoconstriction and secretions associated with nerve agent exposure, effective ventilation may not be initially possible due to high airway resistance (50 to 70 cm H_2O). Adequate atropinization will reverse these muscarinic effects; therefore, atropine should be administered before any other measures are attempted. Endotracheal intubation, followed by positive pressure ventilation with a bag-valve mask, should be performed as quickly as possible. Periodic suctioning of secretions will help to improve ventilation and air exchange. Patients with seizures and respiratory failure can be saved with immediate and adequate intervention.

Antidote administration

Three medications are used to treat the signs and symptoms of nerve agent intoxication: atropine sulfate, pralidoxime chloride, and diazepam. The general indications for use of these antidotes will be presented first, followed by a discussion of their use in the treatment of mild, moderate, or severe nerve agent intoxication.

Atropine

Atropine works to block the effect of the accumulated neurotransmitter, ACh, at muscarinic sites. The more ACh at the sites, the more atropine is required to counteract its effects. Atropine can be administered intravenously (IV), intramuscularly (IM), or endotracheally (ET). Parenteral atropine will reverse muscarinic effects such as rhinorrhea, salivation, sweating, bronchoconstriction, bronchorrhea, nausea, vomiting, and diarrhea.

The IV route of atropine administration is preferred but can be also given intramuscularly (IM) or intrachecally but only until IV access is established.

The initial parenteral dose of atropine is 2 to 6 mg in the adult, with subsequent doses titrated to the severity of the nerve agent signs and symptoms. Treatment for chemical nerve agent exposure might require up to 40 mg of atropine. Patients poisoned with insecticides may require these large doses; over 1,000 mg of atropine have been used. When atropine therapy exceeds the amount necessary to reverse the effect of the cholinergic hyperstimulation, it may cause toxicity manifested by dry mouth, flushing, and diminished sweating, but this would be extremely unlikely in a patient poisoned by an organophosphate (OP) compound. Side effects in unexposed people (not poisoned by OP compounds) include mydriasis, blurred vision, tachycardia, and diminished secretions. The latter (i.e., loss of sweating) may be of concern in a hot environment. Glycopyrrolate is a poor substitute and should not be used if atropine is readily available.

Atropine dosing is guided by the patient's clinical presentation and should be given until secretions are dry or drying and ventilation becomes less labored. When shortness of breath, increased airway resistance, and secretions have abated and the patient is breathing easier, he or she has received enough atropine. Heart rate and pupillary size, ordinarily accurate reflections of atropine dosing, are not useful for clinical monitoring after nerve agent exposure.

Atropine will not reverse nicotinic effects such as fasciculations, twitching, or muscle weakness. Nor are miosis or ciliary body spasm reversed by parenteral atropine; relief of intractable pain in or around the eye requires the installation of one percent homatropine topically.

Pralidoxime chloride (2-PAMCl)

This is an antidote that can specifically break the bond between the nerve agent and the enzyme AChE and thus remove the agent. This will free the enzyme, making it once again available to break down ACh. Clinically, this will decrease muscle twitching, improve muscle strength, and allow the patient to breathe easier; however, it has little effect on the muscarinic effects described previously. The bond between the enzyme and the nerve agent can "age," that is, the enzyme and agent become irreversibly bound. This means that if the antidote is not administered within 4 to 6 hours after sarin exposure (the aging time for the sarin-enzyme complex) or within 60 hours after VX exposure (the aging time for the VX-enzyme complex), the bond becomes permanent. Usually, there is plenty of time to treat patients with 2-PAMCl after exposure to nerve agents with the exception of GD. The soman-enzyme complex ages in about 2 minutes. Since pralidoxime takes time to take effect, atropine administration is the first priority.

MARK I kit

Includes atropine and pralidoxime chloride (2-PAMCl) and is used by the military in autoinjectors which together are called the MARK I kit. The atropine autoinjector contains 2 milligrams (mg) of atropine and is administered IM by pressing the end of the device onto the thigh. A spring pushes the needle into the muscle and causes the atropine to be injected. This device causes atropine to be absorbed more rapidly than when administered by a conventional needle and syringe. The other autoinjector contains 600 mg of 2-PAMCl. The Food and Drug Administration

(FDA) has approved the autoinjectors for civilian use and Los Angeles County first responder EMS units have caches of these for field use. The Mark 1 is for healthy adult use only.

Diazepam
Seizures are treated with benzodiazepines such as diazepam. These medications can be used IV or via an autoinjector which contains 10 mg of diazepam. Some authorities recommend treating all severely exposed patients with diazepam whether they are convulsing or not. If three atropine MARK I kits are required initially, because of the victim's clinical presentation, diazepam should be administered immediately thereafter. Diazepam should be given liberally and dosages may total 40 mg or more.

Treatment

Latent effects
Victims who present to the ED alleging exposure to nerve agents should be considered potentially exposed, triaged for anxiety and other injuries, and observed for up to 1 hour if a vapor exposure is alleged, or up to 18 hours if a liquid exposure is possible (or if the exposure history is uncertain).

Mild effects
If there are mild effects from liquid exposure (localized sweating and fasciculations at the site of liquid contact), give 600 mg 2-PAMCl IM (MARK I kit) or 1 gram (gm) 2-PAMCl IV slowly over 20 to 30 minutes. The presence of miosis and rhinorrhea requires observation only. If the victim is suffering from airway effects (shortness of breath, chest tightness, and profuse airway secretions) that are not improving, then treat with 2 mg of atropine IM or IV, or with the MARK I kit. Supplemental oxygenation will be needed only in those patients with pulmonary or cardiac disease. IM atropine dosing can be repeated at 5 to 10 minute intervals as needed.

> Note: Patients with pinpoint pupils may have severe light sensitivity and pain, but only require reassurance since these symptoms will resolve. At the hospital, these patients should be given a topical eye medication (homatropine) only for relief of severe pain in the eye(s) or head because the drug causes blurred vision. This may be done if miosis occurs as part of moderate or severe systemic effects as well.

Moderate vapor exposure
Be more aggressive with moderate vapor exposures.
Symptoms include those for mild exposures with more severe respiratory distress and may be accompanied by muscular weakness and possibly GI effects (vomiting and diarrhea). Initial dose for these patients is 1 or 2 MARK I kits containing a total of 2 mg atropine and 600 mg 2-PAMCl. Treatment may also be given IV, with 2 to 4 mg Atropine given IV push, and 1 gram of 2-PAMCl given by IV infusion slowly.

This dosing can be followed by repeat doses of 2 mg of Atropine at 5 to 10 minute intervals as needed, and 600 mg of 2-PAMCl for a total of 1,800 mg 2-PAMCl with the MARK I kit IM (or 1 gm 2-PAMCl IV for a total of three doses at hourly intervals).

Antidotes can also be given IV, with Atropine given in 2 mg increments at 5 to 10 minute intervals, and 2-PAMCl given by infusion, 1 gm over 20 to 30 minutes, for a total of 3 doses at hourly intervals.

Moderate liquid exposure

Symptoms will include increasing respiratory distress and nausea, vomiting and diarrhea. For moderate toxicity several hours after liquid exposure, 2 mg of atropine and 600 mg 2-PAMCl should be given initially. Repeated doses of atropine and 2-PAMCl may be necessary. Oxygen may be needed in those with cardiac or pulmonary disease who have severe breathing difficulty.

Severe vapor or liquid exposure

Severe exposure symptoms will include all the above plus unconsciousness, seizures, apnea, or severe effects in two or more systems (excluding the eyes). Give 3 Mark 1s and diazepam and manage the airway. Repeat atropine at 5-10 minute intervals as necessary and 2-PAMCl in one hour.

Treatment for Nerve Agent Exposure

Exposure	Clinical	Treatment
Latent	None	None, observe for 1 hour with vapor and for 18 hours if liquid or unknown exposure
Mild	Miosis with dim and/or blurred vision, rhinorrhea, shortness of breath.	Miosis and rhinorrhea, observation only. Shortness of breath: one MARK 1 kit or Atropine 2 mg IM/IV and 2-PAMCl 600 mg IM or 1 gm IV.
Moderate	Above, but more severe; or vomiting and diarrhea	One MARK 1 Kit or Atropine 2mg IM/IV and 2-PAMCl 600 mg IM or 1 gm IV. Repeat 2 mg Atropine at 5-10 minute intervals until agent effects diminish.
Severe	Above plus unconsciousness, Flaccid paralysis, respiratory distress, cyanosis, seizures or severe effects in two or more organ systems	Oxygen, bag mask, intubate after three MARK 1 kits or Atropine 6 mg IM and 2-PAMCl 1800 mg IM or 1 gm 2-PAMCl IV repeated twice at hourly intervals. Repeat 2 mg Atropine at 3-5 minute intervals until atropinized. Diazepam for seizures.

Age-Related Antidote Administration

Atropine

Certain members of the population may be more sensitive to Atropine. These include infants, young children, and the elderly. Pediatric experts have divided the age groups for IM administration of Atropine. These doses may be repeated as clinically indicated.

Category/Age	Dose
Infant - 0 to 2 years	0.5 mg single dose
Child - 2 to 10 years	1.0 mg single dose
Adolescent - young adult	2.0 mg single dose
Elderly - frail or medically compromised adult	1 mg and repeat as necessary
If Atropine is to be given IV, then the dose for infants through young adults is 0.02 mg/kg.	

If only standard MARK I kits are available, the use of a 2 mg Atropine autoinjector can be used, but infants and small children are at risk of being injured by the autoinjector needle. The most significant adverse effect of high dose Atropine in the younger patient is the inhibition of sweating.

Pralidoxime chloride

(no data available for 2-PAMCl use in children exposed to nerve agents)
Dose may be adjusted in the elderly; frail, hypertensive, or with renal disease, using one-half the usual adult dose of 2-PAMCl (7.5 mg/kg IV). If hypertension becomes significant during the administration of the 2-PAMCl, treat with IV phentolamine as follows: Adults - 5mg IV, Pediatrics - 1 mg IV

Category/Weight	IV dose	Weight	IM dose
Infant ≤ 70 kg	15 mg/kg repeated twice at hourly intervals	< 20 kg	15 mg/kg
Above 70kg	1 gm repeated twice at hourly intervals PRN	> 20 kg	600 mg autoinjector

Diazepam

Recommended Pediatric Dose	
Infants > 30 days to ≤ 5	0.2 to 0.5 mg/kg IV slowly every 2 to 5 minutes to maximum dose of 5 mg
Children > 5 years	1 mg IV every 2 to 5 minutes to maximum dose of 10 mg

BLISTER AGENTS OR VESICANTS

Vesicants cause blistering. They may be plant, animal, chemical, or sunlight. Those discussed are the chemical warfare vesicants, namely sulfur mustard and Lewisite. A close relative of sulfur mustard, nitrogen mustard was the first cancer chemotherapeutic agent.

Sulfur Mustard

Sulfur mustard is a vapor inhalation and liquid contact hazard. Mustard causes injury to the eyes, skin, airways, and some internal organs. This chemical warfare agent has a delayed action, and exposure to it may result in blisters on the skin, temporary blindness, and respiratory distress. More extensive injury can result in death due to respiratory failure from airways injury, sepsis as a result of bone marrow damage, decrease in white blood cells, and impairment of the immune system. There is no specific therapy.

Characteristics

Mustard is an oily liquid yellow to brown in color. Its name comes from its odor of garlic or mustard, but odor should not be relied upon for detection. Mustard is a persistent agent and not volatile at temperate conditions, however at temperatures above 100 ºF it is a definite vapor hazard. Mustard has a relatively high freezing point and is often mixed with similar agents such as Lewisite to lower the freezing point. Because of its oily and persistent nature, mustard poses a definite concern for cross contamination.

Mechanism of action

Mustard is absorbed and causes chemical cellular damage within 1 to 2 minutes, but clinical effects do not begin for hours. There is no immediate pain, there is no immediate skin discoloration, and there is no immediate eye irritation. However, hours later, the casualty realizes that he or she has been exposed and presents to the ED for evaluation and treatment. The onset time for clinical effects ranges from 2 to 24 hours, but the most common interval is 4 to 8 hours.

Despite years of research, the exact mechanism by which mustard damages cells is unknown. It alkylates DNA and clings to proteins and other cellular components. The end result is DNA damage and cellular death. The injury is very similar to that produced by radiation, and mustard is a radiomimetic agent. Topically, three organ systems directly affected by mustard are the eyes, skin, and respiratory tract.

Clinical Effects

Ophthalmic

There is a spectrum of eye involvement. The eye lesion, after a small exposure to mustard, may consist only of mild conjunctivitis. A larger exposure will produce a more severe conjunctivitis, lid inflammation and edema, blepharospasm, and corneal roughening. These casualties will be unable to open their eyes and will be temporarily without sight. A larger exposure, particularly if by liquid, may produce corneal opacification, corneal ulceration, or corneal perforation. Miosis is sometimes observed after mustard exposure and is thought to be due to cholinergic effects.

Integumentary

Skin effects begin hours after exposure with erythema accompanied by burning and itching. This is followed by the development of small vesicles, which later coalesce to form blisters. The size and depth of the lesion depends on the amount of exposure and whether exposure was by vapor or liquid. Coagulation necrosis extending into the dermis may develop under blisters caused by liquid.

Pulmonary

Mustard damages the mucosa or lining of the airways. This damage begins in the upper airways and descends in a dose-dependent manner to the smallest bronchiole. After a small exposure or initially after a large exposure, there may be epistaxis, sinus discomfort, and a mild to moderate pharyngitis with a hacking cough. After a moderate to large exposure, there may be laryngitis with voice loss and a productive cough. If the exposure is large, the agent reaches the smallest airways to cause dyspnea and productive cough, as the mustard will damage not only the mucosa, but the underlying musculature as well. At this stage, there may be hemorrhagic pulmonary edema around the bronchioles, but otherwise, pulmonary edema is rare.

Gastrointestinal

Gastrointestinal effects within the first 24 hours following exposure include nausea and vomiting. These effects are thought to be in part due to cholinergic stimulation. There may be some added effects of mustard on the GI tract from the swallowed tracheal secretions. Gastrointestinal effects seen after 3 to 5 days are thought to be due to tissue destruction in the abdomen.

Hematopoietic or Blood Forming System

Absorption of significant amounts of mustard produces damage to and death of the stem or precursor cells of the bone marrow. If this occurs, the white blood cell count, after an initial increase because of the toxic exposure, starts decreasing on about the third or fourth day after exposure and continues downward until recovery begins. If the amount of mustard absorbed is quite large, there is no recovery and the cell count will reach zero. Survival usually does not occur when this happens. The absence of these cells increases susceptibility to infection and contributes to death. The red blood cells and platelets also decline following the white blood cells.

Medical Management

Decontamination

Decontamination should consist of physical removal of any residual agent by whatever means available. The casualties should remove all clothing, rings, and jewelry. Skin and hair decontamination should be performed with soap and water. Decontamination must be done as quickly as possible since cellular damage occurs in as little as two minutes. Decontamination of the casualty at the ED 30 minutes or more after contact with mustard will not change the clinical course of the patient's illness, but is effective in preventing cross-contamination of providers.

Treatment

Treatment is largely supportive since there is no antidote for the effects of sulfur mustard.

Skin

Soothing creams or lotions might be effective for irritation and itching. Large blisters should be unroofed and denuded areas irrigated several times a day followed by a topical antibiotic (Silvadene, etc.) to prevent skin bacterial superinfection. Oral pain medications will likely be necessary. Fluid requirements should be assessed, less fluid replacement is necessary than with thermal burns. Care must be taken not to over hydrate the patient (burn formula resuscitation is not recommended). Rarely will burns be full thickness requiring skin grafting.

Eyes

Again, mustard fixes to tissues within the first several minutes after exposure. Gentle irrigation with saline or water during this time period will be helpful. Aggressive attempts to pry apart severely painful, blepharospastic eyelids to accomplish an irrigation 30 minutes or more after exposure is of dubious value, since the damage has been done and the agent has evaporated or has been absorbed. With severe eye injuries, homatropine or other mydriatics should be applied topically to prevent synechiae formation. Topical antibiotics should be applied several times a day and petroleum jelly should be applied to lid edges to prevent them from adhering. Topical ophthalmic analgesics may be used to facilitate initial examination. However, oral pain medication is preferred to topical analgesics, since topical agents may damage the cornea and delay healing. Many ophthalmologists feel that the application of topical steroids within the first 24 hours, but not after, might be of benefit. Early involvement of an ophthalmologist is advised, and visual acuity should be obtained before treatment measures are instituted.

Pulmonary

Upper or minor airway symptoms (sore throat, non-productive cough, hoarseness) may be relieved by steam inhalation and cough suppressants. The initial chemical pneumonitis should be treated in the usual manner; however, antibiotics should not be used until an organism is demonstrated, which usually occurs between the third and fifth day post-exposure. A patient with severe airway effects will benefit from oxygen and assisted ventilation, particularly positive end expiratory pressure (PEEP) or continuous positive airway pressure (CPAP). Intubation should be performed if there are signs of severe upper airway involvement, and should be done early, before laryngeal spasm or edema makes it difficult. Bronchodilators may be needed; if they fail to relieve bronchospasm, steroids may be tried. Otherwise, steroids are of questionable benefit.

Lewisite

Characteristics

Lewisite is a vesicant that has been stockpiled militarily, but there have been few human exposures to the chemical.

Clinical Effects

Lewisite is rapidly absorbed by the eyes, skin, and lungs and produces blisters similar to sulfur mustard. In contrast to sulfur mustard, however, lewisite is highly irritating on initial exposure. It also produces visible lesions more quickly. Unlike mustard, it does not damage the bone marrow. Lewisite is an arsenical compound, thus a heavy metal poison.

Integumentary
Lewisite causes greater skin damage than sulfur mustard. A gray area of dead skin can progress to blisters and severe tissue necrosis and sloughing.

Pulmonary
Since lewisite causes immediate irritation to the nose and sinuses, an effort by the victim to evacuate the area of contamination may prevent more severe lung damage. Pseudomembrane formation is common.

Cardiovascular
Lewisite causes increased capillary permeability, leading to volume depletion, hypotension, hepatic and renal injury.

Medical Management

Decontamination
Casualties should remove all clothing and jewelry. Decontamination of skin and hair with soap and water will remove most of the chemical, if performed quickly after contamination.

Treatment
The antidote available is dimercaprol which is called British anti-Lewisite (BAL**). BAL can be administered IM to reduce the systemic effects of the vesicant. Since it is administered parenterally, BAL has no effect on Lewisite damage to the skin and eyes.

***BAL is also used for some other heavy metal poisonings.*

BLOOD AGENTS

Cyanide

Cyanide is a chemical that is widely utilized, manufactured, and transported in the U.S. Over 300,000 tons of cyanide are produced annually. It is used in printing, agriculture, photography, and in the manufacture of paper and plastics. It is also a combustion product of burning synthetic materials. Rail cars with 30,000-gallon tanks of cyanide represent potential transportation and terrorist threats. A large amount of cyanide is needed to cause death on the battlefield; therefore, it is not a very good military weapon. Terrorist use in confined spaces such as a subway care, shopping center, convention center or high-rise building would be far more effective.

Characteristics

Cyanide is stored and utilized in the liquid or solid state. It may have an odor of bitter almonds, but the ability to smell the cyanide exists in only 40 percent of the population.

Three types of cyanide may be encountered: hydrogen cyanide (AC), cyanogen chloride (CK), and cyanide salts. The term cyanide refers to the anion, CN^-, or to its acidic form, hydrocyanic acid (HCN). Cyanogen (CN2) is formed by the oxidation of cyanide anions. However, the term cyanogen has also come to mean a substance that forms cyanide upon metabolism and produces the biological effects of free cyanide. Cyanogen chloride is a pungent, heavier-than-air vapor, which can cause irritation of the eyes, nose, and throat. This is in distinct contrast to hydrogen cyanide, which has no irritant properties.

Cyanide salts (for example, NaCN) are compounds that dissociate into the cyanide anion (CN-) and a cation (Na+). Salts are most dangerous following ingestion; onset of action is slower and more prolonged. Cyanide salts generate hydrogen cyanide gas on contact with a strong acid (e.g., sulfuric acid).

Mechanism of Action

Cyanide exists normally in human tissues and is usually metabolized by sulfur in the presence of a hepatic enzyme, rhodanese, into thiocyanante, which is excreted in the urine.

Under normal conditions, the cyanide anion is attracted to iron in the ferric state (Fe+++). In the mitochondrion of the human cell, cytochrome A3 in the cytochrome oxidase complex contains Fe+++. Cyanide is bound to cytochrome A3 and thus inhibits the effect of cytochrome oxidase. This enzyme complex is responsible for the utilization of oxygen within the cell. In the presence of cyanide, even though there is plenty of dissolved oxygen in the blood, the cells cannot use the available oxygen. As a result, cells must utilize anaerobic metabolism, or the creation of energy without the benefit of oxygen, which causes severe lactic acidosis. When cells cannot get enough energy, they die. Cells in the brain and heart are affected initially.

Acute cyanide poisoning occurs after inhaling the agent, but may also occur after drinking solutions of cyanide (it is sometimes used with suicidal intent) or by skin contact with large amounts of liquid cyanide.

Clinical Effects

After inhalation of a low concentration, the patient may become anxious, will often hyperventilate, and typically develops a headache with dizziness and vomiting. Skin color may initially be flushed but may also be normal or cyanotic. A cherry-red skin color is characteristic of cyanide, but this is not always seen. If a victim is exposed to a low concentration of vapor and removed from the source of the cyanide, the symptoms should not progress.

In about 15 seconds after inhaling a large amount of cyanide, victims become anxious and start to hyperventilate. Thirty seconds after exposure, the patient may begin to convulse. In 3 to 5 minutes, breathing ceases. Asystole, or cessation of heart activity, occurs in 6 to 10 minutes, followed by death. The patient may have normal sized or dilated pupils. Death can occur within 8 minutes of exposure.

Laboratory

A normal oxygen saturation may be noted when using a pulse oximeter, despite the fact that the patient may be in severe respiratory distress. There is high arterial oxygen content to venous blood because oxygen is not extracted from arterial blood by the cells. Metabolic (lactic) acidosis may also be present from the lack of oxygen to the tissues. Cyanide toxicity can be measured at the hospital by checking serum cyanide concentrations. These values may, however, only be available after a delay of several hours and of no value in the initial management of acute severe poisoning.

Medical Management

Patients who have inhaled significant doses of cyanide must be rapidly treated with appropriate antidotes to prevent brain damage. Cyanide is attracted to iron (Fe+++) in a form of hemoglobin called methemoglobin. In fact, cyanide will preferentially leave the cytochrome oxidase enzyme in the cell and bind to circulating methemoglobin. Drugs such as amyl nitrite and sodium nitrite, which are found in the cyanide treatment kit, increase blood concentrations of methemoglobin and are antidotal. Adding sodium thiosulfate completes the detoxification process.

Patients should be treated with IV saline for hydration; sodium bicarbonate and intubation with hyperventilation should be used for the metabolic acidosis. Oxygenation should be maintained with high-flow oxygen by mask or by endotracheal tube. Monitor and treat significant arrhythmias.

The Cyanide Antidote Kit

This kit (formerly known as the Lily Cyanide Kit and now produced by Taylor Pharmaceuticals) contains amyl nitrite, sodium nitrite, and sodium thiosulfate.

Amyl nitrite

Amyl nitrite is available in perles, more commonly referred to as ampules, which are broken and placed in either a gauze bandage, or in the bag-mask, and inhaled for 15 seconds, then taken away for 15 seconds (although, if the patient is breathing, he probably does not need the antidote). This is the initial step in antidote therapy. Amyl nitrite forms methemoglobin and reduces the elevated total peripheral resistance caused by the acidosis and cyanide. This should be used only until the IV drugs can be given. Inhalation of amyl nitrite will cause orthostatic hypotension. However, if the patient can stand, he or she does not need the antidote.

Sodium nitrite

Sodium nitrite is a strong methemoglobin former that is available for IV use in a dose of 300 mg in 10 cc. This dose is injected over 2 to 4 minutes and has the potential side effect of orthostatic hypotension. Normal saline infusion and supine posture can help to correct the hypotension. However, if patients can stand, they do not need the sodium nitrite. The pediatric dosage is 0.2 cc/kg, not to exceed 10 cc.

Sodium thiosulfate

This compound is a co-factor for the enzyme rhodanese for detoxification (to change cyanide to a form that can be excreted by the kidneys). The drug is administered in a 50cc ampule (12.5 gm) over 5 minutes by IV.

Treatment

General:

Remove from the area of exposure and remove clothing

Mild exposure:

If conscious and breathing, give O_2 and IV fluids. Observe and monitor no antidotes are necessary.

Severe exposure:

If unconscious, whether breathing or not, give O_2, and bag-mask ventilate with 100 percent O_2. Cardiac monitor. Oxygen saturation may or may not be normal. Administer the following medications:

Amyl nitrite:

Crush into a 4 x 4 piece of gauze and place over face or in a bag mask (however, if patients are breathing, they probably do not need the drug). Add another ampule every few minutes.
Give only until IV drugs are available.

Sodium nitrite:
When IV established, give 300 mg (10 cc ampule) over 5 minutes for adults. For children, use 0.22 to 0.33 ml/kg of the 3 percent solution. Watch for orthostatic hypotension (however, if patient can stand, they do not need this).

Sodium thiosulfate:
Give 12.5 gm (50 cc) IV (administered after sodium nitrite). For children, use 1.65 ml/kg of the 25 percent solution.

Treatment for Cyanide Exposure				
Patient	**Mild (conscious)**	**Severe (unconscious)**	**Other Treatment**	**Precautions**
Pediatric	If patient is conscious and has no other signs or symptoms, antidotes may not be necessary	Sodium nitrite[1]: 0.12 – 0.33 ml/kg, not to exceed 10 ml of 3% solution[2] slow IV over no less than 5 minutes, or slower if hypotension develops and Sodium thiosulfate: 1.65 ml/kg of 25% solution IV over 10 - 20 minutes	For sodium nitrite induced orthostatic hypotension, normal saline infusion and supine position are recommended. If still apneic after antidote administration, consider sodium bicarbonate for severe acidosis.	Victims whose clothing or skin is contaminated with hydrogen cyanide liquid or solution can secondarily contaminate response and hospital personnel by direct contact or through off-gassing vapors. Avoid dermal contact with cyanide-contaminated victims or with gastric contents of victims who may have ingested cyanide-containing materials. Victims exposed to hydrogen cyanide gas only, do not pose a contamination risk to rescuers or health care providers. If the patient is a victim of recent smoke inhalation (may have high carboxyhemoglobin levels) administer the sodium thiosulfate only.
Adult	If patient is conscious and has no other signs or symptoms, antidotes may not be necessary	Sodium nitrite[1]: 10 - 20 ml of 3% solution[2] slow IV over no less than 5 minutes, or slower if hypotension develops and Sodium thiosulfate: 50 ml of 25% solution IV over 10 - 20 minutes		
If sodium nitrite is unavailable, administer amyl nitrite by inhalation from crushable ampules. Available in Pasadena Cyanide Antidote Kit, formerly Lilly Cyanide Kit.				

PULMONARY INTOXICANTS

Pulmonary intoxicants cause severe life-threatening lung injury after inhalation. These effects are generally delayed several hours after exposure. Treatment is usually supportive and may require advanced intensive care techniques including intubation, use of a mechanical ventilation and PEEP. Pulmonary intoxicants included with this group are phosgene and chlorine.

Phosgene

Phosgene is widely used today in the manufacturing of dyes, coal tar, pesticides, and pharmaceuticals. It was widely used in WWI until mustard was introduced on the battlefield.

The Bhopal, India disaster of 1984, at a Union Carbide plant, involved the release of 50,000 pounds of methylisocyanate. This chemical is composed of phosgene and methylamine. There were 150,000 people affected, 10,000 severely injured, and 3,300 killed. The effects of the release were thought to be due to a combination of isocyanate and phosgene.

Characteristics

Phosgene has a characteristic odor of freshly mown hay and is four times heavier than air. It is a gas above 47 ºF, and is principally a hazard by inhalation.

Mechanism of Action and Clinical Effects

Phosgene dissolves slowly in water to form carbon dioxide and hydrochloric acid (HCl). In contact with the moist mucosa the HCl causes a transient irritation of the eyes, nose, sinuses, and throat. It can also irritate the upper airway and bronchi, causing a dry cough. However, the primary damage from phosgene is from the carbonyl group, which destroys the alveolar capillary membrane. (Perflouroisobutylene, PFIB, the combustion product of burning Teflon, found in many military vehicles, has a similar action as phosgene, but is more toxic.)

Phosgene penetrates poorly into the airways due to its poor water solubility. There is a symptom-free period of 2 to 24 hours. Over the first several hours, the carbonyl group from the phosgene attacks the surface of the alveolar capillaries. Eventually, this causes the leakage of serum from the capillaries in the lung into the alveoli and interstitial space. The fluid fills the tissues, causing severe hypoxia and apnea. As the fluid leaks into the alveoli, massive amounts of fluid (up to 1 liter per hour) pour out of the circulation. The patient develops a severe non-cardiogenic pulmonary edema.

Medical Management and Treatment

The leakage of fluid in the lungs causes volume depletion. Although the patient may clinically look like traditional heart failure, DO NOT USE DIURETICS. These patients are volume depleted. Treat hypotension with fluids. These patients may require intubation and the use of PEEP.

In the hospital, the initial examination of a patient, symptomatic or not, should include – (as a minimum): auscultation, chest x-ray, and arterial blood gases. If the victim develops severe dyspnea due to upper airway irritation, early intubation should be considered to manage oxygen delivery and to prevent laryngeal spasm. The airway should be suctioned frequently to remove secretions. According to some authorities, antibiotic use should be guided by Gram stain and culture results. Another source recommends prophylactic antibiotics, as autopsy studies show uniform evidence of pneumonia and bronchitis.

Ventilator management, PEEP, and oxygen administration might require consultation with a pulmonologist. Fluid hydration may be necessary to treat the hypotension, bradycardia, or impending renal failure. Diuretics such as Lasix are contraindicated because of the hypotension and the noncardiac nature of the pulmonary edema. Standard bronchodilators will usually control bronchospasm, but if not, steroids may be needed for this purpose. Routine steroid use is controversial, but steroids seemed to offer some efficacy after the Bhopal tragedy. Once the patient recovers, there should be little residual pulmonary effect.

Chlorine

Chlorine is a significant irritant to the eyes and respiratory tract. It is widely used in the manufacture of chemicals, plastics, and paper and is commonly used in swimming pools and laboratories. Industrial exposures have produced large numbers of injuries.

Characteristics

Chlorine is a greenish-yellow gas that has a characteristic pungent odor that is irritating to the nasal mucosa. It is transported as a liquid and is less alkaline than ammonia.

Mechanism of Action and Clinical Effects

Chlorine injures cells by reacting with water, producing hydrochloric acid (irritating) and free oxygen radicals (attack cells). It is toxic to any body surface including the eyes, skin, respiratory tract, and GI tract. Chlorine gas is 30 times more irritating to the respiratory mucosa than HCl.

In seconds after the exposure, there are symptoms of irritation to the eyes, nose, and throat. This is followed by irritation of the respiratory tract with coughing, shortness of breath, wheezing, chest pain,

and sputum production. Initial respiratory distress is followed in 12 to 24 hours by noncardiogenic pulmonary edema. Sudden death is usually due to severe hypoxia and cardiac arrest.

Medical Management and Treatment

Move exposed victims away from the source of exposure. If the victim has no complaints, probably no treatment will be necessary.

Toxicity to skin and eyes should be treated with copious flushing with water. Irritation of the respiratory tract is treated with oxygen, cool mist to moisten the damaged mucosa, and bronchodilators to resolve bronchospasm.

Intubation, mechanical ventilation, and assessment of hydration may be required. Bronchoscopy may be useful to remove mucosal plugs.

Ammonia

Characteristics

Ammonia is a colorless, highly water-soluble, alkaline gas that has a pungent odor. It is widely used industrially in the U.S. with over 500,000 workers potentially exposed annually. It is used as an agricultural fertilizer and is used in the manufacture of explosives, dyes, and plastics.

Mechanism of Action

Ammonia is rapidly absorbed by mucosal surfaces and causes damage to the eyes, oral cavity, throat, and lungs. When mixed with water, it forms a corrosive agent, ammonium hydroxide (NH_4OH) that causes considerable damage in the form of liquefaction necrosis. Due to its high water solubility, ammonia penetrates rapidly into tissue. Household ammonia generally has a pH less than 12 and generally causes limited damage to eyes or mucosa. Anhydrous ammonia is an industrial chemical that has a very high pH and is extremely corrosive and can cause severe damage to the eyes, lungs, and skin.

Clinical Effects

Ophthalmic

Initially, ammonia causes burning, tearing, and severe pain. It has a tremendous capacity to penetrate the eye, causing corneal opacification and lens damage leading to cataract formation.

Pulmonary
Mild exposure causes cough, shortness of breath, chest pain, wheezing, and laryngitis. Higher exposure can cause hypoxia, chemical pneumonia (pneumonitis), and hemorrhage. This will gradually improve over 72 hours. If the patient survives the first 24 hours, recovery is probable.

Integumentary
Pain, blister formation, and possibly deep burns similar to frostbite can occur.

Gastrointestinal
If ammonia is ingested, severe mouth pain, cough, abdominal pain, nausea, and vomiting can occur. Severe edema of lips and mouth is seen. The patient should be examined to make certain that laryngeal irritation does not cause airway obstruction. Esophageal stricture and perforation is common.

Medical Management

After the patient has been removed from the area of exposure, decontamination should be started immediately in the field.

