NEW JERSEY HAZMAT EMERGENCY RESPONSE COURSE

STUDENT GUIDE **COURSE NUMBER: 06061**

Emegency Department Operations Hazmat/WMD Hospital Provider

PRESENTED THROUGH:
NEW JERSEY STATE POLICE-HOMELAND SECURITY BRANCH
SPECIAL OPERATIONS SECTION, TECHNCIAL RESPONSE BUREAU
HAZARDOUS MATERIALS RESPONSE UNIT (HMRU)

STUDENT GUIDE **5th Edition** 1004

TABLE OF CONTENTS

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Mr. Eugene J. O'Neill, NREMT-B, University of Medicine & Dentistry of NJ–University Hospital

INSTRUCTIONS FOR FILLING OUT THE REGISTRATION FORM

- Begin on the side that has the words **REGISTRATION FORM** at the top.
- Use #2 pencil only.
- Your name will appear on your certificate exactly as you list it here.

FIRST NAME

1. In the open boxes under the area labeled FIRST NAME, start at the left and spell out your first name by printing letters in the boxes (1 letter per box).

—If you want to use a title in front of your name, start at the left and leave a blank box between the title and the first name. Due to limited space, abbreviations should be used as necessary.

e.g. Lieutenant Richard Tumid can be written as: Lt. Richard Tumid or Lieut. R. Tumid.

—If you don't use all the boxes leave the unfilled ones blank.

e.g. #1 There are eleven boxes available for your first name. If your name is Frank, fill in the first five boxes and leave the last six boxes blank.

e.g. #2 Mary Ellen spells her first name with a space between Mary and Ellen. She should leave a blank box between Mary and Ellen on the form. This would also leave the last space at the end blank.

M.I.

2. If you use a middle initial write it in the open box under M.I.

LAST NAME

3. In the open boxes under the area labeled LAST NAME, start at the left (the box immediately after the M.I. box) and spell out your last name by printing letters in the boxes leaving the extra boxes blank.

—After you have finished printing out your first name, middle initial and last name, go back and darken in the box under each letter that corresponds to that letter.

EXTENSION

4. Find the box labeled EXTENSION and if applicable darken the appropriate square.

HOME STREET ADDRESS

5. In the open boxes under the area labeled HOME STREET ADDRESS, start at the left and fill in your home STREET address only. **DO NOT INCLUDE THE NAME OF THE TOWN NOR YOUR ZIP CODE.** Leave spaces where you would normally leave them when writing it out.

e.g. 123 W. Main St. would use the first three blocks for the numbers 1, 2 & 3 followed by a space, then the letter W followed by a space, then the next four blocks for the letters in Main followed by a space and finally the next two blocks for St. or the next six for Street.

—Please leave the rest blank. Your CITY and STATE will be determined from your ZIP code.

APT. NO.

6. If applicable start at the left and fill in the open boxes under the area labeled APT. NO. If there is less than four letters and/or numbers leave spaces blank.

—After you have finished printing your home street address and if applicable Apt. No., go back and darken in the box under each letter or number that corresponds to that letter or number.

ZIP CODE

7. Find the area labeled ZIP CODE and fill in the open boxes with your **nine digit** ZIP code, if known. If you do not know your 9 digit zip code, then just enter the 5 digit number.

—When completed go back and darken in the box under each number that corresponds to that number.

DATE OF BIRTH

8. In the open boxes under the area labeled DATE OF BIRTH, fill in the month, day & year using two boxes for each. ALL the boxes must be filled out.

e.g. May 27, 1945 should be written as 05 27 45.

—When completed go back and darken in the box under each number that corresponds to that number.

SOCIAL SECURITY NO.

9. In the open boxes under the area labeled SOCIAL SECURITY NO. fill in your social security number.

—When completed go back and darken in the box under each number that corresponds to that number.

FOR BOXES 10, 11, 12 & 13 FILL IN ONLY ONE BUBBLE FOR EACH BOX

SEX

10. In the area labeled SEX, darken in the appropriate box.

ETHNIC GROUP

11. In the area labeled ETHNIC GROUP, darken in the appropriate box.

PRIMARY LANGUAGE

12. In the area labeled PRIMARY LANGUAGE, darken in the appropriate box.

EDUCATION LEVEL

13. In the area labeled EDUCATION LEVEL, darken in the appropriate box. All darken in the highest level of education in which you have completed.

HOME PHONE NUMBER

14. Turn over the form and begin with HOME PHONE NUMBER in the upper left hand corner.

—Fill in the open boxes with your area code and phone number, then go back and darken in the box under each number that corresponds to that number.

WORK PHONE NUMBER

15. Complete the WORK PHONE NUMBER the same as you did the HOME PHONE NUMBER.

COURSE START DATE

16. In the open boxes under the area labeled COURSE START DAY, list the month, day and year of the first day of this course. Every box must have a number entered into it.

—When completed go back and darken in the box under each number that corresponds to that number.

INSTRUCTORS NO.

17. All state certified instructors have an individual four digit number assigned to them. At each course, one instructor will be designated as the lead instructor. Place the lead instructor number in column #1.

—In the open boxes under the area labeled INSTRUCTORS NUMBERS, start on the left and list the number of the lead instructor first, then list the numbers of any additional instructions for this day's training.

—There is only enough room for the listing of three instructors.

—When completed go back and darken in the box under each number that corresponds to that number.

COURSE #

18. In the open boxes under the area labeled COURSE #, start on the left and write the course number that the lead instructor gives you.

—**NOTE:** All Course #s must be five digits. If you have a four digit # add a **0** to the **beginning** of the number.

e.g. Four digit #6007 should be written as five digit #06007.

—When completed go back and darken in the box under each number that corresponds to that number.

JURISDICTION SERVED M-CODES

19. In the open boxes under the area labeled JURISDICTION SERVED M-CODES, start on the left and write the code number that is assigned to the jurisdiction that you serve in your primary emergency service position. The lead instructor will have a list of the M-CODES for all municipalities in the state.

EMERG. SERVICE POSITION

20. The emergency service position that you receive pay from is your primary emergency service position. If you do not receive pay and have two or more volunteer positions then you must choose which one is your emergency service position. You can only choose ONE primary service position.

—In the area labeled EMERGENCY SERVICE POSITIONS, darken **one** "P" box next to your **primary** emergency service position. All other emergency service positions that you hold are secondary and you should darken the "S" box next to any that apply.

ANSWER FOR PRIMARY POSITION

21. The area labeled ANSWER FOR PRIMARY POSITION refers to your primary emergency service position and is divided into two sections.

—Darken in the appropriate box in the status section and the sector section.

QUESTIONS

22. Please answer the three questions as they apply to your emergency service positions.

NOTE: A baseline physical establishes a medical base that can be compared to future physical results to determine changes that may be caused by chemical exposure.

PRE-TEST

23. If your course uses a PRE-TEST follow the instructions of the lead instructor.

PLEASE INSURE THAT ALL AREAS ARE FILLED IN. ASK FOR HELP FROM YOUR INSTRUCTOR IF YOU ARE UNSURE OF HOW TO COMPLETE ANY AREA ON THE FORM.

REMEMBER, IF THIS FORM IS FILLED OUT INCORRECTLY, IT COULD RESULT IN YOU NOT RECEIVING A CERTIFICATE.

PLEASE USE #2 PENCIL ONLY.

REGISTRATION FORM

Fill in name as you wish it to appear on your certificate.

FIRST NAME | MI | LAST NAME

HOME STREET ADDRESS (Do not include city and state) | APT. NO.

EXTENSION

JR. ☐
SR. ☐
III ☐

ZIP CODE

DATE OF BIRTH

SOCIAL SECURITY NO.

FOR EACH BOX CHOOSE ONE ONLY.

ETHNIC GROUP

- BLACK ☐
- HISPANIC ☐
- ASIAN ☐
- CAUCASIAN ☐
- OTHER ☐

PRIMARY LANGUAGE

- ☐ ENGLISH
- ☐ SPANISH
- ☐ OTHER (specify)

_____

EDUCATION LEVEL (Highest level only)

- SOME HIGH SCHOOL ☐
- HIGH SCHOOL GRADUATE ☐
- SOME COLLEGE / TECH. TRAINING ☐
- COLLEGE TECH. SCHOOL DEGREE ☐
- SOME GRADUATE STUDIES ☐
- GRADUATE DEGREE ☐

SEX

- MALE ☐
- FEMALE ☐

HOME PHONE NUMBER

COURSE START DATE

INSTRUCTORS NUMBERS

1 2 3

COURSE #

WORK PHONE NUMBER

Fill in ONLY ONE primary (P) and as many secondary positions (S) that apply

EMERGENCY SERVICE POSITIONS

1. Fire fighter	P S
2. Police	P S
3. Emergency medical service/BLS	P S
4. Emergency medical service/ALS	P S
5. Emergency room staff	P S
6. Health officer	P S
7. Hazmat team	P S
8. Indust. fire brigade	P S
9. Public works	P S
10. Utility Authority	P S
11. Emergency mgmt.	P S
12. NJDEPE responder	P S
13. Govt. official	P S
14. Educator/trainer	P S
15. Other public sector	P S
16. Other private sector	P S

ANSWER FOR PRIMARY POSITION

Volunteer	
Paid Part-Time	
Paid Full-Time	
Public Sector	
Private Sector	

Jurisdiction Served

M-CODES

PLEASE ANSWER THE FOLLOWING QUESTIONS:

1. - Have you ever been ill or injured due to hazardous substances? Y N
2. - Are you enrolled in a medical surveillance program? Y N
   (If yes)
   - Have you had a baseline exam? Y N
   - Were the results explained to you? Y N
   - Are you examined annually? Y N
3. - Has any employer provided you with other health and safety training within the last two years? Y N
   (If yes)
   - Did it cover your rights under Federal OSHA laws? Y N
   - Did it cover your rights under State laws? Y N

PRE-TEST ANSWERS

TEST VERSION

1 A B C D E	11 A B C D E	21 A B C D E	31 A B C D E	41 A B C D E
2 A B C D E	12 A B C D E	22 A B C D E	32 A B C D E	42 A B C D E
3 A B C D E	13 A B C D E	23 A B C D E	33 A B C D E	43 A B C D E
4 A B C D E	14 A B C D E	24 A B C D E	34 A B C D E	44 A B C D E
5 A B C D E	15 A B C D E	25 A B C D E	35 A B C D E	45 A B C D E
6 A B C D E	16 A B C D E	26 A B C D E	36 A B C D E	46 A B C D E
7 A B C D E	17 A B C D E	27 A B C D E	37 A B C D E	47 A B C D E
8 A B C D E	18 A B C D E	28 A B C D E	38 A B C D E	48 A B C D E
9 A B C D E	19 A B C D E	29 A B C D E	39 A B C D E	49 A B C D E
10 A B C D E	20 A B C D E	30 A B C D E	40 A B C D E	50 A B C D E

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GEOGRAPHIC IDENTIFICATION CODE SCHEME

Incorporated Areas of New Jersey

Arranged Alphabetically by County and Municipality

All codes listed in this Manual will be four (4) digit codes

The first two (2) digits being the County Code, the second (2) being the Municipality Code

EXAMPLES:

Counties:

01—Atlantic County
02—Bergen County
03—Burlington County

Municipalities:

01—Absecon City
02—Atlantic City City
03—Brigantine City

Complete Code

0101—Atlantic County, Absecon City
0201—Bergen County, Allendale Borough
0301—Burlington County, Bass River Township

ATLANTIC COUNTY—01

0101 Absecon City
0102 Atlantic City
0103 Brigantine City
0104 Buena Borough
0105 Buena Vista Twsp.
0106 Corbin City
0107 Egg Harbor City
0108 Egg Harbor Twsp.
0109 Estell Manor City
0110 Folsom Borough
0111 Galloway Twsp.
0112 Hamilton Twsp.
0113 Hammonton Town
0114 Linwood City
0115 Longport Borough
0116 Margate City
0117 Mullica Twsp.
0118 Morthfield City
0119 Pleasantville City
0120 Port Republic City
0121 Somers Point City
0122 Ventnor City
0123 Weymouth Twsp.

BERGEN COUNTY—02

0201 Allendale Borough
0202 Alpine Borough
0203 Bergenfield Borough
0204 Bogota Borough
0205 Carlstadt Borough
0206 Cliffside Park Borough
0207 Closter Borough
0208 Cresskill Borough
0209 Demarest Borough
0210 Dumont Borough
0211 Elmwood Park Borough
0212 East Rutherford Borough
0213 Edgewater Borough
0214 Emerson Borough
0215 Englewood City
0216 Englewood Cliffs Borough
0217 Fair Lawn Borough
0218 Fairview Borough
0219 Fort Lee Borough
0220 Franklin Lakes Borough
0221 Garfield City
0222 Glen Rock Borough
0223 Hackensack City
0224 Harrington Park Borough
0225 Hasbrouck Heights Borough
0226 Haworth Borough
0227 Hillsdale Borough
0228 Hohokus Borough
0229 Leonia Borough
0230 Little Ferry Borough
0231 Lodi Borough
0232 Lyndhurst Twsp.
0233 Mahwah Twsp.
0234 Maywood Borough
0235 Midland Park Borough
0236 Montvale Borough
0237 Moonachie Borough
0238 New Milford Borough
0239 North Arlington Borough
0240 Northvale Borough
0241 Norwood Borough
0242 Oakland Borough
0243 Old Tappan Borough
0244 Oradell Borough
0245 Palisades Park Borough
0246 Paramus Borough
0247 Park Ridge Borough
0248 Ramsey Borough
0249 Ridgefield Borough
0250 Ridgefield Park Village
0251 Ridgewood Village
0252 River Edge Borough
0253 River Vale Twsp.
0254 Rochelle Park Twsp.
0255 Rockleigh Borough
0256 Rutherford Borough
0257 Saddle Brook Twsp.
0258 Saddle River Borough
0259 South Hackensack Twsp.
0260 Teaneck Twsp.
0261 Tenafly Borough
0262 Teterboro Borough
0263 Upper Saddle River Borough
0264 Waldwick Borough
0265 Wallington Borough
0266 Washington Twsp.
0267 Westwood Borough
0268 Woodcliff Lake Borough
0269 Wood-Ridge Borough
0270 Wyckoff Twsp.

BURLINGTON COUNTY—03

0301 Bass River Twsp.
0302 Beverly City
0303 Bordentown City
0304 Bordentown Twsp.

0305 Burlington City
0306 Burlington Twsp.
0307 Chesterfield Twsp.
0308 Cinnaminson Twsp.
0309 Delanco Twsp.
0310 Delran Twsp.
0311 Eastampton Twsp.
0312 Edgewater Park Twsp.
0313 Evesham Twsp.
0314 Fieldsboro Borough
0315 Florence Twsp.
0316 Hainesport Twsp.
0318 Lumberton Twsp.
0319 Mansfield Twsp.
0320 Maple Shade Twsp.
0321 Medford Twsp.
0322 Medford Lakes Borough
0323 Moorestown Twsp.
0324 Mount Holly Twsp.
0325 Mount Laurel Twsp.
0326 New Hanover Twsp.
0327 North Hanover Twsp.
0328 Palmyra Borough
0329 Pemberton Borough
0330 Pemberton Twsp.
0331 Riverside Twsp.
0332 Riverton Borough
0333 Shamong Twsp.
0334 Southampton Twsp.
0335 Springfield Twsp.
0336 Tabernacle Twsp.
0337 Washington Twsp.
0338 Westampton Twsp.
0317 Willingboro Twsp.
0339 Woodland Twsp.
0340 Wrightstown Borough

CAMDEN COUNTY—04

0401 Audubon Borough
0402 Audubon Park Borough
0403 Barrington Borough
0404 Bellmawr Borough
0405 Berlin Borough
0406 Berlin Twsp.
0407 Brooklawn Borough
0408 Camden City
0412 Cherry Hill Twsp.
0409 Chesilhurst Borough
0410 Clementon Borough
0411 Collingswood Borough
0413 Gibbsboro Borough
0414 Gloucester City
0415 Gloucester Twsp.
0416 Haddon Twsp.
0417 Haddonfield Borough
0418 Haddon Heights Borough
0419 Hi-Nella Borough
0420 Laurel Springs Borough
0421 Lawnside Borough
0422 Lindenwold Borough
0423 Magnolia Borough
0424 Merchantville Borough
0425 Mount Ephraim Borough
0426 Oaklyn Borough
0427 Pennsauken Twsp.
0428 Pine Hill Borough
0429 Pine Valley Borough
0430 Runnemede Borough
0431 Somerdale Borough
0432 Stratford Borough
0433 Tavistock Borough
0434 Voorhees Twsp.
0435 Waterford Twsp.
0436 Winslow Twsp.
0437 Wood-Lynne Borough

CAPE MAY COUNTY—05

0501 Avalon Borough
0502 Cape May City
0503 Cape May Point Borough
0504 Dennis Twsp.
0505 Lower Twsp.
0506 Middle Twsp.
0507 North Wildwood City
0508 Ocean City
0509 Sea Isle City
0510 Stone Harbor Borough
0511 Upper Twsp.
0512 West Cape May Borough
0513 West Wildwood Borough
0514 Wildwood City
0515 Wildwood Crest Borough
0516 Woodbine Borough

CUMBERLAND COUNTY—06

0601 Bridgeton City
0602 Commercial Twsp.
0603 Deerfield Twsp.
0604 Downe Twsp.
0605 Fairfield Twsp.
0606 Greenwich Twsp.
0607 Hopewell Twsp.
0608 Lawrence Twsp.
0609 Maurice River Twsp.
0610 Millville City
0611 Shiloh Borough
0612 Stow Creek Twsp.
0613 Upper Deerfield Twsp.
0614 Vineland City

ESSEX COUNTY—07

0701 Belleville Town
0702 Bloomfield Town
0703 Caldwell Borough
0705 Cedar Grove Twsp.
0706 East Orange City
0707 Essex Fells Borough
0704 Fairfield Borough
0708 Glen Ridge Borough
0709 Irvington Town
0710 Livingston Twsp.
0711 Maplewood Twsp.
0712 Millburn Twsp.
0713 Montclair Town
0714 Newark City
0715 North Caldwell Borough
0716 Nutley Town
0717 Orange City
0718 Roseland Borough
0719 South Orange Village
0720 Verona Borough
0721 West Caldwell Borough
0722 West Orange Town

GLOUCESTER COUNTY—08

0801 Clayton Borough
0802 Deptford Twsp.
0803 East Greenwich Twsp.
0804 Elk Twsp.
0805 Franklin Twsp.
0806 Glassboro Borough
0807 Greenwich Twsp.
0808 Harrison Twsp.
0809 Logan Twsp.
0810 Mantua Twsp.
0811 Monroe Twsp.
0812 National Park Borough
0813 Newfield Borough
0814 Paulsboro Borough
0815 Pitman Borough
0816 South Harrison Twsp.
0817 Swedesboro Borough
0818 Washington Twsp.
0819 Wenonah Borough
0820 West Deptford Twsp.
0821 Westville Borough
0822 Woodbury City
0823 Woodbury Heights Borough
0824 Woolwich Twsp.