General Management

Remove all clothing and wash skin and hair with soap and large amounts of water for 15 to 20 minutes.

Cover burns with a sterile dressing.

The eyes should be irrigated continuously with water. A Morgan lens device and topical analgesics will enable continuous eye irrigation therapy. Both of these items should be considered part of an antidote/equipment cache. Slit lamp exam after fluorescein staining will reveal the ocular injury.

Damage to the lungs is common after inhaling anhydrous ammonia, often resulting in non-cardiogenic pulmonary edema. Since the victims may quickly develop shortness of breath and laryngeal swelling, early intubation should be considered to protect the airway.

Riot Control Agents

Irritating agents and lacrimators are chemicals that stimulate lacrimal glands to produce tears. Riot control agents, and tear gas are synonyms for a group of aerosol-dispersed chemicals that produce eye, nose, mouth, skin, and respiratory tract irritation. This class of chemical agents causes involuntary eye closing due to irritation. For police, this is an effective weapon as it can

disable an assailant. It is widely used in the civilian arena for self-protection. The deleterious effect is usually transient, about 30 minutes after exposure.

Riot Control Agents include:

CN (Mace7)
OC (oleoresin capsicum or pepper spray)
Adamsite
CS (tear gas)

Characteristics

Riot control agents are solids. They are sometimes dispersed in a solution that is aerosolized and can be dispersed from grenade or bomb.

Some police SWAT teams have small grenades that contain rubber pellets and/or CS. CN (the active ingredient in Mace7) has caused several deaths from pulmonary injury. CS is less toxic.

Capsicum, or pepper spray, is derived from the oleoresin capsicum in certain peppers. It is also used as an over-the-counter topical pain medication.

Adamsite is an irritating and vomiting agent that acts very similarly to CN and CS. The onset of its effects is delayed for minutes, compared to seconds for CN and CS. In addition, adamsite does not cause skin irritation.

Clinical Effects

Pain, burning, and irritations of exposed mucous membranes comprise the clinical picture.

Ophthalmic

Blepharospasm, or spasm of the muscle that causes eyelid closure, causes very transient blindness due to the closed eyelids. Vision, however, is not impaired once the eyes are opened. They cause tearing, conjunctival injection, and redness.

Pulmonary

Upper airway (mouth, nose)
Can cause nasal discharge, sneezing and burning.

Lower airway (lungs, bronchi)
Can cause coughing, shortness of breath, and chest tightness. Bronchospasm and wheezing can occur for hours after the exposure.

Integumentary

All can cause burning and redness, and it is claimed that O. capsicum has fewer tendencies to

cause dermatitis. After exposure to large amounts of CS and CN, the onset of a more severe dermatitis with erythema and blisters may be delayed for 4 to 6 hours after exposure. These more severe effects occur under high temperature conditions with high humidity and large amounts of agent contacting the skin.

Cardiovascular
Increased blood pressure and heart rate are probably a response to anxiety.

Medical Management

The effects of the riot control agents will rarely last longer than 30 minutes, although the skin redness or erythema may last longer. In fact, in non- terrorist situations, most people will not seek medical care. Less than 1 percent will have eye, airway, or skin complaints severe enough to be medically assessed. A higher percentage might seek care because of anxiety and panic.

There is no antidote available for these agents. Treatment is supportive and directed towards alleviating symptoms which are not usually severe.

Ophthalmic
Should be irrigated copiously with water or saline. Remove contact lenses. Utilize slit lamp exam to make certain that all solid particle foreign bodies are removed. Follow-up with ophthalmologist is recommended.

Pulmonary
Treat wheezing with bronchodilators or steroids if standard bronchodilators fail. Oxygen therapy if indicated. Most symptoms should be maximal within an hour or two.

Integumentary
Most skin exposures require little more than reassurance. With prolonged pain, decontaminate with soap and water or a solution containing a carbonate and/or a bicarbonate. Do NOT use bleach. The delayed onset dermatitis should be managed with frequent irrigation and soothing ointments or creams.

TRIAGE OF CHEMICAL AGENT CASUALTIES

	Nerve Agent	Mustard	Pulmonary Intoxicants
Immediate	Unconscious or convulsing casualties, or those with major disorders of two or more body systems are triaged as immediate. Immediate treatment should include antidote administration and positive pressure ventilation to preserve airways. Rapid intervention will result in an improved outcome.	Patients with moderate to severe pulmonary signs and symptoms are classified as immediate.	Patients who require immediate attention are those who develop noncardiogenic pulmonary edema within 6 hours after exposure to a pulmonary intoxicant such as phosgene
Delayed	Nerve agent casualties are categorized as delayed if their initial symptoms are improving. Antidote treatment of these patients depends on the amount of antidote available. If supplies are limited, then immediate patients will be treated first. The delayed category is also used for patients recovering from exposure after treatment who are conscious and have improved respiratory status. These patients may need additional treatment and need to be observed for several hours.	Most mustard casualties are triaged as delayed, including those with burns covering 5 to 50 percent of their body surface area (BSA) or with eye involvement.	Delayed casualties are those who develop cough and dyspnea more than 6 hours after exposure. These casualties should be admitted and observed for the development of latent pulmonary edema.
Minimal	The minimal nerve agent casualty is walking and talking and indicates intact breathing and circulation. These patients may be able to assist with other patients and/or decontamination.	Casualties with burns of less than 5 percent BSA are minimal.	
Expectant	The patient who has been apneic for more than 5 minutes and has no pulse or blood pressure is categorized as expectant.	The expectant casualty is the victim with burns greater than 50 percent BSA or no respiration or pulse.	Expectant casualties are those who develop non-cardiogenic pulmonary edema within 6 hours of exposure, in circumstances where, due to limited resources, ICU support is not readily available (i.e. mass casualty circumstance)

Cyanide

Few signs and symptoms are visible except at very high doses. Severe cyanide exposures require rapid intervention and are categorized as immediate. In these patients, convulsions occur after 30 seconds, respiration ceases after 3 to 5 minutes, and death ensues in 6 to 10 minutes.

Casualties with lower dose exposures have headaches, nausea and vomiting, hyperventilation, and dizziness, and should be categorized as delayed. These symptoms will improve if the patient is removed from the source of exposure.

CHEMICAL AGENT DETECTION

Recognition of a chemical attack is initially based on clinical criteria. This assessment can be augmented by chemical detection. The current technology for the detection of a chemical agent release is limited. Each of the various types of detectors currently available has specific qualifying factors.

In general, chemical agent detection equipment can help to determine the need for and level of PPE required to protect first responders and hospital personnel. It can be used to certify that the victim has been adequately decontaminated to prevent cross-contamination, and as an early warning device to notify authorities and the community of a chemical agent release. Chemical detectors do not, however, replace the need for an adequately supervised decontamination process.

KEY POINTS

To safely respond to a chemical terrorist attack, local communities must develop resources, protocols, and policies that will enable a safe and appropriate response. Patients exposed to hazardous chemicals require evacuation, life-saving intervention e.g., ABCs, antidote therapy if available, and decontamination. First responders and medical personnel must be trained and equipped to safely function in a chemically contaminated environment.

Decontamination

Proper decontamination is the most important first step in treating a patient exposed to chemical agents. Immediate removal of the patient's clothing can remove up to 90 percent of the contaminant. Removed clothing should be bagged, sealed and retained as possible evidence and for proper treatment and/or disposal. After the clothing is removed, the patient's skin and eyes may need to be decontaminated. In most cases, decontamination of skin can be accomplished by gentle and thorough washing with soap and water. For eyes, flush with plenty of water or normal saline solution. Whenever possible, water run-off from decontamination should be contained.

It is important not to abrade the skin during washing and rinsing. This is especially true after exposure to blistering/vesicant agents which bind to skin. These agents may leave the skin compromised and susceptible to further damage. For pulmonary agents or incapacitating agents, a rinse in water alone may be adequate.

Victims contaminated with hydrogen cyanide liquid can secondarily contaminate response personnel by direct contact or through off-gassing vapors. Avoid dermal contact with cyanide-contaminated victims or with the gastric contents of victims who may have ingested cyanide-containing materials. Victims exposed to hydrogen cyanide gas do not pose a contamination risk to rescuers.

Personal protective equipment

Respiratory Protection

Protection from both vapors and particulates may be required when dealing with chemical agent releases. Surgical and N-95 masks will NOT protect against inhalation of vapors. Powered air-purifying respirators (PAPR) are recommended for health care providers performing decontamination procedures.

Dermal Protection

Latex examination gloves provide little protection from most chemical agents and can cause allergies. Chemical resistant suits, nitrile, butyl or neoprene gloves and boots provide splash protection and need to be worn when performing decontamination.

Antidote Therapy for Chemical Weapons Attacks			
Chemical	Antidote	Decontamination (Including removal of clothing)	Other
Nerve Agent	Atropine, 2-PAMCl	Soap and Water	Diazepam (Valium)
Sulfur Mustard	None, Supportive	Soap and Water	Delayed onset, delayed bullae, pulmonary care
Lewisite	BAL, Supportive	Soap and Water	Acute onset, treat acidosis, volume depletion, pseudomembranes
Cyanide	Methemoglobin, Amyl Nitrite, Sodium Nitrite, Sodium Thiosulfate	Soap and Water	Bicarbonate, O_2, fluids, treat acidosis, Sudden loss of consciousness
Phosgene	None, Supportive	Soap and Water	IVF, monitor volume, O_2, early intubation, steroids, watch for pulmonary edema
PFIB	None, Supportive		Monitor, O_2, watch for pulmonary edema
Ammonia	None, Supportive	Irrigate eyes – water only Soap and Water	Milk, bronchodilators, Silvadene, GI endoscopy, watch for mediastinitis, liquefaction
Chlorine	None, Supportive	Irrigate eyes – water only Soap and Water	Bronchodilators, steroids, intubation, bronchoscopy
CN (Mace)	None	Irrigate eyes – water only Soap and Water	Remove foreign body from eye, watch for bronchospasm
CS (Tear gas)	None	Irrigate eyes – water only Soap and Water	
Oleoresin capsicum	None	Irrigate eyes – water only Soap and Water	From chili pepper, dermatitis, eye injury

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Chemical Agents

Biotoxins

Poisons that come from plants or animals

- Abrin
- Ricin
- Saxitoxin
- Tetrodotoxin
- Trichothecene

For the most accurate and up-to-date information, including resources on additional biotoxin agents, please refer to the CDC web site at: http://www.cdc.gov/

TOXIC SYNDROME DESCRIPTION

Ricin or Abrin Poisoning

Summary statement

Ricin is a potent biological toxin that is derived from castor beans. Its mechanism of action in the body is inhibition of protein synthesis. Clinical manifestations are dependent on the route of exposure. Ingestion of ricin typically leads to profuse vomiting and diarrhea followed by multisystem organ failure and possibly death within 36 to 72 hours of exposure. Inhalation of ricin typically leads to respiratory distress, fever, and cough followed by the development of pulmonary edema, hypotension, respiratory failure, and possibly death within 36 to 72 hours.

The amount and route of the exposure to ricin and the premorbid condition of the person exposed will contribute to the time of onset and the severity of illness. For example, the inhalation of ricin would be expected to lead to a quicker onset of poisoning and to cause a more rapid progression of poisoning compared with the ingestion of ricin, given the same exposure amount.

Signs and symptoms of exposure

The following is a more comprehensive list of signs and symptoms that may be encountered in a person exposed to ricin. The list does not convey prioritization or indicate specificity. Also, partial presentations (an absence of some of the following signs/symptoms) do not necessarily imply less severe disease.

Gastrointestinal

- Abdominal pain
- Vomiting
- Diarrhea (nonbloody or bloody)
- Abnormal liver function tests
- Multiple ulcerations and hemorrhages of gastric and small-intestinal mucosa on endoscopy

Respiratory

- Cough
- Chest tightness
- Dyspnea
- Hypoxemia
- Noncardiogenic pulmonary edema

Skin and mucous membranes

- Redness and pain of eyes and skin

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General

- Fever
- Fatigue
- Weakness
- Muscle pain
- Dehydration

Other organs

- Seizures (uncommon)
- Cardiovascular collapse (hypovolemic shock)

Laboratory (nonspecific)

- Metabolic acidosis
- Increased liver function tests
- Increased renal function tests
- Hematuria
- Leukocytosis (two- to five-fold higher than normal value)

Note: The actual clinical manifestations of a ricin or abrin exposure may be more variable than the syndrome described above.

Differential diagnosis

Inhalation:
- Staphylococcal enterotoxin B
- Exposure to pyrolysis byproducts of organofluorines (Teflon, Kevlar)
- Oxides of nitrogen
- Phosgene
- Ozone

Ingestion:
- Enteric pathogens
- Mushrooms
- Caustics
- Iron
- Arsenic
- Colchicine

This toxic syndrome description is based on CDC's best current information.
It may be updated as new information becomes available.

For more information, visit www.bt.cdc.gov/chemical, or call CDC at
800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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FACT SHEET

Facts About Ricin

What ricin is

- Ricin is a poison found naturally in castor beans. If castor beans are chewed and swallowed, the released ricin can cause injury. Ricin can be made from the waste material left over from processing castor beans.
- It can be in the form of a powder, a mist, or a pellet, or it can be dissolved in water or weak acid.
- It is a stable substance under normal conditions, but can be inactivated by heat above 80 degrees Centigrade.

Where ricin is found and how it is used

- Castor beans are processed throughout the world to make castor oil. Ricin is part of the waste "mash" produced when castor oil is made.
- Ricin has been used experimentally in medicine to kill cancer cells.

How you could be exposed to ricin

- It would take a deliberate act to make ricin and use it to poison people. Accidental exposure to ricin is highly unlikely, except through the ingestion of castor beans.
- If made into a partially purified material or refined into a terrorist or warfare agent, ricin could be used to expose people through the air, food, or water.
- In 1978, Georgi Markov, a Bulgarian writer and journalist who was living in London, died after he was attacked by a man with an umbrella. The umbrella had been rigged to inject a poison ricin pellet under Markov's skin.
- In the 1940's the U.S. military experimented with using ricin as a possible warfare agent. In some reports ricin has possibly been used as a warfare agent in the 1980s in Iraq and more recently by terrorist organizations.
    - Ricin poisoning is not contagious. It cannot be spread from person to person through casual contact.

How ricin works

- Ricin works by getting inside the cells of a person's body and preventing the cells from making the proteins they need. Without the proteins, cells die. Eventually this is harmful to the whole body, and death may occur.
- Effects of ricin poisoning depend on whether ricin was inhaled, ingested, or injected.

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Signs and symptoms of ricin exposure

- The major symptoms of ricin poisoning depend on the route of exposure and the dose received, though many organs may be affected in severe cases.
- Initial symptoms of ricin poisoning by inhalation may occur within 8 hours of exposure. Following ingestion of ricin, initial symptoms typically occur in less than 6 hours.
- **Inhalation:** Within a few hours of inhaling significant amounts of ricin, the likely symptoms would be respiratory distress (difficulty breathing), fever, cough, nausea, and tightness in the chest. Heavy sweating may follow as well as fluid building up in the lungs (pulmonary edema). This would make breathing even more difficult, and the skin might turn blue. Excess fluid in the lungs would be diagnosed by x-ray or by listening to the chest with a stethoscope. Finally, low blood pressure and respiratory failure may occur, leading to death. In cases of known exposure to ricin, people having respiratory symptoms that started within 12 hours of inhaling ricin should seek medical care.
- **Ingestion:** If someone swallows a significant amount of ricin, he or she would develop vomiting and diarrhea that may become bloody. Severe dehydration may be the result, followed by low blood pressure. Other signs or symptoms may include hallucinations, seizures, and blood in the urine. Within several days, the person's liver, spleen, and kidneys might stop working, and the person could die.
- Skin and eye exposure: Ricin is unlikely to be absorbed through normal skin. Contact with ricin powders or products may cause redness and pain of the skin and the eyes.
- Death from ricin poisoning could take place within 36 to 72 hours of exposure, depending on the route of exposure (inhalation, ingestion, or injection) and the dose received. If death has not occurred in 3 to 5 days, the victim usually recovers.
- Showing these signs and symptoms does not necessarily mean that a person has been exposed to ricin.

How authorities confirm cases of suspected ricin poisoning

- If we suspect that people have inhaled ricin, a potential clue would be that a large number of people who had been close to each other suddenly developed fever, cough, and excess fluid in their lungs. These symptoms could be followed by severe breathing problems and possibly death.
- If in suspected situations where ricin may have been disseminated, preliminary environmental testing by public health or law enforcement authorities may detect ricin in powders or materials released into the immediate environment. Persons occupying such areas may initially be observed for signs of ricin poisoning.
- No widely available, reliable medical test exists to confirm that a person has been exposed to ricin.

How ricin poisoning is treated

- Because no antidote exists for ricin, the most important factor is avoiding ricin exposure in the first place.
- If exposure cannot be avoided, the most important factor is then getting the ricin off or out of the body as quickly as possible.
- Symptomatic ricin poisoning is treated by giving victims supportive medical care to minimize the effects of the poisoning. The types of supportive medical care would depend on several factors, such as the route by which victims were poisoned (that is, whether poisoning was by inhalation, ingestion, or skin or eye exposure). Care could include such measures as helping victims breathe, giving them intravenous

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fluids (fluids given through a needle inserted into a vein), giving them medications to treat conditions such as seizure and low blood pressure, flushing their stomachs with activated charcoal (if the ricin has been very recently ingested), or washing out their eyes with water if their eyes are irritated.

How you can protect yourself, and what to do if you are exposed to ricin

- First, get fresh air by leaving the area where the ricin was released.
  - If the ricin release was outside, move away from the area where the ricin was released.
  - If the ricin release was indoors, get out of the building.
- If you are near a release of ricin, emergency coordinators may tell you to either evacuate the area or to "shelter in place" inside a building to avoid being exposed to the chemical. For more information on evacuation during a chemical emergency, see "Facts About Evacuation" at http://emergency.cdc.gov/planning/evacuationfacts.asp. For more information on sheltering in place during a chemical emergency, see "Facts About Sheltering in Place" at http://emergency.cdc.gov/planning/shelteringfacts.asp.
- If you think you may have been exposed to ricin, you should remove your clothing, rapidly wash your entire body with soap and water, and get medical care as quickly as possible.
- *Removing your clothing:*
  - Quickly take off clothing that may have ricin on it. Any clothing that has to be pulled over the head should be cut off the body instead of pulled over the head.
  - If you are helping other people remove their clothing, try to avoid touching any contaminated areas, and remove the clothing as quickly as possible.
- *Washing yourself:*
  - As quickly as possible, wash any ricin from your skin with large amounts of soap and water. Washing with soap and water will help protect people from any chemicals on their bodies.
  - If your eyes are burning or your vision is blurred, rinse your eyes with plain water for 10 to 15 minutes. If you wear contacts, remove them and put them with the contaminated clothing. Do not put the contacts back in your eyes (even if they are not disposable contacts). If you wear eyeglasses, wash them with soap and water. You can put your eyeglasses back on after you wash them.
- *Disposing of your clothes:*
  - After you have washed yourself, place your clothing inside a plastic bag. Avoid touching contaminated areas of the clothing. If you can't avoid touching contaminated areas, or you aren't sure where the contaminated areas are, wear rubber gloves, turn the bag inside out and use it to pick up the clothing, or put the clothing in the bag using tongs, tool handles, sticks, or similar objects. Anything that touches the contaminated clothing should also be placed in the bag. If you wear contacts, put them in the plastic bag, too.
  - Seal the bag, and then seal that bag inside another plastic bag. Disposing of your clothing in this way will help protect you and other people from any chemicals that might be on your clothes.
  - When the local or state health department or emergency personnel arrive, tell them what you did with your clothes. The health department or emergency personnel will arrange for further disposal. Do not handle the plastic bags yourself.
- For more information about cleaning your body and disposing of your clothes after a chemical release, see "Chemical Agents: Facts About Personal Cleaning and Disposal of Contaminated Clothing" at http://emergency.cdc.gov/planning/personalcleaningfacts.asp.

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- If someone has ingested ricin, do not induce vomiting or give fluids to drink.
- Seek medical attention right away. Dial 911 and explain what has happened.

How you can get more information about ricin

You can contact one of the following:

- Regional poison control center (1-800-222-1222)
- Centers for Disease Control and Prevention
  - Public Response Hotline (CDC)
    - 800-CDC-INFO
    - 888-232-6348 (TTY)
  - Emergency Preparedness and Response Web site (emergency.cdc.gov/)
  - E-mail inquiries: cdcinfo@cdc.gov

This fact sheet is based on CDC's best current information. It may be updated as new information becomes available.

CASE DEFINITION

Saxitoxin

Clinical description

Exposure to saxitoxin might cause numbness of the oral mucosa within 30 minutes after ingestion. In severe poisoning, signs and symptoms typically progress rapidly, including parasthesias, a floating sensation, muscle weakness, vertigo, and cranial nerve dysfunction. Respiratory failure and death might occur from paralysis (1-5).

Laboratory criteria for diagnosis

- *Biologic*: A case in which saxitoxin in urine is detected, as determined by a commercial laboratory.

-OR-

- *Environmental*: Detection of saxitoxin in ingested compounds or seafood, as determined by a commercial laboratory or FDA.

Case classification

- *Suspected*: A case in which a potentially exposed person is being evaluated by health-care workers or public health officials for poisoning by a particular chemical agent, but no specific credible threat exists.
- *Probable*: A clinically compatible case in which a high index of suspicion (credible threat or patient history regarding location and time) exists for saxitoxin exposure, or an epidemiologic link exists between this case and a laboratory-confirmed case.
- *Confirmed*: A clinically compatible case in which laboratory tests have confirmed exposure.

The case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and nonspecific laboratory evidence of a particular chemical was present or a 100% certainty of the etiology of the agent is known.

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CASE DEFINITION

Tetrodotoxin

Clinical description

The consumption of toxic amounts of tetrodotoxin results primarily in neurologic and gastrointestinal signs and symptoms. In severe poisoning, dysrhythmias, hypotension, and even death might occur (1, 2). If a rapid onset of one of the following neurologic and gastrointestinal signs or symptoms occurs, the clinical description for tetrodotoxin poisoning has been met: 1) oral paresthesias (might progress to include the arms and legs), 2) cranial nerve dysfunction, 3) weakness (might progress to paralysis), or 4) nausea or vomiting.

Laboratory criteria for diagnosis

- *Biologic*: No biologic marker for tetrodotoxin exposure is available.
- *Environmental*: No method for detection of tetrodotoxin in environmental samples is available commercially.

Case classification

- *Suspected*: A case in which a potentially exposed person is being evaluated by health-care workers or public health officials for poisoning by a particular chemical agent, but no specific credible threat exists.
- *Probable*: A clinically compatible case in which a high index of suspicion (credible threat or patient history regarding location and time) exists for tetrodotoxin exposure, or an epidemiologic link exists between this case and a laboratory-confirmed case.
- *Confirmed*: A clinically compatible case in which laboratory tests (not available for tetrodotoxin) are confirmatory.

The case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and nonspecific laboratory evidence of a particular chemical was present or a 100% certainty of the etiology of the agent is known.

Additional resources

1. Sims JK, Ostman DC. Pufferfish poisoning: emergency diagnosis and management of mild human tetrodotoxication. Ann Emerg Med 1986;15:1094-8.
2. Torda TA, Sinclair E, Ulyatt DB. Puffer fish (tetrodotoxin) poisoning: clinical record and suggested management. Med J Aust 1973;1:599-602.

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CASE DEFINITION

Trichothecene Mycotoxins

Clinical description

Trichothecene mycotoxins might be weaponized and dispersed through the air or mixed in food or beverages. Initially, route-specific effects are typically prominent. Dermal exposure leads to burning pain, redness, and blisters, and oral exposure leads to vomiting and diarrhea. Ocular exposure might result in blurred vision, and inhalational exposure might cause nasal irritation and cough. Systemic symptoms can develop with all routes of exposure and might include weakness, ataxia, hypotension, coagulopathy, and death (1).

Laboratory criteria for diagnosis

- *Biologic*: Selected commercial laboratories are offering immunoassays to identify trichothecenes or trichothecene-specific antibodies in human blood or urine (2, 3). However, these procedures have not been analytically validated and are not recommended.
- *Environmental*: Detection of trichothecene mycotoxins in environmental samples, as determined by FDA.

As a result of indoor air-quality investigations involving mold and potentially mold-related health effects, mycotoxin analyses of bulk environmental samples are now commercially available through environmental microbiology laboratories in the United States (4). Studies have not been done to determine the background level of trichothecenes in non-moldy homes and office buildings or nonagricultural outdoor environments. Therefore, the simple detection of trichothecenes in environmental samples does not invariably indicate an intentional contamination.

Case classification

- *Suspected*: A case in which a potentially exposed person is being evaluated by health-care workers or public health officials for poisoning by a particular chemical agent, but no specific credible threat exists.
- *Probable*: A clinically compatible case in which a high index of suspicion (credible threat or patient history regarding location and time) exists for trichothecene mycotoxins exposure, or an epidemiologic link exists between this case and a laboratory-confirmed case.
- *Confirmed*: A clinically compatible case in which laboratory tests of environmental samples have confirmed exposure.

The case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and nonspecific laboratory evidence of a particular chemical was present or a 100% certainty of the etiology of the agent is known.

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Additional resources

1. Wannemacher RW Jr, Wiener SL. Trichothecene mycotoxins. In: Zajtchuk R, Bellamy RF, eds. Textbook of military medicine: medical aspects of chemical and biologic warfare. Washington, DC: Office of the Surgeon General at TMM Publications, Borden Institute, Walter Reed Army Medical Center; 1997:655-77.
2. Croft WA, Jastromski BM, Croft AL, Peters HA. Clinical confirmation of trichothecene mycotoxicosis in patient urine. J Environ Biol 2002;23:301-20.
3. Vojdani A, Thrasher HD, Madison RA, Gray MR, Heuser G, Campbell AW. Antibodies to molds and satratoxin in individuals exposed in water-damaged buildings. Arch Environ Health. 2003;58:421-32.
4. Tuomi T, Reijula K, Johnsson T, et al. Mycotoxins in crude building materials from water-damaged buildings. Appl Environ Microbiol 2000;66:1899-904.

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Chemical Agents

Blister Agents/Vesicants

Chemicals that severely blister the eyes, respiratory tract, and skin on contact

- Mustards
    - Sulfur mustard (H) (mustard gas)

For the most accurate and up-to-date information, including resources on additional vesicant agents, please refer to the CDC web site at: http://www.cdc.gov/

VESICANTS

ALL SUSPECT CASES OF CHEMICAL EXPOSURE MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY DIVISION OF EPIDEMIOLOGY

During Business Hours:	619-515-6620
After Hours (County Communications):	858-565-5255

Agents:

- HD & H - Mustard
- L – Lewisite
- CX – Phosgene Oxime

Biological Effects:

- Vapor and liquid are threats to exposed skin and mucus membranes.
- Cell damage, tissue necrosis, toxic byproducts, metabolic acidosis, secondary infections, pulmonary insufficiency

Clinical:

- Mustard
  - Onset – Delayed 2-48 hours
  - Skin: Erythema, small vesicles: later coalesce, blisters/bulla, possible coagulation necrosis with liquid exposure
  - Eyes: Conjunctivitis, corneal abrasions
  - Airway: Hemorrhage and pain resulting in secretions, cough, shortness of breath, hoarseness and stridor
  - GI effects
  - Bone marrow suppression
- Lewisite
  - Onset – Immediate burning and pain on contact.
  - Skin and mucous membrane irritation, erythema, blisters, skin, eye and airway injury.
- Phosgene Oxime
  - Manifests similar to Lewisite with wheal-like skin lesions.

Diagnosis:

- Primary diagnosis should be made from clinical manifestation.

Decontamination:

- Medical personnel must wear personal protective equipment prior to any contact with patients.
- Early decontamination protects the patient; late decontamination protects the medical personnel and facility.
- Wash with copious amounts of soap and water.

Treatment:

- Immediate decontamination, symptomatic treatment, pulmonary support, analgesics. Topical Silver Sulfadiazine can be used on large de-roofed blistered regions. If Silver Sulfadiazine is unavailable, use sterile petroleum.
- In the case of Lewisite parenteral British Anti-lewisite (BAL) may be used.

TOXIC SYNDROME DESCRIPTION

Vesicant/Blister Agent Poisoning

The purpose of this document is to enable health care workers and public health officials to recognize when a chemical event has poisoned people by exposing them to vesicants/blister agents. Vesicants include distilled mustard (HD), mustard gas (H), lewisite, mustard/lewisite, mustard/T, nitrogen mustard, phosgene oxime, sesqui mustard, and sulfur mustard.

Summary

Vesicants, also referred to as "blister agents," were the most commonly used chemical warfare agents during World War I. The most likely routes of exposure are inhalation, dermal contact, and ocular contact. Vesicants are highly reactive chemicals that combine with proteins, DNA, and other cellular components to result in cellular changes immediately after exposure.

Depending on the vesicant, clinical effects may occur immediately (as with phosgene oxime or lewisite) or may be delayed for 2 to 24 hours (as with mustards). Following exposure, the most commonly encountered clinical effects include dermal (skin erythema and blistering), respiratory (pharyngitis, cough, dyspnea), ocular (conjunctivitis and burns), and gastrointestinal (nausea and vomiting).

The amount and route of exposure to the vesicant, the type of vesicant, and the premorbid condition of the person exposed will contribute to the time of onset and the severity of illness. For example, ingestion of a vesicant leads to gastrointestinal symptoms more prominent than those that would result from inhalation exposure to the same dose and type of vesicant.

Signs and symptoms

The following is a more comprehensive list of signs and symptoms that may be encountered in a person exposed to a vesicant. Signs and symptoms are not listed in order of presentation or specificity. Also, partial presentations (an absence of some of the following signs/symptoms) do not necessarily imply less severe disease.

Respiratory signs and symptoms

- Clear rhinorrhea
- Nasal irritation/pain
- Sore throat
- Cough
- Dyspnea (shortness of breath)
- Chest tightness
- Tachypnea
- Hemoptysis

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Dermal signs and symptoms

- Itching
- Immediate blanching (phosgene oxime)
- Erythema (immediate with lewisite and phosgene oxime, may be delayed for 2 to 24 hours with mustards)
- Blisters (within 1 hour with phosgene oxime, delayed for 2 to 12 hours with lewisite, delayed for 2 to 24 hours with mustards)
- Necrosis and eschar (over a period of 7 to 10 days)

Ocular signs and symptoms

- Conjunctivitis
- Lacrimation
- Eye pain/burning
- Photophobia
- Blurred vision
- Eyelid edema
- Corneal ulceration
- Blindness

Cardiovascular signs

- Hypotension (with high-dose exposure to lewisite)
- Atrioventricular block and cardiac arrest (with high-dose exposure)

Gastrointestinal signs and symptoms (prominent if ingestion is a route of exposure)

- Abdominal pain
- Nausea and vomiting
- Hematemesis
- Diarrhea (sometimes bloody)

Central nervous system signs and symptoms (with exposure to high doses)

- Tremors
- Convulsions
- Ataxia
- Coma

Laboratory findings suggestive of vesicant exposure

Although it is a nonspecific finding, leukopenia can indicate vesicant exposure. It usually begins 3 to 5 days after exposure. With a white blood cell count < 500, the prognosis is poor.

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Differential diagnosis

- Barbiturates
- Chemotherapeutic agents
- Carbon monoxide
- Stevens-Johnson syndrome
- Staphylococcus scalded skin syndrome
- Toxic epidermal necrolysis
- Bullous pemphigoid
- Pemphigus vulgaris
- Other chemical burns (such as with strong acids, bases, or corrosives)

Note: The actual clinical manifestations of a vesicant exposure may be more variable than the syndrome described above.

This toxic syndrome description is based on CDC's best current information.
It may be updated as new information becomes available.

For more information, visit www.bt.cdc.gov/chemical, or call CDC at
800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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FACT SHEET

Facts About Sulfur Mustard

What sulfur mustard is

- Sulfur mustard is a type of chemical warfare agent. These kinds of agents are called vesicants or blistering agents, because they cause blistering of the skin and mucous membranes on contact.
- Sulfur mustard is also known as "mustard gas or mustard agent," or by the military designations H, HD, and HT.
- Sulfur mustard sometimes smells like garlic, onions, or mustard and sometimes has no odor. It can be a vapor (the gaseous form of a liquid), an oily-textured liquid, or a solid.
- Sulfur mustard can be clear to yellow or brown when it is in liquid or solid form.

Where sulfur mustard is found and how it is used

- Sulfur mustard is not found naturally in the environment.
- Sulfur mustard was introduced in World War I as a chemical warfare agent. Until recently, it was available for use in the treatment of a skin condition called psoriasis. Currently, it has no medical use.

How people can be exposed to sulfur mustard

- If sulfur mustard is released into the air as a vapor, people can be exposed through skin contact, eye contact, or breathing. Sulfur mustard vapor can be carried long distances by wind.
- If sulfur mustard is released into water, people can be exposed by drinking the contaminated water or getting it on their skin.
- People can be exposed by coming in contact with liquid sulfur mustard.
- Sulfur mustard can last from 1 to 2 days in the environment under average weather conditions and from weeks to months under very cold conditions.
- Sulfur mustard breaks down slowly in the body, so repeated exposure may have a cumulative effect (that is, it can build up in the body).

How sulfur mustard works

- Adverse health effects caused by sulfur mustard depend on the amount people are exposed to, the route of exposure, and the length of time that people are exposed.
- Sulfur mustard is a powerful irritant and blistering agent that damages the skin, eyes, and respiratory (breathing) tract.
- It damages DNA, a vital component of cells in the body.
- Sulfur mustard vapor is heavier than air, so it will settle in low-lying areas.

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Immediate signs and symptoms of sulfur mustard exposure

- Exposure to sulfur mustard is usually not fatal. When sulfur mustard was used during World War I, it killed fewer than 5% of the people who were exposed and got medical care.
- People may not know right away that they have been exposed, because sulfur mustard often has no smell or has a smell that might not cause alarm.
- Typically, signs and symptoms do not occur immediately. Depending on the severity of the exposure, symptoms may not occur for 2 to 24 hours. Some people are more sensitive to sulfur mustard than are other people, and may have symptoms sooner.
- Showing these signs and symptoms does not necessarily mean that a person has been exposed to sulfur mustard.
- Sulfur mustard can have the following effects on specific parts of the body:
  - *Skin*: redness and itching of the skin may occur 2 to 48 hours after exposure and change eventually to yellow blistering of the skin.
  - *Eyes*: irritation, pain, swelling, and tearing may occur within 3 to12 hours of a mild to moderate exposure. A severe exposure may cause symptoms within 1 to 2 hours and may include the symptoms of a mild or moderate exposure plus light sensitivity, severe pain, or blindness (lasting up to 10 days).
  - *Respiratory tract*: runny nose, sneezing, hoarseness, bloody nose, sinus pain, shortness of breath, and cough within 12 to 24 hours of a mild exposure and within 2 to 4 hours of a severe exposure.
  - *Digestive tract*: abdominal pain, diarrhea, fever, nausea, and vomiting.
- Showing these signs and symptoms does not necessarily mean that a person has been exposed to sulfur mustard.

What the long-term health effects may be

- Exposure to sulfur mustard liquid is more likely to produce second- and third- degree burns and later scarring than is exposure to sulfur mustard vapor. Extensive skin burning can be fatal.
- Extensive breathing in of the vapors can cause chronic respiratory disease, repeated respiratory infections, or death.
- Extensive eye exposure can cause permanent blindness.
- Exposure to sulfur mustard may increase a person's risk for lung and respiratory cancer.

How people can protect themselves and what they should do if they are exposed to sulfur mustard

- Because no antidote exists for sulfur mustard exposure, the best thing to do is avoid it. Immediately leave the area where the sulfur mustard was released. Try to find higher ground, because sulfur mustard is heavier than air and will settle in low-lying areas.
- If avoiding sulfur mustard exposure is not possible, rapidly remove the sulfur mustard from the body. Getting the sulfur mustard off as soon as possible after exposure is the only effective way to prevent or decrease tissue damage to the body.
- Quickly remove any clothing that has liquid sulfur mustard on it. If possible, seal the clothing in a plastic bag, and then seal that bag inside a second plastic bag.
- Immediately wash any exposed part of the body (eyes, skin, etc.) thoroughly with plain, clean water. Eyes need to be flushed with water for 5 to 10 minutes. Do NOT cover eyes with bandages, but do protect them with dark glasses or goggles.
- If someone has ingested sulfur mustard, do NOT induce vomiting. Give the person milk to drink.
- Seek medical attention right away. Dial 911 and explain what has happened.

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How sulfur mustard exposure is treated

The most important factor is removing sulfur mustard from the body. Exposure to sulfur mustard is treated by giving the victim supportive medical care to minimize the effects of the exposure. Though no antidote exists for sulfur mustard, exposure is usually not fatal.

Where people can get more information about sulfur mustard

For more information about sulfur mustard, people can contact the following:

- Regional poison control center (1-800-222-1222)
- Centers for Disease Control and Prevention
  - Public Response Hotline (CDC)
    - 800-CDC-INFO
    - 888-232-6348 (TTY)
  - Emergency Preparedness and Response Web site (http://www.bt.cdc.gov/)
  - E-mail inquiries: cdcinfo@cdc.gov

This fact sheet is based on CDC's best current information. It may be updated as new information becomes available.

Last reviewed on 03/23/05.

Chemical Agents

Blood Agents

Poisons that affect the body by being absorbed into the blood

- Cyanide

For the most accurate and up-to-date information, including resources on additional blood agents, please refer to the CDC web site at: http://www.cdc.gov/

CYANIDE

ALL SUSPECT CASES OF CHEMICAL AGENTS MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY DIVISION OF EPIDEMIOLOGY

During Business Hours:	619-515-6620
After Hours (County Communications):	858-565-5255

Agent:
- AC – Hydrogen cyanide
- CK – Cyanogen chloride

Biological Effects:
- Reacts with metallic complexes, iron, cobalt, and sulfur donor compounds.
- Carried via the blood to tissues.
- Reacts with iron and HbO2 to form HbCN (cyanohemoglobin).
- Binds to mitochondrial enzyme to inactivate cytochrome oxidase preventing cellular uptake and utilization of oxygen.

Clinical:
- Severe exposure onset 15 seconds to 8 minutes
  - Directly stimulates carotid and aortic chemoreceptors – increases heart rate and blood pressure.
  - Loss of consciousness; seizure
  - Hypoventilation; central apnea
  - Cardiac dysrhythmias and death
- Moderate exposure onset is gradual and delayed (minutes to hours)
  - Transcutaneous/Airborne: Nausea, headache, vertigo, muscular weakness ataxia, nystagmus, confusion
  - Ingestion: Hypersalivation, epigastric pain, hyperthermia, nausea, vomiting

Laboratory Diagnosis:
- Elevated blood cyanide concentration
  - 0.5-1 mcg/ml = mild effects
  - > 2.5mcg/ml = coma, seizures, death
- Metabolic anion gap acidosis
- Increased oxygen content of venous blood greater than normal

Decontamination:
- Skin decontamination is usually not indicated because agents are highly volatile.
- Wet contaminated clothing should be removed and underlying skin be washed with soap and water.

Treatment:
- Treatment should be initiated based on clinical suspicion, NOT awaiting lab results.
- Remove to fresh air
- Administer antidote:
  - Sodium Nitrite 10 ml 3% (30mg/ml) = 300mg IV over 3 minutes; may repeat half of original dose if signs reoccur. Pediatric dose 0.22—0.33 mL/kg 3% solution IV. May consult hemoglobin/dose nomogram.
  - Sodium Thiosulfate 50 ml 25% (250mg/ml) = 12.5g IV over 10 minutes; may repeat half of original dose if signs reoccur. Pediatric dose 1.65 mL/kg IV.
  - Consider Hydroxocobalamin (Cyanokit) adult dose 5 gm IV over 15 minutes if available (in place of nitrites/thiosulfate).
- Supportive care
- Correct acidosis

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FACT SHEET

Facts About Cyanide

What cyanide is

- Cyanide is a rapidly acting, potentially deadly chemical that can exist in various forms.
- Cyanide can be a colorless gas, such as hydrogen cyanide (HCN) or cyanogen chloride (CNCl), or a crystal form such as sodium cyanide (NaCN) or potassium cyanide (KCN).
- Cyanide sometimes is described as having a "bitter almond" smell, but it does not always give off an odor, and not everyone can detect this odor.
- Cyanide is also known by the military designations AC (for hydrogen cyanide) and CK (for cyanogen chloride).

Where cyanide is found and how it is used

- Hydrogen cyanide, under the name Zyklon B, was used as a genocidal agent by the Germans in World War II.
- Reports have indicated that during the Iran-Iraq War in the 1980s, hydrogen cyanide gas may have been used along with other chemical agents against the inhabitants of the Kurdish city of Halabja in northern Iraq.
- Cyanide is naturally present in some foods and in certain plants such as cassava. Cyanide is contained in cigarette smoke and the combustion products of synthetic materials such as plastics. Combustion products are substances given off when things burn.
- In manufacturing, cyanide is used to make paper, textiles, and plastics. It is present in the chemicals used to develop photographs. Cyanide salts are used in metallurgy for electroplating, metal cleaning, and removing gold from its ore. Cyanide gas is used to exterminate pests and vermin in ships and buildings.
- If accidentally ingested (swallowed), chemicals found in acetonitrile-based products that are used to remove artificial nails can produce cyanide.

How people can be exposed to cyanide

- Poisoning caused by cyanide depends on the amount of cyanide a person is exposed to, the route of exposure, and the length of time that a person is exposed.
- Breathing cyanide gas causes the most harm, but ingesting cyanide can be toxic as well.
- Cyanide gas is most dangerous in enclosed places where the gas will be trapped.
- Cyanide gas evaporates and disperses quickly in open spaces, making it less harmful outdoors.
- Cyanide gas is less dense than air, so it will rise.
- Cyanide prevents the cells of the body from getting oxygen. When this happens, the cells die.
- Cyanide is more harmful to the heart and brain than to other organs because the heart and brain use a lot of oxygen.

Immediate signs and symptoms of cyanide exposure

- People exposed to a small amount of cyanide by breathing it, absorbing it through their skin, or eating foods that contain it may have some or all of the following symptoms within minutes:

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  - Rapid breathing
  - Restlessness
  - Dizziness
  - Weakness
  - Headache
  - Nausea and vomiting
  - Rapid heart rate
- Exposure to a large amount of cyanide by any route may cause these other health effects as well:
  - Convulsions
  - Low blood pressure
  - Slow heart rate
  - Loss of consciousness
  - Lung injury
  - Respiratory failure leading to death
- Showing these signs and symptoms does not necessarily mean that a person has been exposed to cyanide.

What the long-term health effects may be

Survivors of serious cyanide poisoning may develop heart and brain damage.

How people can protect themselves and what they should do if they are exposed to cyanide

- First, get fresh air by leaving the area where the cyanide was released. Moving to an area with fresh air is a good way to reduce the possibility of death from exposure to cyanide gas.
  - If the cyanide release was outside, move away from the area where the cyanide was released.
  - If the cyanide release was indoors, get out of the building.
- If leaving the area that was exposed to cyanide is not an option, stay as low to the ground as possible.
- Remove any clothing that has liquid cyanide on it. If possible, seal the clothing in a plastic bag, and then seal that bag inside a second plastic bag. Removing and sealing the clothing in this way will help protect people from any chemicals that might be on their clothes.
- If clothes were placed in plastic bags, inform either the local or state health department or emergency coordinators upon their arrival. Do not handle the plastic bags.
- Rinse the eyes with plain water for 10 to 15 minutes if they are burning or vision is blurred.
- Wash any liquid cyanide from the skin thoroughly with soap and water.
- If cyanide is known to be ingested (swallowed), do not induce vomiting or give fluids to drink.
- Seek medical attention right away. Dial 911 and explain what has happened.

How cyanide poisoning is treated

Cyanide poisoning is treated with specific antidotes and supportive medical care in a hospital setting. The most important thing is for victims to seek medical treatment as soon as possible.

How people can get more information about cyanide

People can contact one of the following:

- Regional poison control center (1-800-222-1222)
- Centers for Disease Control and Prevention

- Public Response Hotline (CDC)
  - (800) 232-4636 (English and Spanish)
  - TTY (888) 232-6358
- Emergency Preparedness and Response Web site (http://www.bt.cdc.gov/)
- E-mail inquiries: cdcinfo@cdc.gov

This fact sheet is based on CDC's best current information. It may be updated as new information becomes available.

Last reviewed on 02/26/03.

The Centers for Disease Control and Prevention (CDC) protects people's health and safety by preventing and controlling diseases and injuries; enhances health decisions by providing credible information on critical health issues; and promotes healthy living through strong partnerships with local, national, and international organizations

For more information, visit www.bt.cdc.gov/chemical, or call CDC at 800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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CASE DEFINITION

Cyanide

Clinical description

Inhalation of cyanide gas or ingestion of cyanide salts typically leads to lethargy or coma (possibly sudden collapse), dyspnea, tachypnea, tachycardia, and hypotension. Severe poisoning results in bradypnea, bradycardia, cardiovascular collapse, and death. Nonspecific laboratory findings include metabolic and lactic acidosis (1-3).

Laboratory criteria for diagnosis

Biologic: A case in which cyanide concentration is higher than the normal reference range (0.02--0.05 µg/mL) in whole blood (3), as determined by a commercial laboratory.

-OR-

Environmental: Detection of cyanide in environmental samples, as determined by NIOSH or FDA.

Case classification

- *Suspected*: A case in which a potentially exposed person is being evaluated by health-care workers or public health officials for poisoning by a particular chemical agent, but no specific credible threat exists.
- *Probable*: A clinically compatible case in which a high index of suspicion (credible threat or patient history regarding location and time) exists for cyanide exposure, or an epidemiologic link exists between this case and a laboratory-confirmed case.
- *Confirmed*: A clinically compatible case in which laboratory tests have confirmed exposure.

The case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and nonspecific laboratory evidence of a particular chemical was present or a 100% certainty of the etiology of the agent is known.

Additional resources

1. Curry SC. Hydrogen cyanide and inorganic cyanide salts. In: Sullivan JB, Krieger GR, eds. Hazardous materials toxicology: clinical principles of environmental health. Baltimore, MD: Williams & Wilkins; 1992:698-710.
2. Baskin SI, Brewer TG. Cyanide poisoning. In: Zajtchuk R, Bellamy RF, eds. Textbook of military medicine: medical aspects of chemical and biological warfare. Washington, DC: Office of the Surgeon General at TMM Publications, Borden Institute, Walter Reed Army Medical Center; 1997:271-86.
3. Kerns W II, Isom G, Kirk MA. Cyanide and hydrogen sulfide. In: Goldfrank LR, Flomenbaum NE, Lewin NA, Howland MA, Hoffman RS, Nelson LS, eds. Goldfrank's toxicologic emergencies. 7th ed. New York, NY: McGraw-Hill; 2002:1498-514.

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Chemical Agents

Choking/Lung/Pulmonary Agents

Chemicals that cause severe irritation or swelling of the respiratory tract (lining of the nose, throat, and lungs)

- Chlorine (CL)

For the most accurate and up-to-date information, including resources on additional pulmonary agents, please refer to the CDC web site at: http://www.cdc.gov/

PULMONARY AGENTS

ALL SUSPECT CASES OF CHEMICAL AGENTS MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY DIVISION OF EPIDEMIOLOGY

During Business Hours: 619-515-6620
After Hours (County Communications): 858-565-5255

Agents:

- CG - Phosgene
- Chlorine

Biological Effects:

- Carbonyl reacts with primary amines, hydroxyl groups, enzymes, other groups affecting cell wall stability; particular effect on capillary membrane permeability causing leak of plasma into alveoli to produce pulmonary edema.
- In terminal bronchioles there is a constriction, swelling, necrosis, perivascular edema, dilatation/engorgement of capillaries and veins, interstitial edema, epithelial sloughing and plugging by necrotic cells or mucus.

Clinical:

- Time of onset and rapidity of progression depend on intensity of exposure (10 minutes to 24 hours).
- Signs and symptoms include: eye and airway irritation, dyspnea, chest tightness, and delayed pulmonary edema.
- Sequence of respiratory effects;
  - 10 minutes to 2 hours: nasal irritation, chest tightness, cough, lacrimation
  - 30 minutes to 24 hours: severe cough, laryngospasm, pulmonary edema (NOTE: time of onset of symptoms correlate well with the likelihood of mortality. An exposed individual who is "normal" by symptoms, examination (CXR, ABG) at 4 hours is unlikely to die from the exposure although abnormal pulmonary function may develop up to 24 hours later).

Laboratory Diagnosis:

- Radiological
  - Early CXR may exhibit hyperinflation (20-30 minute).
  - Post exposure 2-12 hours, CXR may reflect pulmonary edema (non-cardiac) / ARDS. Resolution of pulmonary edema by CXR delayed after offset of symptoms by 12-24 hours.
- Arterial Blood Gases
  - Hyperventilation occurs early (5-15 minutes) resulting in a mild respiratory alkalosis (pCO2 3-35).
  - Hypoxemia occurs with progression of interstitial edema generally at the same time course as radiologic changes.

Decontamination:

- Vapor – fresh air
- Liquid – copious water irrigation

Treatment:

- Prophylaxis: None
- Post Exposure
  - No specific chemical therapy.
  - No evidence that steroids are useful after exposure.
  - No evidence that antibiotics prevent bacterial superinfection.
- Acute Therapy
  - Remove from exposure and place patient at rest.
  - Supportive care as need (ABC).
  - Observe for at least 4 hours.
  - If physical exam, CXR and ABG show no symptoms at four hours post exposure, patient is unlikely to die.
  - Recall that abnormalities requiring care may still appear up to 24-48 hours after exposure. Early application of positive airway pressure (PEEP) appears useful; however, intubation and aggressive pulmonary management may be required.

FACT SHEET

Facts About Chlorine

What chlorine is

- Chlorine is an element used in industry and found in some household products.
- Chlorine is sometimes in the form of a poisonous gas. Chlorine gas can be pressurized and cooled to change it into a liquid so that it can be shipped and stored. When liquid chlorine is released, it quickly turns into a gas that stays close to the ground and spreads rapidly.
- Chlorine gas can be recognized by its pungent, irritating odor, which is like the odor of bleach. The strong smell may provide an adequate warning to people that they have been exposed.
- Chlorine gas appears to be yellow-green in color.
- Chlorine itself is not flammable, but it can react explosively or form explosive compounds with other chemicals such as turpentine and ammonia.

Where chlorine is found and how it is used

- Chlorine was used during World War I as a choking (pulmonary) agent.
- Chlorine is one of the most commonly manufactured chemicals in the United States. Its most important use is as a bleach in the manufacture of paper and cloth, but it is also used to make pesticides (insect killers), rubber, and solvents.
- Chlorine is used in drinking water and swimming pool water to kill harmful bacteria. It is also as used as part of the sanitation process for industrial waste and sewage.
- Household chlorine bleach can release chlorine gas if it is mixed with other cleaning agents.

How people can be exposed to chlorine

- People's risk for exposure depends on how close they are to the place where the chlorine was released.
- If chlorine gas is released into the air, people may be exposed through skin contact or eye contact. They may also be exposed by breathing air that contains chlorine.
- If chlorine liquid is released into water, people may be exposed by touching or drinking water that contains chlorine.
- If chlorine liquid comes into contact with food, people may be exposed by eating the contaminated food.
- Chlorine gas is heavier than air, so it would settle in low-lying areas.

How chlorine works

- The extent of poisoning caused by chlorine depends on the amount of chlorine a person is exposed to, how the person was exposed, and the length of time of the exposure.
- When chlorine gas comes into contact with moist tissues such as the eyes, throat, and lungs, an acid is produced that can damage these tissues.

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Immediate signs and symptoms of chlorine exposure

- During or immediately after exposure to dangerous concentrations of chlorine, the following signs and symptoms may develop:
    - Coughing
    - Chest tightness
    - Burning sensation in the nose, throat, and eyes
    - Watery eyes
    - Blurred vision
    - Nausea and vomiting
    - Burning pain, redness, and blisters on the skin if exposed to gas, skin injury similar to frostbite if exposed to liquid chlorine
    - Difficulty breathing or shortness of breath (may appear immediately if high concentrations of chlorine gas are inhaled, or may be delayed if low concentrations of chlorine gas are inhaled)
    - Fluid in the lungs (pulmonary edema) within 2 to 4 hours
- Showing these signs or symptoms does not necessarily mean that a person has been exposed to chlorine.

What the long-term health effects are

- Long-term complications from chlorine exposure are not found in people who survive a sudden exposure unless they suffer complications such as pneumonia during therapy. Chronic bronchitis may develop in people who develop pneumonia during therapy.

How people can protect themselves, and what they should do if they are exposed to chlorine

- Leave the area where the chlorine was released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing exposure to chlorine.
    - If the chlorine release was outdoors, move away from the area where the chlorine was released. Go to the highest ground possible, because chlorine is heavier than air and will sink to low-lying areas.
    - If the chlorine release was indoors, get out of the building.
- If you think you may have been exposed, remove your clothing, rapidly wash your entire body with soap and water, and get medical care as quickly as possible.
- *Removing and disposing of clothing:*
    - Quickly take off clothing that has liquid chlorine on it. Any clothing that has to be pulled over the head should be cut off the body instead of pulled over the head. If possible, seal the clothing in a plastic bag. Then seal the first plastic bag in a second plastic bag. Removing and sealing the clothing in this way will help protect you and other people from any chemicals that might be on your clothes.
    - If you placed your clothes in plastic bags, inform either the local or state health department or emergency personnel upon their arrival. Do not handle the plastic bags.
    - If you are helping other people remove their clothing, try to avoid touching any contaminated areas, and remove the clothing as quickly as possible.
- *Washing the body:*
    - As quickly as possible, wash your entire body with large amounts of soap and water. Washing with soap and water will help protect people from any chemicals on their bodies.
    - If your eyes are burning or your vision is blurred, rinse your eyes with plain water for 10 to 15 minutes. If you wear contacts, remove them before rinsing your eyes, and place them in

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the bags with the contaminated clothing. Do not put the contacts back in your eyes. You should dispose of them even if you do not wear disposable contacts. If you wear eyeglasses, wash them with soap and water. You can put the eyeglasses back on after you wash them.

- If you have ingested (swallowed) chlorine, do not induce vomiting or drink fluids.
- Seek medical attention right away. Dial 911 and explain what has happened.

How chlorine exposure is treated

No antidote exists for chlorine exposure. Treatment consists of removing the chlorine from the body as soon as possible and providing supportive medical care in a hospital setting.

How people can get more information about chlorine

People can contact one of the following:

- Regional poison control center (1-800-222-1222)
- Centers for Disease Control and Prevention
  - Public Response Hotline (CDC)
    - (800) 232-4636 (English and Spanish)
    - TTY (888) 232-6358
  - Emergency Preparedness and Response Web site (http://www.bt.cdc.gov/)
  - E-mail inquiries: cdcinfo@cdc.gov

This fact sheet is based on CDC's best current information. It may be updated as new information becomes available.

Last reviewed on 03/23/05.

The Centers for Disease Control and Prevention (CDC) protects people's health and safety by preventing and controlling diseases and injuries; enhances health decisions by providing credible information on critical health issues; and promotes healthy living through strong partnerships with local, national, and international organizations

For more information, visit www.bt.cdc.gov/chemical, or call CDC at 800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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Chemical Agents

Incapacitating Agents

Drugs that make people unable to think clearly or that cause an altered state of consciousness (possibly unconsciousness)

- Fentanyls & other opioids

For the most accurate and up-to-date information, including resources on additional incapacitating agents, please refer to the CDC web site at: http://www.cdc.gov/

CASE DEFINITION

Opioids (Fentanyl, Etorphine, or Others)

Clinical description

Exposure to opioids typically occurs through ingestion but potentially can result from inhalation, if opioids are aerosolized. Clinical effects of opioid poisoning result from central nervous system and respiratory system depression manifesting as lethargy or coma, decreased respiratory rate, miosis, and possibly apnea (1, 2).

Laboratory criteria for diagnosis

- *Biologic*: A case in which opioids are detected in urine, as determined by hospital or commercial laboratory tests. Fentanyl derivatives and certain other synthetic opioids (e.g., oxycodone) might not be detected by routine toxicologic screens.

- OR-

- *Environmental*: Detection of opioids in environmental samples, as determined by FDA.

Case classification

- *Suspected*: A case in which a potentially exposed person is being evaluated by health-care workers or public health officials for poisoning by a particular chemical agent, but no specific credible threat exists.
- *Probable*: A clinically compatible case in which a high index of suspicion (credible threat or patient history regarding location and time) exists for opioid exposure, or an epidemiologic link exists between this case and a laboratory-confirmed case.
- *Confirmed*: A clinically compatible case in which laboratory tests have confirmed exposure.

The case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and nonspecific laboratory evidence of a particular chemical was present or a 100% certainty of the etiology of the agent is known.

Additional resources

1. Nelson LS. Opioids. In: Goldfrank LR, Flomenbaum NE, Lewin N-A, Howland MA, Hoffman RS, Nelson LS, eds. Goldfrank's toxicologic emergencies. 7th ed. New York, NY: McGraw-Hill; 2002:901-23.
2. Sporer KA. Acute heroin overdose. Ann Intern Med 1999;130:584-90.

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Chemical Agents

Nerve Agents

Highly poisonous chemicals that work by preventing the nervous system from working properly

- G agents
  - Sarin (GB)
  - Soman (GD)
  - Tabun (GA)

For the most accurate and up-to-date information, including resources on additional nerve agents, please refer to the CDC web site at: http://www.cdc.gov/

NERVE AGENTS

ALL SUSPECT CASES OF CHEMICAL AGENTS MUST BE REPORTED IMMEDIATELY TO THE SAN DIEGO COUNTY DIVISION OF EPIDEMIOLOGY

During Business Hours:	619-515-6620
After Hours (County Communications):	858-565-5255

Agents:

- GA (Tabun)
- GB (Sarin)
- GD (Soman)
- GF
- VX
- Organophosphates/Carbamates

Biological Effects:

- Nerve agents are the most toxic of the known chemical agents. They are hazards in their liquid and vapor states and can cause death within minutes after exposure.
- Inhibits acetylcholinesterase.
- Effects are due to excess acetylcholine.

Clinical:

- Vapor onset seconds to 1-2 minutes after exposure.
  - Small exposure – miosis, rhinorrhea, mild SOB
  - Large exposure – sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions, miosis
- Liquid on Skin onset usually < 30 minutes after exposure but can be up to 18 hours after.
  - Small to moderate exposure – localized sweating, nausea, vomiting, feeling of weakness
  - Large exposure – sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions

S - Salivation
L – Lacrimation
U – Urination (incontinence)
D - Diaphoresis
G – Gastro-intestinal (diarrhea, cramping)
E – Emesis

Diagnosis:

- Nerve agent diagnosis in a symptomatic patient needs to be a clinical diagnosis in order to initiate treatment in a timely manner.
- Laboratory diagnosis can be made by measuring cholinesterase activity. Nerve agents inhibit the cholinesterase activity of the blood components, and estimation of this activity is useful in detecting exposure to these agents. The erythrocyte enzyme activity is more sensitive to acute nerve agent exposure than is the plasma enzyme activity.

Decontamination:

- Medical personnel must wear personal protective equipment prior to any contact with patients.
- Remove clothing and decontaminate with copious amounts of soap and water.

Treatment:

- Atopine – 2-6mg IM or IV
  - Give 2mg every 5-10 minutes as needed until reduction in secretions and improvement in ventilation.
  - Usual doses in severe cases 15-20mg
- Pralidoxime Chloride (2-Pam Cl) – 600mg-1800mg IM or 1000mg IV slowly over 20-30minutes
  - Give an additional 600mg IM or 1000mg IV every hour as needed
- Airway support
  - Ventilation
  - Suction

CASE DEFINITION

Nerve Agents or Organophosphates

Clinical description

Nerve agent or organophosphate toxicity might result from multiple routes of exposure and is a cholinergic syndrome consisting of excess respiratory and oral secretions, diarrhea and vomiting, diaphoresis, convulsions, altered mental status, miosis, bradycardia, and generalized weakness that can progress to paralysis and respiratory arrest (1-3).

In certain cases, excessive autonomic activity from stimulation of nicotinic receptors will offset the cholinergic syndrome and will include mydriasis, fasciculations, tachycardia, and hypertension.

Laboratory criteria for diagnosis

- *Biologic*: A case in which nerve agents in urine are detected, as determined by CDC or one of five LRN laboratories that have this capacity. Decreased plasma or red blood cell cholinesterase levels based on a specific commercial laboratory reference range might indicate a nerve agent or organophosphate exposure; however, the normal range levels for cholinesterase are wide, which makes interpretation of levels difficult without a baseline measurement or repeat measurements over time.

-OR-

- *Environmental*: Detection of organophosphate pesticides in environmental samples, as determined by FDA. However, a confirmation test for nerve agents in environmental samples is not available.

Case classification

- *Suspected*: A case in which a potentially exposed person is being evaluated by health-care workers or public health officials for poisoning by a particular chemical agent, but no specific credible threat exists.
- *Probable*: A clinically compatible case in which a high index of suspicion (credible threat or patient history regarding location and time) exists for nerve agent or organophosphate pesticide exposure, or an epidemiologic link exists between this case and a laboratory-confirmed case.
- *Confirmed*: A clinically compatible case in which laboratory tests have confirmed exposure.

The case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and nonspecific laboratory evidence of a particular chemical was present or a 100% certainty of the etiology of the agent is known.

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Additional resources

1. Sidell FR. Soman and sarin: clinical manifestations and treatment of accidental poisoning by organophosphates. Clin Toxicol 1974;7:1-17.
2. Sidell FR. Nerve agents. In: Zajtchuk R, Bellamy RF, eds. Textbook of military medicine: medical aspects of chemical and biological warfare. Washington, DC: Office of the Surgeon General at TMM Publications, Borden Institute, Walter Reed Army Medical Center; 1997:129-79.
3. Holstege CP, Kirk M, Sidell FR. Chemical warfare: nerve agent poisoning. Crit Care Clin 1997;13:923-42.

TOXIC SYNDROME DESCRIPTION

Nerve Agents and Organophosphate Pesticides

The purpose of this document is to enable health care workers and public health officials to recognize an unknown or suspected exposure to a nerve agent or an organophosphate (OP) pesticide. Nerve agents are chemical warfare agents that have the same mechanism of action as OP pesticides. They are potent inhibitors of acetylcholinesterase. Inhibition of acetylcholinesterase leads to an accumulation of acetylcholine in the central and peripheral nervous system. Excess acetylcholine produces a predictable cholinergic syndrome consisting of copious respiratory and oral secretions, diarrhea and vomiting, sweating, altered mental status, autonomic instability, and generalized weakness that can progress to paralysis and respiratory arrest.

The amount and route of exposure to the nerve agent or OP pesticide, the type of agent or pesticide, and the premorbid condition of the exposed person will contribute to the time of onset and the severity of illness. For example, inhalation of a nerve agent or an OP pesticide leads to a quicker onset of poisoning with more severe symptoms compared with dermal exposure, given the same amount of agent.

Signs and symptoms

The following is a more comprehensive list of signs and symptoms that may be encountered in a person exposed to a nerve agent or OP pesticide. Signs and symptoms are not listed in order of presentation or specificity. Also, partial presentations (an absence of some of the following signs/symptoms) do not necessarily imply less severe disease.

Central nervous system signs and symptoms

- Miosis (unilateral or bilateral)
- Headache
- Restlessness
- Convulsions
- Loss of consciousness
- Coma

Respiratory signs and symptoms

- Rhinorrhea (perfuse watery runny nose)
- Bronchorrhea (excessive bronchial secretions)
- Wheezing
- Dyspnea (shortness of breath)
- Chest tightness
- Hyperpnea (increased respiratory rate/depth)—early
- Bradypnea (decreased respiratory rate)—late

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Cardiovascular signs

- Tachycardia (increased heart rate)—early
- Hypertension (high blood pressure)—early
- Bradycardia (decreased heart rate)—late
- Hypotension (low blood pressure)—late
- Dysrhythmias (prolonged QT on EKG, ventricular tachycardia)

Gastrointestinal signs and symptoms

- Abdominal pain
- Nausea and vomiting
- Diarrhea
- Urinary incontinence, frequency

Musculoskeletal signs and symptoms

- Weakness (may progress to paralysis)
- Fasciculations (local or generalized)

Skin and mucous membrane signs and symptoms

- Profuse sweating (local or generalized)
- Lacrimation (tear formation)
- Conjunctival injection

Laboratory finding suggestive of nerve agent poisoning

- Decreased plasma or red blood cell (RBC) cholinesterase activity

Limitations

- Wide normal range for enzyme activity makes interpretation difficult without a baseline measurement.
- Cholinesterase activity correlates poorly with severity of local effects after vapor exposures.
- Plasma or RBC cholinesterase may be disproportionately inhibited depending on the particular nerve agent, amount of exposure and time interval since exposure.

Interpreting cholinesterase activity

- Plasma cholinesterase
  - usually declines faster than RBC cholinesterase;
  - is easier to assay than RBC cholinesterase;
  - regenerates faster than RBC cholinesterase;
  - may have a day-to-day variation in enzyme activity as high as 20%;
  - is less specific than RBC cholinesterase; and
  - can show false depression from liver disease, malnutrition, pregnancy, genetic deficiency, or drugs (e.g., codeine, morphine, cocaine, succinylcholine).
- Red blood cell cholinesterase
  - is a better reflection of CNS cholinesterase activity;
  - is more specific test than plasma cholinesterase;
  - may have a day-to-day variation in enzyme activity as high as 10%; and

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- can show false depression from antimalarial therapy or pernicious anemia.

Differential diagnosis

- Carbamate insecticides
- Medicinal carbamates (eg, pyridostigmine, neostigmine, physostigmine)
- Cholinomimetic compounds (eg, pilocarpine, methacholine, bethanechol)
- Nicotine alkaloids (eg, nicotine, coniine)
- Muscarine-containing mushrooms
- Neuromuscular blocking drugs (eg, atracurim, vecuronium)

Note: The actual clinical manifestations of an exposure to a nerve agent or an organophosphate pesticide may be more variable than the syndrome described in this document.

FACT SHEET

Facts about Chemical Agents: Sarin

Sarin, also known as GB, is a human-made chemical warfare agent classified as a nerve agent. Nerve agents are the most toxic and rapidly acting of the known chemical warfare agents. They are similar to certain kinds of pesticides (insect killers) called organophosphates in terms of how they work and what kind of harmful effects they cause. However, nerve agents are much more potent than organophosphate pesticides. Sarin originally was developed in 1938 in Germany as a pesticide. It is a clear, colorless, and tasteless liquid that has no odor in its pure form. However, sarin can evaporate into a vapor (gas) and spread into the environment.