HUDSON COUNTY—09

0901 Bayonne City
0902 East Newark Borough
0903 Guttenberg Town
0904 Harrison Town
0905 Hoboken City
0906 Jersey City City
0907 Kearny Town
0908 North Bergen Twsp.
0909 Secaucus Town
0910 Union City
0911 Weehawken Twsp.
0912 West New York Town

HUNTERDON COUNTY—10

1001 Alexandria Twsp.
1002 Bethlehem Twsp.
1003 Bloomsbury Borough
1004 Califon Borough
1005 Clinton Town
1006 Clinton Twsp.
1007 Delaware Twsp.
1008 East Amwell Twsp.
1009 Flemington Borough
1010 Franklin Twsp.
1011 Frenchtown Borough
1012 Glen Gardner Borough
1013 Hampton Borough
1014 High Bridge Borough
1015 Holland Twsp.
1016 Kingwood Twsp.
1017 Lambertville City
1018 Lebanon Borough
1019 Lebanon Twsp.
1020 Milford Borough
1021 Raritan Twsp.
1022 Readington Twsp.
1023 Stockton Borough
1024 Tewksbury Twsp.
1025 Union Twsp.
1026 West Amwell Twsp.

MERCER COUNTY—11

1101 East Windsor Twsp.
1102 Ewing Twsp.
1103 Hamilton Twsp.
1104 Hightstown Borough
1105 Hopewell Borough
1106 Hopewell Twsp.

1107 Lawrence Twsp.
1108 Pennington Borough
1109 Princeton Borough
1110 Princeton Twsp.
1111 Trenton City
1112 Washington Twsp.
1113 West Windsor Twsp.

MIDDLESEX COUNTY—12

1201 Carteret Borough
1202 Cranbury Twsp.
1203 Dunellen Borough
1204 East Brunswick Twsp.
1205 Edison Twsp.
1206 Helmetta Borough
1207 Highland Park Borough
1208 Jamesburg Borough
1210 Metuchen Borough
1211 Middlesex Borough
1212 Milltown Borough
1213 Monroe Twsp.
1214 New Brunswick City
1215 North Brunswick Twsp.
1209 Old Bridge Twsp.
1216 Perth Amboy City
1217 Piscataway Twsp.
1218 Plainsboro Twsp.
1219 Sayreville Borough
1220 South Amboy City
1221 South Brunswick Twsp.
1222 South Plainfield Borough
1223 South River Borough
1224 Spotswood Borough
1225 Woodbridge Twsp.

MONMOUTH COUNTY—13

1330 Aberdeen Twsp.
1301 Allenhurst Borough
1302 Allentown Borough
1303 Asbury Park City
1305 Atlantic Highlands Borough
1306 Avon-By-The-Sea Borough
1307 Belmar Borough
1308 Bradley Beach Borough
1309 Brielle Borough
1304 Colts Neck Twsp.
1310 Deal Borough
1311 Eatontown Borough
1312 Englishtown Borough
1313 Fair Haven Borough
1314 Farmingdale Borough
1315 Freehold Borough
1316 Freehold Twsp.
1339 Hazlet Twsp.
1317 Highlands Borough
1318 Holmdel Twsp.
1319 Howell Twsp.
1320 Interlaken Borough
1321 Keansburg Borough
1322 Keyport Borough
1323 Little Silver Borough
1324 Loch Arbour Village
1325 Long Branch City
1326 Manalapan Twsp.
1327 Manasquan Borough
1328 Marlboro Twsp.
1329 Matawan Borough
1331 Middletown Twsp.
1332 Millstone Twsp.
1303 Monmouth Beach Borough
1334 Neptune Twsp.
1335 Neptune City Borough
1337 Ocean Twsp.
1338 Oceanport Borough
1340 Red Bank Borough
1341 Roosevelt Borough
1342 Rumson Borough
1343 Sea Bright Borough
1344 Sea Girt Borough
1345 Shewsbury Borough
1346 Shewsbury Twsp.
1347 South Belmar Borough
1348 Spring Lake Borough
1349 Spring Lake Heights Borough
1336 Tinton Falls Borough
1350 Union Beach Borough
1351 Upper Freehold Twsp.
1352 Wall Twsp.
1353 West Long Branch Borough

MORRIS COUNTY—14

1401 Boonton Town
1402 Boonton Twsp.
1403 Butler Borough
1404 Chatham Borough
1405 Chatham Twsp.
1406 Chester Borough
1407 Chester Twsp.
1408 Denville Twsp.
1409 Dover Town
1410 East Hanover Twsp.
1411 Florham Park Borough
1412 Hanover Twsp.
1413 Harding Twsp.
1414 Jefferson Twsp.
1415 Kinnelon Borough
1416 Lincoln Park Borough
1417 Madison Borough
1418 Mendham Borough
1419 Mendham Twsp.
1420 Mine Hill Twsp.
1421 Montville Twsp.
1422 Morris Twsp.
1423 Morris Plains Borough
1424 Morristown Town
1425 Mountain Lakes Borough
1426 Mount Arlington Borough
1427 Mount Olive Twsp.
1428 Netcong Borough
1429 Parsippany-Troy Hills Twsp.
1430 Long Hill Twsp.
1431 Pequannock Twsp.
1432 Randolph Twsp.
1433 Riverdale Borough
1434 Rockaway Borough
1435 Rockaway Twsp.
1436 Roxbury Twsp.
1437 Victory Gardens Borough
1438 Washington Twsp.
1439 Wharton Borough

OCEAN COUNTY—15

1501 Barnegat Light Borough
1533 Barnegat Twsp.
1502 Bay Head Borough
1503 Beach Haven Borough
1504 Beachwood Borough
1505 Berkeley Twsp.
1506 Brick Twsp.
1507 Dover Twsp.
1508 Eagleswood Twsp.
1509 Harvey Cedars Borough
1510 Island Heights Borough
1511 Jackson Twsp.
1512 Lacey Twsp.
1513 Lakehurst Borough
1514 Lakewood Twsp.
1515 Lavallette Borough
1516 Little Egg Harbor Twsp.
1517 Long Beach Twsp.
1518 Manchester Twsp.
1519 Mantoloking Borough
1520 Ocean Twsp.
1521 Ocean Gate Borough
1522 Pine Beach Borough
1523 Plumsted Twsp.
1524 Point Pleasant Borough
1525 Point Pleasant Beach Borough
1526 Seaside Heights Borough
1527 Seaside Park Borough
1528 Ship Bottom Borough
1529 South Toms River Borough
1530 Stafford Twsp.
1531 Surf City Borough
1532 Tuckerton Borough

PASSAIC COUNTY—16

1601 Bloomingdale Borough
1602 Clifton City
1603 Haledon Borough
1604 Hawthorne Borough
1605 Little Falls Twsp.
1606 North Haledon Borough
1607 Passaic City
1608 Paterson City
1609 Pompton Lakes Borough
1610 Prospect Park Borough
1611 Ringwood Borough
1612 Totowa Borough
1613 Wanaque Borough
1614 Wayne Twsp.
1615 West Milford Twsp.
1616 West Paterson Borough

SALEM COUNTY—17

1701 Alloway Twsp.
1713 Carney's Point Twsp.
1702 Elmer Borough
1703 Elsinboro Twsp.
1704 Lower Alloways Creek Twsp.
1705 Pennsville Twsp.
1706 Mannington Twsp.
1707 Oldmans Twsp.
1708 Penns Grove Borough
1709 Pilesgrove Twsp.
1710 Pittsgrove Twsp.
1711 Quinton Twsp.
1712 Salem City
1714 Upper Pittsgrove Twsp.
1715 Woodstown Borough

SOMERSET COUNTY—18

1801 Bedminster Twsp.
1802 Bernards Twsp.
1803 Bernardsville Borough
1804 Bound Brook Borough
1805 Branchburg Twsp.
1806 Bridgewater Twsp.
1807 Far Hills Borough
1808 Franklin Twsp.

1809 Green Brook Twsp.
1810 Hillsborough Twsp.
1811 Manville Borough
1812 Millstone Borough
1813 Montgomery Twsp.
1814 North Plainfield Borough
1815 Peapack-Gladstone Borough
1816 Raritan Borough
1817 Rocky Hill Borough
1818 Somerville Borough
1819 South Bound Brook Borough
1820 Warren Twsp.
1821 Watchung Borough

SUSSEX COUNTY—19

1901 Andover Borough
1902 Andover Twsp.
1903 Branchville Borough
1904 Byram Twsp.
1905 Frankford Twsp.
1906 Franklin Borough
1907 Fredon Twsp.
1908 Green Twsp.
1909 Hamburg Borough
1910 Hampton Twsp.
1911 Hardyston Twsp.
1912 Hopatcong Borough
1913 Lafayette Twsp.
1914 Montague Twsp.
1915 Newton Town
1916 Ogdensburg Borough
1917 Sandyston Twsp.
1918 Sparta Twsp.
1919 Stanhope Borough
1920 Stillwater Twsp.
1921 Sussex Borough
1922 Vernon Twsp.
1923 Walpack Twsp.
1924 Wantage Twsp.

UNION COUNTY—20

2001 Berkeley Heights Twsp.
2002 Clark Twsp.
2003 Cranford Twsp.
2004 Elizabeth City
2005 Fanwood Borough
2006 Garwood Borough
2007 Hillside Twsp.
2008 Kenilworth Borough
2009 Linden City
2010 Mountainside Borough
2011 New Providence Borough
2012 Plainfield City
2013 Rahway City
2014 Roselle Borough
2015 Roselle Park Borough
2016 Scotch Plains Twsp.
2017 Springfield Twsp.
2018 Summit City
2019 Union Twsp.
2020 Westfield Town
2021 Winfield Twsp.

WARREN COUNTY—21

2101 Allamuchy Twsp.
2102 Alpha Borough
2103 Belvidere Town
2104 Blairstown Twsp.
2105 Franklin Twsp.
2106 Frelinghuysen Twsp.
2107 Greenwich Twsp.
2108 Hackettstown Town
2109 Hardwick Twsp.
2110 Harmony Twsp.
2111 Hope Twsp.
2112 Independence Twsp.
2113 Knowlton Twsp.
2114 Liberty Twsp.
2115 Lopatcong Twsp.
2116 Mansfield Twsp.
2117 Oxford Twsp.
2118 Pahaquarry Twsp.
2119 Phillipsburg Town
2120 Pohatcong Twsp.
2121 Washington Borough
2122 Washington Twsp.
2123 White Twsp.

INTRODUCTION

The presence of hazardous materials or toxic chemicals at an incident location or other emergency situation adds a new dimension of risk to those handling and treating casualties. The fundamental difference between a hazardous materials incident and other emergencies is the potential for acute risk from contamination to both patient and responder. In some cases, traditional practices must be altered to avoid compounding a critical situation.

Hospital emergency departments must protect their personnel and other people within the hospital, while providing the best care for the chemically contaminated patient. **This guide is intended to help hospital emergency departments plan for incidents that involve hazardous materials and improve their ability to respond to these incidents appropriately.**

To ensure appropriate and timely patient care, as well as optimal worker protection, emergency personnel must have an understanding of decontamination procedures and personal protective equipment that they do not generally receive in the course of their routine professional training. They should also be aware of community resources that could be called upon to assist in emergency response.

Current training curricula for emergency physicians, nurses, and emergency medical technicians (EMTs) often do not adequately prepare these professionals to either manage the contaminated individual or decontaminate patients exposed to toxic substances. High-quality, specific, and concise guidance is needed to describe appropriate procedures to be followed by emergency medical personnel to safely care for a patient, as well as to protect equipment, hospital personnel, and others from risk of exposure.

This guide for emergency department personnel is designed to familiarize readers with the concepts, terminology, and key considerations that affect the management of incidents of chemical contamination. It has been developed not only to present uniform guidance for emergency care of chemically contaminated patients, but also to provide basic information critical to advance planning and implementation of emergency medical services' (EMS) strategies. It is intended to illustrate the characteristics of hazardous materials incidents that mandate modifications to traditional emergency response and the preparatory actions that should be taken to respond effectively to hazardous materials incidents.

All hospital and community emergency response systems may not be prepared to respond to a hazardous chemical incident to the same degree. This document may be used to assess capabilities with respect to potential community hazards and to develop response plans using national and community-specific resources. Worker safety and training are also key factors in effective management of medical emergencies. This document is intended to provide source material for developing local training and safety protocols.

PLANNING FOR THE HAZMAT/ WMD INCIDENT

ADVANCE PLANNING FOR A HAZMAT/WEAPONS OF MASS DESTRUCTION (WMD) INCIDENT

Advance pre-planning is the most essential phase of preparation for a HAZMAT/WMD incident.

I. External Pre-planning

External pre-replanning should involve coordination with local agencies such as fire, EMS, and HazMat teams, along with local chemical or industrial sites. It will be important to establish an integrated plan that outlines a common response philosophy AND the roles and responsibilities that should be taken in the event of a HAZMAT/WMD case.
For example, it will be important to know the following types of information in the preparation for a HAZMAT/WMD incident:

1. What are the sources of Hazardous Material accidents in the community?
2. Where is the nearest HazMat team located?
3. Which local EMS organization and fire departments are trained and equipped to respond to a HazMat incident?
4. Do any of the local industrial sites have HazMat teams that can be called upon for assistance in response to a HazMat incident?
5. How will victims from a HAZMAT/WMD incident be handled?
6. Will **all** contaminated/injured patients be decontaminated prior to transport to the hospital? Does this include trauma patients? Are there any exceptions? Who will perform field decontamination?
7. To what extent will patients be decontaminated? (i.e. gross, partial or fully decontaminated)
8. Which local hospitals are trained and equipped to handle hazardous material accident victims?
9. How will a hazardous material incident involving mass casualties be handled? How will the patient load be dispersed in this type of incident?

II. Internal Pre-planning

A formal written HAZMAT/WMD Response Plan should be developed as part of the overall Hospital Disaster Plan. It should be developed in conjunction with the local EMS organization, fire department, HazMat team and local industry. It should be comprehensive in addressing all possible situations involving hazardous material accident patients:

- walk in patient(s)
- announced pre-hospital delivered patients
- unannounced pre-hospital delivered patients
- stable vs. unstable patient
- mass casualties incident

The formal plan should address the following types of issues:

A. Determine the responsibilities of the department ranging from emergency medicine, administration and security to clinical specialist such as toxicology, laboratory medicine and occupational medicine.

B. Determine the responsibilities of emergency department personnel who will be involved in the handling of a hazardous material accident patient.

C. Selection and set-up of an outside or external decontamination area.

D. Selection and set-up of an inside or internal decontamination area.

E. Selection of decontamination equipment and supplies.

F. Selection of proper personal protective clothing.

G. Establish pre-hospital notification procedure.

H. Development of a list of resource organizations that can be contacted in the event of a HazMat incident.

REMEMBER: Coordinate your planning efforts with JCAHO and Office of Emergency Management Standards.

Essential Public Health Services

- Monitor health status to identify community health problems
- Diagnose and investigate health problems and health hazards in the community
- Inform, educate, and empower people about health issues
- Mobilize community partnerships to identify and solve health problems
- Develop policies and plans that support individual and community health efforts
- Enforce laws and regulations that protect health and ensure safety
- Link people to needed personal health services and assure the provision of health care when otherwise unavailable
- Assure a competent public health and personal health care workforce
- Evaluate effectiveness, accessibility, and quality of personal and population-based health services
- Research for new insights and innovative solutions to health problems

From "Public Health in America." Public Health Functions Steering Committee, 1994.

PERSONAL PROTECTIVE EQUIPMENT (PPE)

Introduction

Dealing with a HAZMAT/WMD incident is risky business. The first responder initially deals with unknown factors which can clearly be hazardous to his health. As such, he must handle the incident differently than he would normally and with much more caution.

Greater care should be given to personal protection of the emergency services personnel with more detail given to approach and operational procedures. The availability of proper protective equipment, or the lack of it, has a direct bearing on how and if an approach is made; what the incident mitigation objectives can be; how work area assignments are made and defined; and how the establishment of working limits (operating time, work zones, and personal protection) are determined.

The most critical factor here is the life threat to E.D. personnel. Without knowledge of exactly what personal protection equipment is necessary for the materials involved in the incident and the protective limits of the equipment, the incident response team can get into immediate serious trouble. The first concern should be the proper protection of the first responder.

Federal Regulations Pertaining to the Use of Personal Protective Equipment (PPE)

The term Personal Protective Equipment (PPE) is used in this document to refer to both personal protective clothing and equipment. The purpose of PPE is to shield or isolate individuals from the chemical, physical, and biological hazards that may be encountered at a hazardous materials incident.

OSHA standards mandate specific training requirements (8 hours of initial training or sufficient experience to demonstrate competency) for employees engaged in emergency response to hazardous substances incidents at the first responder operations level. Additionally, each employer must develop a safety and health program and provide for emergency response. These standards also are intended to provide additional protection for those who respond to hazardous materials incidents, such as firefighters, police officers, and EMS personnel. OSHA's March 6, 1989, 29 CFR [1910.120] final rule as it applies to emergency medical personnel states that: "Training shall be based on the duties and functions to be performed by each responder of an emergency response organization (p. 9329).

Training Is Essential Before Any Individual Attempts To Use PPE

No single combination of protective equipment and clothing is capable of protecting against all hazards. Thus, PPE should be used in conjunction with other protective methods. The use of PPE can itself create significant worker hazards, such as heat stress, physical and psychological stress, and impaired vision, mobility, and communication. In general, the greater the level of PPE protection, the greater are the associated risks. For any given situation, equipment and clothing should be selected that provide an adequate level of protection. Over-protection can be as hazardous as under-protection and should be avoided. Personnel should not be expected to use PPE without adequate training. The two basic objectives of any PPE program should be to protect the wearer from safety and health hazard and to prevent injury to the wearer from incorrect use and/or malfunction of the PPE. To accomplish these goals, a comprehensive PPE program should include: hazard identification; medical monitoring; environmental surveillance; selection, use, maintenance, and decontamination of PPE; and training.

Protective Equipment for ED Personnel

The first consideration of all E.D. personnel must be their own ability to survive the incident. That thought sounds simple enough. You would think that if you're out in the E.D., some distance from the incident in fresh air, that you're okay. This may not necessarily be true! The products involved in hazardous materials incidents can be colorless, odorless, tasteless, and, you may not feel their presence as they envelop you in a destructive cloud that may not be noticed until years later. The question of E.D. personnel safety is dependent upon three factors:

1. what products are involved in the incident
2. what are their associated risks under the incident conditions
3. what level of protection should operating personnel have to deal with the incident.

The first and second factors can only be answered through discovery of what products are involved. **If, and as long as, the products remain unknown,** then a worse case probability should be assumed. Once the product or products are known and the risks have been evaluated accordingly, **then** the level of personal protection can be set to match the needs of the operational objectives.