Where sarin is found and how it is used

Sarin and other nerve agents may have been used in chemical warfare during the Iran-Iraq War in the 1980s. Sarin was used in two terrorist attacks in Japan in 1994 and 1995. It is not found naturally in the environment.

How people can be exposed to sarin

Following release of sarin into the air, people can be exposed through:

- skin contact
- eye contact
- by breathing air that contains sarin

Sarin mixes easily with water, so it could be used to poison water. Following release of sarin into water, people can be exposed by touching or drinking water that contains sarin. Following contamination of food with sarin, people can be exposed by eating the contaminated food. A person's clothing can release sarin for about 30 minutes after it has come in contact with sarin vapor, which can lead to exposure of other people. Because sarin breaks down slowly in the body, people who are repeatedly exposed to sarin may suffer more harmful health effects. Because sarin vapor is heavier than air, it will sink to low-lying areas and create a greater exposure hazard there.

How sarin works

The extent of poisoning caused by sarin depends on the amount of sarin to which a person was exposed, how the person was exposed, and the length of time of the exposure. Symptoms will appear within a few seconds after exposure to the vapor form of sarin and within a few minutes up to 18 hours after exposure to the liquid form.

All the nerve agents cause their toxic effects by preventing the proper operation of the chemical that acts as the body's "off switch" for glands and muscles. Without an "off switch," the glands and muscles are constantly being stimulated. They may tire and no longer be able to sustain breathing function.

Sarin is the most volatile of the nerve agents, which means that it can easily and quickly evaporate from a liquid into a vapor and spread into the environment. People can be exposed to the vapor even if they do not come in contact with the liquid form of sarin. Because it evaporates so quickly, sarin presents an immediate but short-lived threat.

(continued)

Immediate signs and symptoms of sarin exposure

People may not know that they were exposed because sarin has no odor. People exposed to a low or moderate dose of sarin by breathing contaminated air, eating contaminated food, drinking contaminated water, or touching contaminated surfaces may experience some or all of the following symptoms within seconds to hours of exposure:

- Runny nose
- Watery eyes
- Small, pinpoint pupils
- Eye pain
- Blurred vision
- Drooling and excessive sweating
- Cough
- Chest tightness
- Rapid breathing
- Diarrhea
- Increased urination
- Confusion
- Drowsiness
- Weakness
- Headache
- Nausea, vomiting, and/or abdominal pain
- Slow or fast heart rate
- Low or high blood pressure

Even a small drop of sarin on the skin can cause sweating and muscle twitching where sarin touched the skin. Exposure to large doses of sarin by any route may result in the following harmful health effects:

- Loss of consciousness
- Convulsions
- Paralysis
- Respiratory failure possibly leading to death

Showing these symptoms does not necessarily mean that a person has been exposed to sarin.

What the long-term health effects are

Mild or moderately exposed people usually recover completely. Severely exposed people are not likely to survive. Unlike some organophosphate pesticides, nerve agents have not been associated with neurological problems lasting more than 1 to 2 weeks after the exposure.

How people can protect themselves, and what they should do if they are exposed to sarin

- Recovery from sarin exposure is possible with treatment, but the antidotes available must be used quickly to be effective. Therefore, the best thing to do is avoid exposure:
  - Leave the area where the sarin was released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing the possibility of death from exposure to sarin vapor.
    - If the sarin release was outdoors, move away from the area where the sarin was released. Go to the highest ground possible, because sarin is heavier than air and will sink to lowlying areas.
    - If the sarin release was indoors, get out of the building.
- If people think they may have been exposed, they should remove their clothing, rapidly wash their entire body with soap and water, and get medical care as quickly as possible.
- *Removing and disposing of clothing:*
  - Quickly take off clothing that has liquid sarin on it. Any clothing that has to be pulled over the head should be cut off the body instead of pulled over the head. If possible, seal the clothing in a plastic bag. Then seal the first plastic bag in a second plastic

(continued)

bag. Removing and sealing the clothing in this way will help protect people from any chemicals that might be on their clothes.
    - If clothes were placed in plastic bags, inform either the local or state health department or emergency personnel upon their arrival. Do not handle the plastic bags.
    - If helping other people remove their clothing, try to avoid touching any contaminated areas, and remove the clothing as quickly as possible.

- *Washing the body:*
    - As quickly as possible, wash any liquid sarin from the skin with large amounts of soap and water. Washing with soap and water will help protect people from any chemicals on their bodies.
    - Rinse the eyes with plain water for 10 to 15 minutes if they are burning or if vision is blurred.
- If sarin has been swallowed, do not induce vomiting or give fluids to drink.
- Seek medical attention immediately. Dial 911 and explain what has happened.

How sarin exposure is treated

Treatment consists of removing sarin from the body as soon as possible and providing supportive medical care in a hospital setting. Antidotes are available for sarin. They are most useful if given as soon as possible after exposure.

For more information on sarin, call XXX-XXX-XXXX or visit the Centers for Disease Control and Prevention's (CDC) website at www.bt.cdc.gov/chemical or call the Regional Poison Control Center at 1-800-222-1222, or the CDC Public Response Hotline at (800) 232-4636 (English and Spanish), or (888) 232-6358 (TTY). The above information has been adapted from the CDC fact sheet "Facts about Sarin."

FACT SHEET

Facts About Soman

What soman is

- Soman is a human-made chemical warfare agent classified as a nerve agent. Nerve agents are the most toxic and rapidly acting of the known chemical warfare agents. They are similar to pesticides (insect killers) called organophosphates in terms of how they work and the kinds of harmful effects they cause. However, nerve agents are much more potent than organophosphate pesticides.
- Soman was originally developed as an insecticide in Germany in 1944.
- Soman is also known as "GD."
- Soman is a clear, colorless, tasteless liquid with a slight camphor odor (for example, Vicks Vapo-Rub®) or rotting fruit odor. It can become a vapor if heated.

Where soman is found and how it is used

- It is possible that soman or other nerve agents were used in chemical warfare during the Iran-Iraq War in the 1980s.
- Soman is not found naturally in the environment.

How people can be exposed to soman

- Following release of soman into the air, people can be exposed through skin contact, eye contact, or inhalation (breathing in the soman).
- Soman mixes easily with water, so it could be used to poison water. Following release of soman into water, people can be exposed by drinking contaminated water or getting contaminated water on their skin.
- Following contamination of food with soman, people can be exposed by eating the contaminated food.
- A person's clothing can release soman for about 30 minutes after contact with soman vapor, which can lead to exposure of other people.
- Soman breaks down slowly in the body, meaning that repeated exposures to soman and/or other nerve agents can have a cumulative effect (build up in the body).
- Because soman vapor is heavier than air, it will sink to low-lying areas and create a greater exposure hazard there.

How soman works

- The extent of poisoning caused by soman depends on the amount of soman to which a person was exposed, how the person was exposed, and the length of time of the exposure.
- Symptoms will appear within a few seconds after exposure to the vapor form of soman, and within a few minutes to up to 18 hours after exposure to the liquid form.
- All the nerve agents cause their toxic effects by preventing the proper operation of the chemical that acts as the body's "off switch" for glands and muscles. Without an "off switch," the glands and muscles are constantly being stimulated. They may tire and no longer be able to sustain breathing function.

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- Compared with other nerve agents, soman is more volatile than VX but less volatile than sarin. The higher a chemical's volatility, the more likely it will evaporate from a liquid into a vapor and disperse into the environment. People can be exposed to the vapor even if they do not come in contact with the liquid form.
- Because of its high volatility, soman is an immediate but short-lived threat and does not last a long time in the environment.
- Because soman is more volatile than the nerve agent VX (the most potent nerve agent), it will remain on exposed surfaces for a shorter period of time compared with VX.

Immediate signs and symptoms of soman exposure

- Although soman has a camphor or fruity odor, the odor may not be noticeable enough to give people sufficient warning against a toxic exposure.
- People exposed to a low or moderate dose of soman by inhalation, ingestion (swallowing), or skin absorption may experience some or all of the following symptoms within seconds to hours of exposure:
    - Runny nose
    - Watery eyes
    - Small, pinpoint pupils
    - Eye pain
    - Blurred vision
    - Drooling and excessive sweating
    - Cough
    - Chest tightness
    - Rapid breathing
    - Diarrhea
    - Increased urination
    - Confusion
    - Drowsiness
    - Weakness
    - Headache
    - Nausea, vomiting, and/or abdominal pain
    - Slow or fast heart rate
    - Abnormally low or high blood pressure
- Even a tiny drop of nerve agent on the skin can cause sweating and muscle twitching where the agent touched the skin.
- Exposure to a large dose of soman by any route may result in these additional health effects:
    - Loss of consciousness
    - Convulsions
    - Paralysis
    - Respiratory failure possibly leading to death
- Showing these signs and symptoms does not necessarily mean that a person has been exposed to soman.

What the long-term health effects are

Mild or moderately exposed people usually recover completely. Severely exposed people are not likely to survive. Unlike some organophosphate pesticides, nerve agents have not been associated with neurological problems lasting more than 1 to 2 weeks after the exposure.

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Facts About Soman
(continued from previous page)

How people can protect themselves, and what they should do if they are exposed to soman

- Recovery from soman exposure is possible with treatment, but the antidotes available must be used quickly (within minutes) to be effective. Therefore, the best thing to do is avoid exposure:
    - Leave the area where the soman was released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing the possibility of death from exposure to soman vapor.
        - If the soman release was outdoors, move away from the area where the soman was released. Go to the highest ground possible, because soman is heavier than air and will sink to low-lying areas.
        - If the soman release was indoors, get out of the building.
- If people think they may have been exposed, they should remove their clothing, rapidly wash their entire body with soap and water, and get medical care as quickly as possible.
- *Removing and disposing of clothing:*
    - Quickly take off clothing that has liquid soman on it. Any clothing that has to be pulled over the head should be cut off the body instead of pulled over the head. If possible, seal the clothing in a plastic bag. Then seal the first plastic bag in a second plastic bag. Removing and sealing the clothing in this way will help protect people from any chemicals that might be on their clothes.
    - If clothes were placed in plastic bags, inform either the local or state health department or emergency personnel upon their arrival. Do not handle the plastic bags.
    - If helping other people remove their clothing, try to avoid touching any contaminated areas, and remove the clothing as quickly as possible.
- *Washing the body:*
    - As quickly as possible, wash any liquid soman from the skin with large amounts of soap and water. Washing with soap and water will help protect people from any chemicals on their bodies.
    - Rinse the eyes with plain water for 10 to 15 minutes if they are burning or if vision is blurred.
- If soman has been ingested (swallowed), do not induce vomiting or give fluids to drink.
- Seek medical attention right away. Dial 911 and explain what has happened.

How soman exposure is treated

Treatment consists of removing soman from the body as soon as possible and providing supportive medical care in a hospital setting. Antidotes are available for soman. They are most useful if given as soon as possible after exposure.

How people can get more information about soman

People can contact one of the following:

- Regional poison control center (1-800-222-1222)
- Centers for Disease Control and Prevention
    - Public Response Hotline (CDC)
        - (800) 232-4636 (English and Spanish)
        - TTY (888) 232-6358
    - Emergency Preparedness and Response Web site (http://www.bt.cdc.gov/)
    - E-mail inquiries: cdcinfo@cdc.gov

This fact sheet is based on CDC's best current information. It may be updated as new information becomes available.

Last reviewed on 03/23/05

Nerve Agents
Tabun (GA) CAS 77-81-6; Sarin (GB) CAS 107-44-8; Soman (GD) CAS 96-64-0; and VX CAS 5078269-9

Synonyms:

GA: ethyl dimethylamidocyanophosphate; ethyl N,N-dimethylphosphoramidocyanidate; ethyl dimethyl-phosphoramidocyanidate; dimethylaminoethoxy-cyanophosphine oxide; dimethyl-amidoethoxy-phosphoryl cyanide; EA1205; dimethylphosphoramidocyanidic acid ethyl ester

GB: isopropyl methylphosphonofluoridate; isopropoxymethylphosphoryl fluoride; trilone; MFI; TL1 618; isopropylmethanefluorophosphonate; T144; T2106; fluoro(isopropoxy)methylphosphine oxide; methylisopropoxyfluorophosphine oxide; zarin

GD: pinacolyl methylphosphonofluoridate; 1,2,2-trimethylpropyl methylphosphonofluoridate; methyl-pinacolyloxyfluorophosphine oxide; pinacolyloxymethylphosphonyl fluoride; pinacolylmethyl-fluorophosphonate; 1,2,2-trimethylpropoxyfluoro(methyl)phosphine oxide; pinacolyl methyl-phosphonyl fluoride

VX: O-ethyl S-(2-diisopropylaminoethyl) methylphosphonothiolate; methylphosphonothioic acid; S-2-(diisopropylamino)ethyl O-ethyl methylphosphonothioate; O-ethyl S-(2-diisopropyl-aminoethyl)methylphosphonothioate; O-ethyl S-(2-diisopropoylaminoethyl) methylthiol-phosphonoate; O-ethyl S-diisopropylaminoethyl methylphosphonothiolate

- **Persons whose skin or clothing is contaminated with nerve agent can contaminate rescuers by direct contact or through off-gassing vapor. Persons whose skin is exposed only to nerve agent vapor pose no risk of secondary contamination; however, clothing can trap vapor.**
- **G-type nerve agents (GA, GB, and GD) are clear, colorless liquids that are volatile at ambient temperatures. VX is an amber-colored, oily liquid with low volatility unless temperatures are high.**
- **Nerve agents are readily absorbed by inhalation, ingestion, and dermal contact. Rapidly fatal systemic effects may occur.**

Description

Nerve agents are the most toxic of the known chemical warfare agents. **They are chemically similar to organophosphate pesticides and exert their biological effects by inhibiting acetylcholinesterase enzymes.** G-type agents are clear,

colorless, and tasteless liquids that are miscible in water and most organic solvents. GB is odorless and is the most volatile nerve agent; however, it evaporates at about the same rate as water. GA has a slightly fruity odor, and GD has a slight camphor-like odor. VX is a clear, amber-colored, odorless, oily liquid. It is miscible with water and soluble in all solvents. It is the least volatile nerve agent. Table 1 lists selected physical properties for each of the nerve agents.

Routes of Exposure

Inhalation

Nerve agents are readily absorbed from the respiratory tract. Rhinorrhea and tightness in the throat or chest begin within seconds to minutes after exposure. Nerve agent vapors are heavier than air. Odor does not provide adequate warning of detection. The estimated LCt_{50} (the product of concentration times time that is lethal to 50% of the exposed population by inhalation) ranges from 10 mg-min/m^3 for VX to 400 mg-min/m^3 for GA.

Skin/Eye Contact

Nerve agent liquids are readily absorbed from the skin and eyes. Vapors are not absorbed through the skin except at very high concentrations. Ocular effects may result from both direct contact and systemic absorption. The nature and timing of symptoms following dermal contact with liquid nerve agents depend on exposure dose; effects may be delayed for several hours. As little as one drop of VX on the skin can be fatal and 1 to 10 mL of GA, GB, or GD can be fatal.

Ingestion

Ingestion of nerve agents is expected to be relatively rare compared to inhalation exposure or skin contact; however, they are readily absorbed from the GI tract and are highly toxic.

Sources/Uses

Most of the nerve agents were originally synthesized in a search for insecticides, but because of their toxicity, they were evaluated for military use. GA was synthesized in 1936 by a German scientist who synthesized GB 2 years later. During World War II, Germany developed chemical weapons using both GA and GB but never used them. GD was synthesized in 1944 by a German chemist, and VX was synthesized in the early 1950s by a British scientist. Although related organophosphate chemicals are used in medicine, pharmacology, and agriculture, these are not as toxic as the nerve agents. Nerve agents were used by Iraq against Iran and have been used by terrorists. They are known to be included in military stockpiles of several nations, including the United States.

Standards and

Guidelines

Workplace time-weighted average: GA and GB, 0.0001 mg/m^3; GD, 0.00003 mg/m^3; VX, 0.00001 mg/m^3

General population limits: 0.000003 mg/m^3 (all) over an 8-hour workshift)

Physical Properties

Table 1. Physical Properties of Nerve Agents

	Nerve Agent			
Property	**Tabun (GA)**	**Sarin (GB)**	**Soman (GD)**	**VX**
Description	clear, colorless, and tasteless liquid	clear, colorless, tasteless, and odorless liquid	pure liquid is clear, colorless, and tasteless; discolors with aging to dark brown	amber colored, tasteless, and odorless oily liquid
Warning properties	Although GA has a slight fruit odor, this cannot be relied on to provide sufficient warning against toxic exposure	none	Although GD has a slight fruity or camphor odor, this cannot be relied on to provide sufficient warning against toxic exposure.	none
Molecular weight	162.3 daltons	140.1 daltons	182.2 daltons	267.4 daltons
Boiling point	(760 mm Hg) = 428 to 475 °F) (220 to 246 °C)	(760 mm Hg) = 316 °F (158 °C)	(760 mm Hg) = 332.6 to 392 °F (167 to 200 °C)	(760 mm Hg) = 568.4 °F (298 °C)
Freezing point	-58 °F (-50 °C)	-68.8 °F (-56 °C)	-43.6 °F (-42 °C)	-59.8 °F (-51 °C)
Specific gravity	1.073 g/mL (water = 1.0)	1.089 (water = 1.0)	1.022 (water = 1.0)	1.008 (water = 1.0)
Vapor pressure	0.037 mm Hg at 68 °F (20 °C); 0.057 mm Hg at 77 °F (25 °C)	2.1 mm Hg at 68 °F (20 °C); 2.9 mm Hg at 77 °F (25 °C)	0.4 mm Hg at 77 °F (25 °C)	0.0007 mm Hg at 77 °F (25 °C)
Vapor density	5.6 (air = 1.0)	4.9 (air = 1.0)	6.33 (air = 1.0)	9.2 (air = 1.0)
Liquid density	1.08 g/mL at 77 °F (25 °C)	1.10 g/mL at 68 °F (20 °C)	1.02 g/mL at 77 °F (25 °C)	1.008 g/mL at 68 °F (20 °C)
Flash point	172.4 °F (78 °C)	nonflammable	249.8 °F (121 °C)	318.2 °F (159 °C)
Solubility in water	9.8 g/100 g at 77 °F (25 °C)	miscible	2.1 g/100g at 68 °F (20 °C)	3 g/100 g (miscible below 48.9 °F 9.4 °C)
Volatility	490 mg/m^3 at 77 °F (25 °C)	22,000 mg/m^3 at 77 °F (25 °C)	3,900 mg/m^3 at 77 °F (25 °C)	10.5 mg/m^3 at 77 °F (25 °C)
NAERG#	153	153	153	153

Incompatibilities

Decomposition of GA may produce HCN, oxides of nitrogen, oxides of phosphorus, carbon monoxide, and hydrogen cyanide. Under acid conditions GB and GD hydrolyze to form HF. GB decomposes tin, magnesium, cadmium plated steel, and aluminum. Hydrolysis of VX produces a class B poison.

Health Effects

Manifestations of nerve agent exposure include rhinorrhea, chest tightness, pinpoint pupils, shortness of breath, excessive salivation and sweating, nausea, vomiting, abdominal cramps, involuntary defecation and urination, muscle twitching, confusion, seizures, flaccid paralysis, coma, respiratory failure, and death.

- **Nerve agents are potent acetylcholinesterase inhibitors causing the same signs and symptoms regardless of the exposure route. However, the initial effects depend on the dose and route of exposure.**

Acute Exposure

Nerve agents alter cholinergic synaptic transmission at neuroeffector junctions (muscarinic effects), at skeletal myoneural junctions and autonomic ganglia (nicotinic effects), and in the CNS. Initial symptoms depend on the dose and route of exposure.

Muscarinic effects include pinpoint pupils; blurred or dim vision; conjunctivitis; eye and head pain; hypersecretion by salivary, lacrimal, sweat, and bronchial glands; narrowing of the bronchi; nausea, vomiting, diarrhea, and crampy abdominal pains; urinary and fecal incontinence; and slow heart rate.

Nicotinic effects include skeletal muscle twitching, cramping, and weakness. Nicotinic stimulation can obscure certain muscarinic effects and produce rapid heart rate and high blood pressure.

Relatively small to moderate vapor exposure causes pinpoint pupils, rhinorrhea, bronchoconstriction, excessive bronchial secretions, and slight to moderate dyspnea. Mild to moderate dermal exposure results in sweating and muscular fasciculations at the site of contact, nausea, vomiting, diarrhea, and weakness. The onset of these mild to moderate signs and symptoms following dermal exposure may be delayed for as long as 18 hours. Higher exposures (any route) cause loss of consciousness, seizures, muscle fasciculations, flaccid paralysis, copious secretions, apnea, and death.

Children do not always respond to chemicals in the same way that adults do. Different protocols for managing their care may be needed.

CNS

Nerve agents cause behavioral and psychological changes in humans. CNS effects include irritability, nervousness, fatigue, insomnia, memory loss, impaired judgment, slurred speech, and depression. High exposures may produce loss of consciousness, seizures, and apnea.

Respiratory Inhalation of nerve agent vapors causes respiratory tract effects within seconds to minutes. Symptoms include excessive rhinorrhea and bronchial secretions, chest tightness, and difficulty breathing due to constriction of bronchial muscles and mucous secretions. Respiratory failure may occur due to CNS depression.

Cardiovascular Vagal stimulation may produce bradycardia, but pulse rate may be increased due to ganglionic stimulation, and the effects of hypoxia. Bradyarrhythmias and hypertension may occur.

Gastrointestinal Abdominal pain, nausea and vomiting are common manifestations of exposure by any route but may be the first systemic effects from liquid exposure on skin. If these symptoms occur within an hour of dermal exposure, severe intoxication is indicated. Diarrhea and fecal incontinence may also occur.

Skeletal muscles Nerve agents stimulate skeletal muscle producing fasciculations and twitching leading to fatigue and flaccid paralysis. Muscle twitching/fasciculations are clinical identifiers that indicate possible nerve agent exposure.

Metabolic Profuse sweating may occur.

Ocular Symptoms may occur from local effects of vapor exposure and from systemic absorption. Pinpoint pupils and spasm of the muscle of visual accommodation (i.e., ciliary muscle) leading to blurred and dim vision, aching pain in the eye, and conjunctivitis are typical effects.

Potential Sequelae CNS effects such as fatigue, irritability, nervousness and impairment of memory may persist for as long as 6 weeks after recovery from acute effects. Although exposure to some organophosphate compounds may cause a delayed mixed sensory-motor peripheral neuropathy, there are no reports of this condition among humans exposed to nerve agents.

Chronic Exposure Very little information is available regarding prolonged exposures to low levels of nerve agents.

Carcinogenicity No information is available regarding the potential carcinogenicity of nerve agents in humans. Limited animal data indicate that nerve agents are not likely to be carcinogenic.

Reproductive and

Developmental Effects The limited data available indicate that nerve agents are not reproductive or developmental toxicants.

Prehospital Management

Victims whose skin or clothing is contaminated with liquid nerve agent can contaminate rescuers by direct contact or through off-gassing vapor.

Nerve agents are extremely toxic and can cause loss of consciousness and convulsions within seconds and death from respiratory failure within minutes of exposure.

Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity; however, pralidoxime must be administered within minutes to a few hours following exposure (depending on the specific agent) to be effective. Treatment consists of supportive measures and repeated administration of antidotes.

Hot Zone

Responders should be trained and appropriately attired before entering the Hot Zone. If the proper personal protective equipment (PPE) is not available, or if the rescuers have not been trained in its use, call for assistance in accordance with local Emergency Operational Guides (EOG). Sources of such assistance include local HAZMAT teams, mutual aid partners, the closest metropolitan strike system (MMRS) and the U.S. Soldier and Biological Chemical Command (SBCCOM)-Edgewood Research Development and Engineering Center. SBCCOM may be contacted (from 0700-1630 EST call 410-671-4411 and from 1630-0700 EST call 410-278-5201), ask for the Staff Duty Officer.

Rescuer Protection

Nerve agent vapor is readily absorbed by inhalation and ocular contact and produces rapid local and systemic effects. The liquid is readily absorbed thorough the skin; however, effects may be delayed for several minutes to up to18 hours.

Respiratory protection: Pressure-demand, self-contained breathing apparatus (SCBA) is recommended in response situations that involve exposure to any nerve agent vapor or liquid.

Skin protection: Chemical-protective clothing and butyl rubber gloves are recommended when skin contact is possible because nerve agent liquid is rapidly absorbed through the skin and may cause systemic toxicity.

ABC Reminders

Chemical casualty triage is based on walking feasibility, respiratory status, age, and additional conventional injuries. The triage officer must know the natural course of a given injury, the medical resources immediately available, the current and likely

casualty flow, and the medical evacuation capabilities. General principles of triage for chemical exposures are presented in the box on the following page. There are four triage categories: immediate (priority 1), delayed (priority 2), minimal (priority 3), and expectant (priority 4). Clinical signs and effects of nerve agents associated with each of these categories are presented in Table 2.

Before transport, all casualties must be decontaminated. If needed, consult with the base station physician or the regional poison control center for advice concerning management of multiple casualties.

General principles of triage for chemical exposures

(1) Check triage tag/card for any previous treatment or triage.
(2) Survey for evidence of associated traumatic/blast injuries.
(3) Observe for sweating, labored breathing, coughing/vomiting, secretions.
(4) Severe casualty triaged as immediate if assisted breathing is required.
(5) Blast injuries or other trauma, where there is question whether there is chemical exposure, victims must be tagged as immediate in most cases. Blast victims evidence delayed effects such as ARDS, etc.
(6) Mild/moderate casualty: self/buddy aid, triaged as delayed or minimal and release is based on strict follow up and instructions.
(7) If there are chemical exposure situations which may cause delayed but serious signs and symptoms, then overtriage is considered appropriate to the proper facilities that can observe and manage any delayed onset symptoms.
(8) Expectant categories in multi-casualty events are those victims who have experienced a cardiac arrest, respiratory arrest, or continued seizures immediately. Resources should not be expended on these casualties if there are large numbers of casualties requiring care and transport with minimal or scant resources available.

1. *Immediate:* casualties who require lifesaving care within a short time, when that care is available and of short duration. This care may be a procedure that can be done within minutes at an emergency treatment station (e.g., relief of an airway obstruction, administering antidotes) or may be acute lifesaving surgery.

2. *Delayed:* casualties with severe injuries who are in need of major or prolonged surgery or other care and who will require hospitalization, but delay of this care will not adversely affect the outcome of the injury (e.g., fixation of a stable fracture).

3. *Minimal:* casualties who have minor injuries, can be helped by nonphysician medical personnel, and will not require hospitalization.

4. *Expectant:* casualties with severe life-threatening injuries who would not survive with optimal medical care, or casualties whose injuries are so severe that their chance of survival does not justify expenditure of limited resources. As circumstances permit, casualties in this category may be reexamined and possibly be retriaged to a higher category.

Table 2. Triage for Nerve Agent Casualties

Category (Priority)	Effects	Clinical Signs
Immediate (1)	Unconscious, talking but not walking, or moderate to severe effects in two or more systems (e.g., respiratory, GI, muscular, CNS)	Seizing or post-ictal, severe respiratory distress or apneic. Recent cardiac arrest.
Delayed (2)	Recovering from agent exposure or antidote	Diminished secretions, improving respiration.
Minimal (3)	Walking and talking	Miosis, rhinorrhea, mild to moderate dyspnea.
Expectant (4)	Unconscious	Cardiac/respiratory arrest of long duration.

ABC Reminders

Quickly ensure that the victim has a patent airway. Maintain adequate circulation. If trauma is suspected, maintain cervical immobilization manually and apply a decontaminable cervical collar and a backboard when feasible. Apply direct pressure to stop arterial bleeding, if present.

Antidotes

Administration of antidotes is a critical step in managing a nerve agent victim; however, this may be difficult to achieve in the Hot Zone, because the antidotes may not be readily available, and procedures or policies for their administration while in the Hot Zone may be lacking. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl. Otherwise, administer antidotes as described in Table 3.

Victim Removal

If victims can walk, lead them out of the Hot Zone to the Decontamination Zone. Dependant upon available resources, triage of remaining victims should be performed. Victims who are unable to walk may be removed on backboards or gurneys. If these are not available, carefully carry or drag victims to safety. Should there be a large number of casualties, and if decontamination resources permit, separate decontamination corridors should be established for ambulatory and non-ambulatory victims.

Table 3. Recommendations for Nerve Agent Therapy -- Prehospital Management.

Patient Age	Antidotes[1]		Other Treatment
	Mild/Moderate Symptoms[2]	Severe Symptoms[3]	
Infant (0 - 2 yrs)	Atropine: 0.05 mg/kg IM; 2-PAM Cl: 15 mg/kg IM	Atropine: 0.1 mg/kg IM; 2-PAM Cl: 25 mg/kg IM	**Assisted ventilation** should be started after administration of antidotes for severe exposures. **Repeat atropine (2 mg IM)** at 5 - 10 minute intervals until secretions have diminished and breathing is comfortable or airway resistence has returned to near normal.
Child (2 - 10 yrs)	Atropine: 1 mg IM; 2-PAM Cl: 15 mg/kg IM	Atropine: 2 mg IM; 2-PAM Cl: 25 mg/kg IM	
Adolescent (>10 yrs)	Atropine: 2 mg IM; 2-PAM Cl: 15 mg/kg IM	Atropine: 4 mg IM; 2-PAM Cl: 25 mg/kg IM	
Adult	Atropine: 2 to 4 mg IM; 2-PAM Cl: 600 mg IM	Atropine: 6 mg IM; 2-PAM Cl: 1800 mg IM	
Elderly, frail	Atropine: 1 mg IM; 2-PAM Cl: 10 mg/kg IM	Atropine: 2 to 4 mg IM; 2-PAM Cl: 25 mg/kg IM	

1. **2-PAMCl solution needs to be prepared** from the ampule containing 1 gram of desiccated 2-PAMCl: **inject** 3 ml of saline, 5% distilled or sterile water into ampule and shake well. Resulting solution is 3.3 ml of 300 mg/ml.
2. **Mild/Moderate symptoms** include localized sweating, muscle fasciculations, nausea, vomiting, weakness, dyspnea.
3. **Severe symptoms** include unconsciousness, convulsions, apnea, flaccid paralysis.

Decontamination Zone

Rapid decontamination is critical to prevent further absorption by the patient and to prevent exposure to others. Decontaminable gurneys and back boards should be used if possible when managing casualties in a contaminated area. Decontaminable gurneys are made of a monofilament polypropylene fabric that allows drainage of liquids, does not absorb chemical agents, and is easily decontaminated. Fiberglass back boards have been developed specifically for use in HAZMAT incidents. These are nonpermeable and readily decontaminated. The **Chemical Resuscitation Device** is a bag-valve mask equipped with a chemical agent cannister that can be used to ventilate casualties in a contaminated environment.

Rescuer Protection

Personnel should continue to wear the same level of protection as required in the Hot Zone (see *Rescuer Protection* under Hot Zone, above).

ABC Reminders

Quickly ensure that the victim has a patent airway. Maintain adequate circulation. Stabilize the cervical spine with a decontaminable collar and a backboard if trauma is suspected. Antidote administration may be required to allow ventilation. Suction oral and bronchial secretions. Administer supplemental oxygen if cardiopulmonary compromise is suspected. Assist ventilation with a bag-valve-mask device equipped with a cannister or air filter if necessary. Direct pressure should be applied to control heavy bleeding, if present.

Antidotes

Administer antidotes if they have not been administered. If possible, a system should be employed to track antidotes administered. If atropine was previously administered and signs and symptoms have not diminished within 5 to 10 minutes, give a second dose of atropine (2 mg for adults or 0.05 to 0.1 mg/kg for children) (see *Antidotes* under *Hot Zone*, Table 3).

Basic Decontamination

The eyes must be decontaminated within minutes of exposure to liquid nerve agent to limit injury. Flush the eyes immediately with water for about 5 to 10 minutes by tilting the head to the side, pulling eyelids apart with fingers, and pouring water slowly into eyes. There is no need to flush the eyes following exposure to nerve agent vapor. Do not cover eyes with bandages.

If exposure to liquid agent is suspected, cut and remove all clothing and wash skin immediately with soap and water. If shower areas are available, a thorough shower with soap and water should be used. However, if water supplies are limited, and showers are not available, an alternative form of decontamination is to use 0.5% sodium hypochlorite solution, or absorbent powders such as flour, talcum powder, or Fuller's earth If exposure to vapor only is certain, remove outer clothing and wash exposed skin with soap and water or 0.5% sodium hypochlorite. Place contaminated clothes and personal belongings in a sealed double bag.

In cases of ingestion, **do not induce emesis**. If the victim is alert and able to swallow, immediately administer a slurry of activated charcoal.

Transfer to Support Zone

As soon as basic decontamination is complete, move the victim to the Support Zone.

Support Zone

All victims must be decontaminated properly before entering the Support Zone (see *Decontamination Zone*, above).

ABC Reminders

Quickly ensure that the victim has a patent airway. If trauma is suspected, maintain cervical immobilization manually and apply a cervical collar and a backboard when feasible. Ensure adequate respiration; administer supplemental oxygen if cardiopulmonary compromise is suspected. **In a severely exposed casualty (unconscious, gasping, or not breathing), the antidotes will be required to allow ventilation**. Suction oral and bronchial secretions. Maintain adequate circulation. Establish intravenous access if necessary. Attach a cardiac monitor, as needed. Direct pressure should be applied to stop bleeding, if present.

Antidotes

Administer antidotes if they have not been administered (see *Antidotes* under *Hot Zone*, Table 3). Administer atropine (2 mg for adults and 0.05 to 0.1 mg/kg for children) every 5 to 10 minutes until dyspnea, resistence to ventilation, and secretions are minimized.

Additional Decontamination

In cases of ingestion, **do not induce emesis**. If the victim is alert and able to swallow, immediately administer a slurry of activated charcoal if not given previously.

Advanced Treatment

Intubate the trachea in cases of coma or respiratory compromise, or to facilitate removal of excessive pulmonary secretions. When the patient's condition precludes endotracheal intubation, perform cricothyrotomy if equipped and trained to do so. Frequent suctioning of the airways will be necessary to remove mucous secretions.

When possible, atropine and 2-PAM Cl should be given under medical supervision to symptomatic patients who have known or strongly suspected nerve agent toxicity (see *Antidote* sections, above).

Patients who are comatose, hypotensive, or seizing or have cardiac dysrhythmias should be treated according to advanced life support (ALS) protocols. Diazepam (5 to 10 mg in adults and 0.2 to 0.5 mg/kg in children) should be used to control convulsions. Lorazepam or other benzodiazepines may be used but barbiturates, phenytoin, and other anticonvulsants are not effective.

Transport to Medical Facility

Report to the base station and the receiving medical facility the condition of the patient, treatment given, and estimated time of arrival at the medical facility.

Emergency Department Management

Patients whose skin or clothing is contaminated with liquid nerve agent can contaminate rescuers by direct contact or through off-gassing vapor.

Nerve agents are extremely toxic and can cause death within minutes to hours after exposure from respiratory failure.

Atropine and pralidoxime (2-PAM Cl) are antidotes for nerve agent toxicity; however, pralidoxime must be administered within minutes to a few hours following exposure (depending on the specific agent) to be effective. Treatment consists of supportive measures and repeated administration of antidotes.

Decontamination Area

Previously decontaminated patients may be treated or held for observation. Others require decontamination as described below.

ABC Reminders

Evaluate and support the airway, breathing, and circulation. If the patient is apneic, give antidotes immediately (see *Antidote* section below). Intubate the trachea in cases of respiratory compromise. Suctioning may be required for excessive bronchial secretions. If the patient's condition precludes intubation, surgically create an airway. Antidote administration may be required to allow ventilation.

Personal Protection

If contaminated patients arrive at the Emergency Department, they must be decontaminated before being allowed to enter the facility. Decontamination can only take place inside the hospital if there is a decontamination facility with negative air pressure and floor drains to contain contamination. Personnel should wear the same level of protection required in the Hot Zone (see *Rescuer Protection* under *Hot Zone*, above).

Basic Decontamination

Patients who are able and cooperative may assist with their own decontamination. Remove and double bag contaminated clothing and all personal belongings.

For patients exposed to nerve agent vapor only, remove outer clothing and wash exposed areas including the head and hair with soap and water. For patients exposed to liquid agent, remove all clothing and wash entire body and hair with soap and water or 0.5% hypochlorite followed by a water rinse.

Irrigate exposed eyes with plain water or saline for about 5 to 10 minutes (see Basic Decontamination under Decontamination Zone, above). Remove contact lenses if present and easily removable without additional trauma to the eye.

In cases of ingestion, **do not induce emesis**. If the patient is able to swallow, immediately administer a slurry of activated charcoal if not given previously. (More information is provided in *Ingestion Exposure* above.)

Treatment Area

All patients should undergo decontamination before entering the treatment area (see *Decontamination Area*, above).

ABC Reminders

Evaluate and support the airway, breathing, and circulation (as in *ABC Reminders*, above). Establish intravenous access in seriously ill patients. Continuously monitor cardiac rhythm.

Triage

Patients who are conscious and have full muscular control will need minimal care. Patients who may have been exposed to liquid must be kept under observation for at least 18 hours.

Patients with a history of possible exposure to vapor only (with no possibility of liquid exposure) who have no signs of exposure by the time they reach the medical facility have not been exposed (because these effects occur within seconds to minutes after exposure). They can be discharged.

Antidotes and Other Treatments

Patients exposed to vapor who have miosis and rhinorrhea will need no care unless (a) they have eye or head pain or nausea and vomiting; under these circumstances topical atropine or homatropine in the eye should relieve the symptoms and the patient can be discharged within an hour or so; or (b) the rhinorrhea is very severe; under these circumstances, atropine IM (2 mg in adults and 0.05 mg/kg in children) should relieve this and the patient can be discharged in an hour or so. Topical atropine and homatropine should not be used routinely for miosis because they cause visual impairment for about 24 hours. See Table 4 for other antidote and treatment recommendations.

Table 4. Recommendations for Nerve Agent Therapy -- Emergency Department Management.

Patient Age	Antidotes		Other Treatment
	Mild/Moderate Symptoms[1]	Severe Symptoms[2]	
Infant (0 - 2 yrs)	Atropine: 0.05 mg/kg IM or 0.02 mg/kg IV; 2-PAM Cl: 15 mg/kg IV slowly	Atropine: 0.1 mg/kg IM or 0.02 mg/kg IV; 2-PAM Cl: 15 mg/kg IV slowly	**Assisted ventilation** as needed. **Repeat atropine (2 mg IM or 1 mg IM for infants)** at 5 - 10 minute intervals until secretions have diminished and breathing is comfortable or airway resistence has returned to near normal. **Phentolamine** for 2-PAM induced hypertension: (5 mg IV for adults; 1 mg IV for children) **Diazepam** for convulsions: (0.2 to 0.5 mg IV for infants 5 years; 1 mg IV for children >5 years; 5 mg IV for adults)
Child (2 - 10 yrs)	Atropine: 1 mg IM; 2-PAM Cl: 15 mg/kg IV slowly	Atropine: 2 mg IM; 2-PAM Cl: 15 mg/kg IV slowly	
Adolescent (>10 yrs)	Atropine: 2 mg IM; 2-PAM Cl: 15 mg/kg IV slowly	Atropine: 4 mg IM; 2-PAM Cl: 15 mg/kg IV slowly	
Adult	Atropine: 2 to 4 mg IM; 2-PAM Cl: 15 mg/kg (1 g) IV slowly	Atropine: 6 mg IM; 2-PAM Cl: 15 mg/kg (1 g) IV slowly	
Elderly, frail	Atropine: 1 mg IM; 2-PAM Cl: 5 to 10 mg/kg IV slowly	Atropine: 2 mg IM; 2-PAM Cl: 5 to 10 mg/kg IV slowly	

1. **Mild/Moderate symptoms** include localized sweating, muscle fasciculations, nausea, vomiting, weakness, dyspnea.
2. **Severe symptoms** include unconsciousness, convulsions, apnea, flaccid paralysis.

Inhalation Exposure

Ventilatory support is essential. Following low-dose exposure, administration of antidotes and supplemental oxygen may be adequate. Suction secretions from the nose, mouth, and

respiratory tract. Marked resistance to ventilation is expected due to bronchial constriction and spasm. Resistance lessens after administration of atropine.

Skin Exposure

Skin must be decontaminated within minutes following exposure to nerve agent. Because of the high toxicity, rapid absorption, and volatility, it is unlikely that a patient brought to a medical facility will have nerve agent on the skin. However, some nerve agent may remain in the hair
or clothing and should be decontaminated if not previously done (see *Basic Decontamination*, above).

Eye Exposure

Severity of miosis cannot be used as an indicator of the amount of exposure or effectiveness of the antidotes. Maximum miosis may not occur until an hour or more after exposure. If severe eye pain or nauseaand vomiting occur, protect eyes from bright light and consider topical administration of atropine or homatropine. Test visual acuity.

Ingestion Exposure

Do not induce emesis because of the risk of pulmonary aspiration of gastric contents which may result from abrupt respiratory arrest, seizures, or vomiting. If the patient is alert and charcoal has not been given previously, administer a slurry of activated charcoal. If the patient's condition is evaluated within 30 minutes after ingestion, consider gastric lavage. (Gastric contents should be considered potentially hazardous by skin contact or inhalation and should be quickly isolated.)

Laboratory Tests

Routine laboratory studies for all admitted patients include CBC, glucose, and serum electrolyte determinations. Chest X-ray and pulse oximetry (or ABG measurements) are recommended for severe exposures. Symptomatic and asymptomatic patients suspected of significant exposure should have determinations of red blood cell (RBC) cholinesterase activity, the most useful test for nerve agent poisoning. Severe symptoms of toxicity are usually present when more than 70% of RBC cholinesterase is inhibited. However, there is no correlation between cholinesterase activity and severity of topical signs and symptoms (e.g., miosis, rhinorrhea, dyspnea). If this test is not available, plasma cholinesterase can be measured.

Disposition and Follow-up

Patients exposed to nerve agent vapor who have only miosis and/or mild rhinorrhea when they reach the medical facility do not need to be admitted. All other patients who have had exposure to nerve agent should be hospitalized and observed closely.

Delayed Effects

Effects from skin exposure to liquid nerve agent may not develop for up to 18 hours following exposure.

Patients who have inhalation exposure and who complain of chest pain, chest tightness, or cough should be observed and examined periodically for 6 to 12 hours to detect delayed-onset bronchitis, pneumonia, pulmonary edema, or respiratory failure.

Formaldehyde poisoning can cause permanent alterations of nervous system function, including problems with memory, learning, thinking, sleeping, personality changes, depression, headache, and sensory and perceptual changes.

Follow-up

Patients who have severe exposure should be evaluated for persistent CNS sequelae. Patients should be advised to avoid organophosphate insecticide exposure until sequential RBC cholinesterase activity (measured at weekly to monthly intervals) has stabilized in the normal range, a process that may take 3 to 4 months after severe poisoning (see *Follow-up Instructions*, included with the *Nerve Agent Patient Information Sheet* below).

Reporting

Other persons may still be at risk in the setting where this incident occurred. If a public health risk exists, notify your state or local health department or other responsible public agency.

Nerve Agents
Patient Information Sheet

This handout provides information and follow-up instructions for persons who have been exposed to nerve agents.

What are nerve agents?
Nerve agents are chemical warfare agents, similar to but much more potent than organophosphate insecticides. They are colorless to amber-colored, tasteless liquids that may evaporate to create a gas. GB and VX are odorless, while GA has a slight fruity odor, and GD has a slight camphor odor.

What immediate health effects can result from exposure to nerve agents?
Nerve agents are extremely toxic chemicals that attack the nervous system. As little as one drop to a few milliliters of nerve agent contacting the skin can cause death within 15 minutes. Nerve agent exposure can cause runny nose, sweating, blurred vision, headache, difficulty breathing, drooling, nausea, vomiting, muscle cramps and twitching, confusion, convulsions, paralysis, and coma. Symptoms occur immediately if you inhale nerve agent vapor but may be delayed for several hours if you get nerve agent liquid on your skin.

Can nerve agent poisoning be treated?
There are antidotes for nerve agent poisoning but they must be administered quickly after exposure. Immediate decontamination is critical and hospitalization may be needed.

Are any future health effects likely to occur?
Complete recovery may take several months. After a severe exposure with prolonged seizures, permanent damage to the central nervous system is possible.

What tests can be done if a person has been exposed to nerve agents?
Activity of a blood enzyme called acetylcholinesterase can be measured to assess exposure and recovery.

Where can more information about nerve agents be found?
More information about nerve agents can be obtained from your regional poison control center; the Agency for Toxic Substances and Disease Registry (ATSDR); your doctor; or a clinic in your area that specializes in toxicology or occupational and environmental health. Ask the person who gave you this form for help locating these telephone numbers.

Follow-up Instructions

Keep this page and take it with you to your next appointment. Follow *only* the instructions checked below.

[ ] Call your doctor or the Emergency Department if you develop any unusual signs or symptoms within the next 24 hours, especially:
dizziness, loss of coordination, loss of memory
coughing, wheezing, or shortness of breath
nausea, vomiting, cramps, or diarrhea
muscle weakness or twitching
blurred vision

[ ] No follow-up appointment is necessary unless you develop any of the symptoms listed above.
[ ] Call for an appointment with Dr. _____ in the practice of _____.
When you call for your appointment, please say that you were treated in the Emergency Department at _____ Hospital by _____ and were advised to be seen again in _____ days.
[ ] Return to the Emergency Department/ _____ Clinic on (date) _____at __ _____ AM/PM for a follow-up examination.
[ ] Do not perform vigorous physical activities for 1 to 2 days.
[ ] You may resume everyday activities including driving and operating machinery.
[ ] Do not return to work for _____ days.
[ ] You may return to work on a limited basis. See instructions below.
[ ] Avoid exposure to cigarette smoke for 72 hours; smoke may worsen the condition of your lungs.
[ ] Avoid drinking alcoholic beverages for at least 24 hours; alcohol may worsen injury to your stomach or have other effects.
[ ] Avoid taking the following medications: _____
[ ] You may continue taking the following medication(s) that your doctor(s) prescribed for you: __
_____
_____
[ ] Other instructions: _____
_____
_____

- Provide the Emergency Department with the name and the number of your primary care physician so that the ED can send him or her a record of your emergency department visit.

- You or your physician can get more information on the chemical by contacting: _____ _____ or _____, or by checking out the following Internet Web sites: _____; _____.

Signature of patient _____ Date _____

Signature of physician _____ Date_____

Chemical Agents

Riot Control Agents/Tear Gas

Highly irritating agents normally used by law enforcement for crowd control or by individuals for protection (for example, mace)

For the most accurate and up-to-date information, including resources on the specific riot control agents, please refer to the CDC web site at: http://www.cdc.gov/

FACT SHEET
Interim Document

Facts About Riot Control Agents

What riot control agents are

- Riot control agents (sometimes referred to as "tear gas") are chemical compounds that temporarily make people unable to function by causing irritation to the eyes, mouth, throat, lungs, and skin.
- Several different compounds are considered to be riot control agents. The most common compounds are known as chloroacetophenone (CN) and chlorobenzylidenemalononitrile (CS). Other examples include chloropicrin (PS), which is also used as a fumigant (that is, a substance that uses fumes to disinfect an area); bromobenzylcyanide (CA); dibenzoxazepine (CR); and combinations of various agents.

Where riot control agents are found and how they are used

- Riot control agents are used by law enforcement officials for crowd control and by individuals and the general public for personal protection (for example, pepper spray).
- CS is also used in military settings to test the speed and ability of military personnel to use their gas masks.

How you could be exposed to riot control agents

- Because they are liquids or solids (for example, powder), riot control agents such as CN and CS could be released in the air as fine droplets or particles.
- If agents are released into the air, people could be exposed to them through skin contact, eye contact, or breathing.

How riot control agents work

- The extent of poisoning caused by riot control agents depends on the amount of riot control agent to which a person was exposed, the location of exposure (indoors versus outdoors), how the person was exposed, and the length of time of the exposure.
- Riot control agents work by causing irritation to the area of contact (for example, eyes, skin, nose) within seconds of exposure.
- The effects of exposure to a riot control agent are usually short-lived (15–30 minutes) after the person has been removed from the source and decontaminated (cleaned off).

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Immediate signs and symptoms of exposure to a riot control agent

People exposed to riot control agents may experience some or all of the following symptoms immediately after exposure:

- Eyes: excessive tearing, burning, blurred vision, redness
- Nose: runny nose, burning, swelling
- Mouth: burning, irritation, difficulty swallowing, drooling
- Lungs: chest tightness, coughing, choking sensation, noisy breathing (wheezing), shortness of breath
- Skin: burns, rash
- Other: nausea and vomiting

Long-lasting exposure or exposure to a large dose of riot control agent, especially in a closed setting, may cause severe effects such as the following:

- Blindness
- Glaucoma (a serious eye condition that can lead to blindness)
- Immediate death due to severe chemical burns to the throat and lungs
- Respiratory failure possibly resulting in death

Showing these signs and symptoms does not necessarily mean that a person has been exposed to riot control agents.

Long-term health effects of exposure to riot control agents

- Prolonged exposure, especially in an enclosed area, may lead to long-term effects such as eye problems including scarring, glaucoma, and cataracts, and may possibly cause breathing problems such as asthma.
- If symptoms go away soon after a person is removed from exposure to riot control agents, long-term health effects are unlikely to occur.

How you can protect yourself, and what to do if you are exposed to riot control agents

- Since inhalation is likely to be the primary route of exposure, leave the area where the riot control agents were released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing exposure to riot control agents.
  - If the riot control agents were released outdoors, move away from the area where the agents were released. Avoid dense, low-lying clouds of riot control agent vapor.
  - Go to the highest ground possible, because riot control agents will form a dense vapor cloud that can travel close to the ground.
  - If the release of riot control agents was indoors, get out of the building.
- If you are near a release of riot control agent, emergency coordinators may tell you to either evacuate the area or "shelter in place" inside a building to avoid being exposed to the chemical. For more information on

evacuation during a chemical emergency, see "Facts About Evacuation" at http://www.bt.cdc.gov/planning/evacuationfacts.asp. For more information on sheltering in place during a chemical emergency, see "Facts About Sheltering in Place" at http://www.bt.cdc.gov/planning/Shelteringfacts.asp.

- If you think you may have been exposed to riot control agent, you should remove your clothing, rapidly wash your entire body with soap and water, and get medical care as quickly as possible.
- *Removing your clothing:*
  - Quickly take off clothing that may have riot control agent on it. Any clothing that has to be pulled over the head should be cut off the body instead of pulled over the head.
  - If you are helping other people remove their clothing, try to avoid touching any contaminated areas, and remove the clothing as quickly as possible.
- *Washing yourself:*
  - As quickly as possible, wash any riot control agent from your skin with large amounts of soap and water. Washing with soap and water will help protect people from any chemicals on their bodies.
  - If your eyes are burning or your vision is blurred, rinse your eyes with plain water for 10 to 15 minutes. If you wear contacts, remove them and put them with the contaminated clothing. Do not put the contacts back in your eyes (even if they are not disposable contacts). If you wear eyeglasses, wash them with soap and water. You can put your eyeglasses back on after you wash them. If you are wearing jewelry that you can wash with soap and water, you can wash it and put it back on. If it cannot be washed, it should be put with the contaminated clothing.
- *Disposing of your clothes:*
  - After you have washed yourself, place your clothing inside a plastic bag. Avoid touching contaminated areas of the clothing. If you can't avoid touching contaminated areas, or you aren't sure where the contaminated areas are, wear rubber gloves, turn the bag inside out and use it to pick up the clothes (inverting the bag over the clothes when you have all the clothes picked up), or put the clothes in the bag using tongs, tool handles, sticks, or similar objects. Anything that touches the contaminated clothing should also be placed in the bag. If you wear contacts, put them in the plastic bag, too.
  - Seal the bag, and then seal that bag inside another plastic bag. Disposing of your clothing in this way will help protect you and other people from any chemicals that might be on your clothes.
  - When the local or state health department or emergency personnel arrive, tell them what you did with your clothes. The health department or emergency personnel will arrange for further disposal. Do not handle the plastic bags yourself.
- For more information about cleaning your body and disposing of your clothes after a chemical release, see "Chemical Agents: Facts About Personal Cleaning and Disposal of Contaminated Clothing" at http://www.bt.cdc.gov/planning/personalcleaningfacts.asp.
- Seek medical attention right away. Dial 911 and explain what has happened.

How exposure to riot control agents is treated

- Treatment consists of helping the affected person get more oxygen in his or her blood and of stopping agent-caused chemical burns from getting worse. Medications that are used to treat asthma (such as bronchodilators and steroids) may also be used to help the person breathe.

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- Eye exposures are treated by rinsing the eyes with water until there is no evidence of riot control agents in the eyes.
- No antidote exists for poisoning from riot control agents.
- Burn injuries to the skin are treated with standard burn management techniques, including use of medicated bandages.

How you can get more information about riot control agents

You can contact one of the following:

- Regional poison control center (1-800-222-1222)
- Centers for Disease Control and Prevention
    - Public Response Hotline (CDC)
        - (800) 232-4636 (English and Spanish)
        - TTY (888) 232-6358
    - Emergency Preparedness and Response Web site (http://www.bt.cdc.gov/)
    - E-mail inquiries: cdcinfo@cdc.gov

This fact sheet is based on CDC's best current information. It may be updated as new information becomes available.

For more information, visit www.bt.cdc.gov/chemical, or call CDC at 800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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NUCLEAR/RADIOLOGICAL TERRORISM

The following section, “Section III: Nuclear/Radiological Terrorism Information and Treatment Guidelines For Hospitals and Clinicians” is an excerpt from the July 2006 *Terrorism Agent Information and Treatment Guidelines for Clinicians and Hospitals* published, and approved for reprinting, by the County of Los Angeles Public Health, Emergency Medical Services Agency.

Please refer to local, state, and federal resources for updates and event-specific information.

SECTION III: NUCLEAR/RADIOLOGICAL TERRORISM INFORMATION AND TREATMENT GUIDELINES FOR HOSPITALS AND CLINICIANS

INTRODUCTION

There was a time when many people feared a possible nuclear attack by the former Soviet Union. In response to federal civil defense recommendations, families built bomb shelters in their basements, and stocked cellars with food and supplies in hope of surviving the nuclear fallout that was sure to follow. With the end of the Cold War, the threat of thermonuclear war has lessened considerably. Now, a new type of threat is confronting society in which nuclear/ radiological weapons may be directed against civilian targets by terrorists.

It is important that first responders [police, fire, EMS personnel, physicians, nurses, and other health care providers] understand radiation exposure and the consequences of conventional explosives to spread radioactive materials so that they may better respond to and treat victims of this type of incident. In responding to such an event, rescue should not be attempted until the incident scene is secured, routine monitoring is performed, and the responder is dressed in appropriate personal protective equipment (PPE).

TERRORIST USE OF NUCLEAR MATERIALS

Terrorist use of radioactive materials or a nuclear device constitutes a plausible threat. Such an incident could occur in one of five ways:

- Simple radiological device
- Radiological dispersal device
- Reactor sabotage
- Improvised nuclear device
- Nuclear weapon

The medical consequences will be dependent on the type of device used.

Simple Radiological Device (SRD)

This is the deliberate act of spreading radioactive material without the use of an explosive device. An example would be the placement of a high activity radioactive isotope in a public place exposing numerous individuals to various levels of radiation. Sealed sources could also be used to expose individuals near the source.

Radiological Dispersal Device (RDD)

A radiological dispersal device does not cause a nuclear reaction. Such a device is formed by combining an explosive agent (TNT or a plastic explosive) with radioactive materials that may have been stolen, for example, from a hospital or local industry. The initial explosion kills or injures those closest to the bomb, while the radioactive substances remain to expose and contaminate survivors and emergency responders.

A similar type of event could also occur from a failed nuclear weapon detonation. (Under these circumstances, only the conventional component of the bomb explodes, rather than a nuclear reaction, causing widespread release of plutonium).

Nuclear Reactor Sabotage

Most people are aware of the reactor accidents of Three Mile Island and Chernobyl. The accident at Chernobyl was caused as the result of approximately eight safety systems being bypassed.

In the Western World, probability of terrorism involving a reactor is low. This is due to the high security surrounding a reactor together with the safety systems incorporated into the reactor. There is extensive shielding around a reactor; therefore, a significant amount of explosives would be required to breach this containment. This is a low probability event.

Improvised Nuclear Device (IND)

This is any device designed to cause a nuclear detonation. Construction of such a device would be difficult as it is not easy to get the weapon to detonate correctly. In some cases only the conventional high explosives in the IND will detonate. In this event, the IND is effectively a radiological dispersal device.

It is unlikely that terrorists will have the engineering sophistication and access to high-grade nuclear materials that are required to build an IND, but any detonation of an improvised or stolen device would generate high levels of radiation.

Nuclear Weapon

The probability of stealing a nuclear weapon in the Western World is very remote because of the high security surrounding these devices. However, a Russian general stated publicly that 50-100 one kiloton, suitcase nuclear weapons are unaccounted for in the former Soviet Union.

THE BASICS OF RADIATION

General Concepts

Examples of radiations include visible light, sound, radio waves, microwaves, heat, ionizing radiation, etc. Each example mentioned here has measurable physical properties and interacts with matter it comes in contact with. This module on the nuclear hazard will be concerned primarily with ionizing radiation.

Ionizing Radiation

As particles or energy emitted from a radioactive/radiation source pass through matter they can strip electrons from atoms/molecules causing them to become electrically charged or ionized, thus ionizing radiation. In living tissues/cells it is these ions produced by radiation that cause cellular damage and affect normal biological processes.

Ionizing radiation can be machine generated (i.e., X-rays), or it can come from radioactive atoms. Radioactive atoms are atoms with too much energy or mass. The basic building block of all matter is the atom. The atom consists of a central nucleus with shells of electrons orbiting around this nucleus. The nucleus is made up of neutrons and protons. These protons, which are positively charge, have the tendency to repel each other. The protons are held together within the nucleus by a force (a super nuclear glue) that has three characteristics: it acts over a very short range, independent of charge, and is very strong. There is a ratio of protons to neutrons (1 to 1.2) for stability of an element. Each element has a defined number of protons. When an element is radioactive, usually there is an imbalance of this ratio of protons to neutrons; often the imbalance is due to an excess of neutrons.

With respect to a radioactive nuclide, for it to become stable, the nucleus has the ability to change a neutron into a proton with the ejection of a negative electron or, conversely, has the ability to change a proton into a neutron with the ejection of a positive electron known as a positron. The nucleus also has the capability of ejecting large particles, consisting of two protons and two neutrons, known as an alpha particle. Therefore, a radioactive nuclide achieves stability by ejecting particles until it has the correct ratio of protons to neutrons, remembering that the resultant element will be different than the original one. Although the final element concerned has the correct number of protons and neutrons, it is still not stable. The reason for this is that within the nucleus there is an excess of energy. This excess energy is given off as electromagnetic energy of very short wave length. It is called gamma radiation. When all this excess energy is given off, the resultant element finally becomes stable.

The most common types of ionizing radiations are alpha particles, beta particles, gamma rays or X-rays, and neutrons. Gamma rays and X-rays may also be referred to as photons.

The basic building block of any tissue is the cell, and damage to the cell changes its chemistry or DNA. The chemical damage is instantaneous, but the clinical expression of this damage can take hours to years to express itself. At high doses, clinical expression can be within hours [e.g., the acute radiation syndrome (ARS)]. However, at lower doses or even after recovery from ARS, there is the probability, although low, of developing cancer 20-30 years later.

Alpha particles

Alpha particles are composed of two neutrons and two protons. Alpha particles do not penetrate the skin and can be shielded by a thin layer of paper or clothing. Because the outer layer of skin is dead and several microns thick, the alpha particle is unable to penetrate through the dead layers of skin to reach the lower layers of living cells and generally will not cause any skin damage. If, however, an alpha emitter gets inside the body through inhalation, ingestion, or via a wound, the alpha emissions are near live tissue, and localized damage could occur.

Beta particles

Beta radiation may travel meters in air and is moderately penetrating. Beta radiation can penetrate human skin to the germinal layer, where new skin cells are produced. If beta emitting contaminants are allowed to remain on the skin for a prolonged period of time, they may cause skin injury. Beta emitting contaminants may be harmful if deposited internally. A sheet of aluminum foil a few millimeters thick will stop beta radiation, and PPE provides some protection against most beta radiation.

Gamma rays (photons)

Gamma radiation is able to travel many meters in air and many centimeters in human tissue. It readily penetrates most materials and is sometimes called penetrating radiation. X-rays are like gamma rays. They too, are penetrating radiation. Radioactive materials that emit gamma radiation constitute both an external and internal hazard to humans. Dense materials are needed to shield against gamma radiation. PPE provides little shielding from gamma radiation but will prevent contamination of the skin. Gamma radiation frequently accompanies the emission of alpha and beta radiation.

Neutrons

Neutrons are neutral particles emitted from the nucleus of an atom. Neutrons lose most of their energy through collisions with other atomic nuclei. An analogy that could be used is the billiard ball effect (i.e., when one billiard ball strikes another, energy is transferred from one ball to the other). Under certain circumstances, neutrons can be captured by a stable nucleus, making the nucleus radioactive. An example of this is Na-23 being changed (transmuted) into Na-24.

Radiation Detection

Unfortunately our body senses cannot detect radiation. We cannot see, smell, taste, feel, or hear radiation, but we have very good instrumentation to detect it. Radiation monitoring instruments detect the presence of radiation, usually by collecting charged particles (ions). The radiation measured is usually expressed as exposure per unit time, using various units of measure, including the curie (Ci), the becquerel (Bq), and counts per minute (CPM). The most commonly used instruments to detect the presence of radiation include:

Geiger Mueller Survey Meter or Geiger Counter

The Geiger-Mueller (GM) survey meter will detect low levels of gamma and most beta radiation. The instrument typically has the capability to distinguish between gamma and beta radiation. This instrument is used to measure background radiation levels and to quickly evaluate potentially contaminated victims. If a greater level of radiation emission is anticipated, a higher range instrument (such as an ionization chamber) should be used. At higher levels, the GM meter will often display incorrectly low or off-scale readings. All healthcare facilities must have staff that knows how to use a GM survey meter.

Ionization Chamber Survey Meter

This device measures gamma ray dose/rate when high level radiation hazards are suspected. Low level gamma contamination is not detected.

Alpha Monitors
Alpha Monitors are designed to measure the presence of alpha particles. Since alpha particles travel short distances, they might not be detected in wounds because blood and tissue fluids may shield the particles from reaching the monitor's surface. Because of these factors experienced persons such as the Radiation Safety Officer (RSO) should do alpha surveys.

Dose Rate Meters
These measure mrad/hour or rad/hour units of personal radiation. To find the dose an individual received, multiply the dose rate by the time. Dose = Dose Rate X Time

Pocket or Personal Dosimeters
These simple devices measure accumulated radiation to gamma rays. Some devices basically contain a piece of film embedded in a badge of varying densities.

Other Devices Used
TLD (lithium fluoride) and QFD Quartz Fiber Dosimeters, and Electronic Readout Dosimeters.

Radiation Units

The basic unit for measuring radiation is the rad (radiation absorbed dose). The rad is defined as the deposition of 0.01 joule of energy per kilogram (kg) of tissue. To quantify the amount of damage that is suspected from a radiation exposure, rads are converted into rems (which at one time stood for Roentgen Equivalent Man). The rem is adjusted to reflect the type of radiation absorbed and the likelihood of damage to tissues/cells. For beta, gamma, and X-rays the rad will be equivalent to the rem (1 rem = 1,000 millirem).

The rem was introduced to take into account this variation in potential tissue damage. This is important because radiation may be of mixed type. For example, a standard X-ray machine was used to deliver 100 rads of radiation and to compare the biological endpoint with other types of radiation. It was found that 100 rads of gamma and beta radiation produced the same effect as 100 rads of X-ray. However, it was found that only 20 rads of neutrons and 5 rads of alpha were found to produce the same effect as 100 rads of X-ray. Therefore, neutron and alpha radiations were more potent and required fewer rads to produce the same effect. (This concept applies only to occupational exposure.) Now weighting factors are used for each organ/tissue.

Radioactive Materials

Radioactive materials, materials that emit ionizing radiation, are used in diagnosis (nuclear medicine), therapy (cancer treatment), industry (non-destructive testing), and for research purposes. A number of radioactive materials, including radioactive waste, are commercially shipped in specialized containers.

Radioactive materials are chemically and physically identical to their non-radioactive counterparts and behave in the body the same as their non-radioactive counterparts (for example, radioactive iodine behave the same as stable iodine). For practical purposes, after 10 half-lives, most of the radioactivity in a particular quantity of radioactive material is gone.

Radioactivity has existed for millions of years in the crust of the earth, in building materials, in the food we eat, the air we breathe, and in virtually everything that surrounds us. Radiation from these materials, as well as cosmic radiation from the sun and universe, makes up the background radiation to which we are constantly exposed.
Most individuals are exposed to about 360 millirems per year through natural causes and manmade sources. Smoking 1.5 packs of cigarettes a day for 1 year produces an accumulative radiation doses of 16 rem to the bifurcation of the bronchus. If an individual is exposed to more than 100 rads at one time, predictable signs and symptoms will develop within a few hours, days, or weeks depending on the dose. Fifty percent of individuals exposed to a single dose of 450 millirems will die without medical intervention.

Radiation Protection Guidelines

Time
The shorter the time in a radiation field, the less the radiation exposure. Work quickly and efficiently. A rotating team approach can be used to keep individual radiation exposures to a minimum.

Distance
The farther a person is from a source of radiation, the lower the radiation dose. Do not touch radioactive materials. Use shovels, brooms, etc., to move materials to avoid physical contact.

Shielding
Although not always practical in emergency situations, shielding offered by barriers can reduce radiation exposure.

Quantity
Limit the amount of radioactive material in the working area to decrease exposure.

RADIATION INJURY

Types of Radiation Injury

Regardless of where or how an accident involving radiation happens, *three types of radiation-induced injury can occur: external irradiation, contamination with radioactive materials, and incorporation of radioactive material into body cells, tissues, or organs.*

External Irradiation

External irradiation occurs when all or part of the body is exposed to penetrating radiation from an external source. During exposure this radiation can be absorbed by the body or it can pass completely through. A similar thing occurs during an ordinary chest x-ray. Following external exposure, an individual is not radioactive and can be treated like any other patient.

Contamination

The second type of radiation injury involves contamination with radioactive materials. Contamination means that radioactive materials in the form of gases, liquids, or solids are released into the environment and contaminate people externally, internally, or both. An external surface of the body, such as the skin, can become contaminated. These victims pose a threat to health care providers and must be decontaminated. If radioactive materials get inside the body through the lungs, gut, or wounds, the contaminant can become deposited internally.