All personnel **must** therefore understand what constitutes personal protection or personal protective equipment. This includes all personnel who work in or near the incident site, regardless of whether they be the nurses, physicians, emergency medical services personnel, x-ray, lab or respiratory services.

The typical first responder will arrive on the scene in the least acceptable level of protection. If it is a police officer it will be a blue uniform with a badge and a side arm for personal protection. Emergency medical personnel will report to the scene with a medical kit, a stethoscope and other implements sticking out of numerous pockets. The firefighter will roll onto the scene in full structural fire fighting gear with hoses and tools ready at hand. **Unless the product exposure risk is known and determined to be no risk at all, or of little risk to personnel, these people should be denied access to the incident site, and their proximity to the operational area should be clearly defined and closely monitored.**

The psychological feeling of invulnerability is a significant factor when dealing with emergency services personnel. The danger must always be in the mind of the incident commander as a concern during operations.

Everyone is vulnerable unless they:

1. are properly protected before they enter the incident site
2. are aware of the risks present at the site
3. know what objectives can be realistically attained.

It should also be noted here that **no one type of personal protection will satisfy every condition encountered** at hazardous materials incidents. The Mayor may find his sport jacket disintegrating; the police officer may find his badge and side arm turning green; the EMS personnel may find themselves unable to dispense medical treatment because they cannot see or breathe, and the firefighter may be running away from the scene at the best possible speed. Obviously, selecting the appropriate level of personal protective equipment necessary for the incident and properly wearing it, is the key to a safe and effective operation.
Clothing which is specifically designed for hazardous materials incidents, and for use with specific types of chemicals, falls into four categories: Level A, Level B, Level C, and Level D. The predominant physical, chemical, and toxic properties of a chemical, or chemicals, involved in a hazardous materials incident will dictate the specific type of chemical protection required. The guidelines for the use of these various levels of protection are as follows:

Level A: MAXIMUM PROTECTION

Should be worn when the highest level of respiratory, skin, and eye protection is required.

Level A Conditions:

- Unknown gas concentrations.
- Known extremely toxic or corrosive gases.
- Possible or expected skin exposure to toxic or corrosive liquids, gases or solids.
- IDLH Atmospheres

Level A Configuration:

- Fully-encapsulating chemical resistant suit completely encloses user and SCBA.

Level B: HIGH RESPIRATORY PROTECTION

Should be worn when the highest level of respiratory protection is needed but a lesser level of skin protection is required. (SPLASH PROTECTION)

Level B Conditions:

- Known contaminant levels below IDLH concentrations.
- Atmosphere with less than 19.5% oxygen.
- Chemical concentrations which are above the TLV level.

Level B Configuration:

- Chemical resistant clothing including boots and gloves, that generally do not fully enclose user and SCBA.

Level C: LIMITED RESPIRATORY PROTECTION

Should be worn when the criteria for using air-purifying/respirators has been met.

Level C Conditions:

- Greater than 19.5% oxygen.
- Contaminant level below IDLH and above TLV.
- Skin contact hazards are minimal or do not exist.

Level C Configuration:

- Level B and Level C differ only in type of respiratory protection required. The chemical protective clothing requirements are the same.

Level D: MINIMUM PROTECTION

Should be worn only as a work uniform and not on any site with a respiratory or skin hazard.

Level D Conditions:

- No possibility of respiratory exposure.
- No possibility of skin exposure.
- No contaminant levels below TWA.

Level D Configuration:

- Standard Work Uniform, including structural firefighter protective equipment.

Note: OSHA Final Rule 29 CFR Part 1910(q)(3)(iv)

(iv) Employees engaged in emergency response and exposed to hazardous substances presenting an inhalation hazard or potential inhalation hazard shall wear positive pressure self-contained breathing apparatus while engaged in emergency response, until such time that the individual in charge of the ICS determines through the use of air monitoring that a decreased level of respiratory protection will not result in hazardous exposures to employees.

NFPA CHEMICAL PROTECTIVE CLOTHING STANDARDS

The National Fire Protection Association has completed the development and publishing of three (3) national standards regarding chemical protective clothing for use during hazardous chemical emergencies.

NFPA 1991 Standard:

This standard is for specifying the design and performance criteria for a chemical protective garment that is intended to be used in a gaseous or vapor atmosphere of chemicals. This garment must be totally encapsulating.

NFPA 1992 Standard:

This standard is for the design and manufacture of a garment that did not have to meet the rigid permeation resistance requirements found in the 1991 standard. In the 1992 standard, Standard on Liquid Splash-protective Suits for Hazardous Chemical Emergencies, the emphasis was basically on two things:

1. single to multi-piece garments
2. suitable chemical test that reflected resistance to liquids.

Its use is for liquid splash environments only.

NFPA 1993 Standard:

This standard deals with support functions and is described as hazardous chemical operations involving controlled chemical uses or exposures in non-flammable atmospheres with minimum threats to loss of life, personnel injury, or damage to property or to the environment. Functions include, but are not limited to, decontamination, remedial cleanup, and training.

PROTECTION FROM CHEMICAL WARFARE AGENTS

Military-issued equipment to protect against these agents varies widely based on the level of anticipated exposure. Civilian activities in the presence of these materials are regulated by HAZWOPER, which is more stringent than military standards. As in any chemical emergency, *use the highest level of protection available until the chemical is identified*. Modify that level of protection as appropriate after determining what chemicals are present. For example, nerve and blister agents require SCBA with Level A protection. Other toxic chemicals may require a lower level of protection.

The level of protection necessary for the hazardous materials responder at an incident should be based on the following factors which must be critically assessed:

A. The type and measured concentration of the chemical substance in the ambient atmosphere and its toxicity.

B. The potential for exposures to substances in the air; to splashes of liquids; and to direct contact with materials for substances due to the work being done at the incident site.

When To Remove Personal Protective Equipment (PPE) If It Has Been Contaminated

There is always the possibility that circumstances will cause PPE to become contaminated despite all precautions. Personnel should continually check each other to detect any contamination. The question of exactly when it is safe to remove contaminated PPE is dependent on several factors which can become quite complicated. The scope of the incident and the probability that multiple chemicals are involved must be considered. The dilemma goes beyond "when" to: where can protective equipment be removed; why should it always be removed when you leave the incident area; what should be removed based on the conditions; and who should do the removal of the equipment. Who, what, when, where, why and how are all critical questions which must be answered when dealing with the removal of personal protective equipment.

The removal of personal protective equipment should never be done within the incident "hot zone" or in any contaminated area until recognized professionals have determined, through the use of appropriate equipment, that the hazard risk has been removed. The incident commander is responsible for insuring that incident operations in the work area, the decontamination area, and any other areas used during the incident are safe.

If personal protective clothing and equipment is removed within the incident site, even where it has been declared safe, incident commanders must continue to monitor personnel, who should be checking each other, to insure that symptoms of exposure are not becoming apparent. There

is always the chance that something was missed. A test may have been performed incorrectly or a testing device may fail. The final responsibility again lies with the incident commander.

Regardless of the type of contaminated protective clothing, the removal of the protective envelope should be a closely monitored and planned exercise. It should only be done when it has been declared safe to do so, and only in an area which has been specifically designated and designed for the purpose. Where the risks to health are unknown or found to be serious—great care must be taken in removal supervision and personnel safety. Personnel are not safe until they have removed their protective clothing an equipment, and are returned to a safe and clean environment.

Respiratory Protection

The use of respiratory protection at a hazardous materials incident is mandatory. The level (degree) of respiratory protection must be in compliance with both OSHA regulations, NIOSH guidance documents, standard operating procedures, and, most of all, be suited for the hazard and the wearer. Air purifying respirators (APR's) and self-contained breathing apparatus (SCBA) are the only two forms of respiratory protection that is addressed.

Air Purifying Respirators (APR's)

The use of APR's is limited to the available approved cartridges or canisters. Both cartridges and canisters have very limited use, if used at all, during a hazardous materials incident. This is due to several very critical factors:

1. APR's are negative system, thus allowing for infiltration of contaminated air into the mask,
2. APR's have very limited use times, which does not afford the wearer any substantial protection,
3. APR's require individual fit testing prior to actual use and wearing,
4. APR's do not protect the wearer from unknown air contaminants,
5. APR's ARE NOT APPROVED FOR USE BY EMERGENCY RESPONDERS AT A HAZARDOUS MATERIALS INCIDENT.
6. You cannot be sure that the contaminants at the emergency will not elevate nor control the oxygen content of the atmosphere.

As stated in 4 above, in order for APR's to provide the safe and proper level of protection necessary for the wearer to be protected, the wearer must know both the contaminant type and concentration. This is not the case for the emergency worker or hazardous materials responder.

As such, this form of respiratory protection is reserved for use by those workers that are outside both the hot and warm zone, and who have been properly fit tested as well as supplied with the appropriate canister or cartridge, based upon verifiable air monitoring. ONLY UNDER THE DIRECT SUPERVISION OF THE ON SCENE COORDINATOR OR OTHER HEALTH OR SAFETY OFFICER CAN THESE DEVICES BE USED AT A HAZARDOUS MATERIALS INCIDENT.

Self-Contained Breathing Apparatus (SCBA)

The SCBA affords the wearer the best, and highest, level of respiratory protection (Level B and Level A). It provides the wearer with his or her personal air supply, totally segregated from the environmental air.

Although there exist various makes, models, styles, and manufacturers, the important thing to remember is that there exists only three types:

1. Re-breathers,
2. Demand,
3. Pressure Demand.

RE-BREATHERS are basically air generators. The wearer is supplied with a closed, recirculating system, whereby exhaled air is sent through a carbon dioxide scrubber, and returned, after a small "injection" of oxygen. The wearer also carries a small canister of oxygen in the unit. THESE UNITS ARE NOT TOTALLY POSITIVE PRESSURE AND ARE NOT APPROVED FOR HAZARDOUS MATERIAL RESPONDERS.

The DEMAND type, NOT APPROVED FOR USE AT HAZARDOUS MATERIAL INCIDENTS, only provides positive pressure to the user upon exhalation. As such, the possibility exists that the wearer may breathe contaminated air.

The PRESSURE DEMAND type is the ONLY TYPE APPROVED FOR USE AT HAZARDOUS MATERIAL INCIDENTS, since positive pressure is always present in the system, thus preventing the wearer from inhaling environmental air.

Lastly, the hazardous materials responder may be subject to or required to work using an air line system. In this set-up, the worker is "tethered" to a fixed air supply with a pressurized airline. The wearer may also be equipped with a dual mode operating SCBA or an escape pack. However, UNDER ANY AIRLINE SET-UP THE WORKER MUST BE SUPPLIED WITH A MINIMUM OF A FIVE MINUTE ESCAPE PACK. Normally, however, this operational set-up is used during extensive containment or remedial operations.

The Hazardous Materials Involved:

The form of hazardous material involved will have a direct bearing on the choice of personal protective equipment, or the decision to withdraw from the area. These are three broad categories of hazardous material to consider: chemical materials, biological (etiologic) materials, and radioactive materials.

These categories can be defined as follows:

1. **Chemical Materials:** Are materials which are hazardous because of their chemical and physical properties.
2. **Biological Materials:** Are organisms which can have a dangerous effect on life or the environment, and they can exist in normal ambient environments.
3. **Radioactive Materials:** These are materials which emit ionizing radiation.

Each of these categories and the risks associated with that particular category of hazardous material will influence the choice of personal protective equipment. In addition, the type of material, as referenced to these categories, can have far reaching effects on how personal protective equipment is used (operationally), how and whether it can be decontaminated, and whether it can be reused during the operation. An incident involving radioactive material, for example, can lead to the disposal of all personal protective equipment utilized during the incident—**and it can never be used again**. This can certainly be an expensive proposition for many communities.

The physical state of the hazardous material involved is also a factor of concern in choosing protective equipment. Materials, or elements, can be classified into three basic states of matter: gases, liquids, and solids. Each of these states can affect your choice of equipment and how you wear it. As an example, large solids are not as much of a problem as liquids, gases or fine dusts (solid particles) and vapors, which can permeate or penetrate protective clothing as well as contaminate it.

The Means By Which Personal Protective Equipment Performance May Become Compromised

Personal protective equipment used in the E.D. may become defective leaving incident personnel vulnerable to the life threatening effects of hazardous chemicals. Personal protective equipment must be inspected on a regular basis to determine if its reliability meets the minimum protection requirements to sustain the protective envelope.

Personal protective equipment may be affected in the following ways:

Chemical resistance is the ability of the chemical material or materials which make up the protective clothing and equipment to prevent or reduce degradation and permeation of the fabric by the attack chemical. In the case of structural fire fighting clothing this ability is extremely limited as compared to the numerous chemical products which may affect its integrity.

Degradation is a chemical action involving the molecular breakdown of the material due to contact with a chemical.

Permeation is a chemical action involving the movement of chemicals, on a molecular level, through intact material. There usually is no indication that this process is occurring.

Penetration is the movement of material through a suit's closures, such as zippers, buttonholes, seams, flaps or other design features. This also includes loose stitching, and rips and tears in personal protective clothing.

PROTECTIVE CLOTHING AND DEVICE FOR HAZARDOUS MATERIALS/WMD WORKER SURVEY

EPA LEVEL	DEGREE OF EXPOSURE	NATURE OF EXPOSURE	RESPIRATORY PROTECTION	PROTECTIVE CLOTHING	EXAMPLES OF WORK ENVIRONMENTS
A	HIGHEST LEVEL OF EYE, SKIN AND RESPIRATORY PROTECTION NEEDED	IDLH IN AIR AND SKIN IS MET. ABSORPTION THROUGH SKIN & CONTACT CAUSES SEVERE INJURY OR DEATH.	S.C.B.A. OR AIR LINE WITH ESCAPE BOTTLE	FULLY ENCAPSULATED SUIT (CHEMICAL SPECIFIC RESISTANT) GLOVES } (CHEMICAL RESISTANT) BOOTS } 2 WAY INTRINSICALLY SAFE RADIO	CONFINED SPACES AND OXYGEN DEFICIENT ATMOSPHERE. HIGH EXPOSURE POTENTIAL. EXPLOSIVE OR HIGH VISIBLE OR SUSPECTED TOXIC OR CORROSIVE VAPORS AND GASES. USED WHEN EXCAVATING LEAKING DRUMS (CYANIDE POISONS, ARSENICS AND PESTICIDES) EMERGENCIES WITH KNOWN TOXIC ENVIRONMENTS.
B	HIGHEST LEVEL OR RESPIRATORY PROTECTION BUT LESSER SKIN PROTECTION	AIR CONCENTRATIONS REQUIRE HIGH PROTECTION (IDLH IN AIR), 19.5% OXYGEN OR LESS. NO SEVERE SKIN HAZARD (SPLASH)	S.C.B.A. OR AIR LINE WITH ESCAPE BOTTLE	CHEMICAL RESISTANT COVERALL (SPLASH SUIT) DISPOSAL CHEMICAL SUITS COVERALLS AND LONG UNDERWEAR 2 PR. CHEMICAL RESISTANT GLOVES BOOTS AND COVERS HARD HAT ESCAPE MASK 2 WAY INTRINSICALLY SAFE RADIO	TOXIC SUBSTANCES HAVE BEEN IDENTIFIED. RESPIRATORY PROTECTION IS A PROBLEM. NO SEVERE SKIN DAMAGE. USED WHEN HANDLING DRUMS, TEMPORARY STORAGE FACILITY WHERE HIGHER LEVEL OF PROTECTION IS NOT REQUIRED. POSSIBLE POTENTIAL OF TOXIC VAPORS OR OXYGEN DEFICIENCY.
C	WHEN AIR PURIFYING RESPIRATOR IS NEEDED	OXYGEN IS GREATER THAN 19.5% IN AIR. UP TO TLV VALUE IS MET.	FULL FACE AIR PURIFYING RESPIRATOR	CHEMICAL RESISTANT COVERALL (SPLASH SUIT) DISPOSAL CHEMICAL SUITS COVERALLS AND LONG UNDERWEAR 2 PR. CHEMICAL RESISTANT GLOVES BOOTS AND GLOVES HARD HAT ESCAPE MASK 2 WAY INTRINSICALLY SAFE RADIO	S.C.B.A. NOT NEEDED BUT RESPIRATOR IS REQUIRED. NO ADVERSE AFFECTS TO SKIN IF SPLASHED. DIRECT READING INSTRUMENTS SHOW SLIGHTLY ABOVE BACKGROUND OR TLV VALUES. PARTICLES OR DUST IN AIR.
D	NO PROTECTION NEEDED AGAINST HAZARDS	LESS THAN TLV LEVEL	NOT NECESSARY TO BE WORN	NORMAL WORK ATTIRE	ADMINISTRATION AREA OF WORK SITE. NO CONTAMINATION. LOW THREAT OF HAZARD IMPACT.

EMERGENCY DEPARTMENT DECONTAMINATION

Introduction

DEFINITION

Decontamination is the process of removing potentially harmful contaminants from exposed individuals and equipment in order to reduce the spread of contamination in the work area and to prevent inadvertent and unnecessary contact with contaminated materials.

Personnel should not handle a contaminated suit, tool, or person without proper protective equipment. Failure to do so may lead to skin absorption or inhalation of the contaminant, resulting in injury, illness, or death.

Not every patient you deal with will be contaminated. In fact, the *majority* of your patients **will not** be contaminated and can be handled in the routine fashion. However, until proven otherwise, you must *assume that every patient is contaminated.*Every attempt must be made to keep contaminated patients separated from those who are uncontaminated. This is best achieved by the use of *at least two* EMS units at the site of the emergency. One unit should be available to be dedicated to the treatment, care, and transportation of contaminated patients (if required) while the other should treat only those who have not been contaminated and do not require special handling procedures. A third may be needed for medical monitoring of personnel.

Units that have been set up and designated to handle contaminated patients need to be identified to EMS personnel and others on the site. One of the more popular means in use is to place a *red "X"* over the Star-of-Life symbol on the four sides of the vehicle. Red duct tape, or any red plastic or cloth tape can serve this purpose. This identification also becomes important upon arrival *at the medical facility.* Special entry locations may have been set up to deal with the contaminated patient so traffic control officers can direct marked units to the appropriate locations.

THE CONCEPT OF SECONDARY CONTAMINATION

An essential question to ask is, "What is the risk of *secondary contamination* (to rescuing personnel, transport vehicles, hospital emergency departments) from this chemical?" It is traditionally axiomatic in hazardous materials emergency management that chemicals should be considered both highly toxic and highly contaminating to personnel, vehicles, and the environment. However, a great many chemicals are very highly toxic *only* in the high concentrations found in the immediate exposure area (hot zone) but pose *little or no risk* to persons outside the hot zone. Small amounts of some chemicals may produce relatively little acute toxicity, but because they are suspected of causing cancer or other chronic disease they are considered to create a risk of secondary contamination.