Incorporation

The third type of radiation injury that can occur is incorporation of radioactive material. Incorporation refers to the uptake of radioactive materials by body cells, tissues, and target organs such as bone, liver, thyroid, or kidney. In general, radioactive materials are distributed throughout the body based upon their chemical properties. Incorporation cannot occur unless contamination has occurred.

Severity of Injury

In general, the higher the dose, the more severe the early effects will be and the greater the possibility of delayed effects. Obviously, one can increase the dose until the cell is killed outright. However, it is found that a much lower dose can stop cell division.

For example, if we consider the hematopoietic system, an individual hematopoietic stem cell has the capability of producing millions of mature cells. Preventing stem cell division means the loss of these cells. The importance of this is that a sub-lethal dose produces these effects.

Two important organ systems that have rapidly dividing cell lines are the hematopoietic and gastrointestinal systems.

After the dropping of the atomic bombs in Japan, experiments were carried out on various animals to determine the dose that would kill 50 percent of the experimental animal population within a set time period. Accident data on humans that were not treated indicate the lethal dose (LD) 50 was in the region of 350 rads to 450 rads.

Acute Radiation Syndrome (ARS)

Definition

An acute illness, which follows a roughly predictable course over a period of time ranging from a few hours to several weeks after exposure to ionizing radiation. The acute radiation syndrome is produced if enough radiation reaches enough sensitive tissue. Important factors are:

- High dose
- High dose rate
- Whole body exposure
- Penetrating irradiation

Signs and Symptoms

The signs and symptoms that develop in ARS occur through four distinct phases:

Prodromal phase

Depending on the total amount of radiation absorbed, patients may experience a variety of symptoms including loss of appetite, nausea, vomiting, fatigue, and diarrhea. After high radiation doses, additional symptoms such as prostration, fever, respiratory difficulties, and increased excitability may develop. This is the stage at which most victims seek medical care.

Latent phase

This is the transitional period in which many of the initial symptoms resolve, and may last for up to 3 weeks depending on the original radiation dose. This time interval decreases as the initial dose increases.

Illness phase

The period of time when overt illness develops, often characterized by infection, bleeding, electrolyte imbalance, diarrhea, changes in mental status, and shock.

Recovery or death phase

This follows the period of overt illness, which may take weeks or months to resolve.

AFFECTED SYSTEMS

Hematopoietic or Blood Forming System

This system shows the earliest indication of the severity of the radiation exposure through the rapidity and degree of drop in the cell count (lymphocytes, granulocytes, thrombocytes, and reticulocytes). This reduction in the cell count is commonly associated with fever, sepsis, and hemorrhagic complications.

The absolute lymphocyte count at 48 hours after exposure is a good predictor for prognosis. For example, if the total lymphocyte count is greater than 1,200 it is unlikely that the patient has received a lethal dose. If at 48 hours the lymphocyte cell count is between 300 and 1,200, a significant exposure has occurred and the patient should be hospitalized with barrier protection isolation. Lymphocyte levels of less than 300 cells per/ml are usually critical and warrant the consideration of the use of colony-stimulating factors on an individual basis.

Gastrointestinal System

Symptoms in this system are regularly seen at acute doses greater than 600 rads and result from damage to the epithelial cells lining the intestinal tract. The higher the exposure, the sooner the symptoms of nausea and vomiting develop. The presence of these symptoms typically overlap with the drop in the cell count described previously. As a result, sepsis, loss of fluids, electrolytes and opportunistic infections complicate the picture. Persistent high fevers and bloody diarrhea is an ominous sign despite fluid and electrolyte replacement.

Central Nervous System (CNS)

Central nervous system symptoms are seen with acute radiation doses in excess of 1000 rads and are probably due to diffuse microvascular leaks within the brain. Damage to these blood vessels result in the loss of fluids and electrolytes, edema, increased intracranial pressure, and death. This injury is irreversible and the victim rarely lives long enough to suffer any hematological or gastrointestinal symptoms. Symptoms of shock may develop quickly in these patients. There is also associated cardiovascular collapse in this kind of patient.

Integumentary

Various skin changes occur depending on the radiation dose. The injuries tend to progress with dose level and there appears to be a threshold effect for these clinical signs. Early erythema is an important sign to look for. At doses around 300 rads, erythema will develop within a few hours, but more importantly, it can disappear within a few hours only to reappear at a later time. Therefore, the patient should be examined on an hourly basis for this sign and ideally photographs should be taken to document this sign. If local radiation dermatitis develops with this sign, the dose is in the region of 1,000 rads. If blistering occurs then the dose is in the range of 1,500 rads. Also if necrosis develops, the dose is in the region of greater than 5,000 rads. Therefore, by noting these clinical signs, one is able to establish the approximate dose range the patient was subjected to and these doses would be confirmed by dosimetry at a later stage.

>300 rads: Epilation 17-21 days
>600 rads: Erythema that may disappear within a few hours

>1000 rads: Dry desquamation 2 - 4 weeks
>1500 rads: Moist desquamation 2 - 8 weeks
>5000 rads: Necrosis few days to months

Trauma and Radiation

Patients who have suffered trauma (from an explosive or burn) combined with an acute exposure to penetrating radiation will have an increased chance of dying as compared to patients who have suffered from the same dose of radiation without trauma. All combined injuries are worse than radiation alone. If a patient has received an acute dose greater than 200 rads, effort must be made to close wounds, cover burns, reduce fractures, and perform surgical stabilizing and definitive treatments within the first 48 hours after injury. After 48 hours, surgical interventions should be delayed for 2 to 3 months.

Triage of Radiation Casualties

Triage of victims from a radiological event should follow the same principles used in sorting victims of a hazardous material incident. Victims are classified with regard to their need for treatment and will be classified as requiring minimal treatment, immediate care, delayed care, or as expectant. Since the degree of radiation injury will not be initially apparent, triage criteria will need to be based on associated injuries and complaints. The triage method used will vary according to local practices.

Patients who require immediate attention are those with traumatic injuries such as crushing extremity wounds, incomplete amputations, severe burns of face and upper respiratory tract, and difficulty breathing due to mechanical problems.

Delayed casualties include those with traumatic injuries that are not life-threatening such as simple fractures, or second and third degree burns less than 25 percent of body surface area (BSA).

Minimal casualties are those with burns less than 10 percent of BSA, but not involving critical areas or those who have received short-term body ionizing radiation doses of 100 to 150 rads. When this dose of irradiation is combined with burns, then the prognosis is much more severe.

Expectant casualties have severe burns greater than 30 percent BSA, critical injuries to the respiratory or nervous system, or have received lethal doses of total body radiation, as indicated by a combination of clinical signs, including high fever, disorientation, bloody diarrhea, or vomiting.

Classification, Treatment, and Disposition

Once the radiological survey and decontamination procedure is complete, patients may be classified into one of 3 categories, based on their presenting signs and symptoms.

Survival Probable Group

This group of patients who present without any initial symptoms, or whose symptoms are so minimal (i.e., nausea and vomiting), that they resolve in a few hours. These individuals have most probably not received a lethal radiation dose, and most likely are exposed to < 100 rads. The initial CBC and sequential studies will not show a significant decrease in the lymphocyte or granulocyte counts. These patients can be safely sent home and instructed to return if symptoms redevelop.

Survival Possible Group

These victims present with nausea and vomiting, which typically last 24 to 48 hours followed by an asymptomatic period. During this latent phase, laboratory evaluations will show a drop in various cell counts (lymphocytopenia, leukocytopenia, and thrombocytopenia). If vomiting is severe, these patients should be admitted for fluid and electrolyte therapy and treated with antiemetics.

If the absolute lymphocyte count is less that 1,200 (or 50 percent of the baseline), protective isolation precautions should be implemented. These patients will have typically received a radiation dose in the range of 200 to 800 rads. The LD50 in mass casualty situations is in the range of 350 to 450 rads. Treatment is primarily supportive.

Blood replacement products, hyperalimentation, antibiotics, antivirals, and antifungal medications should only be administered after consultation with a hematologist, oncologist, or infectious disease specialist. Colony-stimulating factors will probably be indicated for pancytopenia.

Survival Improbable Group

Patients in this group have been exposed to whole-body irradiation in doses exceeding 800 rads. These victims present an acute onset of fulminating vomiting, diarrhea, and shock, requiring aggressive fluid and electrolyte therapy. The presence of any CNS symptoms (confusion, a change in mental status, etc.) signals that the patient has received a lethal dose of radiation. These victims will develop bone marrow suppression, leading to aplasia and pancytopenia that is uniformly fatal unless a successful hematopoietic stem cell transplant and/or colony stimulating factors are used. Treatment outcomes in some recent accidents suggest that exposure in the 800 to 1,200 rad range can be successfully managed through the hematopoietic crisis, although the individuals treated often succumbed to residual lung damage 6 to 12 months following exposure. In mass casualty situations, these victims are provided with comfort measures only (i.e., pain management).

CASUALTY MANAGEMENT IN A DISASTER

Activate the Hospital Plan

- Establish triage area outside the hospital
- Set up decontamination corridor, preferable outside the Emergency Department
- Plan to control contamination:
- Set up a controlled area large enough to hold the anticipated number of victims. Demarcate controlled area with tape or floor markings.
- Prevent tracking of contaminants by covering floor areas
- Restrict access to the controlled area
- Monitor anyone or anything leaving the controlled area
- Use strict isolation precautions, including protective clothing and double bagging
- Use a buffer zone or secondary control line for added security
- Control waste by using plastic-lined container for clothing, linens, dressings, etc.
- Control ventilation
- Change instruments, outer gloves, drapes, etc. when they become contaminated
- Use waterproof materials to limit the spread of contaminated liquids; for example, waterproof aperture drapes
- Protective clothing for staff: gowns (preferably water-resistant), caps, masks, boots, and two pairs of gloves. Staff should wear dosimetry (if it is available) and eye protection.
- Notify Radiation Safety Officer (RSO) to issue dosimeters, prepare instruments and mobilize nuclear medicine staff to assist with surveys
- Designate storage area for waste (outside hospital)

Patient Arrival

- Determine if incident involves contamination. If so, each patient should be surveyed, checking for the presence of radioactive contamination. If contaminated, the patient should be directed to the decontamination area.
- Upon arrival, all patient clothing should be removed under the guidance/direction of staff, so that further contamination of the patient is limited. Clothing and personal belongings are placed in a labeled biohazard bag.
- Patients suspected of being contaminated should be decontaminated. If open wounds are present, they should be irrigated first. Then covered with a sterile, waterproof dressing prior to the total body washing. After decontamination, each patient should be re-surveyed.
- After decontamination, biological samples should be taken: nasal swabs, throat swabs, etc.
- Collect blood for CBC and differential.
- Note past medical history of patient. Important questions are history of renal disease, allergies, or nuclear medicine procedures.

Internal Contamination

Once radioactive materials cross cell membranes, they are said to be incorporated. Incorporation is a time-dependent, physiological phenomenon related to both the physical and chemical natures of the contaminant. The rate of incorporation can be quite rapid, occurring in minutes, or it can take days to months. Thus, time can be critical and treatment (decorporation) urgent. Several methods of preventing incorporation (e.g., catharsis, gastric lavage) might be applicable and can be prescribed by a physician. Some of the medications or preparations used in decorporation might not be available at the facility but should be stocked locally.

If internal contamination is suspected or has occurred, the physician, or RSO should request samples of urine, feces, vomitus, wound secretions, etc. Whole-body counting and radioassay also can help evaluate the magnitude of the problem and the effect of any treatment. The contaminated patient admitted with an airway device or endotracheal tube must be considered to be internally contaminated.

KEY POINTS

Hospital personnel should be prepared for a nuclear reactor accident, industrial incident, or terrorist event. It will present many unique challenges to hospital personnel. A radiation-contaminated patient should be handled in the same manner as any hazardous material accident victim.

Hospital emergency department personnel should always use proper priorities in caring for accident victims where potential radiation hazards exist: treat life-threatening problems first, limit the radiation dose to both victim and personnel, and control the spread of radioactive contaminants. *Serious medical problems have priority over the concerns about radiation, such as radiation monitoring, contamination control, and decontamination.*

Irradiation of the whole body or some specific body part does not constitute a medical emergency even if the amount of radiation received is high. The effects of irradiation usually are not evident for days to weeks and while medical treatment is needed, it is not needed on an emergency basis. On the other hand, contamination accidents must be considered medical emergencies since they *might* lead to internal contamination and subsequent incorporation. Incorporation can result in adverse health effects several years later if the amount of incorporated radioactive material is high.

Carefully evaluating the initial presenting signs and symptoms (such as nausea, vomiting, diarrhea, changes in mental status, shock, and lymphocyte count over the first 48 hours) becomes the most reliable indicator of the radiation dose and patient's ultimate prognosis.

Since no antidote exists for radiation exposure, treatment is primarily supportive with more specialized care directed towards patients with high dose irradiation and those with internal contamination. Consultation with specialists in hematology, oncology, radiation, and infectious disease should be obtained.

The need for initial treatment for internally contaminated patients is determined based on the patient's medical condition, history, biological samples (nasal swabs), and definitive evaluation of internal contamination. Whole body counting may be needed if internal contamination is suspected.

MEDICATIONS AND MECHANISMS OF DECORPORATION

(Modified Form Safety Series 47, IAEA)

Radionuclide	Medication	Ingestion/Inhalation	Wound	Principle of Action
Iodine	KI	130 mg (tab.) stat, followed by 130 mg q.d. x 7 if indicated	Same	Blocking
Rare earths Plutonium Transplutonics Yttrium	DTPA	1 gm Ca-DTPA in 500 ml 5 percent D/W IV over 60 min; or 1 gm (4ml) in 6 ml 5 percent D/W by slow IV injection (1 min)		Chelation
Polonium Mercury Arsenic Bismuth Gold	BAL	One ampule (=300 mg) IM q 4 hrs for 3 days - (first test for sensitivity with 3 amp.)	Same	Promotes excretion
Uranium	Bicarbonate	Slow IV infusion of bicarbonated physiological solution (250 ml at 14 percent)	Slow IV infusion of bicarbonated physiological solution (250 ml at 14 percent) and wash with bicarbonate	Alkalinization of urine; reduces chance of ATN
Cesium Rubidium Thallium	Prussian Blue [Ferrihexacyano-Ferrate(II)]	1 gm in 100-200 ml water p.o. t.i.d. for several days	Same	Mobilization from organs and tissues - reduction and absorption
Radium	Ca-gluconate	May be tried; 20 percent Ca-gluconate 10 ml IV once or twice daily	Same	Displacement
Strontium	Ammonium chloride	3 gm t.i.d. p.o.	Same	Demineralizing agent
Tritium	Water	Force liquid	Same	Isotopic dilution
Strontium Radium	$BaSO_4$	100 gm $BaSO_4$ in 250 ml of water	Same	Reduces absorption
Calcium Barium	Sodium Alignate	10 gm in a large glass of water	Same	Inhibits absorption
Copper Polonium Lead Mercury Gold	D-penicillamine	1 gm IV q.d. or 0.9 gm p.o. 14 - 6 hours	Same	Chelation

FACT SHEET

Acute Radiation Syndrome: A Fact Sheet for Physicians

Acute Radiation Syndrome (ARS) (sometimes known as radiation toxicity or radiation sickness) is an acute illness caused by irradiation of the entire body (or most of the body) by a high dose of penetrating radiation in a very short period of time (usually a matter of minutes). The major cause of this syndrome is depletion of immature parenchymal stem cells in specific tissues. Examples of people who suffered from ARS are the survivors of the Hiroshima and Nagasaki atomic bombs, the firefighters that first responded after the Chernobyl Nuclear Power Plant event in 1986, and some unintentional exposures to sterilization irradiators.

The required conditions for Acute Radiation Syndrome (ARS) are:

- **The radiation dose must be large** (i.e., greater than 0.7 Gray (Gy)[1,2] or 70 rads).
  - o Mild symptoms may be observed with doses as low as 0.3 Gy or 30 rads.
- **The dose usually must be external** (i.e., the source of radiation is outside of the patient's body).
  - o Radioactive materials deposited inside the body have produced some ARS effects only in extremely rare cases.
- **The radiation must be penetrating** (i.e., able to reach the internal organs).
  - o High energy X-rays, gamma rays, and neutrons are penetrating radiations.
- **The entire body** (or a significant portion of it) must have received the dose.[3]
  - o Most radiation injuries are local, frequently involving the hands, and these local injuries seldom cause classical signs of ARS.
- **The dose must have been delivered in a short time** (usually a matter of minutes).
  - o Fractionated doses are often used in radiation therapy. These large total doses are delivered in small daily amounts over a period of time. Fractionated doses are less effective at inducing ARS than a single dose of the same magnitude.

The three classic ARS Syndromes are:

- Bone marrow syndrome (sometimes referred to as hematopoietic syndrome): the full syndrome will usually occur with a dose greater than approximately 0.7 Gy (70 rads) although mild symptoms may occur as low as 0.3 Gy or 30 rads.[4]
  - o The survival rate of patients with this syndrome decreases with increasing dose. The primary cause of death is the destruction of the bone marrow, resulting in infection and hemorrhage.
- Gastrointestinal (GI) syndrome: the full syndrome will usually occur with a dose greater than approximately 10 Gy (1000 rads) although some symptoms may occur as low as 6 Gy or 600 rads.

1 The Gray (Gy) is a unit of absorbed dose and reflects an amount of energy deposited into a mass of tissue (1 Gy = 100 rads). In this document, the referenced absorbed dose is that dose inside the patient's body (i.e., the dose that is normally measured with personal dosimeters).

2 The referenced absorbed dose levels in this document are assumed to be from beta, gamma, or x radiation. Neutron or proton radiation produces many of the health effects described herein at lower absorbed dose levels.

3 The dose may not be uniform, but a large portion of the body must have received more than 0.7 Gy (70 rads).

4 Note: although the dose ranges provided in this document apply to most healthy adult members of the public, a great deal of variability of radiosensitivity among individuals exists, depending upon the age and condition of health of the individual at the time of exposure. Children and infants are especially sensitive.

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  - o Survival is extremely unlikely with this syndrome. Destructive and irreparable changes in the GI tract and bone marrow usually cause infection, dehydration, and electrolyte imbalance. Death usually occurs within 2 weeks.
- **Cardiovascular (CV)/ Central Nervous System (CNS) syndrome:** the full syndrome will usually occur with a dose greater than approximately 50 Gy (5000 rads) although some symptoms may occur as low as 20 Gy or 2000 rads.
  - o Death occurs within 3 days. Death likely is due to collapse of the circulatory system as well as increased pressure in the confining cranial vault as the result of increased fluid content caused by edema, vasculitis, and meningitis.

The four stages of ARS are:

- **Prodromal stage (N-V-D stage):** The classic symptoms for this stage are nausea, vomiting, as well as anorexia and possibly diarrhea (depending on dose), which occur from minutes to days following exposure. The symptoms may last (episodically) for minutes up to several days.
- **Latent stage:** In this stage, the patient looks and feels generally healthy for a few hours or even up to a few weeks.
- **Manifest illness stage:** In this stage, the symptoms depend on the specific syndrome (see Table 1) and last from hours up to several months.
- **Recovery or death:** Most patients who do not recover will die within several months of exposure. The recovery process lasts from several weeks up to two years.

These stages are described in more detail in Table 1.

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Table 1. Acute Radiation Syndromes

Syndrome	Dose*	Prodromal Stage	Latent Stage	Manifest Illness Stage	Recovery
Hematopoietic (Bone marrow)	> 0.7 Gy (> 70 rads) (mild symptoms may occur as low as 0.3 Gy or 30 rads)	• Symptoms are anorexia, nausea and vomiting. • Onset occurs 1 hour to 2 days after exposure. • Stage lasts for minutes to days.	• Stem cells in bone marrow are dying, although patient may appear and feel well. • Stage lasts 1 to 6 weeks.	• Symptoms are anorexia, fever, and malaise. • Drop in all blood cell counts occurs for several weeks. • Primary cause of death is infection and hemorrhage. • Survival decreases with increasing dose. • Most deaths occur within a few months after exposure.	• In most cases, bone marrow cells will begin to repopulate the marrow. • There should be full recovery for a large percentage of individuals from a few weeks up to two years after exposure • Death may occur in some individuals at 1.2 Gy (120 rads). • The $LD_{50/60}$† is about 2.5 to 5 Gy (250 to 500 rads).
Gastrointestinal (GI)	> 10 Gy (> 1000 rads) (some symptoms may occur as low as 6 Gy or 600 rads)	• Symptoms are anorexia, severe nausea, vomiting, cramps, and diarrhea. • Onset occurs within a few hours after exposure. • Stage lasts about 2 days.	• Stem cells in bone marrow and cells lining GI tract are dying, although patient may appear and feel well. • Stage lasts less than 1 week.	• Symptoms are malaise, anorexia, severe diarrhea, fever, dehydration, and electrolyte imbalance. • Death is due to infection, dehydration, and electrolyte imbalance. • Death occurs within 2 weeks of exposure.	• The LD_{100}‡ is about 10 Gy (1000 rads).
Cardiovascular (CV)/ Central Nervous System (CNS)	> 50 Gy (5000 rads) (some symptoms may occur as low as 20 Gy or 2000 rads)	• Symptoms are extreme nervousness and confusion; severe nausea, vomiting, and watery diarrhea; loss of consciousness; and burning sensations of the skin. • Onset occurs within minutes of exposure. • Stage lasts for minutes to hours.	• Patient may return to partial functionality. • Stage may last for hours but often is less.	• Symptoms are return of watery diarrhea, convulsions, and coma. • Onset occurs 5 to 6 hours after exposure. • Death occurs within 3 days of exposure.	• No recovery is expected.

* The absorbed doses quoted here are "gamma equivalent" values. Neutrons or protons generally produce the same effects as gamma, beta, or X-rays but at lower doses. If the patient has been exposed to neutrons or protons, consult radiation experts on how to interpret the dose.
† The $LD_{50/60}$ is the dose necessary to kill 50% of the exposed population in 60 days.
‡ The LD_{100} is the dose necessary to kill 100% of the exposed population.

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Cutaneous Radiation Syndrome (CRS)

The concept of cutaneous radiation syndrome (CRS) was introduced in recent years to describe the complex pathological syndrome that results from acute radiation exposure to the skin.

ARS usually will be accompanied by some skin damage. It is also possible to receive a damaging dose to the skin without symptoms of ARS, especially with acute exposures to beta radiation or X-rays. Sometimes this occurs when radioactive materials contaminate a patient's skin or clothes.

When the basal cell layer of the skin is damaged by radiation, inflammation, erythema, and dry or moist desquamation can occur. Also, hair follicles may be damaged, causing epilation. Within a few hours after irradiation, a transient and inconsistent erythema (associated with itching) can occur. Then, a latent phase may occur and last from a few days up to several weeks, when intense reddening, blistering, and ulceration of the irradiated site are visible.

In most cases, healing occurs by regenerative means; however, very large skin doses can cause permanent hair loss, damaged sebaceous and sweat glands, atrophy, fibrosis, decreased or increased skin pigmentation, and ulceration or necrosis of the exposed tissue.

Patient Management

Triage: If radiation exposure is suspected:

- Secure ABCs (airway, breathing, circulation) and physiologic monitoring (blood pressure, blood gases, electrolyte and urine output) as appropriate.
- Treat major trauma, burns, and respiratory injury if evident.
- In addition to the blood samples required to address the trauma, obtain blood samples for CBC (complete blood count), with attention to lymphocyte count, and HLA (human leukocyte antigen) typing prior to any initial transfusion and at periodic intervals following transfusion.
- Treat contamination as needed.
- If exposure occurred within 8 to 12 hours, repeat CBC, with attention to lymphocyte count, 2 or 3 more times (approximately every 2 to 3 hours) to assess lymphocyte depletion.

Diagnosis

The diagnosis of ARS can be difficult to make because ARS causes no unique disease. Also, depending on the dose, the prodromal stage may not occur for hours or days after exposure, or the patient may already be in the latent stage by the time they receive treatment, in which case the patient may appear and feel well when first assessed.

If a patient received more than 0.05 Gy (5 rads) and three or four CBCs are taken within 8 to 12 hours of the exposure, a quick estimate of the dose can be made (see Ricks, et. al. for details). If these initial blood counts are not taken, the dose can still be estimated by using CBC results over the first few days. It would be best to have radiation dosimetrists conduct the dose assessment, if possible.

If a patient is known to have been or suspected of having been exposed to a large radiation dose, draw blood for CBC analysis with special attention to the lymphocyte count, every 2 to 3 hours during the first 8 hours after exposure (and every 4 to 6 hours for the next 2 days). Observe the patient during this time for symptoms and consult with radiation experts before ruling out ARS.

If no radiation exposure is initially suspected, you may consider ARS in the differential diagnosis if a history exists of nausea and vomiting that is unexplained by other causes. Other indications are bleeding, epilation, or white blood count (WBC) and platelet counts abnormally low a few days or weeks after unexplained nausea and vomiting. Again, consider CBC and chromosome analysis and consultation with radiation experts to confirm diagnosis.

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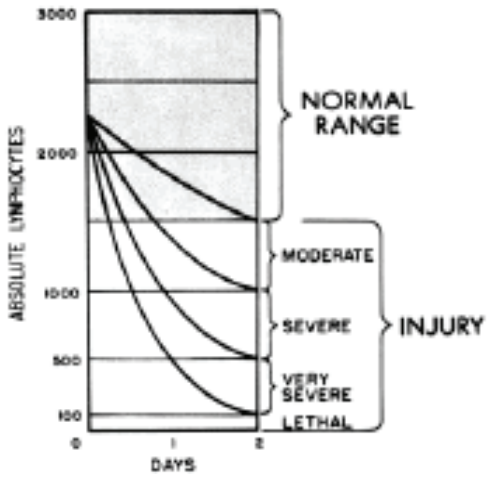
Initial Treatment and Diagnostic Evaluation

Treat vomiting[5] and repeat CBC analysis with special attention to the lymphocyte count every 2 to 3 hours for the first 8 to 12 hours after exposure (and every 4 to 6 hours for the following 2 or 3 days). Sequential changes in absolute lymphocyte counts over time are demonstrated below in the Andrews Lymphocyte Nomogram (see Figure 1). Precisely record all clinical symptoms, particularly nausea, vomiting, diarrhea, and itching, reddening or blistering of the skin. Be sure to include time of onset.

Figure 1: Andrews Lymphocyte Nomogram



From Andrews GA, Auxier JA, Lushbaugh CC. *The Importance of Dosimetry to the Medical Management of Persons Exposed to High Levels of Radiation*. In *Personal Dosimetry for Radiation Accidents*. Vienna: International Atomic Energy Agency; 1965.

Note and record areas of erythema. If possible, take color photographs of suspected radiation skin damage. Consider tissue, blood typing, and initiating viral prophylaxis. Promptly consult with radiation, hematology, and radiotherapy experts about dosimetry, prognosis, and treatment options. Call the Radiation Emergency Assistance Center/Training Site (REAC/TS) at (865) 576-3131 (M-F, 8 am to 4:30 am EST) or (865) 576-1005 (after hours) to record the incident in the Radiation Accident Registry System.

After consultation, begin the following treatment (as indicated):

- supportive care in a clean environment (if available, the use of a burn unit may be quite effective)
- prevention and treatment of infections
- stimulation of hematopoiesis by use of growth factors
- stem cell transfusions or platelet transfusions (if platelet count is too low)
- psychological support
- careful observation for erythema (document locations), hair loss, skin injury, mucositis, parotitis, weight loss, or fever
- confirmation of initial dose estimate using chromosome aberration cytogenetic bioassay when possible. Although resource intensive, this is the best method of dose assessment following acute exposures.
- consultation with experts in radiation accident management

5 Collect vomitus in the first few days for later analysis.

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For More Help

Technical assistance can be obtained from the Radiation Emergency Assistance Center/Training Site (REAC/TS) at (865) 576-3131 (M-F, 8 am to 4:30 pm EST) or (865) 576-1005 (after hours), or on their web site at www.orau.gov/reacts, and the Medical Radiobiology Advisory Team (MRAT) at (301) 295-0316.

Also, more information can be obtained from the CDC Health Alert Network at www.bt.cdc.gov or by calling (800) 311-3435.

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For more information, visit www.bt.cdc.gov/radiation,
or call CDC at 800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

FACT SHEET

Cutaneous Radiation Injury: Fact Sheet for Physicians

Injury to the skin and underlying tissues from acute exposure to a large external dose of radiation is referred to as cutaneous radiation injury (CRI). Acute radiation syndrome (ARS)[1] will usually be accompanied by some skin damage; however, CRI can occur without symptoms of ARS. This is especially true with acute exposures to beta radiation or low-energy x-rays, because beta radiation and low-energy x-rays are less penetrating and less likely to damage internal organs than gamma radiation is. CRI can occur with radiation doses as low as 2 Gray (Gy) or 200 rads[2] and the severity of CRI symptoms will increase with increasing doses. Most cases of CRI have occurred when people inadvertently came in contact with unsecured radiation sources from food irradiators, radiotherapy equipment, or well depth gauges. In addition, cases of CRI have occurred in people who were overexposed to x-radiation from fluoroscopy units.

Early signs and symptoms of CRI are itching, tingling, or a transient erythema or edema without a history of exposure to heat or caustic chemicals. Exposure to radiation can damage the basal cell layer of the skin and result in inflammation, erythema, and dry or moist desquamation. In addition, radiation damage to hair follicles can cause epilation. Transient and inconsistent erythema (associated with itching) can occur within a few hours of exposure and be followed by a latent, symptom-free phase lasting from a few days to several weeks. After the latent phase, intense reddening, blistering, and ulceration of the irradiated site are visible. Depending on the radiation dose, a third and even fourth wave of erythema are possible over the ensuing months or possibly years.

In most cases, healing occurs by regenerative means; however, large radiation doses to the skin can cause permanent hair loss, damaged sebaceous and sweat glands, atrophy, fibrosis, decreased or increased skin pigmentation, and ulceration or necrosis of the exposed tissue.

With CRI, it is important to keep the following things in mind:

- The visible skin effects depend on the magnitude of the dose as well as the depth of penetration of the radiation.
- Unlike the skin lesions caused by chemical or thermal damage, the lesions caused by radiation exposures do not appear for hours to days following exposure, and burns and other skin effects tend to appear in cycles.
- The key treatment issues with CRI are infection and pain management.[3]

[1] See “Acute Radiation Syndrome: A Fact Sheet for Physicians” at http://www.bt.cdc.gov/radiation/arsphysicianfactsheet.asp.
[2] Both the Gray (Gy) and the rad are units of absorbed dose and reflect the amount of energy deposited in a mass of tissue (1 Gy = 100 rads). In this document, the absorbed dose refers to that dose received by at least 10 cm^2 of the basal cell layer of the skin. The referenced absorbed dose levels in this document are assumed to be from beta, gamma, or x-radiation. Neutron or proton radiation produces many of the health effects described herein at lower absorbed dose levels.
[3] On occasion a patient might also be contaminated with radioactive material. To address patient decontamination, please go to the following Web site: http://www.orau.gov/reacts/emergency.htm.

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Stages and Grades of CRI
CRI will progress over time in stages and can be categorized by grade, with characteristics of the stages varying by grade of injury, as shown in Table 1. **Appendix A** gives a detailed description of the various skin responses to radiation, and **Appendix B** provides color photographs of examples of some of these responses.

Prodromal stage (within hours of exposure)—This stage is characterized by early erythema (first wave of erythema), heat sensations, and itching that define the exposure area. The duration of this stage is from 1 to 2 days.

Latent stage (1–2 days postexposure)—No injury is evident. Depending on the body part, the larger the dose, the shorter this period will last. The skin of the face, chest, and neck will have a shorter latent stage than will the skin of the palms of the hands or the soles of the feet.

Manifest illness stage (days to weeks postexposure)—The basal layer is repopulated through proliferation of surviving clonogenic cells. This stage begins with main erythema (second wave), a sense of heat, and slight edema, which are often accompanied by increased pigmentation. The symptoms that follow vary from dry desquamation or ulceration to necrosis, depending on the severity of the CRI (see Table 1).

Third wave of erythema (10–16 weeks postexposure, especially after beta exposure)—The exposed person experiences late erythema, injury to blood vessels, edema, and increasing pain. A distinct bluish color of the skin can be observed. Epilation may subside, but new ulcers, dermal necrosis, and dermal atrophy (and thinning of the dermis layer) are possible.

Late effects (months to years postexposure; threshold dose ~10 Gy or 1000 rads)—Symptoms can vary from slight dermal atrophy (or thinning of dermis layer) to constant ulcer recurrence, dermal necrosis, and deformity. Possible effects include occlusion of small blood vessels with subsequent disturbances in the blood supply (telangiectasia); destruction of the lymphatic network; regional lymphostasis; and increasing invasive fibrosis, keratosis, vasculitis, and subcutaneous sclerosis of the connective tissue. Pigmentary changes and pain are often present. Skin cancer is possible in subsequent years.

Recovery (months to years)

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Table 1. Grades of cutaneous radiation injury

	Skin dose*	Prodromal stage	Latent stage	Manifest illness stage	Third wave of erythema†	Recovery	Late effects
I	> 2 Gy (200 rads) ‡	1–2 days postexposure or not seen	no injury evident for 2–5 weeks postexposure§	• 2–5 weeks postexposure, lasting 20–30 days: redness of skin, slight edema, possible increased pigmentation • 6–7 weeks postexposure, dry desquamation	not seen	complete healing expected 28–40 days after dry desquamation (3–6 months postexposure)	• possible slight skin atrophy • possible skin cancer decades after exposure
II	> 15 Gy (1500 rads)	6–24 hours postexposure with immediate sensation of heat lasting 1–2 days	no injury evident for 1–3 weeks postexposure	• 1–3 weeks postexposure; redness of skin, sense of heat, edema, skin may turn brown • 5–6 weeks postexposure, edema of subcutaneous tissues and blisters with moist desquamation • possible epithelialization later	• 10–16 weeks postexposure, injury of blood vessels, edema, and increasing pain • epilation may subside, but new ulcers and necrotic changes are possible	healing depends on size of injury and the possibility of more cycles of erythema	• possible skin atrophy or ulcer recurrence • possible telangiectasia (up to 10 years postexposure) • possible skin cancer decades after exposure

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Cutaneous Radiation Injury: Fact Sheet for Physicians
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Grade	Skin dose*	Prodromal stage	Latent stage	Manifest illness stage	Third wave of erythema†	Recovery	Late effects
III	> 40 Gy (4000 rads)	4–24 hours	none or less than 2 weeks	• 1–2 weeks postexposure: redness of skin, blisters, sense of heat, slight edema, possible increased pigmentation • followed by erosions and ulceration as well as severe pain	• 10–16 weeks	can involve ulcers that are extremely difficult to treat and that can require months to years to heal fully	• possible skin atrophy, depigmentation, constant ulcer recurrence, or deformity • possible occlusion of small vessels with subsequent disturbances in the blood supply, destruction of the lymphatic network, regional lymphostasis, and increasing fibrosis and sclerosis of the connective tissue • possible telangiectasia • possible skin cancer decades after exposure

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Grade	Skin dose*	Prodromal stage	Latent stage	Manifest illness stage	Third wave of erythema†	Recovery	Late effects
IV	> 550 Gy (55,000 rads)	Occurs minutes to hours	none	•1–4 days postexposure accompanied by blisters •early ischemia (tissue turns white, then dark blue or black with substantial pain) in most severe cases •tissue becomes necrotic within 2 weeks following exposure, accompanied by substantial pain	does not occur due to necrosis of skin in the affected area	recovery possible following amputation of severely affected areas and possible skin grafts	•continued plastic surgery may be required over several years •possible skin cancer decades after exposure

*Absorbed dose to at least 10 cm^2 of the basal cell layer of the skin
†Especially with beta exposure
‡The Gray (Gy) is a unit of absorbed dose and reflects an amount of energy deposited in a mass of tissue (1 Gy = 100 rads).
§Skin of the face, chest, and neck will have a shorter latent phase than the skin of the palms of the hands and the skin of the feet.

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Patient Management

Diagnosis

The signs and symptoms of CRI are as follows:

- Intensely painful burn-like skin injuries (including itching, tingling, erythema, or edema) without a history of exposure to heat or caustic chemicals
  Note: Erythema will not be seen for hours to days following exposure, and its appearance is cyclic.
- Epilation
- A tendency to bleed
- Possible signs and symptoms of ARS

As mentioned previously, local injuries to the skin from acute radiation exposure evolve slowly over time, and symptoms may not manifest for days to weeks after exposure. Consider CRI in the differential diagnosis if the patient presents with a skin lesion without a history of chemical or thermal burn, insect bite, or skin disease or allergy. If the patient gives a history of possible radiation exposure (such as from a radiography source, x-ray device, or accelerator) or a history of finding and handling an unknown metallic object, note the presence of any of the following: erythema, blistering, dry or wet desquamation, epilation, ulceration.

Regarding lesions associated with CRI be aware that,

- days to weeks may pass before lesions appear;
- unless patients are symptomatic, they will not require emergency care; and
- lesions can be debilitating and life threatening after several weeks.

Medical follow-up is essential, and victims should be cautioned to avoid trauma to the involved areas.

Initial Treatment

Localized injuries should be treated symptomatically as they occur, and radiation injury experts should be consulted for detailed information. Such information can be obtained from the Radiation Emergency Assistance Center/Training Site (REAC/TS) at www.orau.gov/reacts/ or (865) 576-1005.

As with ARS, if the patient also has other trauma, wounds should be closed, burns covered, fractures reduced, surgical stabilization performed, and definitive treatment given within the first 48 hours after injury. After 48 hours, surgical interventions should be delayed until hematopoietic recovery has occurred.

A baseline CBC and differential should be taken and repeated in 24 hours. Because cutaneous radiation injury is cyclic, areas of early erythema should be noted and recorded. These areas should also be sketched and photographed, if possible, ensuring that the date and time are recorded. The following should be initiated as indicated:

- Supportive care in a clean environment (a burn unit if one is available)
- Prevention and treatment of infections
- Use of the following:
  - Medications to reduce inflammation, inhibit protealysis, relieve pain, stimulate regeneration, and improve circulation
  - Anticoagulant agents for widespread and deep injury
- Pain management
- Psychological support

Recommendations for Treatment by Stage

The following recommendations for treatment by stage of the illness were obtained by summarizing recommendations from Ricks et al. (226) and Gusev et al. (231), but they do not represent official recommendations of CDC.

Prodromal Stage—Use antihistamines and topical antipruriginous preparations, which act against itch and also might prevent or attenuate initiation of the cycle that leads to the manifestation stage. Anti-inflammatory medications such as corticosteroids and topical creams, as well as slight sedatives, may prove useful.
Latent Stage—Continue anti-inflammatory medications and sedatives. At midstage, use proteolysis inhibitors, such as Gordox®.
Manifestation Stage—Use repeated swabs, antibiotic prophylaxis, and anti-inflammatory medications, such as Lioxasol®, to reduce bacterial, fungal, and viral infections

- o Apply topical ointments containing corticosteroids along with locally acting antibiotics and vitamins.
- o Stimulate regeneration of DNA by using Lioxasol® and later, when regeneration has started, biogenic drugs, such as Actovegin® and Solcoseril®.
- o Stimulate blood supply in third or fourth week using Pentoxifylline® (contraindicated for patients with atherosclerotic heart disease).
- o Puncture blisters if they are sterile, but do not remove them as long as they are intact.
- o Stay alert for wound infection. Antibiotic therapy should be considered according to the individual patient's condition.
- o Treat pain according to the individual patient's condition. Pain relief is very difficult and is the most demanding part of the therapeutic process.
- o Debride areas of necrosis thoroughly but cautiously.

Treatment of Late Effects

After immediate treatment of radiation injury, an often long and painful process of healing will ensue. The most important concerns are the following:

- Pain management
- Fibrosis or late ulcers
  Note: Use of medication to stimulate vascularization, inhibit infection, and reduce fibrosis may be effective Examples include Pentoxifylline®, vitamin E, and interferon gamma. Otherwise, surgery may be required.
- Necrosis
- Plastic/reconstructive surgery
  Note: Surgical treatment is common. It is most effective if performed early in the treatment process. Full-thickness graft and microsurgery techniques usually provide the best results.
- Psychological effects, such as posttraumatic stress disorder
- Possibility of increased risk of skin cancer later in life

For More Assistance

Technical assistance can be obtained from the Radiation Emergency Assistance Center/Training Site (REAC/TS) at (865) 576-3131 (M-F, 8 AM to 4:30 PM EST) or (865) 576-1005 (after hours), or at http://www.orau.gov/reacts/, and from the Medical Radiobiology Advisory Team (MRAT) at (301) 295-0316.

Also, more information can be obtained from the CDC Health Alert Network at http://www.bt.cdc.gov or 1-800-311-3435.

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Appendix A: Responses of the Skin to Radiation

Acute epidermal necrosis (time of onset: < 10 days postexposure; threshold dose: ~550 Gy or 55,000 rads)—
Interphase death of postmitotic keratinocytes in the upper visible layers of the epidermis (may occur with high-dose, low-energy beta irradiation)

Acute ulceration (time of onset: < 14 days postexposure; threshold dose: ~20 Gy or 2000 rads)—
Early loss of the epidermis—and to a varying degree, deeper dermal tissue—that results from the death of fibroblasts and endothelial cells in interphase

Dermal atrophy (time of onset: > 26 weeks postexposure; threshold dose: ~10 Gy or 1000 rads)—
Thinning of the dermal tissues associated with the contraction of the previously irradiated area

Dermal necrosis (time of onset > 10 weeks postexposure; threshold dose: ~20 Gy or 2000 rads)—
Necrosis of the dermal tissues as a consequence of vascular insufficiency

Dry desquamation (time of onset: 3–6 weeks postexposure; threshold dose: ~8 Gy or 800 rads)—
Atypical keratinization of the skin caused by the reduction in the number of clonogenic cells within the basal layer of the epidermis

Early transient erythema (time of onset: within hours of exposure; threshold dose: ~2 Gray [Gy] or 200 rads)—
Inflammation of the skin caused by activation of a proteolytic enzyme that increases the permeability of the capillaries

Epilation (time of onset: 14–21 days; threshold dose: ~3 Gy or 300 rads)—
Hair loss caused by the depletion of matrix cells in the hair follicles

Late erythema (time of onset: 8–20 weeks postexposure; threshold dose: ~20 Gy or 2000 rads)—
Inflammation of the skin caused by injury of blood vessels. Edema and impaired lymphatic clearance precede a measured reduction in blood flow.

Invasive fibrosis (time of onset: months to years postexposure; threshold dose: ~20 Gy or 2000 rads)—
Method of healing associated with acute ulceration, secondary ulceration, and dermal necrosis that leads to scar tissue formation

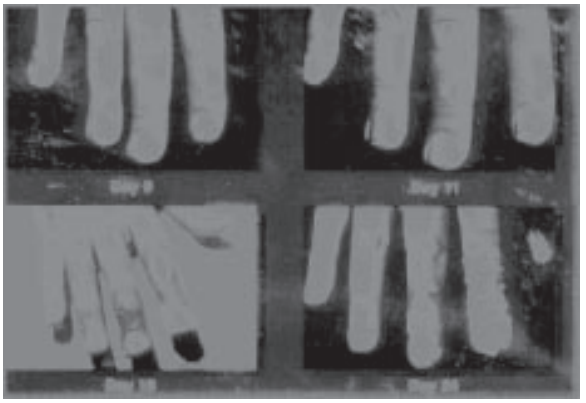
Main erythema (time of onset: days to weeks postexposure; threshold dose: ~3 Gy or 300 rads)—
Inflammation of the skin caused by hyperaemia of the basal cells and subsequent epidermal hypoplasia (see photos 1 and 2)

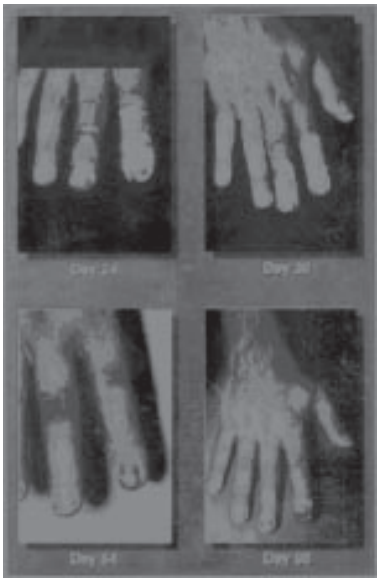
Moist desquamation (time of onset: 4–6 weeks postexposure; threshold dose: ~15 Gy or 1500 rads)—
Loss of the epidermis caused by sterilization of a high proportion of clonogenic cells within the basal layer of the epidermis

Secondary ulceration (time of onset: > 6 weeks postexposure; threshold dose: ~15 Gy or 1500 rads)—
Secondary damage to the dermis as a consequence of dehydration and infection when moist desquamation is severe and protracted because of reproductive sterilization of the vast majority of the clonogenic cells in the irradiated area

Telangiectasia (time of onset: > 52 weeks postexposure; threshold dose for moderate severity at 5 years: ~40 Gy or 4000 rads)—
Atypical dilation of the superficial dermal capillaries

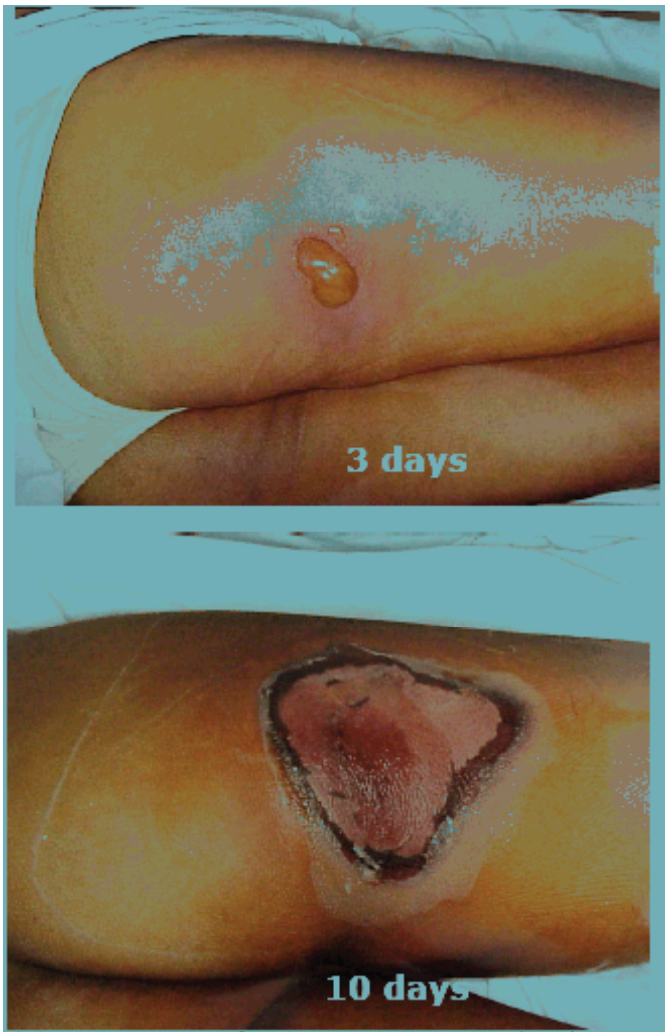
Appendix B: Images





Figures 1 & 2. Erythema. These photos display the progression of erythema in a patient involved in an x-ray diffraction accident, 9 days to 96 days postexposure. The day following the exposure (not shown), the patient displayed only mild diffuse swelling and erythema of the fingertips. On day 9, punctuate lesions resembling telangiectasias were noted in the subungal region of the right index finger, and on day 11, blisters began to appear. Desquamation continued for several weeks. The patient developed cellulitis in the right thumb approximately 2 years following exposure. The area of the right fingertip and nail continued to cause the patient great pain when even minor trauma occurred to the fingertip, and he required occasional oral narcotic analgesics to manage this pain. He continued to experience intense pain resulting from minor trauma to the affected areas for as long as 4 years postexposure.

(photos courtesy of Gusev IA and reprinted with permission)



(photos courtesy of Ricks RC and reprinted with permission)

Figures 3 & 4. Acute ulceration. These photos show acute ulceration in a Peruvian patient who inadvertently placed a 26-Ci (0.962-TBq) irridiun-192 (^{192}Ir) source in his back pocket, 3 days and 10 days postexposure. The source remained in the patient's pocket for approximately 6.5 hours, at which time he complained to his wife about pain in his posterior right thigh. He sought medical advice and was told he probably had been bitten by an insect. In the meantime, his wife sat on the patient's pants (her case appears on the next page) while breastfeeding the couple's 1½-year-old child. The source was recovered several hours later by nuclear regulatory authorities, and the patient was transported to Lima for treatment. This patient exhibited a drastic reduction in lymphocyte count by day 3 postexposure, and a 4-by-4-cm lesion appeared on day 4. Eventually he suffered with a massive ulceration and necrosis of the site with infection, and his right leg was amputated. Grade II and III CRI was also evident on his hands, left leg, and perineum, but he survived and returned to his family.

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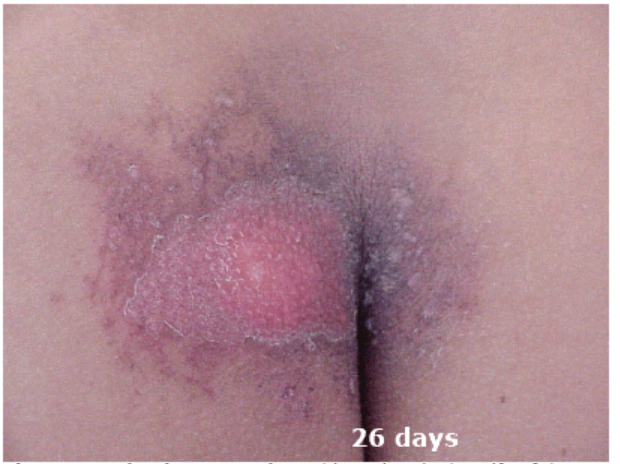

Figure 5. Moist desquamation. This patient is the wife of the previous case study, 26 days postexposure. She was exposed to the ^{192}Ir source when she sat on her husband's pants (still containing the source) for approximately 20 minutes after he had changed clothes that evening.

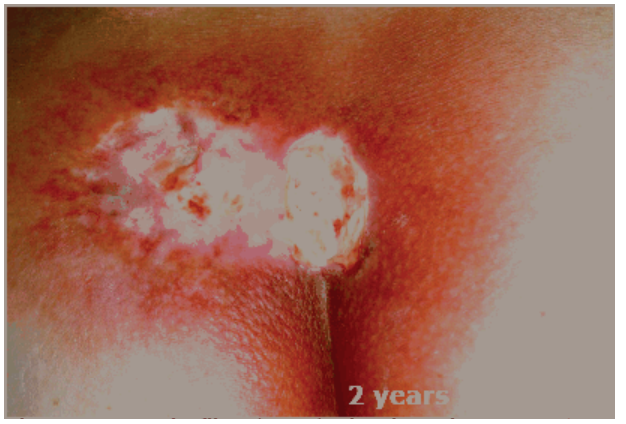

Figure 6. Necrosis, fibrosis, and telangiectasia. Same patient, 2 years following exposure.
(photos courtesy of Ricks RC and reprinted with permission)

For more information, visit www.bt.cdc.gov/radiation,
or call CDC at 800-CDC-INFO (English and Spanish) or 888-232-6348 (TTY).

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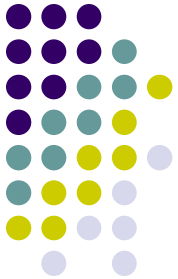
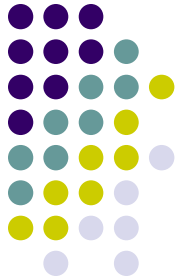
Radiation Information



- Ionizing Radiation
  - Radiation capable of imparting its energy to the body and causing chemical changes
- Contaminated Person
  - A person who has radioactive material on their skin or inside their body (e.g., inhalation, ingestion or wound contamination)
- Types of ionizing radiation
  - Alpha Particles
    - Stopped by a sheet of paper
  - Beta Particles
    - Stopped by a layer of clothing or less than an inch of a substance
  - Gamma Rays
    - Stopped by inches to feet of concrete or less than an inch of lead

Adapted from SONGS overview, presented by OES, Southern California Edison & Department of Environmental Health (July, 2009)

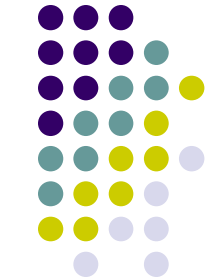
Radiation Hazards



- Types
  - External Exposure
    - Whole-body or partial-body (no radiation hazard to EMS staff)
  - Contaminated
    - External radioactive material: on the skin
    - Internal radioactive material: inhaled, swallowed, absorbed through skin or wounds
- Causes
  - Accidents
    - Nuclear reactor
    - Medical radiation therapy
    - Industrial irradiator
    - Lost/stolen medical or industrial sources
    - Transportation
  - Terrorist Events
    - Radiological dispersal device (dirty bomb)
    - Low yield nuclear weapon

Adapted from SONGS overview, presented by OES, Southern California Edison & Department of Environmental Health (July, 2009)

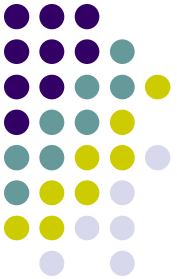
Planning for Nuclear Power Plant Emergencies



- Emergency conditions can fall into 4 categories:
  - Unusual Event
    - Least serious, minor plant problem; no public action necessary
  - Alert
    - An event could reduce the level of safety at the plant, but back-up systems are available; no public action necessary
  - Site Area Emergency
    - Potential or actual safety system problem at the plant; response centers are activated and staffed
  - General Emergency
    - Events that have occurred or are occurring which involve actual or imminent core degradation or melting with potential for loss of containment integrity; offsite agencies to initiate planned public protective actions

Information courtesy of San Diego County Nuclear Power Plant Emergency Response Plan (July, 2009)

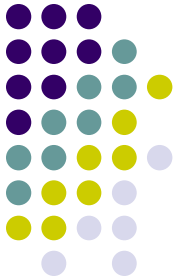
Protective Actions to Limit Exposure



- Sheltering
  - Use of building material for protection
- Evacuation
  - Protection by avoidance
- Decontamination
  - Of a person, object or area
- Use of Thyroid-Blocking Agents
  - Limit uptake of radiation by thyroid gland
- Allowing for Radioactive Decay
  - Keeping population away from contaminated areas

Information courtesy of San Diego County Nuclear Power Plant Emergency Response Plan (July, 2009)

Resources



- www.radiationanswers.org
- www.epa.gov/epahome/topics.htm
- 211 San Diego (public inquiry hotline)

Patient KI Pre-Screening Questionnaire

The purpose of this form is to determine whether a person should ingest KI and, if so, to determine the proper dosage.

The use of Potassium Iodide is voluntary. You are not required to accept it or use it.

Yes	No	
☐	☐	**Have you consumed Potassium Iodide (KI) today?**
☐	☐	**Are you allergic to shellfish, iodine, potassium tablets or dyes used in medical tests? (Example: angiograms)**
☐	☐	**Do you have a thyroid disorder? Do you have a rare skin disease named Dermatitis Herpetiformis (not a herpes virus infection) or Hypocomplementemic Vasculitis?**
☐	☐	**Do you wish to speak to our medical professional for more information?**
_____		**If the KI is for a child, what is the age of the child?**
_____		**If the KI is for an adolescent between 12 and 18 years, what is the weight of the adolescent?**

Explosions and Blast Injuries: A Primer for Clinicians

Key Concepts

- Bombs and explosions can cause unique patterns of injury seldom seen outside combat.
- The predominant post explosion injuries among survivors involve standard penetrating and blunt trauma. Blast lung is the most common fatal injury among initial survivors.
- Explosions in confined spaces (mines, buildings, or large vehicles) and/or structural collapse are associated with greater morbidity and mortality.
- Half of all initial casualties will seek medical care over a one-hour period. This can be useful to predict demand for care and resource needs.
- Expect an "upside-down" triage - the most severely injured arrive after the less injured, who bypass EMS triage and go directly to the closest hospitals.

Background

Explosions can produce unique patterns of injury seldom seen outside combat. When they do occur, they have the potential to inflict multi-system life-threatening injuries on many persons simultaneously. The injury patterns following such events are a product of the composition and amount of the materials involved, the surrounding environment, delivery method (if a bomb), the distance between the victim and the blast, and any intervening protective barriers or environmental hazards. Because explosions are relatively infrequent, blast-related injuries can present unique triage, diagnostic, and management challenges to providers of emergency care.

Few U.S. health professionals have experience with explosive-related injuries. Vietnam era physicians are retiring, other armed conflicts have been short-lived, and until this past decade, the U.S. was largely spared of the scourge of mega-terrorist attacks. This primer introduces information relevant to the care of casualties from explosives and blast injuries.

Classification of Explosives

Explosives are categorized as **high-order explosives** (HE) or **low-order explosives** (LE**)**. HE produce a defining supersonic over-pressurization shock wave. Examples of HE include TNT, C-4, Semtex, nitroglycerin, dynamite, and ammonium nitrate fuel oil (ANFO). LE create a subsonic explosion and lack HE's over-pressurization wave. Examples of LE include pipe bombs, gunpowder, and most pure petroleum-based bombs such as Molotov cocktails or aircraft improvised as guided missiles. HE and LE cause different injury patterns.

Explosive and incendiary (fire) bombs are further characterized based on their source. "Manufactured" implies standard military-issued, mass produced, and quality-tested

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weapons. “Improvised” describes weapons produced in small quantities, or use of a device outside its intended purpose, such as converting a commercial aircraft into a guided missile. Manufactured (military) explosive weapons are exclusively HE-based. Terrorists will use whatever is available – illegally obtained manufactured weapons or improvised explosive devices (also known as “IEDs”) that may be composed of HE, LE, or both. Manufactured and improvised bombs cause markedly different injuries.

Blast Injuries

The four basic mechanisms of blast injury are termed as primary, secondary, tertiary, and quaternary (Table 1). “Blast Wave” (primary) refers to the intense over-pressurization impulse created by a detonated HE. Blast injuries are characterized by anatomical and physiological changes from the direct or reflective over-pressurization force impacting the body’s surface. The HE “blast wave” (over-pressure component) should be distinguished from “blast wind” (forced super-heated air flow). The latter may be encountered with both HE and LE.

Table 1: Mechanisms of Blast Injury

Category	Characteristics	Body Part Affected	Types of Injuries
Primary	Unique to HE, results from the impact of the over pressurization wave with body surfaces.	Gas filled structures are most susceptible - lungs, GI tract, and middle ear	- Blast lung (pulmonary barotrauma) - TM rupture and middle ear damage - Abdominal hemorrhage and perforation - Globe (eye) rupture - Concussion (TBI without physical signs of head injury)
Secondary	Results from flying debris and bomb fragments	Any body part may be affected	- Penetrating ballistic (fragmentation) or blunt injuries -Eye penetration (can be occult)
Tertiary	Results from individuals being thrown by the blast wind	Any body part may be affected	- Fracture and traumatic amputation - Closed and open brain injury
Quaternary	- All explosion-related injuries, illnesses, or diseases not due to primary, secondary, or tertiary mechanisms. - Includes exacerbation or complications of existing conditions.	Any body part may be affected	- Burns (flash, partial, and full thickness) - Crush injuries - Closed and open brain injury - Asthma, COPD, or other breathing problems from dust, smoke, or toxic fumes - Angina - Hyperglycemia, hypertension

LE are classified differently because they lack the self-defining HE over-pressurization wave. LE's mechanisms of injuries are characterized as due from ballistics (fragmentation), blast wind (not blast wave), and thermal. There is some overlap between LE descriptive mechanisms and HE's Secondary, Tertiary, and Quaternary mechanisms.

Table 2: Overview of Explosive-related Injuries

System	Injury or Condition
Auditory	TM rupture, ossicular disruption, cochlear damage, foreign body
Eye, Orbit, Face	Perforated globe, foreign body, air embolism, fractures
Respiratory	Blast lung, hemothorax, pneumothorax, pulmonary contusion and hemorrhage, A-V fistulas (source of air embolism), airway epithelial damage, aspiration pneumonitis, sepsis
Digestive	Bowel perforation, hemorrhage, ruptured liver or spleen, sepsis, mesenteric ischemia from air embolism
Circulatory	Cardiac contusion, myocardial infarction from air embolism, shock, vasovagal hypotension, peripheral vascular injury, air embolism-induced injury
CNS injury	Concussion, closed and open brain injury, stroke, spinal cord injury, air embolism-induced injury
Renal Injury	Renal contusion, laceration, acute renal failure due to rhabdomyolysis, hypotension, and hypovolemia
Extremity injury	Traumatic amputation, fractures, crush injuries, compartment syndrome, burns, cuts, lacerations, acute arterial occlusion, air embolism-induced injury

Note: Up to 10% of all blast survivors have significant eye injuries. These injuries involve perforations from high-velocity projectiles, can occur with minimal initial discomfort, and present for care days, weeks, or months after the event. Symptoms include eye pain or irritation, foreign body sensation, altered vision, periorbital swelling or contusions. Findings can include decreased visual acuity, hyphema, globe perforation, subconjunctival hemorrhage, foreign body, or lid lacerations. Liberal referral for ophthalmologic screening is encouraged.

Selected Blast Injuries

Lung Injury

"Blast lung" is a direct consequence of the HE over-pressurization wave. It is the most common fatal primary blast injury among initial survivors. Signs of blast lung are usually present at the time of initial evaluation, but they have been reported as late as 48 hours after the explosion. Blast lung is characterized by the clinical triad of apnea, bradycardia, and hypotension. Pulmonary injuries vary from scattered petechae to confluent hemorrhages. Blast lung should be suspected for anyone with dyspnea, cough, hemoptysis, or chest pain following blast exposure. Blast lung produces a characteristic "butterfly" pattern on chest X-ray. A chest Xray is recommended for all exposed persons and a prophylactic chest tube (thoracostomy) is recommended before general anesthesia or air transport is indicated if blast lung is suspected.

Ear Injury

Primary blast injuries of the auditory system cause significant morbidity, but are easily overlooked. Injury is dependent on the orientation of the ear to the blast. TM perforation is the most common injury to the middle ear. Signs of ear injury are usually present at time of initial evaluation and should be suspected for anyone presenting with hearing loss, tinnitus, otalgia, vertigo, bleeding from the external canal, TM rupture, or mucopurulent otorhea. All patients exposed to blast should have an otologic assessment and audiometry.

Abdominal Injury

Gas-containing sections of the GI tract are most vulnerable to primary blast effect. This can cause immediate bowel perforation, hemorrhage (ranging from small petechiae to large hematomas), mesenteric shear injuries, solid organ lacerations, and testicular rupture. Blast abdominal injury should be suspected in anyone exposed to an explosion with abdominal pain, nausea, vomiting, hematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolemia, or any findings suggestive of an acute abdomen. Clinical findings may be absent until the onset of complications.

Brain Injury

Primary blast waves can cause concussions or mild traumatic brain injury (MTBI) without a direct blow to the head. Consider the proximity of the victim to the blast particularly when given complaints of headache, fatigue, poor concentration, lethargy, depression, anxiety, insomnia, or other constitutional symptoms. The symptoms of concussion and post traumatic stress disorder can be similar.

Emergency Management Options

- Follow your hospital's and regional disaster system's plan.
  Expect an "upside-down" triage - the most severely injured arrive after the less injured, who by-pass EMS triage and go directly to the closest hospitals.
  Double the first hour's casualties for a rough prediction of total "first wave" of casualties.
- Obtain and record details about the nature of the explosion, potential toxic exposures and environmental hazards, and casualty location from police, fire, EMS, ICS Commander, regional EMA, health department, and reliable news sources.
- If structural collapse occurs, expect increased severity and delayed arrival of casualties.

Medical Management Options

- Blast injuries are not confined to the battlefield. They should be considered for any victim exposed to an explosive force.
- Clinical signs of blast-related abdominal injuries can be initially silent until signs of acute abdomen or sepsis are advanced.
- Standard penetrating and blunt trauma to any body surface is the most common injury seen among survivors. Primary blast lung and blast abdomen are associated with a high mortality rate. “Blast Lung” is the most common fatal injury among initial survivors.
- Blast lung presents soon after exposure. It can be confirmed by finding a “butterfly” pattern on chest X-ray. Prophylactic chest tubes (thoracostomy) are recommended prior to general anesthesia and/or air transport.
- Auditory system injuries and concussions are easily overlooked. The symptoms of mild TBI and posttraumatic stress disorder can be identical.
- Isolated TM rupture is not a marker of morbidity; however, traumatic amputation of any limb is a marker for multi-system injuries.
- Air embolism is common, and can present as stroke, MI, acute abdomen, blindness, deafness, spinal cord injury, or claudication. Hyperbaric oxygen therapy may be effective in some cases.
- Compartment syndrome, rhabdomyolysis, and acute renal failure are associated with structural collapse, prolonged extrication, severe burns, and some poisonings.
- Consider the possibility of exposure to inhaled toxins and poisonings (e.g., CO, CN, MetHgb) in both industrial and criminal explosions.
- Wounds can be grossly contaminated. Consider delayed primary closure and assess tetanus status. Ensure close follow-up of wounds, head injuries, eye, ear, and stress-related complaints.
- Communications and instructions may need to be written because of tinnitus and sudden temporary or permanent deafness.

Selected Readings

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This Explosives Primer was developed from published and unpublished sources. If quoted, please cite date and time as changes will be made as new information becomes available or is cleared for public distribution.