Tables 1 and 2 list selected examples of hazardous substances which carry a high vs. a low risk for *secondary contamination*The lists are meant to be illustrative, not exhaustive. Note that highly toxic chemicals may be found in *either* list. The Haz Mat Team, Regional Poison Control Center or County Health Department can assist you in determining the potential for secondary contamination of other hazardous materials.

SUBSTANCES WITH SERIOUS POTENTIAL FOR SECONDARY CONTAMINATION:

Unless the victim has been properly decontaminated, substances like those listed in Table 1 may persist in significant amounts on the victim's clothing, skin, hair, or personal belongings, and may jeopardize health care workers or other attendants. Recommended protective gear should be worn. Reducing the potential for chemical exposure from any form of mouth-to-mouth resuscitation, including use of pocket one-way valve mouth-to-mouth resuscitation devices should be carefully considered when the victim has been exposed to one of the listed gases. If resuscitation efforts are necessary, a bag valve mask with reservoir device connected to oxygen, should be applied to the patient. Contact with even lightly contaminated skin or clothing should be minimized prior to decontamination. *Proper decontamination by adequately protected personnel must be carried out before the victim is treated by prehospital or emergency department personnel.*

Table 1: Substances with a High Risk for Secondary Contamination

Examples:

- Acids, alkali & corrosives (if concentrated)
- Asbestos (large amounts, crumbling)
- Cyanide salts & related compounds (e.g., nitriles) and hydrogen cyanide gas
- Hydrofluoric acid solutions
- Nitrogen-containing and other oxidizers which may produce methemoglobinemia (aniline, aryl amines, aromatic nitro-compounds, chlorates, etc.)
- Pesticides (organophosphates)
- PCBs (polychlorinated biphenyls)
- Phenol and phenolic compounds
- Many other oily or adherent toxic dusts and liquids
- Radioactive material

SUBSTANCES WITH LITTLE RISK FOR SECONDARY CONTAMINATION:

Many of the substances listed in Table 2 are highly toxic. However, even if they persist in the victim's clothing, skin, hair, or personal belongings after removal from hot zone, they are not likely to jeopardize health care workers or rescuers and are not likely to secondarily contaminate vehicles or the emergency department. *On-scene decontamination, if indicated, is desirable, especially clothing removal and victim wash, but not essential.*

Table 2: Substances with a Low Risk for Secondary Contamination

Examples:

- Most gases and vapors unless they condense in significant amounts on the clothing, skin or hair
- Weak acids, weak alkali and weak corrosives in low concentrations (excluding hydrofluoric acid)
- Weak acid or weak alkali vapors (unless clothing soaked and excluding hydrofluoric acid vapor)
- Arsine gas
- Carbon monoxide gas
- Gasoline, kerosene & related hydrocarbons
- Phosphine gas
- Smoke/combustion products (excluding chemical fires)
- Small quantities of common hydrocarbon solvents (e.g., toluene, xylene, paint thinner, ketones, chlorinated degreasers)

Decontamination Area Preparation

Any victim of a hazardous materials incident must be considered to be contaminated until demonstrated otherwise.

Security personnel should be stationed at the main entrance of the emergency department close to the decontamination area to prevent unauthorized entry, and to direct the vehicle transporting the patient to the appropriate area. A reception area should be set up just outside the emergency department entrance, where arriving contaminated patients can be screened for adequate decontamination.

A decontamination area should be large enough to facilitate decontamination of more than one patient and accommodate the many personnel involved in patient treatment and contamination reduction. The ventilation system should either be separate from the rest of the hospital or turned off in order to prevent spread of airborne contaminants throughout the facility. The best place (weather permitting) to evaluate and initially treat contaminated patients is outside where ambient ventilation will keep cross-exposure low. Some hospitals have radiation decontamination facilities that can be used with minor changes. An outside or portable decontamination system is a viable substitute and would aid in preventing contamination of the emergency department and other patients. A practical alternative for facilities with limited resources is to have a warm shower nozzle, soap, a wading pool, and plastic garbage bags in a predesignated area outside the emergency department back door. The patient may be able to remove his or her own contaminated clothing, place it in a double bag, and do his or her own soap and water decontamination. A partial tent or curtain can provide privacy for the patients. In most circumstances, ordinary hospital gowns, plastic goggles, and plain latex gloves will adequately protect hospital staff in case they have to assist the patient in removing soaked clothing, wash exposed skin and hair, or perform eye irrigation. With large amounts of concentrated corrosives or very oily materials, such as pesticides, disposable CPC and unmilled nitrile gloves will offer additional protection. If it is anticipated that your facility is likely to receive heavy contaminated patients who have not received prior decontamination, then it may be appropriate to purchase appropriate protective gear and to fit and train emergency department staff in its use. However, no person should wear and use specialized PPE, especially respiratory protective gear, without prior training.

To prevent unnecessary contamination, all nonessential and nondisposable equipment should be removed from the decontamination area. A "clean" member of the staff should stand on the clean side of the decontamination area to hand in supplies and receive medical specimens.

DECONTAMINATION PROCEDURES

Hazardous materials incidents involve numerous on-site problems and operational concerns. Common to all these responses is the threat of contamination. Decontamination must be considered an essential part of hazardous materials response operations. This module will detail the purpose and steps taken in field decon operations.
Personnel may become contaminated in a number of ways including:

- contacting vapors, gases, mists or particles in the air
- being splashed by materials during rescue or containment operations
- walking through puddles of liquids or on contaminated soil
- treating contaminated patients
- using contaminated instruments or equipment

Decontamination is the process of making response personnel, victims and equipment free from contamination by eliminating or reducing harmful substances to a safe level. Response team personnel must undergo decon prior to removing their protective equipment. Victims need to be decontaminated before being turned over to EMS transport personnel. Equipment must be thoroughly cleaned so that its subsequent use will not lead to a spread of contamination.

Different chemical threats require varying levels of decon. In cases of extremely hazardous or unknown substances, the following minimum decon procedures should be complied with:

1. Establishment of an entry/exit point: This point will be used by all personnel to enter and exit the area of contamination. The use of one entrance will reduce the chance of contamination leaving the area. An emergency exit should also be established. This will allow for a secondary exit should conditions deteriorate and demand immediate evacuation.
2. Primary Decontamination: This step may actually entail many intermediate steps. The personnel should undergo water rinsing and soap or solution washes to remove as much contaminant as possible. The number of washes will depend on the nature of the contaminant.
3. Removal and isolation of protective clothing: Outer protective clothing should be removed at this station. Outer gloves and overboots should be removed first. The protective clothing can then be removed with special care taken to reduce the risk of contaminating the worker. Inner gloves are the last piece of protective equipment to be removed.
4. Removal of personal clothing: With extreme hazardous substances, the removal and isolation of the worker's personal clothing is necessary. All clothing should be isolated for future cleaning or disposal.
5. Personnel shower: In order to ensure complete decontamination, all personnel should shower. Liquid soaps work best. Special attention should be directed to the hair, fingernails and areas such as the underarms and groin. With known exposure, all run-off should be contained if possible.
6. Drying off and redressing: Disposable towels should be used for drying. Clean clothes can then be worn. Many teams use disposable coveralls or hospital scrubs.
7. Medical evaluation: All personnel with potential exposure must undergo a medical evaluation. Entry personnel should have received a pre-entry exam as a baseline. Vital signs, indications of exposure and signs of heat stress should all be evaluated. Personnel should be transported to a hospital for further evaluation if necessary.

The extent of this process will depend on the nature of the contaminant and the level of exposure. Steps 4 through 7 may not necessarily take place in this order. For example, the medical exam can follow primary decon and protective clothing removal. Or, if contaminants are not extremely hazardous, personnel may shower at an off-site location.

Water is an essential component of decon, and can be used to dilute many products. Water can be sprayed from garden hoses adapted for use or special deluge showers. Inexpensive showers can be made from PVC pipe with holes drilled to provide for water flow. Water massage shower heads are excellent because of their variable flow and spray patterns. Water sprays should be kept to a minimum to reduce overspray from the contaminant pools and to reduce the amount of contaminated water to be dealt with later.

PVC (plastic) pipe becomes brittle in cold temperatures and the glue used to hold the PVC together becomes much less adhesive. All PVC piping must be inspected and tested prior to use.

Decon usually requires the use of soaps or solutions. Usually a mild detergent and water may be sufficient. In special cases, a specific decon solution will be required. Depending on the contaminant, a special base, acid, solvent or bleach solution may be used. **These solutions are only used with equipment and should never be applied to skin.**

Water from decon procedures needs to be contained and possibly disposed of as hazardous waste. Numerous devices are available to contain run-off water; children's wading pools, fire department drafting tanks, hose lines covered by visquene, containment areas fashioned from ladders and salvage covers, and commercially available portable decontamination tanks are all possible alternatives. The decision of which option to choose should be governed by how easy it is to assemble and use. Remember that there is a chance that the pool may need to be disposed of.

Personnel conducting decon operations must be properly protected. This should include the use of positive-pressure SCBA and chemical-resistant outer clothing.

Decon procedures must cover any equipment that has been inadvertently contaminated, such as protective equipment, SCBA, tools and possibly even vehicles. If vehicles have been contaminated, procedures should include a thorough washing with special attention paid to tires and other contact surfaces. A mechanic should thoroughly inspect the vehicle after decon. Equipment may need to be steam-cleaned or sandblasted to ensure that it is clean. Resources such as Chemtrec, computer data bases and the DEP can be contacted for assistance in determining the extent of decon that is necessary.

Personnel should carry out a gross decon and isolation process on all equipment prior to completing procedures on themselves. Protective equipment and tools should be isolated for further cleaning and testing. Occasionally protective equipment cannot be totally decontaminated and must be disposed of. After the decon process is complete, the waste water and equipment (i.e., pools) can be disposed of as hazardous waste.

The personnel who are conducting the decon operations must also go through a cleaning process. Personnel should work their way through the decon area, becoming cleaner as they progress. The object is to be absolutely clean when leaving the contamination reduction corridor. They should decon each other, with the last person finishing procedures on himself.

A trend in the hazardous waste industry is to move toward "dry decontamination." While the term may be misleading, this process does allow for a minimum of liquid waste by-products.

This concept requires the use of layered, disposable protective clothing. A water/solution may be necessary for the areas of gross contamination such as overboots and gloves. Most clothing should be removed and disposed of without extensive washing and rinsing. This allows for easier cleanup and reduces the chance of secondary contamination from toxins trapped in reusable protective clothing.

Unfortunately, there is no method to immediately determine how effective decon procedures have been in removing contaminants. Discolorations, stains, corrosive effects and substances adhering to objects can indicate that the contaminants have not been removed. However, observable effects only point to surface contamination and not permeation (absorption) into the clothing. Also, many contaminants are not easily detected.

Two methods of measuring the effectiveness of decon procedures are swipe and permeation testing. Cloth or paper patches (swipes) are wiped over decontaminated surfaces and sent to a laboratory for analysis. Swipe tests can be done on protective clothing, equipment and skin. Permeation tests require that a piece of protective clothing be sent for analysis. However, both swipe and permeation testing provides after-the-fact confirmation. Along with visual observations, the test results can help evaluate the effectiveness of the completed decon procedures.

CONTAMINATED VICTIMS

Special attention needs to be devoted to contaminated patients. These patients pose a risk of secondary exposure to the transport personnel and vehicle. Also at risk is the receiving hospital and ED staff. Every effort must be made to decontaminate the patient prior to transport. **Gross decontamination can be accomplished by simply removing the patient's clothing and using a water rinse.** A more complete decon can be accomplished with a soap and water wash.

The process of patient decon should start with the removal of all clothing, jewelry and shoes. Then any visible contaminants should be removed from the patient. Dry particles can be gently brushed away, while liquids should be blotted away with absorbent cloth. This will reduce the chance of water reacting with the chemicals or increasing the absorption of a nonsoluble liquid. Care must be taken not to scrape the skin during this process. Soft tissue damage (burns, bruises, abrasions or lacerations) increases skin permeability and the absorption rate of the toxin.

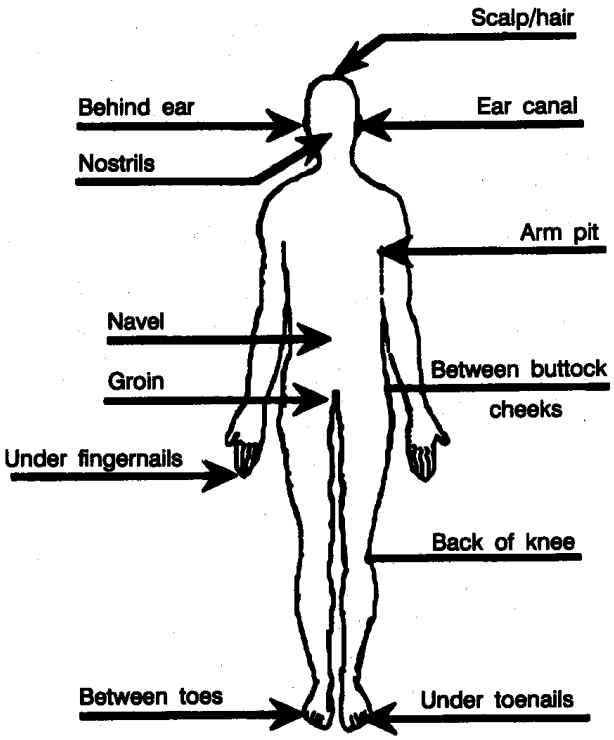
Soaps used for patient decon should be mild and non-abrasive. Tincture of green soap is desirable because of its slightly alkaline nature that approximates the body's pH level. Its alcohol base also helps to remove hydrocarbons and solvents from the skin. If green soap is not available, any mild liquid soap such as Dawn dishwashing detergent will work. Never use decon solutions on skin. The patient should be washed with soft sponges to reduce the chance of skin abrasion.

Water spray should be mild to avoid aggravating any soft tissue damage. The temperature should be warm—never hot. If cold water must be used, there is a risk of hypothermia. Try to contain the run-off as hazardous waste, but do not delay treatment in life-threatening situations if containment is not available. In these cases, try instead to avoid allowing run-off to enter drains or water sources.

Patient decon should begin at the head and then proceed to any areas where skin is damaged. Care must be taken not to flush contaminants into wounds. Carefully wash and rinse the wound area from the center out. After the wound area is clean, cover it with a water-occlusive dressing or plastic wrap to preclude any further contamination. Once all wound areas are clean, procedures can progress to other areas of the body. Ear/nose cavities should be irrigated, hair washed and fingernails cleaned. Special attention should be focused on opposing surface areas, such as the underarms and groin. Eyes should be flushed at the scene and irrigation continued during transport, preferably with saline.

Privacy is an important consideration in field decontamination activities. In order to obtain cooperation from the patient, steps to assure patient privacy must be undertaken. Tarps, salvage covers, sheets, blankets, and other such items may be used to construct privacy screens on-site. Remember, that both male and female decontamination areas may be needed.

Clothing must be provided to ambulatory patients following field decontamination activities. Disposable clothing, such as Tyvek(tm) coveralls, may be used for this purpose. If such clothing is not available, blankets, disposable sheets, etc., can be used. The Red Cross or Salvation Army may be able to assist in this task.



Frequently missed body zones during decontamination

Initial patient stabilization should be carried out simultaneously with decon. This will mandate that the person providing patient care is trained in the use of and provided with proper protective equipment. Under no circumstances should personnel be allowed to use protective equipment without proper training. If proper training and equipment are unavailable, arrangements should be made with the local fire department for a co-response to all possible chemical emergencies.

Under ideal circumstances, patients should be fully decontaminated prior to transport. In most cases, this will eliminate the chance of secondary contamination of response personnel. However, hazardous materials incidents are unlike many of our standard responses. Often, the incident will continue to escalate and can endanger an entire community. In such cases, total commitment cannot be focused on complete patient decontamination. Patient care is only one aspect of these incidents, and manpower may be limited. As a result, patient decon may be less than optimal.

CONSIDERATIONS FOR AMBULATORY DECONTAMINATION

- Remove any signs of contamination by scraping, sweeping or blotting the material away. Remember, that you must be protected from cross contamination.
- Have patient remove clothing rapidly but cautiously. Direct patient not to have outer surface of garments come in contact with their skin. Removal should be from top to bottom. (Patient's clothing could have absorbed most of the contaminant. Just think, normal clothing covers about 85% of the human body).
- Remove all external items from having contact with body. These items may include hearing aids, jewelry, watches, toupees, wigs and artificial limbs. Eye glasses, if needed by patient, must be washed prior to being worn.
- If the patient wore glasses or contact lenses, flush the eyes with large amounts of water.
- Gently wash face and hair with soap and lukewarm water, followed by a thorough rinse. Try not to have runoff contact any other part of the patient's body.
- Begin to decon other body surfaces starting from the neck down. Try to blot the skin instead of swabbing or wiping. Get into areas such as underneath the fingernails.
- Put patient into uncontaminated clothing.

CONSIDERATIONS FOR NON-AMBULATORY DECONTAMINATION

- Remove any signs of gross contamination. Remember, you must be protected from cross contamination.
- Cut away patient's clothing and remove all personal property. All property should be bagged, secured and clearly identified.
- Make sure your hands (the rescuer or health care provider) are decontaminated and thoroughly rinsed with water before removing contact lenses. Contact lenses should be removed to decrease the risk of cross contamination.
- Eyeglasses from patient must be decontaminated. Eyeglasses in metal frames can be decontaminated in a bath of solution for 5 minutes followed by a thorough rinsing. Eyeglasses in a composite or plastic frame should be secured in an impermeable bag for later decontamination.

- The victim's skin excluding the face should be blotted with the solution of 0.5% hypochlorite. Superficial wounds are flushed with a 0.5% hypochlorite solution and new dressings applied as needed. Splints are not removed but saturated to the skin with 0.5% solution. If the splint cannot be saturated it must be removed sufficiently so that everything under the splint can be saturated with a 0.5% hypochlorite solution.
- The victim should then be showered or otherwise washed with copious amounts of water, starting with the face and hands and then the rest of the body.
- Medical screening (triage) should now continue.
- Patient should receive new clothing (i.e., hospital scrubs) and continue to be observed for further signs of exposure.
- Each individual, having been processed through decontamination, should be marked and identified as such. This can be accomplished with a triage tag or by marking victim's forehead. During processing, each individual should receive a certificate indicating:
  —Description of decontamination actions taken;
  —Time decontamination was completed;
  —Time released from observation area; and
  —Any medical treatment performed in conjunction with decontamination.

A copy should also go to decontamination record management.

TRANSPORT OF CONTAMINATED PATIENTS

Other situations may necessitate the transport of patients before they are completely clean. Inclement weather can also be a major factor. Patient condition may require that only a gross decontamination be undertaken before rapid transport. Perhaps a more realistic approach is to attempt to get the patient as clean as possible (ACAP). In these cases, patient isolation principles should be instituted. Depending on the contaminant and the level of contamination, protective equipment may be necessary during transport. Keep ventilation to as high a level as weather conditions permit. Remember that airflow in the patient compartment of ambulances is usually minimal at best.

In extreme cases, the ambulance may need to be protected by covering surfaces with plastic and removing non-essential equipment prior to transport. Due to the slippery nature of wet plastic, cover the floor with a sheet or blanket. An alternative to covering the ambulance surfaces is to encapsulate the patient in blankets, sheets or plastic. Some response teams have excellent results using zip-front body bags. These allow for the rapid containment of a patient yet still provide quick access to the patient via the zipper. Obviously, the bag should only be zipped to the chest level. With highly absorptive contaminants, toxicity can actually be increased by the use of plastic or body bags. This can be reduced by placing a disposable blanket in the bag before the patient. The blanket will keep the plastic from touching the patient's skin. There are also commercially available products on the market to contain contaminated run off from a patient during transport.

It is important to note that many patients may come into the ED by private vehicle with no decon prior to the arrival. Thus, it is essential that all hospitals be able to provide decontamination and immediate treatment. Routine decon should be carried out if there is any question regarding contamination status. EDs are required to have a means to decon and contain run-off in order to be accredited by the Joint Commission on the Accreditation of Health Care Organizations. Some hospitals have fully contained decon rooms, while many others are starting to use portable containment tanks or special decon tables.

The possibility of secondary contamination from patients, response team members and equipment is a dangerous and real threat. Decontamination, correctly removing personal protective equipment and using site response zones can minimize cross-contamination to personnel and other areas. This module only provides general guidance on methods and techniques. The exact decon procedure must be determined after evaluating the factors specific to the incident.

Haz Mat Incident Team Members

PERSONNEL	RESPONSIBILITIES
Chemical Safety Officer (CSO):	Provides technical consultation/information for handling the incident. Overall management of non-medical aspects at the scene. A Haz Mat team member might be a possible backup.
Security:	Sets up decon area. Restricts access to ER. Directs traffic. Restricts access to news media.
Decontamination Team:	Decontaminates patient. Makes sure all equipment is properly decontaminated and disposed of. Provides for safe handling of all waste. Cleans up decon area when procedures are completed.
Medical Team:	Provides for treatment of patient.
Public Information Officer (PIO):	Meets with members of the news media. Provides all press releases.

Decontamination Area Set Up

The decontamination area is set up by both security and members of the decon team.

Security:

Marks off restricted area with barrier cones and warning tape to designated restricted area depending on hospital.

All personnel not associated with decontamination of the patients are to be restricted from the area by security.

Security directs all ambulances, rescue units, other transportation vehicles with CONTAMINATED PATIENTS to the decon area.

Decon Team:

Assists in setting up the decon containment pool and shower setup.

Prepares decontamination supplies, wash solutions, attaches hose to water supply.

Tests water quantity and quality before the patient arrives.

Determines if any additional supplies or materials are needed.

Decon team suits up and waits for patients.

Once patients are in the area, only properly protected decon members or medical staff, if in PPE, are permitted in the area.

Shutting Down the DECON Area

At the conclusion of the decon process, it is important that the decon area itself be decontaminated to prevent the spread of any contaminated material.

1. Clean up is to be done by **Decon Team** in **PPE**. Your local hazmat team should handle the clean up.
2. All solid waste that is contaminated is to be collected and placed in a "Contamination Bag." (Double lined plastic garbage bags will work.)

   If it is determined to be a hazard, it will be disposed of by a hazardous waste company. For WMD events, be careful to protect and secure evidence.
3. Waste water is to be held as follows:
   a. if it is determined not to be hazardous, it can be disposed of in the sewer system.
   b. if hazardous, the waste water must be sealed in drums and arrangements made for pick up by a hazardous waste disposal company.
   c. the **Chemical Safety Officer** will make these determinations and arrangements.

4. The entire decon area is to be straightened up and cleaned down.

5. All supplies and decon equipment is to be properly put away. Inventory is to be taken as to what is to be needed.

6. Haz Mat supplies are to be relocated to storage area.

Common Sense Techniques

When performing decontamination, the goals, as well as the tasks needed to accomplish these goals, should be kept simple. There are "common sense" techniques that could be used to help protect the health and safety of all personnel involved and to prevent the spread of the hazardous material. Some of the "common sense" techniques to be considered are the following:

1. Check your own hands and feet (both should be protected upon arrival at the incident) for any signs of contamination.
2. Observe each other. Do a complete visual check of other personnel for signs of contamination. If a substance is noted, decontamination procedures must be employed.
3. If you are unsure that any piece of protective clothing or equipment has been completely decontaminated, carefully remove articles and leave them behind to be properly collected. YOUR SAFETY COMES FIRST. EQUIPMENT CAN BE REPLACED.
4. While decontaminating, avoid direct contact with the contaminated item.

Hazardous Materials/WMD Incident Decontamination Equipment and Supplies

Many of these items are available in the hospital. It would be advisable to set up an area where these supplies can be stored so that they are readily available when an incident occurs.

PERSONNEL PROTECTION EQUIPMENT

Face Shield
Chemical goggles
Surgical gloves
Chemical protection suit with hood
Chemical resistant boots
Duct tape
ID badges

DECONTAMINATION SUPPLIES

Sheets (Disposable)
Surgical Scrub brushes
Cotton tip applicators
Sterile water (for irrigation)
Wraps
Wash cloths (Disposable)
Spray container for soap
Soap
Scrub suits (Disposable)—For redress of ambulatory patient

DECON EQUIPMENT

The following supplies will be needed to set up the decontamination area:

Long handled scrub brushes (for decontamination of suits)
Warning tape
Warning signs
Cones
Containment pools
Decontamination table
Plastic floor covering
Hazardous Material labels for waste containers
Garden hose
Nozzle
Hazardous Materials Bags/Garbage bags
Markers
Scissors
Buckets
Waste containers

Recommended Decontamination Supplies

1. **Patient Decontamination System** that provides for the medical treatment and decontamination of a patient. This system should include a means of collecting waste water.
2. **Protective Floor Covering** constructed of a non-skid chemically resistant material.
3. **Waste Container** with a dolly, lid and liner. All contaminated articles such as the patient's clothes, dressing and medical supplies should be placed in this container for proper disposal.
4. **Sample Collection Kit** that contains all the instructions and necessary supplies for collecting samples. Should be done by qualified individuals.
5. **Decontamination Kit** that contains the necessary procedures, as well as fluids and materials, for patient decontamination.
6. **Antidotes** for use in specific cases.

7. **Contamination Control Measures** used to control access to contaminated area thereby minimizing the spread of the contamination.
    - warning rope
    - warning signs
    - boundary cones
    - step-off pad

    Additional Supplies

    - hose with splash reducing spray nozzle
    - EMT scissors
    - tincture of green soap
    - waterproof drapes (i.e., Chucks)
    - Irri-jet
    - adhesive tape
    - towels
    - soft scrub brush

CHEMICAL WARFARE AGENT DECONTAMINATION

Few HAZMAT teams are equipped to provide the extensive level of decontamination called for in these situations. Residual contamination on the CPC components can cause injury long after the incident itself. Therefore, response teams need to consider how to safely dispose of the contaminated equipment.

In addition, commercial cleanup companies that typically clean up chemical spills may not be prepared to deal with these chemical agents. Specialized government teams would be involved in the cleanup and decontamination of any chemical warfare incident.

PREVENTING HOME CONTAMINATION

Contamination of worker's homes with hazardous chemicals and substances transported from the workplace is a world wide problem. So says the National Institute for Occupational Safety and Health (NIOSH), which has released a report on this issue.

NIOSH found that workers can inadvertently carry hazardous materials home from work on:

- clothes
- hair
- in their vehicles
- skin
- tools

The incidents of home contamination have resulted in a wide range of diseases and, in some cases, death among workers' families.

Here are some tips to prevent contamination at work and at home:

- Change clothes before going home and leave soiled clothing at work to be laundered by the employer
- Store street clothes in separate areas of the workplace to prevent their contamination
- Shower before leaving work
- Prohibit removal of toxic substances or contaminated items from the workplace
- Do not allow family members to visit the workplace
- Inform workers of the risk to family members and of preventive measures
- If contaminated clothing must be laundered at home, keep it separate from family laundry

WATCH OUT FOR YOURSELF AND EVERYONE ELSE!

Emergency Department ignition sources:

A. Most of the electrical/equipment switches.

B. Radios.

C. Electrical clocks.

D. Cigarettes, pipes, cigars, lighters, matches, etc.

E. Flashlights (including penlights) that are not intrinsically safe.

F. Portable radios that are not intrinsically safe.

G. Pagers that are not intrinsically safe.

H. Telemetry equipment that is not intrinsically safe.

I. Use of striking tools causing sparks.

J. Static electricity sparks *(nylon jackets act like batteries for static electricity)*

K. Battery operated hearing aids, watches, etc.

L. Defibrillators.

TOXICOLOGY

INTRODUCTION

• The toxicity of a substance is its ability to cause harmful effects. These effects can strike a single cell, a group of cells, an organ system, or the entire body. A toxic effect may be visible damage, or a decrease in performance or function measurable only by a test. All chemicals can cause harm. When only a very large amount of the chemical can cause damage, the chemical is considered to be relatively non-toxic. When a small amount can be harmful, the chemical is considered toxic.

• The toxicity of a substance depends on three factors:

(1) its chemical structure,

(2) the extent to which the substance is absorbed by the body,

(3) and the body's ability to detoxify the substance (change it into less toxic substances) and eliminate it from the body.

• The toxicity of a substance is the potential of that substance to cause harm, and is only one factoring determining whether a hazard exists. The hazard of a chemical is the practical likelihood that the chemical will cause harm. A chemical is determined to be a hazard depending on the following factors:

(1) toxicity: how much of the substance is required to cause harm,

(2) route of exposure: how the substance enters your body,

(4) dose: how much enters your body,

(5) duration: the length of time you are exposed,

(6) reaction and interaction: other substances you are exposed to,

(7) sensitivity: how your body reacts to the substance compared to others.

• Some chemicals are hazardous because of the risk of fire or explosion. These are important dangers, but are considered to be safety rather than toxic hazards. The factors of a toxic hazard are more fully explained below.

• The longer you are exposed to a chemical, the more likely you are to be affected by it. The dose is still important-at very low levels you may not experience any effects no matter how long you are exposed. At higher concentrations you may not be affected following a short-term exposure, but repeated exposure over time may cause harm. Chemical exposure which continues over a long period of time is often particularly hazardous because some chemicals can accumulate in the body or because the damage does not have a chance to be repaired. The combination of dose and duration is called the rate of exposure.

• The body has several systems, most importantly the liver, kidneys and lungs, that change chemicals to a less toxic form (detoxify) and eliminate them. If your rate of exposure to a chemical exceeds the rate at which you can eliminate it some of the chemical will accumulate in your body. For example, if you work with a chemical for eight hours each day, you have the rest of the day (16 hours) to eliminate it from your body before you are exposed again the next day. If your body can't eliminate all the chemical in 16 hours and you continue to be exposed, the amount in the body will accumulate each day you are exposed. Illness that affects the organs for detoxification and elimination, such as hepatitis (inflammation of the liver), can also decrease their ability to eliminate chemicals from the body.

• Accumulation does not continue indefinitely. There is a point where the amount in the body reaches a maximum and remains the same as long as your exposure remains the same. This point will be different for each chemical. Some chemicals, such as ammonia and formaldehyde, leave the body quickly and do not accumulate at all. Other chemicals are stored in the body for long periods. For instance, lead is stored in the bone, calcium is stored in the liver and kidneys. There are a few substances, such as asbestos fibers, that, once deposited, remain in the body forever.

• The effects of toxic substances may appear immediately or soon after exposure, or they may take many years to appear. Acute exposure is a single exposure or a few exposures. Acute effects are those which occur following acute exposures. Acute effects can occur immediately, or be delayed and occur days or weeks after exposure.

PREVENTION & CONTROL

• Prevention and control measures include, but are not limited to, the following:

(1) Elimination/substitution and process modification;

(2) Engineering controls;

(3) Administrative controls; and

(4) Use of personal protective equipment.

• In certain circumstances, personal protection of the individual employee is necessary. Personal protective devices should be regarded as being supplementary to substitution and engineering control and should not be used in preference to the latter because they do nothing to eliminate the hazard.

• Personal protective equipment must be appropriately selected, individually fitted and workers trained in their correct use and maintenance. The equipment must be regularly checked and maintained to ensure that the worker is being protected.

• Monitoring may be used for the evaluation of a hazard and for assessing the effectiveness of control measures. The design and implementation of a monitoring program should be carried out by, or in consultation with, a properly qualified person. Monitoring of the work environment involves the measurement of atmospheric contaminants at selected locations in the workplace (static, positional monitoring).

• Biological monitoring involves measurement of the concentration of a contaminant, its metabolites or other indicators in the tissues or body fluids of the worker. In some cases, biological monitoring may be required to supplement static or personal monitoring.
f. In the control of health hazards due to a specific contaminant, where it has been demonstrated that the exposure of the employee to the contaminant is approaching the relevant exposure standard, or where biological monitoring indicates that an unacceptable exposure is occurring, immediate action must be taken to reduce the health hazard and intensive monitoring should continue.

ROUTES OF ENTRY

• Injury can be caused by chemicals only if they reach sensitive parts of a person or other living organism at a sufficiently high concentration and for a sufficient length of time. Thus, injury depends upon the physicochemical properties of the potentially toxic substances, the exact nature of the exposure circumstances, and the health and developmental state of the person or organism at risk.

• Major routes of exposure are through the skin (topical), through the lungs (inhalation), or through the gastrointestinal tract (ingestion). In general, for exposure to any given concentration of a substance for a given time, inhalation is likely to cause more harm than ingestion which, in turn, will be more harmful than topical exposure faster.

a. Inhalation Route

Inhalation is the most significant route of entry by which harmful substances enter the human body at work. Toxic atmospheric contaminants may have local or systematic effects. Local effects harm only the part of the body they come in contact with, for example, inhalation of silica dust causing pneumoconiosis. Systemic effects, cause changes to the function of other organs, as in the case of inhaled particles that are soluble in the fluid of the tissues that line the lung, for example, lead and mercury fumes. Inhalation results in the introduction of toxic compounds into the respiratory system. Most of the compounds that are commonly inhaled are gases or vapors of volatile liquids; however, solids and liquids can be inhaled as dusts or aerosols. Inhalation of toxic agents generally results in a rapid and effective absorption of the compound.

When you inhale a toxic chemical, the dose you receive depends on four factors:

(1) The level (concentration) of chemical in the air;

(2) How hard (fast and deep) you are breathing, which depends on your degree of physical exertion;

(3) How much of the chemical that is inhaled stays in your lungs and is absorbed into your bloodstream; and

(4) How long the exposure lasts.

b. Absorption Route

Some atmospheric contaminants may be absorbed through the skin without any noticeable change to the skin, while others may cause serious damage to the skin itself. Ingestion is of relatively minor significance in occupational exposure to toxic materials.

(1) Skin contact exposure does not typically result in as rapid systemic dosage as Inhalation, although some chemicals are readily absorbed through the skin. Many organic compounds are lipid (fat) soluble and can therefore be rapidly absorbed through the skin. Some materials that come in contact with the eyes can also be absorbed. Ingestion is a less common route of exposure for emergency response personnel at hazardous materials incidents. However, incidental hand-to-mouth contact, smoking, and swallowing of saliva and mucus containing trapped airborne contaminants can cause exposure by this route. In addition, emergency medical personnel in both hospital or prehospital settings will see chemical exposures in patients who have ingested toxic substances as a result of accidental poisonings or suicide attempts.

(2) Many people do not realize that chemicals can penetrate healthy intact skin and so this fact should be emphasized.

c. Ingestion

Airborne particles breathed through the mouth or cleared by the cilia of the lungs will be ingested. Otherwise, ingestion of potentially toxic substances in the work, domestic, or natural environment is likely to be accidental and commonsense precautions should minimize this. The nature of the absorption processes following ingestion is discussed elsewhere. The importance of concentration and time of exposure has already been pointed out. It should be remembered that exposure may be continuous or repeated at intervals over a period of time; the consequences of different patterns of exposure to the same amount of a potentially toxic substance may vary considerably in their seriousness.

d. Injection

The injection of hazardous materials into the body sounds, at first, like a bad joke. Who in his right mind would inject themselves—especially when it is not required by a doctor? However, it can occur by stepping on a sharp object, or impaling yourself on or being cut by a sharp object while working at an incident site. It will happen before you even realize it has occurred and the reality of possibly being internally contaminated sinks in. The best precaution for this eventuality is to have protective clothing on, including steel shank and toed foot protection, and by strictly instituting and observing safe work habits.

EXPOSURE LIMITS

These limits are established by health and safety authorities to control exposure to hazardous substances. Exposure limits usually represent the maximum amount (concentration) of a chemical which can be present in the air without presenting a health hazard. However, exposure limits may not always be completely protective, for the following reasons:

- Although exposure limits are usually based on the best available information, this information, particularly for chronic (long-term) health effects, may be incomplete. Often we learn about chronic health effects only after workers have been exposed to a chemical for many years, and then as new information is learned, the exposure limits are changed.
- Exposure limits are set to protect most workers. However, there may be a few workers who will be affected by a chemical at levels below these limits (see "Sensitivity"). Employees performing extremely heavy physical exertion breathe in more air and more of a chemical, and so may absorb an excessive amount.
- Exposure limits do not take into account chemical interactions. When two or more chemicals in the workplace have the same health effects, industrial hygienists use a mathematical formula to adjust the exposure limits for those substances in that workplace.
- When toxic chemicals are present in the workplace, your exposure can be estimated by measuring the concentration of a given chemical in the air and the duration of exposure. This measurement is called air or environmental monitoring or sampling and is usually done by industrial hygienists, using various types of instruments. The air is collected from your breathing zone (the air around your nose and mouth) so that the concentrations measured will accurately reflect the concentration you are inhaling. The exposure levels calculated from this monitoring can then be compared to the Permissible Exposure Level for that chemical.

- Environmental monitoring is the most accurate way to determine your exposure to most chemicals. However, for chemicals that are absorbed by routes other than inhalation, such as through the skin and by ingestion, air monitoring may underestimate the amount of chemical you absorb. For these and some other chemicals, the levels of the chemical (or its breakdown products) in the body can sometimes be measured in the blood, urine or exhaled air. Such testing is called biological monitoring, and the results may give an estimate of the actual dose absorbed into the body. For one substance, lead, biological monitoring is required by law when air monitoring results are above a certain level. The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended the exposure limits for biological monitoring for a small number of chemicals. These are called Biological Exposure Indices (BEIs) and are published together with TLVs.
- If you smell a chemical, you are inhaling it. However, some chemicals can be smelled at levels well below those that are harmful, so that detecting an odor does not mean that you are inhaling harmful amounts. On the other hand, if you cannot smell a chemical, it may still be present. Some chemicals cannot be smelled even at levels that are harmful. The odor threshold is the lowest level of a chemical that can be smelled by most people. If a chemical's odor threshold is lower than the amount that is hazardous, the chemical is said to have good warning properties.
- If you or your co-workers experience symptoms known to be caused by a chemical during or its use, you may have been overexposed. Symptoms might include tears in your eyes; a burning sensation of skin, nose, or throat; a cough; dizziness or a headache.