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BLAST INJURIES

Essential Facts

Essential Facts

Key Concepts

- Bombs and explosions can cause unique patterns of injury seldom seen outside combat
- Expect half of all initial casualties to seek medical care over a one-hour period
- Most severely injured arrive after the less injured, who bypass EMS triage and go directly to the closest hospitals
- Predominant injuries involve multiple penetrating injuries and blunt trauma
- Explosions in confined spaces (buildings, large vehicles, mines) and/or structural collapse are associated with greater morbidity and mortality
- Primary blast injuries in survivors are predominantly seen in confined space explosions
- Repeatedly examine and assess patients exposed to a blast
- All bomb events have the potential for chemical and/or radiological contamination
- Triage and life saving procedures should never be delayed because of the possibility of radioactive contamination of the victim; the risk of exposure to caregivers is small
- Universal precautions effectively protect against radiological secondary contamination of first responders and first receivers
- For those with injuries resulting in nonintact skin or mucous membrane exposure, hepatitis B immunization (within 7 days) and age-appropriate tetanus toxoid vaccine (if not current)

Blast Injuries

- Primary: Injury from over-pressurization force (blast wave) impacting the body surface
  - TM rupture, pulmonary damage and air embolization, hollow viscus injury
- Secondary: Injury from projectiles (bomb fragments, flying debris)
  - Penetrating trauma, fragmentation injuries, blunt trauma
- Tertiary: Injuries from displacement of victim by the blast wind
  - Blunt/penetrating trauma, fractures, and traumatic amputations
- Quaternary: All other injuries from the blast
  - Crush injuries, burns, asphyxia, toxic exposures, exacerbations of chronic illness

Primary Blast Injury

Lung Injury

- Signs usually present at time of initial evaluation, but may be delayed up to 48 hours
- Reported to be more common in patients with skull fractures, >10% BSA burns, and penetrating injury to the head or torso
- Varies from scattered petechiae to confluent hemorrhages
- Suspect in anyone with dyspnea, cough, hemoptysis, or chest pain following blast
- CXR: “butterfly” pattern
- High flow O2 sufficient to prevent hypoxemia via NRB mask, CPAP, or ET tube

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Essential Facts

Primary Blast Injury (continued)

- Fluid management similar to pulmonary contusion; ensure tissue perfusion but avoid volume overload
- Endotracheal intubation for massive hemoptysis, impending airway compromise or respiratory failure
  - Consider selective bronchial intubation for significant air leaks or massive hemoptysis
  - Positive pressure may risk alveolar rupture or air embolism
- Prompt decompression for clinical evidence of pneumothorax or hemothorax
- Consider prophylactic chest tube before general anesthesia or air transport
- Air embolism can present as stroke, MI, acute abdomen, blindness, deafness, spinal cord injury, claudication
  - High flow O2; prone, semi-left lateral, or left lateral position
  - Consider transfer for hyperbaric O2 therapy

Abdominal Injury

- Gas-filled structures most vulnerable (esp. colon)
- Bowel perforation, hemorrhage (small petechiae to large hematomas), mesenteric shear injuries, solid organ lacerations, and testicular rupture
- Suspect in anyone with abdominal pain, nausea, vomiting, hematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolemia
- Clinical signs can be initially subtle until acute abdomen or sepsis is advanced

Ear Injury

- Tympanic membrane most common primary blast injury
- Signs of ear injury usually evident on presentation (hearing loss, tinnitus, otalgia, vertigo, bleeding from external canal, otorrhea)

Other Injury

- Traumatic amputation of any limb is a marker for multi-system injuries
- Concussions are common and easily overlooked
- Consider delayed primary closure for grossly contaminated wounds, and assess tetanus immunization status
- Compartment syndrome, rhabdomyolysis, and acute renal failure are associated with structural collapse, prolonged extrication, severe burns, and some poisonings
- Consider possibility of exposure to inhaled toxins (CO, CN, MetHgb) in both industrial and terrorist explosions
- Significant percentage of survivors will have serious eye injuries

Disposition

- No definitive guidelines for observation, admission, or discharge
- Discharge decisions will also depend upon associated injuries
- Admit 2nd and 3rd trimester pregnancies for monitoring
- Close follow-up of wounds, head injury, eye, ear, and stress-related complaints
- Patients with ear injury may have tinnitus or deafness; communications and instructions may need to be written

This fact sheet is part of a series of materials developed by the Centers for Disease Control and Prevention (CDC) on blast injuries. For more information, visit CDC on the Web at: ***www.emergency.cdc.gov/BlastInjuries****.*

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BLAST INJURIES

Blast Lung Injury

Background

Blast lung injury (BLI) presents unique triage, diagnostic, and management challenges and is a direct consequence of the blast wave from high explosive detonations upon the body. BLI is a major cause of morbidity and mortality for blast victims both at the scene and among initial survivors. The blast wave's impact upon the lung results in tearing, hemorrhage, contusion, and edema with resultant ventilation-perfusion mismatch. BLI is a clinical diagnosis and is characterized by respiratory difficulty and hypoxia, which may occur without obvious external injury to the chest.

Current patterns in worldwide terrorist activity have increased the potential for casualties related to explosions, yet few civilian health care providers in the United States have experience treating patients with explosion-related injuries. Emergency care providers are urged to learn more about the physics of explosions and other types of injuries that can result. Basic clinical information is provided here to inform practitioners of the presentation, evaluation, management, and outcomes of BLIs. Please see the reference list below for more information about how to treat injuries from explosions.

Clinical Presentation

- Symptoms may include dyspnea, hemoptysis, cough, and chest pain.
- Signs may include tachypnea, hypoxia, cyanosis, apnea, wheezing, decreased breath sounds, and hemodynamic instability.
- Associated pathology may include bronchopleural fistula, air emboli, and hemothoraces or pneumothoraces.
- Other injuries may be present.

Diagnostic Evaluation

- Chest radiography is necessary for anyone who is exposed to a blast. A characteristic "butterfly" pattern may be revealed upon x-ray.
- Arterial blood gases, computerized tomography, and doppler technology may be used.
- Most laboratory and diagnostic testing can be conducted per resuscitation protocols and further directed based upon the nature of the explosion (e.g. confined space, fire, prolonged entrapment or extrication, suspected chemical or biologic event, etc).

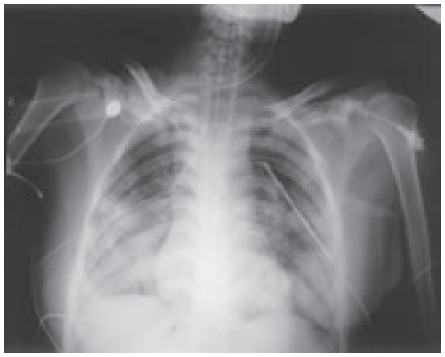

Photo courtesy of Chest, 1999

Management

- Initial triage, trauma resuscitation, treatment, and transfer should follow standard protocols; however some diagnostic or therapeutic options may be limited in a disaster or mass casualty situation.
- In general, managing BLI is similar to caring for pulmonary contusion, which requires judicious fluid use and administration ensuring tissue perfusion without volume overload.
- Clinical interventions

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Management (continued)

- All patients with suspected or confirmed BLI should receive supplemental high flow oxygen sufficient to prevent hypoxemia (delivery may include non-rebreather masks, continuous positive airway pressure, or endotracheal intubation).
- Impending airway compromise, secondary edema, injury, or massive hemoptysis requires immediate intervention to secure the airway. Patients with massive hemoptysis or significant air leaks may benefit from selective bronchus intubation.
- Clinical evidence of or suspicion for a hemothorax or pneumothorax warrants prompt decompression.
- If ventilatory failure is imminent or occurs, patients should be intubated; however, caution should be used in the decision to intubate patients, as mechanical ventilation and positive end pressure may increase the risk of alveolar rupture and air embolism.
- High flow oxygen should be administered if air embolism is suspected, and the patient should be placed in prone, semi-left lateral, or left lateral positions. Patients treated for air emboli should be transferred to a hyperbaric chamber.

Disposition and Outcome

- There are no definitive guidelines for observation, admission, or discharge following emergency department evaluation for patients with possible BLI following an explosion.
- Patients diagnosed with BLI may require complex management and should be admitted to an intensive care unit. Patients with any complaints or findings suspicious for BLI should be observed in the hospital.
- Discharge decisions will also depend upon associated injuries, and other issues related to the event, including the patient's current social situation.
- In general, patients with normal chest radiographs and ABGs, who have no complaints that would suggest BLI, can be considered for discharge after 4-6 hours of observation.
- Data on the short and long-term outcomes of patients with BLI is currently limited. However, in one study conducted on survivors one year post injury, no patients had pulmonary complaints, all had normal physical examinations and chest radiographs, and most had normal lung function tests.

Photo Source: Reprinted by permission from Chest. X-ray Figure I in "Recovery from Blast Lung Injury: One year follow-up", by Hirshberg, Boaz, MD, et al. Dec 1999, Vol 116(6), p 1683-88.

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BLAST INJURIES

Crush Injury and Crush Syndrome

Background

In a terrorist attack, crush injury and crush syndrome may result from structural collapse after a bombing or explosion. Crush injury is defined as compression of extremities or other parts of the body that causes muscle swelling and/or neurological disturbances in the affected areas of the body. Typically affected areas of the body include lower extremities (74%), upper extremities (10%), and trunk (9%). Crush syndrome is localized crush injury with systemic manifestations. These systemic effects are caused by a traumatic rhabdomyolysis (muscle breakdown) and the release of potentially toxic muscle cell components and electrolytes into the circulatory system. Crush syndrome can cause local tissue injury, organ dysfunction, and metabolic abnormalities, including acidosis, hyperkalemia, and hypocalcemia.

Previous experience with earthquakes that caused major structural damage has demonstrated that the incidence of crush syndrome is 2-15% with approximately 50% of those with crush syndrome developing acute renal failure and over 50% needing fasciotomy. Of those with renal failure, 50% need dialysis.

Clinical Presentation

Sudden release of a crushed extremity may result in reperfusion syndrome—acute hypovolemia and metabolic abnormalities. This condition may cause lethal cardiac arrhythmias. Further, the sudden release of toxins from necrotic muscle into the circulatory system leads to myoglobinuria, which causes renal failure if untreated.

Hypotension

- Massive third spacing occurs, requiring considerable fluid replacement in the first 24 hours; patients may sequester (third space) >12 L of fluid in the crushed area over a 48-hour period
- Third spacing may lead to secondary complications such as compartment syndrome, which is swelling within a closed anatomical space; compartment syndrome often requires fasciotomy
- Hypotension may also contribute to renal failure

Renal Failure

- Rhabdomyolysis releases myoglobin, potassium, phosphorous, and creatinine into the circulation
- Myoglobinuria may result in renal tubular necrosis if untreated
- Release of electrolytes from ischemic muscles causes metabolic abnormalities

Metabolic Abnormalities

- Calcium flows into muscle cells through leaky membranes, causing systemic hypocalcemia
- Potassium is released from ischemic muscle into systemic circulation, causing hyperkalemia
- Lactic acid is released from ischemic muscle into systemic circulation, causing metabolic acidosis
- Imbalance of potassium and calcium may cause life-threatening cardiac arrhythmias, including cardiac arrest; metabolic acidosis may exacerbate this situation

Secondary Complications

- Compartment syndrome may occur, which will further worsen vascular compromise

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Initial Management

Prehospital setting:

- Administer intravenous fluids before releasing the crushed body part. (This step is especially important in cases of prolonged crush [>4 hours]; however, crush syndrome can occur in crush scenarios of <1 hour)
- If this procedure is not possible, consider short-term use of a tourniquet on the affected limb until intravenous (IV) hydration can be initiated

Hospital setting:

Hypotension

- Initiate (or continue) IV hydration—up to 1.5 L/hour

Renal Failure

- Prevent renal failure with appropriate hydration, using IV fluids and mannitol to maintain diuresis of at least 300 cc/hr
- Triage to hemodialysis as needed

Metabolic Abnormalities

- Acidosis: Alkalinization of urine is critical; administer IV sodium bicarbonate until urine pH reaches 6.5 to prevent myoglobin and uric acid deposition in kidneys
- Hyperkalemia/Hypocalcemia: Consider administering the following (adult doses): calcium gluconate 10% 10cc or calcium chloride 10% 5cc IV over 2 minutes; sodium bicarbonate 1 meq/kg IV slow push; regular insulin 5-10 U and D50 1-2 ampules IV bolus; kayexalate 25-50g with sorbitol 20% 100mL PO or PR
- Cardiac Arrhythmias: Monitor for cardiac arrhythmias and cardiac arrest, and treat accordingly

Secondary Complications

- Monitor casualties for compartment syndrome; monitor compartmental pressure if equipment is available; consider emergency fasciotomy for compartment syndrome
- Treat open wounds with antibiotics, tetanus toxoid, and debridement of necrotic tissue
- Apply ice to injured areas and monitor for the 5 P's: pain, pallor, parasthesias, pain with passive movement, and pulselessness
- Observe all crush casualties, even those who look well
- Delays in hydration >12 hours may increase the incidence of renal failure; delayed manifestations of renal failure can occur

Disposition

Patients with acute renal failure may require up to 60 days of dialysis treatment; unless sepsis is present, patients are likely to regain normal kidney function.

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BLAST INJURIES

Abdominal Blast Injuries

Background

Abdominal blast injuries are a significant cause of injury and death. The actual incidence of abdominal blast injury is unknown. Incidence and clinical presentation of abdominal blast injury will vary significantly depending upon the patient and the nature of the blast. Underwater blasts carry a significantly greater risk of abdominal injury. Children are more prone to abdominal injuries in blast situations due to their unique anatomy. (For further information please refer to CDC's "Blast Injuries: Pediatrics" fact sheet.)

Clinical Presentation

Gas-containing sections of the GI tract are most vulnerable to primary blast effect. This can cause immediate bowel perforation, hemorrhage (ranging from small petechiae to large hematomas), mesenteric shear injuries, solid organ lacerations, and testicular rupture. Blast abdominal injury should be suspected in anyone exposed to an explosion with abdominal pain, nausea, vomiting, hematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolemia, or any findings suggestive of an acute abdomen. Clinical findings may be absent until the onset of complications:

- Clinical presentation of abdominal blast injury may be overt, or subtle and variable, and may include: abdominal pain, rebound tenderness, guarding, absent bowel sounds, nausea and vomiting, fever, and signs and symptoms of hypovolemia or hemorrhage. Victims of closed space bombings are at risk for more primary blast injuries, including abdominal injury.
- Predominant post-explosion abdominal injuries among survivors involve standard penetrating and blunt trauma (secondary and tertiary blast injury), but include primary blast injuries, including ischemia secondary to arterial gas embolism.
- Abdominal injuries are particularly severe in underwater blasts; the lethal radius of an underwater explosion is about three times that of a similar explosion in air because waves propagate faster and are slower to lose energy with distance due to the relative incompressibility of water.
- Children are more prone to abdominal blast injury
  - smaller and more pliable walls offer less protection
  - thin abdominal walls offer less protection
  - proportionately larger organs render children more vulnerable to injuries, especially to liver and spleen
- Most common abdominal blast injuries include:
  - Primary: abdominal hemorrhage and perforation (colon most vulnerable to perforation)
  - Secondary: penetrating and blunt abdominal trauma
  - Tertiary: blunt and penetrating abdominal trauma
  - Quaternary: crush injury to abdomen and abdominal wall

Diagnostic Evaluation

- Work-up similar to standard blunt and penetrating abdominal trauma
  - Serial abdominal examinations, as presentation may be delayed; serial exams may be difficult in young children
  - Laboratory studies
  - Radiological studies: free air, unexplained ileus, intra-abdominal hematoma/hemorrhage, solid organ contusion/laceration, intra-abdominal abscess

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Initial Management

- ABCs (airway, breathing, circulation) as for all trauma patients
- Nothing by mouth
- Avoid removal of penetrating objects in emergency room (operative intervention due to risk of hemorrhage)
- Antibiotics and tetanus immunization
- Serial exams and laboratory monitoring
- Radiological studies: plain abdominal films, computed tomography [CT] scan, Focused Abdominal Sonography for Trauma (FAST)

Disposition

- High degree of suspicion for missed or delayed abdominal injuries, including serial exams, close follow-up, and strict return instructions should signs or symptoms of abdominal injury manifest after discharge
- Appropriate referral to trauma center as needed

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BLAST INJURIES

Blast Extremity Injuries

Background

The soft tissue and musculoskeletal systems have the highest incidence of bodily injury in survivors of bombings. The most extreme of these injuries, the traumatic amputation, is reported to occur in 1%–3% of blast victims.

Clinical Presentation

Traumatic amputation from ***primary blast injury*** is often considered a marker for a lethal injury. Blast-induced amputations primarily occur through the bony shaft rather than joint disarticulations and may result from the combination of the blast wave and blast wind.

Secondary blast injury to the extremities is marked by penetrating trauma from the bomb casing fragments, materials implanted within the bomb (e.g., nails, screws), flying glass, or from local materials made airborne by proximity to the explosion.

- Wound contamination may occur from the traumatic implantation of biologic material (e.g., bone fragments) from the bomber or from victims in proximity to the explosion
- Irregular projectiles result in extensive tissue damage
- Even with small entrance wounds, surgeons should maintain a low threshold for performing a thorough debridement, as deep contamination and devitalized tissue can produce highly morbid infectious complications

Tertiary and quaternary blast injury to extremities more closely resembles civilian trauma. Victims suffer from blunt impact forces when propelled against surrounding structures.

Building collapse may produce crush injury and the potential for compartment syndrome. (For further information please refer to CDC's "Crush Injury and Crush Syndrome: What Clinicians Need to Know" fact sheet)

Diagnostic Evaluation

- Document a systematic musculoskeletal, neurological, and vascular exam for each extremity
- Extremities should be thoroughly evaluated from a vascular perspective; physical examination is less reliable for detecting vascular injuries from blast than from routine civilian trauma
- Although diligence is warranted in assessing the vascular status of the blast-injured extremity, institutional protocols incorporating mandatory arteriogram have not been published
- Each open wound should be well documented—noting size, exposed bone, and type of contamination—and, ideally, photographed
- Radiological examination of injured extremities should be liberally utilized to identify deep foreign bodies and to characterize bony injuries
- The initial absence of plantar sensation in the blast-injured extremity is not predictive for amputation; 50% of patients will regain this protective sensation over time
- Lower extremity injury scores do not accurately predict the need for amputation

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Initial Management

- Even when blast victims have small entrance wounds, surgeons should maintain a low threshold for performing thorough debridement
- All open fractures are considered contaminated and should receive early antibiotic treatment (first generation cephalosporin and/or aminoglycocide, extended spectrum penicillin)
- Obviously contaminated wounds should be irrigated with sterile saline and dressed with iodophore (Betadine)-soaked sponges; once dressed, re-exposure should wait until operative exploration
- Tetanus prophylaxis should be administered unless immunization within five years can be documented
- Extremity fractures should be splinted to provide mechanical stability and relieve pain

Surgical Management

- Initial debridement and bony stabilization should be done in the operating room to preserve life and limb; wounds should be enlarged with extensive longitudinal incisions and debrided in systematic fashion
- The zone of injury will extend well beyond initial skin wounds and fracture sites; aggressive debridement of necrotic and contaminated tissue is critical because there is a tendency to underestimate the soft tissue injury
- Following debridement, low-pressure pulsatile lavage may be employed to thoroughly irrigate the wound
- Bony stabilization is often provided by external fixation with secondary conversion to definitive plate or intramedullary fixation
- When treating vascular injuries, avoiding prosthetic grafts or repairs/reconstruction within contaminated zones of injury is important; where vessels may not be ligated, autologous vein grafts for critical reconstructions should be used
- Following debridement and bony stabilization, soft tissue injury is generally addressed with creation of an antibiotic bead pouch or application of a vacuum wound dressing
- Cultures are generally not useful during this acute injury management
- Repeat debridement is planned every 24–72 hours, depending on the injury extent, until a stable soft tissue bed is attained
- Literature on the management of small, imbedded foreign bodies is limited; it may be the case that small fragments involving soft tissue only, with small wounds and no active infection or gross contamination, may be treated expectantly
- Before and during each operative procedure, limb viability and feasibility of continued efforts to save the limb must be considered; the overall goal is to preserve potentially functional limbs without jeopardizing the patient's overall health

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BLAST INJURIES

Eye Blast Injuries

Background

Ocular injury is a frequent cause of morbidity in terrorist blast victims, occurring in up to 28% of survivors. The eye, with its protective orbit, tarsal plates, and tough sclera, is resistant to traumatic rupture resulting from a blast overpressure wave. Given enough force, however, rupture can occur. Only one case of pure ***primary blast injury*** to the eye has been reported in the literature. Lesser force can result in internal ocular disruption. ***Secondary blast injury***, caused by flying debris or fragments, is a particular threat to exposed and unprotected heads and eyes. Rapidly accelerated sharp particles, large or small, can lacerate or rupture the cornea or sclera and enter the eye.

Glass is a major source of lacerations and foreign bodies (FBs) affecting the eye. Concrete, metal, wood, and other materials from explosions in buildings can create FB eye injuries. Explosions in open spaces tend to accelerate metallic fragments from the bomb and may also propel soil and organic matter. Ocular injuries occurring from terrorist bombs may be extensive, and may involve blunt or penetrating trauma injury to the tissues of the globe, lids, orbit, or ocular adnexa. Frequently, injuries are bilateral and may range from minor corneal abrasions and foreign bodies to extensive eyelid lacerations, open globe injuries, intraocular foreign bodies (IOFB), or orbital fractures.

Clinical Presentation

- Blast eye injuries may present with a wide range of symptoms, from minimal discomfort to severe pain or loss of vision
- It is critical to appreciate that significant eye damage may be present with normal vision and minimal symptoms; these may include eye irritation or pain, foreign body sensation, decreased or altered vision, bleeding, or periorbital swelling or bruising
- Minor blast-related eye injuries include corneal abrasions, conjunctivitis, and superficial foreign bodies
- Open globe injuries, including penetrating and perforating injuries to the cornea or sclera, are the most common serious blast-related eye injuries (up to 20%–50% of those with eye injuries)
- Eyelid lacerations, often extensive, account for 20%–60% of blast-related eye injuries
- Serious non-penetrating eye injuries include hyphema, traumatic cataract, vitreous hemorrhage, retinal detatchment, choroidal rupture, and optic nerve injuries

Diagnostic Evaluation

- Assume all eye injuries harbor a rupture of the globe
- Ruptured globes or IOFBs may be very subtle—signs of a ruptured globe include 360-degree conjunctival hemorrhage; misshapen pupil; brown or pigmented tissue outside the globe; clear, gel-like tissue outside the globe; or abnormally deep or shallow anterior chamber
- Intraocular foreign bodies may be large and obvious, or small and difficult to detect; may be located in any part of the eye

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Diagnostic Evaluation (continued)

- Obtain visual acuity of each eye if possible; test for light perception (LP), hand motion (HM), and count fingers (CF)
- Thin-cut computed tomography (CT) of the orbits may be helpful in identifying foreign bodies
- Magnetic resonance imaging (MRI) is contraindicated until it is proven no metallic FBs are present; MRI may be helpful in identifying non-metallic (wood, plastic, organic) foreign bodies

Initial Management

- Do not force the lids open to examine the eye; defer examining the eye if there is massive swelling or hematoma of the lids
- Assume all eye injuries harbor a ruptured globe; do not put any pressure on an eye that may be ruptured
- Do not apply a patch or bandage to the eye—use a convex plastic or metal shield, or the bottom of a clean paper or Styrofoam cup be taped to the surrounding bones to protect the globe
- Do not remove impaled foreign bodies; the distal aspect of the foreign body may be in a location that requires special extraction techniques
- Administer tetanus if warranted
- Administer anti-emetics to reduce nausea and vomiting
- Administer intravenous (IV) broad-spectrum antibiotics if a ruptured globe is suspected; current suggestions include the combination ceftazadime/vancomycin; consider IV clindamycin for dirty soil/organic material-contaminated wounds

Disposition

- The examination of blast victims should be approached with a high level of suspicion for occult eye injuries and a low threshold for referral; consult an ophthalmologist as early as possible
- After initial stabilization of the patient and protection of the eye, rapid transport to facilities with ophthalmic operating room (OR) capabilities should be the main goal

This fact sheet is part of a series of materials developed by the Centers for Disease Control and Prevention (CDC) on blast injuries. For more information, visit CDC on the Web at: ***www.emergency.cdc.gov/BlastInjuries****.*

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BLAST INJURIES

Thermal Injuries

Background

Thermal injuries from explosions of conventional weapons are classified as quaternary blast injuries. The rapidly expanding fireball from the explosion may cause flash burns over exposed body parts (e.g., hands, neck, and head). Confined space explosions can enhance thermal effects and increase the risk of inhalation injury. Effectively managing thermal injuries associated with primary blast injury, particularly blast lung injury, may be challenging due to conflicting fluid requirements.

Clinical Presentation

- Most bomb-related burns cover <20% of the total body surface area (TBSA), but occur in combination with other blast injuries.
- Inhalation injury is relatively common (18%) among those who survive explosions in confined spaces.

Initial Prehospital Management

- Stop the burning process; remove restrictive and smoldering clothing.
- Rapidly cooling the isolated burn (cool water irrigation, no ice) will reduce the zone of stasis associated with initial thermal injury; avoid hypothermia and freezing tissue.
- Apply simple dressings to limit secondary wound contamination.

Initial Hospital Management

Immediate Steps

- Remove restrictive and smoldering clothing to stop burning, allow for a thorough examination, and prevent secondary fires in the presence of high flow oxygen.
- Irrigate thermal injuries with cool water to help reduce the area contained in the zone of stasis. Injuries in this area are potentially reversible. Do not use ice and be cognizant of the potential for hypothermia.

Airway/Inhalation Injuries

- Inhalation injury can result from the explosion's extinction of available oxygen and creation of particulate matter, smoke, superheated gases, and toxic by-products.
- Suspect an inhalation injury with a:
  - Closed space explosion;
  - Singed nasal vibrissae or carbonaceous sputum; or
  - Elevated CO or CN levels (obtain only if victim numbers are low— indiscriminant ordering will overwhelm the laboratory resources).
- If airway injury exists, intubate early. Inhalation injury can be fatal if a patient's airway is blocked due to mucosal swelling and progressive edema that obliterates normal airway structures.

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Initial Hospital Management (continued)

- At admission, consider evaluating the fiber optic airway to determine if subsequent airway intervention or aggressive pulmonary toilet is needed.
- Among patients with primary blast injury to the lung, mechanical ventilation and positive pressure may increase the risk of alveolar rupture and air embolism. Patients with inhalational injury may be at a higher risk of barotrauma.

Fluid Resuscitation

- Fluid resuscitation is required for victims with burns that cover >15% of TBSA.
- The goal is to replace the loss of intravascular volume and to maintain tissue perfusion in the first 48 hours post-injury, when capillary leak and relative hypovolemia occur.
- Inadequate fluid resuscitation increases morbidity and mortality**.**
- Fluid resuscitation for significant thermal injury that is initiated more than four hours post-injury is associated with almost 100% mortality.
- Give Lactated Ringers (LR):
  - 4cc/kg/%TBSA in the first 24 hours
  - Give half in the first eight hours starting from the time of the burn insult itself, and the remaining half during the next 16 hours.
- Effective fluid resuscitation is demonstrated by adequate urine output.
- Take care when treating burn victims who have also suffered a blast lung injury. The risk of aggressive hydration to the blast injured lung must be balanced with the need to provide IV fluids to manage the burn.

Pain Management

- Give narcotics for pain.
- Recognize when resources may be limited (e.g., a Rhode Island nightclub fire exhausted a three-month narcotic supply during the acute resuscitation phase at a Level I trauma and burn center).

Other Considerations

- Administer tetanus toxoid if patient did not receive a booster in the last five years, or if date of booster is unknown.
- Full-thickness burns of thorax and extremities may cause the constriction of underlying structures and require an escharotomy.

Disposition

- Inhalation injury is an independent predictor of prolonged ICU care and mortality.
- Burns covering >30% of TBSA are associated with increased death rates.
- Death from burns is dependent on: the percentage of TBSA affected, presence or absence of significant airway and lung involvement, and the age of the victim.
- Patients diagnosed with primary blast lung injury should be admitted to the hospital, regardless of the extent of any associated burn.

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BLAST INJURIES

Bombings and Mental Health

Background

Intentional mass casualty events such as bombings are designed to cause death, destruction, fear, and confusion. In comparison to natural disasters, intentional mass casualty events are associated with higher rates of long term psychological symptoms. The level of fear and distress after a bombing depends on several factors, including injury of self and/or injury or death of family members and friends; separation from or lack of knowledge about loved ones; and the witnessing of horrific and frightening scenes.

Emergency responders and other health care providers may also experience psychological symptoms resulting from continued exposure to death and devastation.

Most fear and distress reactions are normal, expected, and can be managed using principles of good psychological patient care. Clinicians should take all reports of physical, emotional, cognitive, and behavioral reactions seriously.

Clinical Presentation

- Physical reactions: fatigue/exhaustion, gastrointestinal distress, tightening in throat/chest/stomach, headache, worsening chronic conditions, somatic complaints, or racing heartbeat.
- Emotional reactions: depression/sadness, irritability/anger/resentment, anxiety/fear, despair/hopelessness, guilt/self-doubt, unpredictable mood swings, emotional numbness, or inappropriately flat affect.
- Cognitive reactions: confusion/disorganization, recurring dreams or nightmares, preoccupation with the disaster, trouble concentrating/remembering things, difficulty making decisions, questioning spiritual beliefs, disorientation, indecisiveness, worry, shortened attention span, memory loss, unwanted memories, or self-blame.
- Behavioral reactions: sleep problems, crying easily, excessive activity level, increased conflicts with others, hypervigilance/startle reactions, isolation/social withdrawal, distrust, irritability, feeling rejected or abandoned, being distant, judgmental, or over-controlling. Abuse of substances and/or alcohol is also a common symptom.

Initial Management

Provide psychological first aid (PFA) to patients, family members, and emergency response personnel as needed:

- Establish contact and engagement
- Provide/ensure safety and security
- Stabilize, as necessary
- Gather information regarding current needs and concerns
- Avoid encouraging patient to talk about the event as this may intensify symptoms
- Provide practical assistance
- Provide information and education regarding signs of distress and how to cope
- Link with appropriate/needed follow-up services
- Provide family members with accurate, timely, and credible information about patient status and what will be happening next

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Initial Management (continued)

- Provide family members a quiet location away from distressing signs and sounds
- Minimize separation of pediatric and other patients where separation increases distress
- Optimize services of hospital social services and chaplains

Refer to a behavioral health specialist when the following signs occur:

- Disorientation: inability to know date, location, or recent events
- High anxiety and hyper-arousal: highly agitated, unable to sleep, frequent nightmares, flashbacks, or intrusive thoughts
- Dissociation: emotional disconnection, sense of seeing self from another perspective, seeing the environment as unreal, or time distortion
- Severe depression: hopelessness and despair, unrelenting feelings of worthlessness or guilt, frequent crying for no apparent reason, or withdrawal
- Psychosis: hearing voices, seeing things that are not there, appearing out of touch with reality, or excessive preoccupation with ideas or thoughts
- Inability to care for one self: does not eat or bathe, isolated from others, or unable to manage tasks of daily living
- Suicidal or homicidal thoughts or plans
- Problematic use of alcohol or drugs
- Domestic violence: child, spouse, elder, or animal abuse

Address emergency response personnel concerns as needed:

- Be aware of personal stress vulnerabilities in emergency responders
- Identify physical, emotional, cognitive, and behavioral signs in self and coworkers, and practice self-care
- Enforce breaks
- Use a buddy system to identify stress
- Provide PFA as needed
- Seek help from a mental health specialist if necessary
- Be aware of stress and fears in your family resulting from your work/role

Disposition

- Most fear and distress reactions are normal and will resolve without the intervention of a mental health specialist; however, referral services should be made available to all patients, families, and emergency response personnel
- Individuals who belong to strong social networks, such as families and faith communities, tend to do better than those who do not
- Individuals and families that exhibit continuing signs of distress, and those exhibiting signs of mental illness, including psychosis, severe anxiety, and depression, should be referred to a mental health specialist for ongoing care

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COUNTY OF SAN DIEGO
HEALTH AND HUMAN SERVICES AGENCY
PUBLIC HEALTH SERVICES
EMERGENCY MEDICAL SERVICES

Zebra Packet Information Sources

Disease Reporting:
County of San Diego, Health and Human Services Agency, Epidemiology and Immunization Services Branch

Confidential Morbidity Report:
California Health and Human Services Agency, Department of Public Health

Detecting Bioterrorism: The Clinicians Role:
County of San Diego, Health and Human Services Agency, Epidemiology and Immunization Services Branch

Reporting Suspected Bioterrorism Related Illness:
County of San Diego, Emergency Medical Services

California Health Alert Network (CAHAN): CAHAN

California Disaster Healthcare Volunteers (DHV):
DHV & County of San Diego, Emergency Medical Services

Ten Critical Steps for Handling Possible Bioterrorism Events:
California Department of Public Health

Isolation Guidelines:
LTC Suzanne E. Johnson, RN, MSN, CIC, Walter Reed Army Medical Center; Revised by Center for the Study of Bioterrorism and Emerging Infections

Bioterrorism Overview: Centers for Disease Control

Category A List: Centers for Disease Control

Anthrax Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Hughes, J. M. (2002). Anthrax Bioterrorism: Lessons Learned and Future Directions. *Emerging Infectious Diseases* , 1013-1018.

Smallpox Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Evaluating Patients for Smallpox Worksheet: Centers for Disease Control

Breman, J. G. (2002). Diagnosis and Management of Smallpox. *The New England Journal of Medicine* , 1300-1308.

Plague Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Botulism Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Tularemia Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Viral Hemorrhagic Fever Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

(2006). Chemical Terrorism Information and Treatment Guidelines for Hospitals and Clinicians. In *Terrorism Agent Information & Treatment Guidelines for Clinicians & Hospitals.* Los Angeles County Public Health, Emergency Medical Services Agency.

Biotoxin Fact Sheets: Centers for Disease Control

Vesicant Agent Fact Sheets: Centers for Disease Control

Blood Agent Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Pulmonary Agent Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Incapacitating Agent Fact Sheets:
Centers for Disease Control
County of San Diego, Public Health Services

Nerve Agent Fact Sheets:
Centers for Disease Control
Agency for Toxic Substances and Disease Registry
County of San Diego, Public Health Services

Riot Control Agent Fact Sheets: Centers for Disease Control

(2006). Nuclear/Radiological Terrorism Information and Treatment Guidelines for Hospitals and Clinicians. In *Terrorism Agent Information & Treatment Guidelines for Clinicians & Hospitals.* Los Angeles County Public Health, Emergency Medical Services Agency.

Radiation Information:
California Office of Emergency Services, S. C. (2009). *SONGS Overview.*

Mass Casualty Event Fact Sheets: Centers for Disease Control