OCCUPATIONAL EXPOSURE LIMITS

Occupational exposure limits exist to serve one main purpose: protect workers from excessive exposure to toxic chemicals in the workplace. They were designed for healthy adults, usually for an exposure duration of a day's workshift (8 hours). They were not meant to be used for protection of the public, since the general public includes:

- Sensitive groups such as the very young and very old, people with respiratory diseases and other illnesses, and people who are hypersensitive to some chemicals. Occupational exposure limits were also not designed to compare toxicity of chemicals, or to be the fine line between "safe" and "unsafe."
- The current definition has no exposure duration associated with it. Workers should not be in an IDLH environment for any length of time unless they are equipped and protected to be in that environment. They may be found in the NIOSH Pocket Guide to Chemical Hazards.

EXPOSURE LIMITS

The various occupational exposure limits found in the literature or in an MSDS are based primarily on time-weighted average limits, ceiling values, or ceiling concentration limits to which the worker can be exposed to without adverse effects. Examples of these are listed in (Table 8). These values were established to provide worker protection in occupational settings. Because the settings in which these values are appropriate are quite different than an uncontrolled spill site, it is difficult to interpret how these values should be used by emergency medical personnel dealing with a hazardous materials incident. At best, TLV, PEL, IDLH, and REL.

a. Lethal Concentration 50 (LC_{50})

Is the concentration of a material in air that on the basis of respiratory exposure in laboratory tests is expected to kill 50% of test animals when administered as a single exposure (usually 1 hour). A dose of 3,000-3,800 mg/kg tetrachloroethylene is lethal to 50% of rats that received the compound orally; however, only 6.4 to 10 mg/kg of sodium cyanide is required to produce the same effect. Therefore, compounds with low LD_{50} values are more acutely toxic than substances with larger LD_{50} values. The LD_{50} values that appear in an MSDS or in the literature must be used with caution by emergency medical personnel. These values are an index of only one type of response and give no indication of the ability of the compound to cause nonlethal, adverse or chronic effects. Furthermore, LD_{50} values typically come from experimental animal studies. Because of the anatomical and physiological differences between animals and humans, it is difficult to compare the effects seen in experimental animal studies to the effects expected in humans exposed to hazardous materials in the field. Therefore, emergency medical personnel should remember that the LD_{50} and LC_{50} values are only useful for comparing the relative toxicity of compounds and should only be used to determine if one chemical is more toxic than another.

b. Lethal Dose 50 (LD_{50})

Toxicity information is often expressed as the dose of the compound that causes an effect in a percentage of the exposed subjects, which are mostly experimental animals. These dose-response terms are often found in Material Safety Data Sheets (MSDS) and other sources of health information. One dose-response term that is commonly used is the lethal dose of 50 (LD_{50}), the dose which is lethal to 50% of an animal population from exposure by any route other than inhalation when given all in one dose. Another similar term is the lethal concentration 50 (LC_{50}), which is the concentration of a material in air that on the basis of respiratory exposure in laboratory tests is expected to kill 50% of a group of test animals when administered as a single exposure (usually 1 hour). Exhibit I lists a number of chemicals that may be encountered in dealing with hazardous materials incidents, and the reported acute LD_{50} values of these compounds when they are administered orally to rats.

From Exhibit I, it can be seen that a dose of 3,000-3,800 mg/kg tetrachloroethylene is lethal to 50% of rats that received the compound orally; however, only 6.4 to 10 mg/kg of sodium cyanide is required to produce the same effect. Therefore, compounds with low LD_{50} values are more acutely toxic than substances with larger LD_{50} values.

The LD_{50} values that appear in an MSDS or in the literature must be used with caution by emergency medical personnel. These values are an index of only one type of response and give no indication of the ability of the compound to cause nonlethal, adverse or chronic effects. Furthermore, LD_{50} values typically come from experimental animal studies. Because of the anatomical and physiological differences between animals and humans, it is difficult to compare the effects seen in experimental animal studies to the effects expected in humans exposed to hazardous materials in the field. Therefore, emergency medical personnel should remember that the LD_{50} and LC_{50} values are only useful for comparing the relative toxicity of compounds and should only be used to determine if one chemical is more toxic than another.

Exhibit I
Acute LD_{50} Values for Representative Chemicals When Administered Orally to Rats

Chemical	Acute Oral LD_{50} (mg/kg)*
Sodium cyanide	6.4-10
Pentachlorophenol	50-230
Chlordane	83-560
Lindane	88-91
Toulene	2,600-7,000
Tetrachloroethylene	3,000-3,800

*Milligrams of the compound administered per kilogram body weight of the experimental animal.

Responses to toxic chemicals may differ among individuals because of the physiological variability that is present in the human population. For example, an individual may be more likely to experience an adverse health effect after exposure to a toxic chemical because of a reduced ability to metabolize that compound. The presence of preexisting medical conditions can also increase one's susceptibility to toxic chemicals. Respiratory distress in patients or workers with asthma may be triggered by exposure to toxic chemicals at lower concentrations than might be expected to produce the same effect in individuals without respiratory disease. Factors such as age, personal habits (i.e., smoking, diet), previous exposure to toxic chemicals, and medications may also increase one's sensitivity to toxic chemicals. Therefore, exposure to concentrations of toxic compounds that would not be expected to result in the development of a toxic response.

The values listed in Exhibit II were established to provide worker protection in occupational settings. Because the settings in which these values are appropriate are quite different than an uncontrolled spill site, it is difficult to interpret how these values should be used by emergency medical personnel dealing with a hazardous materials incident. At best, TLV, PEL, IDLH, and REL values can be used as a benchmark for determining relative toxicity, and perhaps assist in selecting appropriate levels of Personal Protective Equipment (PPE). Furthermore, these occupational exposure limits are only useful if the appropriate instrumentation is available for measuring the levels of toxic chemicals in the air at the chemical spill site. Of the above occupational exposure limit values, only the OSHA values are regulatory limits. The ACGIH values are for guidance only and are not regulatory limits. In addition, the ACGIH limits have certain caveats that may or may not affect the usefulness of the values. Some of these conditions are individual susceptibility or aggravation of a preexisting condition. Nevertheless, all emergency medical personnel responsible for the management of chemically contaminated patients should be familiar with these concepts because they will be encountered in various documents dealing with patient care or the selection of PPE.

This brief discussion highlights some fundamental concepts of toxicology. Emergency medical personnel responsible for managing chemically contaminated patients are encouraged to obtain further training in recognizing and treating health effects related to chemical exposures. Also, a list of general references in toxicology is provided at the end of this section that will allow emergency medical personnel to undertake a more in-depth examination of the principles of toxicology.

Acute LD_{50} Values for Representative Chemicals When Administered Orally to Rats

Chemical	Acute Oral LD_{50} (mg/kg)*
Sodium cyanide	6.4-10
Pentachlorophenol	50-230
Chlordane	83-560
Lindane	88-91
Toluene	2,600-7,000
Tetrachloroethylene	3,000-3,800

*Milligrams of the compound administered per kilogram body weight of the experimental animal.

How Much (Or How Little) is 1 Part Per Million

To give you an example of how small an amount we are talking about: 1 drop of liquid from an eye dropper is 1 millionth of a tank of gas in an average compact car (13 gallon tank), or 1/8 inch is 1 millionth of a mile.

A milligram is 1/1000 of a gram and there are 28.3 grams in an ounce.

c. Immediately Dangerous to Life and Health (IDLH)

The Immediately Dangerous to Life and Health (IDLH) guidelines are not occupational exposure limits similar to the TLV or PEL. They were developed in the 1970s by NIOSH to guide respirator selection. A recent revision in 1994 updated many of the IDLH concentrations, and changed the IDLH definition. The current (1994) definition of the IDLH is a condition "that poses a threat of exposure to airborne contaminants when that exposure is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from such an enviroment." For concentrations above the IDLH, a self-contained breathing apparatus (SCBA) is required. Below that level, air-purifying respirators may be used, if appropriate.

- Notice that unlike the previous definition of the IDLH, which incorporated a 30-minute time period, the new definition does not have an exposure duration associated with it. If you are in an IDLH condition, you need to get out of there immediately! IDLHs are based on analysis of human and animal studies and were developed for fewer than 100 substances. The rationale for each standard is not clearly stated. Unlike the TLVs, no formal updating mechanism exists for IDLH.

d. Ceiling

A concentration that should not be exceeded at any time. Note that both TWA and STEL permit limited excursion if, in the end, the average is below the exposure limit. The ceiling value, however, may not be exceeded.

e. TLV-STEL

(1) Some substances in the TLV booklet have a short-term exposure limit (STEL). The STEL is a 15-minute exposure limit that should not be exceeded even if the 8-hour TLV remains within the limit. Such limits were assigned to substances exerting toxic effects even over a short period of time. Where a STEL limit is not available (but is believed to be justified), the TLV committee recommends using a limit three times as high as the TLV for a 15-minute exposure.

(2) These values can be used as a benchmark for determining relative toxicity, and perhaps assist in selecting appropriate levels of Personal Protective Equipment (PPE). Furthermore, these occupational exposure limits are only useful if the appropriate instrumentation is available for measuring the levels of toxic chemicals in the air at the chemical spill site. Of the above occupational exposure limit values, only the OSHA values are regulatory limits.

(3) Of the above occupational exposure limit values, only OSHA values are regulatory limits. The ACGIH values are for guidance only and are not regulatory limits. In addition, the ACGIH limits have certain caveats that may or may not affect the usefulness of the values. Some of these conditions are individual susceptibility or aggravation of a preexisting condition. Nevertheless, all emergency medical personnel responsible for the management of chemically contaminated patients should be familiar with these concepts because they will be encountered in various documents dealing with patient care or the selection of PPE.

f. TWA (Time-Weighted Average)

Unless otherwise mentioned, it is the concentration of contaminants over an 8-hour period. It is determined by sampling the breathing zone of the worker for 8 hours.

g. Permissible Exposure Limits

PELs are values set by OSHA. These limits are a legal requirement for occupational exposures, and exceeding them is a violation of the law, for which fines may be imposed. Most of the PELs are based on older TLVs, and some have STELs as well. OSHA's PELs are published yearly in the Code of Federal Regulations (CFR). A recent PEL update was invalidated by a judge in the summer of 1993. Therefore, many of the currently valid PELs date back to 1968. Many states, however, have established their own occupational standards. These are as strict or stricter than OSHA's, were not affected by the recent ruling, and are enforced as before.

h. Threshold Limit values

1. Threshold Limit Values-Time Weighted Average (TLV-TWA) are exposure limits recommended by a committee of the American Conference of Governmental Industrial Hygienists (ACGIH), and are published yearly in a little booklet. All the substances in the TLV booklet have an 8-hour Time-Weighted Average (TWA) exposure which is the level to which workers may be exposed for an 8-hour work shift without suffering an adverse effect. The rationale for setting the limits is explained in a separate publication, "Documentation of the Threshold Limit Values and Biological Exposure Indices" (ACGIH, Cincinnati, Ohio). The TLVs are derived from human studies including epidemiological research and exposure studies with volunteers, occupational accidents, animal studies, and "similar structure analysis" (based on the assumption that compounds similar in structure are similar in toxicity). The TLV committee meets several times every year, and the TLVs are updated regularly.
2. TLV-TWA is meant to regulate exposure over an 8-hour period. Don't extrapolate to shorter periods of time. Don't assume that if a certain limit applies for 8 hours, then eight times that limit may be applied if the exposure lasts for only 1 hour. It simply doesn't work that way. Therefore, the 8-hour limits may not be very useful for spill response, where exposure durations are usually much shorter than 8 hours.

i. Recommended Exposure Limits (RELs)

The Recommended Exposure Limits (RELs) were developed by the National Institute of Occupational Safety and Health (NIOSH). Those standards are similar to TLVs in the way that they were derived, but are often stricter. The RELs are published in the *NIOSH Pocket Guide to Chemical Hazards* which is updated every few years, and in other NIOSH publications.

Occupational Exposure Limits

Value	Abbreviation	Definition
Threshold Limit Value (3 Types) (ACGIH)*	TLV	Refers to airborne concentrations of substances and represents conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect.
1) Threshold Limit Value—Time-Weighted Average (ACGIH)*	TLV-TWA	The time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek, to which nearly all wokers may be repeatedly exposed, day after day, without adverse effect.
2) Threshold Limit Value—Short-Term Exposure Limit (ACGIH)*	TLV-STEL	The concentration to which workers can be exposed continuously for a short period of time without suffering from: 1) irritation, 2) chronic or irreversible tissue damage, or 3) narcosis of sufficient degree to increase the likeihood of accidental injury, impair self-rescue or materially reduce work efficiency, and provided that the daily TLV-TWA is not exceeded.
3) Threshold Limit Value—Ceiling (ACGIH)*	TLV-C	The concentration that should not be exceeded during any part of the working exposure.
Permissible Exposure Limit (OSHA)**	PEL	Same as TLV-TWA.
Immediately Dangerous To Life and Health (OSHA)**	IDLH	A maximum concentration (in air) from which one could escape within 30 minutes without any escape-impairing symptoms or any irreversible health effects.
Recommended Exposure Limit (NIOSH)***	REL	Highest allowable airborne concentration is not expected to injure a worker; expressed as a ceiling limit or time-weighted average for an 8- or 10-hour work day.

*American Conference of Governmental Industrial Hygienists

**Occupational Safety and Health Administration

***National Institute for Occupational Safety and Health

TREATMENT PROTOCOLS

FOR THE HAZARDOUS MATERIALS (INDUSTRIAL CHEMICALS) CONTAMINATED PATIENT

Patient Management

When a hospital receives a call that a patient exposed to hazardous materials is to be received, *a planned course of action* should be implemented. Steps in a protocol must be practiced before a hazardous materials emergency occurs. All staff members of an emergency department should know their responsibilities and how to perform them. All required equipment should be immediately available or readily accessed.

Individuals receiving a potential hazardous materials call should obtain as much information as possible. A checklist should be developed and made available for all telephone or radio communication centers. Information that will aid in initiating appropriate actions includes:

- Type and nature of incident
- Caller's telephone number
- Number of patients
- Signs/symptoms being experienced by the patients
- Nature of injuries
- Name of chemical(s) involved
- Extent of patient decontamination in the field
- Estimated time of arrival

After the above information is received, a predesignated resource center (e.g., New Jersey poison control center, ATSDR) should be contacted for information regarding definitive care procedures, which should include decontamination methods that need to be performed. Communications should be kept open with on-site response personnel to obtain as much advance information as possible.

If incident notification comes from other than usual emergency communication channels, the call should be verified before a hazardous materials response plan is initiated. EMS personnel should be notified of any special approach or entrance to the emergency department and also advised not to bring the patient into the emergency department until the patient has been assessed and accepted by the emergency department.

Often patients contaminated by hazardous materials may be brought into the emergency department unannounced or not through regular EMS channels. This could be an ambulatory patient or a patient transported by private vehicle. The ideal response to this is to call a fire department which is properly trained and equipped or a hazmat team to come to the hospital and set up a decontamination area outside the ambulance entrance. In any event, these patients should be isolated from other patients and assessed and decontaminated as soon as possible.

Emergency Department Preparation

Every member of the emergency department should be familiar with the hospital's hazardous materials response plan and be required to participate in scheduled drills. Preparation for arrival of a contaminated patient should include: notification of all services involved, preparation of a Decontamination Area, and suiting up of the Decontamination Team.

Emergency Department Mobilization

The person receiving a call of incoming victims should notify the Nursing Supervisor who will in turn notify appropriate personnel according to the hospital's response plan. The hospital operator should be instructed to notify security and maintenance, and the nurse on duty should contact the predesignated resource center.

Patient Arrival

The emergency physician-in-charge or an emergency department nurse should meet the ambulance upon arrival and assess the condition of the patients as well as the degree of contamination. Personnel should keep in mind that the actual contamination may be (or become) a life-threatening condition. Triage procedures should also be initiated at this point, if necessary. During initial patient survey and stabilization, contamination reduction should simultaneously be performed. This consists of cutting away or otherwise removing all suspected contaminated clothing, including jewelry and watches, and brushing or wiping off any contamination. Care should be taken to protect any open wounds from contamination. **Emergency department personnel must make every effort to avoid contact with any potentially hazardous substance, and avoid cross contamination.**

Ideally, decontamination should be performed before patient transport; however, field decontamination facilities are limited and emergency department personnel should consider that all hazardous materials patients need decontamination. **If a patient's clothing was not removed at the incident site, it should be removed outside the ambulance but before entry into the emergency department.** This will reduce further exposure to the patient and lessen the extent of contamination introduced to the emergency department. Contaminated clothing should be double bagged in plastic bags, sealed, and labeled. The decontamination team should bring the prepared stretcher to the ambulance, transfer the patient, and take him or her directly to the decontamination area along the predesignated route.

Priority should be given to the ABC (Airway, Breathing, and Circulation) and simultaneous contamination reduction. Once life-threatening matters have been addressed, emergency department personnel can then direct attention to thorough decontamination and secondary patient assessment. Identification of hazardous materials involved can be simultaneously performed by other personnel. It is important to remember that appropriate person protective clothing must be worn until personnel are no longer in danger. Therefore, the sooner the patient becomes decontaminated the sooner personnel may reduce protective measures.

Effective decontamination consists of making the patient As Clean As Possible (ACAP). This means that the contamination has been reduced to a level that is no longer a threat to the patient or the responder. The recorder notes on a diagram of the body the areas found by the physician to be contaminated.

Considerations for Patient Treatment

Primary goals for emergency department personnel in handling a contaminated patient include termination of exposure to the patient, patient stabilization, and patient treatment—while not jeopardizing the safety of emergency department personnel. Termination of exposure can best be accomplished by removing the patient from the area of exposure and by removing contaminants from the patient. Basically, a contaminated patient is like any other and may be treated as such except that staff must protect themselves and others from dangers due to contamination.

Personnel must first address life-threatening issues and then decontamination and supportive measures. Priority should be given to the ABC with simultaneous contamination reduction. Once life-threatening matters have been addressed, emergency department personnel can then direct attention to thorough decontamination, secondary patient assessment, and identification of materials involved. It is important to remember that appropriate personal protective clothing must be worn until personnel are no longer in danger. Therefore, the sooner the patient becomes decontaminated the sooner personnel may reduce protective measures or downgrade the level of protection. Primary and secondary surveys should be completed as conditions allow. In treating patients, personnel should consider the chemical-specific information received from the hazardous materials response resources. In multiple patient situations, proper triage procedures should be implemented. Presenting signs and symptoms should be treated as appropriate and when conditions allow. The sooner a patient has been decontaminated the sooner he or she can be treated like a “normal” patient. Orders of the designated poison control center and attending

physician should be administered. Invasive procedures, such as IVs or intubation, should be performed only for life-threatening conditions, until decontamination is performed. These procedures may create a direct route for introducing the hazardous material into the patient. The patient should be frequently re-assessed because many hazardous materials have latent physiological effects.

Note: The attending staff must remember that since exposure to some substances can result in seri ous delayed effects, sustained observation and monitoring are required.

Patient Management Under Mass Casualty Conditions Involving Hazardous Chemicals

Basic medical procedures in a large-scale hazardous materials incident are not substantially different from life-saving measures in other mass casualty disasters. Primary attention to the ABC continues to have first priority with decontamination performed at the same time. A chemical disaster may overwhelm any one hospital, particularly if it occurs along with another disaster such as an earthquake. Hospitals need to preplan what they will do if they are overwhelmed with hazmat patients.

There are, however, several important differences in disasters involving hazardous materials. Such differences include the need for the effective decontamination of exposed patients and response personnel, and the need for effective safety measures to protect response personnel. Training in the appropriate procedures to be followed is essential for potential responders to a hazardous materials incident involving mass casualties. Standard principles of triage apply in chemical disasters, except in exposures to very toxic substances. The patient, injured or not, must be decontaminated before being transported to the emergency department to protect EMS and emergency department staff.

Exposure to Toxic Chemicals

Exposure to toxic chemicals is frequently the primary concern at Haz Mat incidents. Many scenes contain a variety of chemicals in solid, liquid or gaseous form.

Contaminants can enter the body through the four pathways consisting of inhalation, injection, ingestion and absorption. Inhalation and absorption are considered to be the most common routes of entry and protective equipment is available to minimize the risk. Remember, that the first responder with operational training will be limited in both operation and types of protective equipment.

We must always be aware of the ingestion route and how contaminants enter the body in this manner. The act of smoking, drinking, eating or rubbing ones face with the hands may introduce contaminants to the body through ingestion. For this reason, one must always be aware of the presence of contaminants at a scene and the need for safety, proper protection, and decontamination.

Exposure and Dose

Exposure is the act of coming in contact with a contaminant.

Dose is the amount of contaminant taken into the body.

Warning Properties are the physical characteristics of a chemical identified by the senses.

Carcinogen—substance that causes cancer.

Mutagen—a substance that causes mutations. A mutation is a change in the genetic material in a body cell. Mutations can lead to birth defects, miscarriages, or cancer.

Teratogen—a substance that causes birth defects by damaging a fetus.
A contaminant could cause damage at the point of contact (local) or at another point in the body (systemic). Effects may be immediate **(acute)**, in the case of a large concentrated dose, or more commonly, delayed **(chronic)** and will not be detected for over a period of time. These are known as acute and chronic doses.

LOCALIZED EFFECTS

Toxic Inhalation Injuries

Chemical properties that affect site & types of injuries

PHYSICAL PROPERTIES

- Concentration
- Duration
- Dose
- Solubility

PHYSICAL FORMS

- Gases and Vapors
- Mists
- Fumes

CHEMICAL PROPERTIES

- Reactivity
- pH
- Direct Acting Chemicals
- Indirect Acting Chemicals

Patients baseline condition contributes to injury severity

- Rate and pattern of respirations
- Hx of lung disease
- Pts Age
- Nutrition Status
- Thyroid Function

Upper Airway Injuries (laryngeal edema/spasm)

Most often caused by water soluble/highly reactive chemicals

- Acids (chlorine & hydrogen chloride)
- Alkalis (ammonia)
- Vesicants (formaldehyde)

Lower Airway Injuries (bronchiolar obstruction)

Most often caused by chemical not very water soluble, inhaled in high concentrations, with prolonged exposure to dust/fumes.

- Clinically presents as wheezing/respiratory distress

Bronchospasm & Asthma

- Allergic (sensitized to industrial chemicals)
- Non-allergic (irritants)
- Attacks can be immediate or delayed up to 6 hours
- Anaphylaxis

Pulmonary Edema

- Caused by damage to lung vs. cardiogenic edema

  Pulmonary capillaries become more permeable

  Alveolar lining cells

  Surfactant disrupted
- May develop rapidly or delayed even days later

Chemical Injuries to the Eye

Effects of Acid and Alkali

- Most common cause of serious chemical eye injuries
- Alkali generally more severe than acids
- Acids
    - Coagulation necrosis
    - Coagulum serves as a barrier to deep penetration
    - At pH <2, acids penetrate easy and deep
- Alkali
    - Liquefaction necrosis
    - Creates no barrier, facilitating penetration
- Acids/Alkalis can cause vessel thrombosis
- Ischemic necrosis of the cornea can lead to eye loss
- Severity cannot be judged until 48+ hours
- Alkalis progress for weeks to months

Management of Acid and Alkali Eye Exposures

- Prompt and continuous irrigation with water
    - Acids for at least 15 mins
    - Alkali for at least 20 mins
- Remove by washing foreign bodies/solid particles
- Remove contact lens
- Be gentle in handling the eye

Solvents
Detergents and surfactants
Lacrimators
Delayed corneal injuries

Chemical Injuries to the Skin

Mechanisms of chemical skin injuries:

- Thermal
    - Hot
    - Cold
- Mechanical
- Ischemic
- Irritation
    - Contact dermatitis
    - Vesiculation
- Chemical burns and corrosion
    - Coagulation necrosis
    - Liquefaction necrosis
    - Corrosion

Determinants of severity:

- Nature and reactivity of chemical
- Concentration of the chemical
- Integrity of the skin
- Duration of the exposure
    - Remove chemical by prompt/adequate washing
    - Water should not be used on chemical such as water reactive metals.

Complications:

- Do not underestimate severity
- Severity difficult to judge for first 48 hrs.
- Aggressive fluid replacement (dehydration)
- Infectious complications

Hydrogen fluoride and hydrofluoric acid:

- Sequestration of calcium
- Seizures and cardiac dysrhythmias
- Need to replace calcium in tissues

  Calcium gel/subcutaneously

Phosphorus:

- Spontaneously ignites at temps. >86 degrees F
- Vapors if burning are toxic if inhaled
- Treatment: extinguish burning

  Submerge burning part in cool water

  Keep moist and cool

Water-reactive metals (alkaline metals):

- React with water yielding heat and strong alkali
- Do NOT wash these chemicals with water
- Extinguish with class-D fire extinguisher or sand
- During transport, cover metal with cooking or mineral to prevent air contact and burning

Phenol:

- Strong corrosive agents that are weak acids
- Water washing contributes to absorption and systemic intoxication
- Wipe skin with polyethylene glycol first

Systemic Effects

Systemic routes of exposure:

- Inhalation
    - Most common
    - Water and fat soluble chemicals
    - Reactive chemical may be poorly absorbed
- Percutaneous absorption
    - Fat soluble chemical are better absorbed
    - Other determinants
        - Concentration
        - Duration of exposure
        - Size of exposed surface
        - Skin moisture and temperature
        - Skin integrity
    - Eye exposure
- Ingestion
- Injection

Onset of action:

- Immediate for chemicals which require no transformation to be toxic
- Delayed onset of action with chemicals that are transformed into a toxic form
- Some chemicals have both an immediate and delayed toxic effect

Asphyxiants:

- Injury to organs that require large amounts of oxygen
    - Central Nervous System
    - Kidneys
    - Liver
- Chemical induced
    - Inhibition of cellular respiration by enzyme poisons
        - Intracellular hypoxia
    - Production of abnormal hemoglobin
        - Carboxyhemoglobin
        - Methemoglobin
    - Acute hemolysis
    - Interference of ventilation
        - Heavier than air gases

Central Nervous System depressants:

- Many chemicals have anesthetic or narcotic effects
    - Usually fat soluble chemicals
    - Act on nerve cell membranes
- Clinical effects (mild/moderate/severe)
- Effects similar to common medical conditions

Management:

- Airway/Oxygen
- Cardiac monitoring

Cardiac sensitizers:

- Chemicals can sensitize the heart to the effects of catecholamines released by the body potentially resulting in V-Fib and cardiac arrest.
- V-Fib and cardiac arrest can result from sensitization and catecholamine release

Management:

- Keep victims at rest
- Cardiac monitoring
- Consider reducing doses of catecholamines (when indicated)

Neurotoxic insecticides:

- Activity of organophosphates and carbamates
  - Inhibit cholinesterase
  - Excessive activity of parasympathetic nervous system
- Clinical effects
  - Bradycardia leading to heart block
  - Chest tightness and wheezing
  - Constricted pupils
  - Weakness, twitching, muscle tremors, cramps
  - SLUDGE
  - Seizures

Management:

- Airway/Oxygen
- Antidotes/atropine in large dosages

Electrolyte disorders:

- Sequestration agents (hydrogen fluoride, oxalic acid, phosphorus) bind calcium

  Clinical effects of hypocalcemia

  - Twitching, muscle spasms, seizures
  - Cardiac dysrhythmias and cardiac arrest

  Management

  - Suspect problem from history of exposure
  - Measurement of calcium in blood
  - Replacement of calcium by IV

ACUTE (IMMEDIATE) VS CHRONIC (DELAYED) EFFECTS

Important factors to consider when determining the toxicity of a material are the relationships between concentration, exposure time and the threshold sensitivity of the person exposed.

Generally, a serious exposure refers to a large, single dose received over a short period of time and an immediate response occurs **(acute)**.

A serious exposure may result from a small, single dose over a short period of time and there is no **immediate** effect. This small dose may exceed the threshold sensitivity of the individual causing a serious **delayed** effect **(chronic)**. The classic example of this is cancer.

Doses from several small exposures over a period of time **(chronic exposure)**, causing no immediate effect may also result in a **delayed** effect. This cumulative effect may be serious or minor **(chronic)**.

Examples of Adverse Health Effects from Exposure to Toxic Chemicals

	Target Organ	**Example of Causative**	**Health Effect**	
Toxic End Point	**Systems**	**Agent**	**Acute**	**Chronic**
Carcinogenicity	Multiple Sites	Benzene	Dermatitis Tightness in Chest	Aleukemia Myeloblastic leukemia
Hepatotoxicity	Liver	Carbon Tetrachloride	Vomiting Vessication Dizziness	Liver Necrosis Fatty liver
Neurotoxicity	Nervous System	Lead	Nausea Vomiting Abdominal Pain	Wrist Drop IQ Deficits Encephalopathy
Nephrotoxicity	Kidney	Cadmium	Vomiting Diarrhea Chest Pain	Kidney Damage Anemia

Heat Stress

Heat stress can occur very rapidly—within as little as 15 minutes. It can pose a far greater danger to worker health than chemical exposure. In its early stages, heat stress can cause rashes, cramps, discomfort and drowsiness, resulting in impaired functional ability. Continued heat stress can lead to heat cramps, heat exhaustion, heat stroke, and even death.

Personnel exhibiting the following symptoms require immediate medical attention.

Treatment of Heat Stress

Initial treatment for heat stress includes removing the victim to a cool area, at the very least out of the sun. Heavy clothing should be removed and fluids administered.

Heat stroke is a life threatening emergency and Advanced Life Support (ALS) treatment is required as soon as possible. The same procedures used for heat stress should be followed until ALS care is available. Rapid cooling is essential for a victim with heat stroke.

Frostbite

Frostbite usually occurs on the face or the extremities. Signs and symptoms include pain followed by numbness and white chalky appearance. Use of metal tools or working on metal surfaces which are cold exacerbate the problem.

Personnel experiencing frostbite should be given immediate medical attention.

Treatment of Frostbite

A person with frostbite or hypothermia should be taken to a warm area indoors.

The frostbitten area should be covered with a soft cloth and gradually warmed. It **should not be rubbed**. Rubbing will further damage the frozen tissue. Frostbite is a serious injury which, if improperly treated, may result in loss of the affected body part.

MEDICAL PROTOCOLS

NOTE: FOR ADVICE ON CLINICAL MANAGEMENT CALL THE NEW JERSEY POISON CONTROL CENTER (800) 962-1253

TREATMENT PROTOCOLS

FOR THE HAZMAT CONTAMINATED PATIENT

ACIDS & ACID MISTS
(NOT Including Hydrofluoric Acid)

FORMS:

Gas, liquid (variable concentrations), mixtures with water, and aerosolized dusts.

BACKGROUND:

Acids act as direct irritants and corrosive agents to moist mucous membranes, and to intact skin to a lesser extent. Generally, these substances have very good warning properties: even fairly low airborne concentrations of acids produce rapid onset of eye, nose and throat irritation. Higher concentrations can produce cough, stridor, wheezing, chemical pneumonia or non-cardiogenic pulmonary edema. Occasionally, pulmonary edema may be delayed for several hours, especially with low-solubility gases such as nitrogen oxides. Ingestion of acids can result in severe injury to the upper airway, esophagus and stomach.

POTENTIAL FOR SECONDARY CONTAMINATION:

Small amounts of acid mists can be trapped in clothing after an overwhelming exposure but are not usually sufficient to create a hazard for health care personnel away from the scene. However, clothing which has become soaked with concentrated acid may be corrosive to rescuers. Once the victim has been stripped and flushed with water, there is no significant risk of secondary contamination.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol if indicated, and if not previously decontaminated. Health care personnel should don gloves, gowns, goggles, and other protective clothing until decontamination is completed. Activation of decontamination protocol is probably not needed for acid exposures unless the victim's clothing has been soaked with acid liquid.
2. Evaluate ABC's (airway, breathing, and circulation).
3. O_2 by mask. Intubate if patient manifests severe respiratory distress from pulmonary edema or upper airway swelling. Obtain arterial blood gases and chest x-ray if respiratory distress is present. Severe upper airway edema may necessitate cricothyroidotomy or tracheostomy.

4. Irrigate the eyes copiously with saline or water for at least 15 to 20 minutes if eye irritation is present.
5. Remove and double-bag clothing if not already done. Wash skin copiously with water.
6. If respiratory distress is present or anticipated, admit and observe 24 to 48 hours for possible delayed onset pulmonary edema. Bronchodilators may be helpful.
7. Examine eyes using slit-lamp and/or fluorescein strips, if corneal injury is suspected.
8. If a significant ingestion occurred, consider endoscopy to evaluate the esophagus and stomach.
9. Advise patient that full recovery is generally the rule, but cases of chronic airway disease have been reported following severe exposures. Advise and arrange for follow-up in case victim begins to experience respiratory distress. After exposure to oxides of nitrogen, sudden severe relapse may occur two to three weeks later.

AMMONIA (LIQUID AND GAS)

FORMS:

Gas (anhydrous) and liquid (aqueous solutions, variable concentrations).
NOTE: liquified compressed gas may produce cryogenic (freezing) hazard as it is released into the atmosphere.

BACKGROUND:

Ammonia (NH_3) is a direct irritant and alkaline corrosive agent to moist mucous membranes and, to a lesser extent, to intact skin. Ammonia has very good warning properties. Even fairly low airborne concentrations produce rapid onset of eye, nose and throat irritation. Higher concentrations can produce cough, stridor, wheezing, chemical pneumonitis or non-cardiogenic pulmonary edema. The onset of pulmonary edema is usually rapid but may occasionally be delayed for 12-24 hours. Ingestion of concentrated ammonia solutions (e.g., >5%) may cause serious corrosive injury to the esophagus and stomach and poses an aspiration risk.

POTENTIAL FOR SECONDARY CONTAMINATION:

Small amounts of ammonia vapor can be trapped in clothing after an overwhelming exposure but are not usually sufficient to create a hazard for health care personnel away from the scene. However, clothing which has become soaked with concentrated liquid ammonia may be corrosive to rescuers. Once the victim has been stripped and flushed with water, there is no significant risk of secondary contamination.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol for liquid exposure (and if not previously decontaminated). Health care personnel should don gloves, gowns, goggles or protective clothing until decontamination is completed.
2. Evaluate and support ABC's (airway, breathing, and circulation).

3. O_2 by mask. Intubate if patient manifests severe respiratory distress from pulmonary edema or upper airway swelling. Watch for signs of airway closure and laryngeal edema, such s hoarseness, stridor or retractions. Obtain arterial blood gases and chest x-ray if respiratory distress is present. Severe upper airway edema may necessitate cricothyroidotomy or tracheostomy.
4. Irrigate the eyes copiously with saline or water for at least 15 to 20 minutes if eye irritation is present.
5. Remove and double-bag clothing if not already done. Wash skin copiously with water.
6. Cardiac monitor; 12-lead EKG.
7. If severe respiratory distress is present, admit and observe for 24 hours for delayed-onset pulmonary edema.
8. Examine eyes using slit-lamp and/or fluorescein strips, if corneal injury is suspected.
9. Advise that full recovery is generally the rule, but cases of chronic airway disease have been reported following severe exposures.

ARSINE GAS

FORMS:

Gas may be generated in metal ore processing and electronic component manufacturing.

BACKGROUND:

Arsine (AsH_3) is an extremely toxic and nearly odorless gas (it has a slight odor of garlic). It is used widely in the microelectronics industry and occasionally occurs as a by-product in metallurgy and pesticide manufacturing. Arsine's effects are quite distinct from other arsenic compounds; even in very small quantities, inhaled arsine produces acute hemolysis (rupture of red blood cells), which can result in cardiac decompensation due to anemia, or renal failure due to massive kidney deposition of hemoglobin. Symptoms may be delayed for 2-24 hours, and include weakness, abdominal and flank pain, brown urine, and jaundice. Massive acute exposure appears capable of causing immediate death by an unknown mechanism.

POTENTIAL FOR SECONDARY CONTAMINATION:

Very small amounts of arsine can be trapped in a victim's clothing after an overwhelming exposure but are not usually sufficient to create a hazard for health care personnel away from the scene.

MANAGEMENT IN THE HOSPITAL:

1. Evaluate and support ABC's (airway, breathing, and circulation).
2. Provide O_2 by mask.
3. Monitor cardiac rhythm; obtain 12-lead EKG.
4. Laboratory Tests: Perform urine dipstick for occult blood and hemoglobin. Send for CBC, plasma free hemoglobin (PFHgb), urine hemoglobin, electrolytes, BUN and/or creatinine, bilirubin, blood type and screen, and other laboratory tests as appropriate. Urinary arsenic levels may be elevated for a few weeks after exposure.

5. If there is evidence of acute hemolysis, alkalinize urine with sodium bicarbonate, 50-100 mEq in or added to 1 (one) liter of 5% dextrose or 1/2 NS administered IV at a rate to maintain urine output at 2-3 cc/kg/hr. Consider furosemide or mannitol. Follow electrolytes, BUN, creatinine and fluid status closely because renal failure may result in acute fluid overload.
6. If PFHgb exceeds 1.5 gm/dl, there has been a significant rapid drop in hematocrit (e.g., from 40 to 30 without other explanation) or there are other indications of intravascular hemolysis (severe abdominal pain, jaundice, shock), consider exchange transfusion after consultation with a medical toxicologist. Prepare for dialysis in the event of renal failure. Shock may occur and should be treated appropriately.

NOTE: BAL and other chelating agents are not effective for arsine exposure. Arsine does not produce the classical symptoms of arsenic poisoning.

CARBON MONOXIDE

FORMS:

Gas.

BACKGROUND:

Carbon monoxide (CO) is a colorless, odorless gas. It is a common product of combustion of any organic material and is a major toxic component in cases of smoke inhalation. Carbon monoxide causes poisoning by interfering with the binding of oxygen to hemoglobin in the blood, myoglobin in heart and muscle tissue, and by interfering with oxygen utilization in the cell. Symptoms of progessively worse exposure include, in order, headache, dizziness, giddiness, tinnitus, nausea, muscle weakness, chest pain, dyspnea, syncope, seizures, and coma. **Cherry-red skin coloration is not commonly seen (except post-mortem)** and should not be relied upon for diagnosis. The half-life of CO in the blood is from 5 to 9 hours when the victim is breathing room air, compared to 60-90 minutes when breathing 100% oxygen.

POTENTIAL FOR SECONDARY CONTAMINATION:

Very small amounts of CO can be trapped in victim's clothing after an overwhelming exposure, but are not usually sufficient to create a hazard for health care personnel away from the scene.

MANAGEMENT IN THE HOSPITAL:

1. Evaluate and support ABC's (airway, breathing, and circulation).
2. Provide 100% O_2 by a tight-fitting mask, preferably with oxygen reservoir.
3. Monitor cardiac rhythm, and obtain 12-lead EKG. Watch for ischemic changes.
   NOTE: Carbon Monoxide poisoning will cause a falsely elevated O_2 saturation (SPO_2) and may give a false sense of security to the health care provider that the patient is not hypoxic.
4. Laboratory tests—Send for carboxyhemoglobin level (COHb), arterial blood gases, Hct, electrolytes, and other tests as appropriate.
5. Treat cerebral edema with fluid restriction, hyperventilation, and/or mannitol.

6. Admit to the hospital if any of the following are present:
   a. Mental status changes are present or were present.
   b. COHb >25%.
   c. COHb >15% in a patient with coronary disease, or current symptoms suggestive of coronary disease.
   d. Any EKG change thought to be acute, particularly ST segment depression, regardless of COHb level.
   e. Metabolic acidosis or disordered thermoregulation.
   f. Patient is pregnant and symptomatic or has COHb >10%.
7. A hyperbaric chamber may be helpful in pregnant patients, those with an altered level of consciousness, or the patient that does not rapidly respond to 100% O_2. Consultation with a medical toxicologist is advised. Speed in instituting therapy is very important, and anticipation of hyperbaric oxygen treatment should not delay intubation and the delivery of 100% O_2. In cases of severe exposure, delayed 100% oxygen treatment may increase the risk of permanent brain damage or prolonged (many months) convalescence, although this is not definitely established in the medical literature.

CHLORINE GAS

FORMS:

Gas (anhydrous) or liquid (aqueous chlorine usually in the form of hypochlorite, variable concentrations). The liquid hypochlorite solutions are very unstable and react with acids to release chlorine gas. **NOTE: liquified compressed gas may produce cryogenic (freezing) hazard as it is released into the atmosphere.**

BACKGROUND:

Chlorine is a highly irritating gas which rapidly forms hydrochloric acid after contact with moist mucous membranes in the upper airway and in the lungs. Symptoms occur rapidly and provide good warning properties for exposure. Low concentrations produce eye, noise and throat irritation. Higher concentrations produce cough, wheezing, choking, chemical pneumonitis, or pulmonary edema. Ingestion of concentrated hypochlorite solutions can cause serious corrosive esophageal or stomach injury.

POTENTIAL FOR SECONDARY CONTAMINATION:

Small amounts of chlorine gas can be trapped in clothing after an overwhelming exposure but are not usually sufficient to create a hazard for health care personnel away from the scene. However, clothing which has become soaked with concentrated hypochlorite solution may be corrosive to rescuers. Once the victim has been stripped and flushed with water, there is no significant risk of secondary contamination.

MANAGEMENT IN THE HOSPITAL:

1. Evaluate and support ABC's (airway, breathing, and circulation).
2. Provide O_2 by mask. Intubation may be required for severe respiratory distress.
3. Irrigate eyes copiously with water or saline if eye irritation is present.
4. Monitor cardiac rhythm if clinically indicated.
5. Obtain chest x-ray, arterial blood gases. Obtain other laboratory tests as appropriate.
6. Observe 6-12 hours for delayed-onset pulmonary edema for symptomatic patients.
7. Advise that full recovery is generally expected, but may take several months. Cases of chronic airways disease have been reported following severe exposure.

CYANIDE

FORMS:

Gas (hydrogen cyanide), liquid (solutions of cyanide salts), and solid (cyanide salts). Hydrogen cyanide gas may be formed when acid is added to a cyanide salt or a nitrile.

BACKGROUND:

Cyanide (CN) is an extremely toxic compound which is widely used in industry in a variety of forms (gas, liquid, solid). CN gas (HCN) is a major toxic component in cases of smoke inhalation. CN produces toxicity by interfering with cellular oxygen utilization. Symptoms and signs include headache, dizziness, vomiting, tachypnea, tachycardia, and coma. There may be a distinctive odor ("bitter almonds") on the victim's clothing or breath. Death can occur within minutes of exposure. If exposure is by inhalation of CN gas, peak toxic effects are seen within minutes, but after ingestion of a CN salt, effects may be delayed until the CN is absorbed from the stomach.

POTENTIAL FOR SECONDARY CONTAMINATION:

If the exposure was by inhalation of HCN gas, even though there may be small amounts of gas trapped in clothing after an overwhelming exposure, this is not usually sufficient to create a hazard for health care personnel away from the scene. The risk of secondary contamination to rescuers is greater if there are large amounts of liquid or solid material on the victim's clothing or skin.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol (unless decontaminated before arrival); medical personnel to don gloves, gown, and goggles until decontamination is completed.
2. Evaluate and support ABC's (airway, breathing, and circulation).
3. Administer O_2 by mask or endotracheal tube. Reducing the potential for chemical exposure from any form by mouth-to-mouth resuscitation, including use of pocket one-way valve mouth-to-mouth resuscitation, should be carefully considered.
4. If not decontaminated before arrival, remove and double-bag clothing; wash skin with soap and water if dermal exposure is suspected. Of note, liquid chlorine bleach will decontaminate contaminated equipment and should be used when laundering contaminated clothing.
5. Monitor cardiac rhythm, and obtain 12-lead EKG.

6. Laboratory Tests: Serum thiocyanate, blood cyanide, CBC, electrolytes, arterial blood gases, lactate, and other laboratory tests as appropriate. **Appropriate treatment should not be delayed.**
7. Respiratory Exposure: If the patient arrives asymptomatic, probably no treatment will be needed. If the patient is ill, begin (8) below.

   Ingestion or Skin Contact: ER staff should be ready to initiate therapy immediately, regardless of the presence of symptoms on arrival. Be prepared to intubate quickly.
8. In the symptomatic patient with a significant exposure, administer treatment in the following order (use Cyanide Antidote Kit):
   a. Amyl nitrite—break pearls into gauze sponge and hold under patient's nose or Ambu intake valve for 15 to 30 seconds/minute, until sodium nitrite solution is ready.
   b. Sodium nitrate ($NaNO_2$) 3% IV solution:

      Adults: 10 ml at 2.5 to 5 ml/minute, or 0.35 ml/kg.

      Children: 0.2 ml/kg, not to exceed 10 ml.
   c. Through the same IV line in (B) above, give sodium thiosulfate ($NA_2S_2O_2$), 25%.

      Adults: 12.5 gm (50 cc of 25% solution).

      Children: 1.6 to 1.8 ml/kg of a 25% solution.
   d. Repeat antidote at 50% of initial dose if symptoms persist after 20 minutes. If symptoms worsen after treatment, consider nitrite toxicity causing methemoglobinemia greater than 25%.

   WARNING: Methemoglobinemia may be particularly dangerous in children. Also, be extremely cautious in treating with nitrite if there has also been carbon monoxide exposure. The same dose of nitrites can cause excessive methemoglobinemia. Normal therapeutic amounts of methemoglobinemia in the face of carbon monoxide poisoning can be a problem and should be closely watched.
9. If ingestion is suspected, perform gastric lavage and administer activated charcoal.
10. Admit and observe 24-48 hours. Watch for metabolic acidosis; treat with sodium bicarbonate if needed. Watch for hypotension; treat with fluid and pressors if needed. Hyperbaric oxygen may be helpful in displacing cyanide ion from cellular enzymes.
11. Hyperbaric oxygen may be indicated for victims of smoke inhalation who have had both cyanide and carbon monoxide exposures and who do not respond to treatment.

HYDROFLUORIC ACID

FORMS:

Gas, liquid (variable concentrations), and fluoride salts in the presence of acids may generate toxic quantities of hydrogen fluoride.

BACKGROUND:

Hydrofluoric acid (HF) produces toxicity quite distinct from other mineral acids. The "acid" moiety (hydrogen ion) is relatively unimportant, producing little burning sensation on initial contact. In contrast, the highly toxic fluoride ion has the ability to penetrate tissue and produce indolent ulceration or bony destruction. Solutions of greater than 10-20% are particularly destructive. Inhalation may cause eye, nose and throat irritation, cough, tracheobronchitis, and delayed onset pulmonary edema. Ingestion may produce severe corrosive burns of the esophagus and stomach. Systemic absorption of fluoride (i.e., from a burn or after ingestion) may result in severe hypocalcemia, hypomagnesemia, and hyperkalemia, resulting in tetany and cardiac arrest.

POTENTIAL FOR SECONDARY CONTAMINATION:

Until the soaked clothing has been removed and the affected body part has been flushed, there is some hazard to treating health care personnel, depending on the concentration. Following basic decontamination, there is usually no significant risk of secondary contamination.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol unless the victim was decontaminated before arrival. Medical personnel should don gown, gloves, and goggles until decontamination is completed.
2. Evaluate and support ABC's (airway, breathing, and circulation).
3. If not decontaminated before arrival, remove and double-bag clothing; wash skin with soap and water. Continue to irrigate copiously. HF will continue to leach from exposed skin and tissues for at least 15 minutes. **DILUTION IS BETTER THAN NEUTRALIZATION IN THE FIRST CRITICAL MINUTES!!**

4. Evaluate the extent of skin exposure and following irrigation, administer additional treatment:
   a. If the HF concentration was >20% or is unknown or exposure was prolonged:
      (1) Infiltrate affected area with 10% calcium gluconate, using a 25-30 gauge needle and multiple injections of 0.5 ml per square centimeter, taking care to prevent damaging underlying structures. Pain should resolve with the injection.
      DO NOT USE calcium chloride, which is extremely painful and may further injure tissues.
         a. Repeat after several hours if pain recurs.
         b. Avoid local anesthetics, which may mask clinical findings but do give morphine IV for pain. HF burns can be intensly painful and pain is out of proportion to physical findings.
         c. Limit injection to 0.5 ml per phalanx.
      (2) Remove blisters and debride underlying tissues, as these may contain HF.
      (3) Remove nails if evidence of periungual or ungual tissue involvement. Use a regional anesthesia proximal to the site of injury.
   b. If the HF concentration was <20% and the duration of exposure was brief (less than a few minutes), administer calcium gluconate gel (2.5%) or 30%-50% magnesium sulfate solution by massage or soaks to affected area for at least 30 minutes. This treatment binds HF as the insoluble CaF_2 or MgF_2 salts. If pain persist, go to step 4A. If more than an hour or two has elapsed since the time of initial decontamination topical soaks are probably of marginal benefit.
   c. For extremity burns where topical agents are ineffective in relieving pain or as alternative modality to fingernail removal, an intra arterial injection of calcium gluconate may be effective. Consultation with a medical toxicologist should be obtained.
   d. 2.5-5 cc of a 2.5% calcium gluconate solution may be administered via nebilizer for inhalational exposure.
5. Ingestion—treat as severely corrosive agent. Consider endoscopy to evaluate extent of damage. Consider lavage with calcium containing solution.
6. Additional steps for all patients:
   a. Admit to burn unit or intensive care unit if the total extent of the burn is greater than 2%-3% BSA, or if there is significant respiratory distress.
   b. Observe for hypocalcemia, hyperkalemia or other systemic effects if HF concentration was greater than 20% or if there was prolonged contact with a significant percent of BSA (2% to 3% or more).

c. Obtain 12 lead EKG and provide continuous cardiac monitoring to look for QT prolongation which may be early sign of hypocalcemia. Consider giving IV calcium 1 gram (10 ml) 10% calcium gluconate or calcium chloride prophylactically for higher concentration exposures to greater than 5% BSA or for dilute exposures to larger surface areas.

7. Establish baseline and serial electrolytes, Ca, Mg. Follow blood gases in the event of respiratory exposure.

HYDROGEN SULFIDE, SULFIDES & MERCAPTANS

FORMS:

Gas (hydrogen sulfide, methyl & short-chain alkyl mercaptans) and liquid (other mercaptans).

BACKGROUND:

Hydrogen sulfide (H_2S) is a highly toxic gas with an odor of rotten eggs at low concentrations. At higher concentrations olfactory fatigue rapidly occurs, making odor a poor warning symptom of danger. Mercaptans are sulfur-containing, highly malodorous compounds. All of these compounds are direct irritants, but their major toxicity is due to interference with cellular oxygen utilization. Low-level exposures produce irritation of the eyes, nose and throat, cough, headache, nausea, and dizziness. Higher exposures can cause syncope, seizures, coma, tracheobronchitis, and pulmonary edema (which may occur up to 48-72 hours later). Death may occur within minutes of acute massive exposure.

POTENTIAL FOR SECONDARY CONTAMINATION:

Small amounts of H_2S can be trapped in clothing after an overwhelming exposure but are not usually sufficient to create a hazard for health care personnel away from the scene. However, clothing which has become soaked with concentrated liquid sulfide solutions or mercaptans may pose a risk to rescuers. Once the victim has been stripped and flushed with water, there is no significant risk of secondary contamination. Sulfides are highly water soluble.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol, unless decontaminated before arrival; medical personnel should don gown, gloves, and goggles until decontamination is completed. Odor will provide a warning about the need for decontamination. *A WELL-VENTILATED AREA WILL BE VERY HELPFUL.*
2. Evaluate and support ABC's (airway, breathing, and circulation).
3. Provide O_2 by mask, if the victim has respiratory distress or altered mental status.

4. If not decontaminated before arrival, remove and double-bag clothing; wash skin with soap and water if clothing had been wet. Irrigate the eyes with saline or water if there is eye irritation.
5. Monitor cardiac rhythm and obtain 12-lead EKG. Tachyarrhythmias may occur.
6. Laboratory tests: CBC, electrolytes, creatinine and/or BUN, blood gases, liver function studies, urinalysis, and other laboratory tests as appropriate.
7. If the patient is severely affected with coma or cardiovascular collapse, administer treatment in the following order, using the Cyanide Antidote Kit:
   a. Amyl nitrite: break pearls into gauze sponge and hold under patient's nose or Ambu intake valve for 15 to 30 seconds/minute, until sodium nitrite solution is ready.
   b. Sodium nitrate ($NaNO_2$) 3% IV solution:

      Adults: 10 ml at 2.5 to 5 ml/minute or 0.35 ml/kg.

      Children: 0.2 ml/kg, not to exceed 10 ml.
   c. Sodium thiosulfate is not effective for H_2S exposure.
   d. Repeat antidote at 50% of initial dose if symptoms persist after 20 minutes. If symptoms worsen after treatment, consider the possibility of nitrite toxicity causing methemoglobinemia greater than 25%.
   e. Continue O_2 for at least 2 hours afterward.
8. If symptoms are mild, including eye and throat irritation, headache, nausea, or dizziness, supportive care will suffice.
9. In severe cases, observe for delayed onset pulmonary edema, liver toxicity, or hematuria/proteinuria.
10. Hyperbaric oxygen may be helpful, although the medical literature on this point is still somewhat controversial.

NITROGEN-CONTAINING COMPOUNDS AND OTHER CHEMICALS CAUSING METHEMOGLOBINEMIA

FORMS:

Gas, liquid and solid. Substances tend to be brown or yellow in color, especially when impure.

BACKGROUND:

A wide variety of nitrogen-containing compounds, including anilines, aryl amines, and aromatic nitrogen compounds, are potent oxidizing agents which can produce methemoglobinemia. Methemoglobin is unable to transport oxygen. Patients with methemoglobinemia greater than 15% will appear grey or cyanotic, and their blood will appear chocolate brown. With higher levels signs and symptoms of hypoxia are present, including headache, dizziness, nausea, dyspnea, syncope, seizures, and coma. These methemoglobin-forming compounds may also produce direct systemic effects such as skin or respiratory irritation, vasodilation, hypotension, headache, nausea and CNS depression. Many of the liquid compounds are highly volatile and may be inhaled, and many are well-absorbed through the skin.

POTENTIAL FOR SECONDARY CONTAMINATION:

Depending on the individual compound, these agents may pose a significant health hazard for rescuers and health care personnel. Many are well-absorbed through intact skin. Simple water washing may be insufficient to remove oil compounds.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol, unless decontaminated before arrival or until decontamination is completed. Health care personnel should don neoprene gloves *(do not use canvas, cotton, rubber, or latex gloves)* and protective clothing. If the victim's clothing is wet, or dusty material is present, respiratory protection is appropriate.
2. Evaluate and support ABC's (airway, breathing, and circulation).

3. If not decontaminated before arrival, remove and double-bag contaminated clothing, and wash skin with soap and water.
4. O_2 by mask.
5. Monitor cardiac monitor; obtain 12-lead EKG.
6. The following laboratory tests should be performed: Send methemoglobin (MetHb) level STAT *(MUST be done within 1 hour to be accurate and helpful).* **Note that waiting for this result may jeopardize the patient.** Chocolate brown blood suggests that significant methemoglobinemia is present.
7. Additional laboratory tests: CBC, electrolytes, BUN and/or creatinine. Obtain other laboratory tests as appropriate.
8. Administer methylene blue if MetHb >40% or if MetHb is between 25% and 40% AND the patient is symptomatic. Worrisome symptoms include severe headache, disorientation, tachypnea, tachycardia, or other indications of cardiovascular decompensation.
   a. Give methylene blue, 1% solution (10 mg/ml), 1 to 2 mg/kg IV over 5–10 minutes (equivalent to 0.1 to 0.2 mL/kg, or total of about 5 to 20 mL). Observe for elevated BP, nausea, disorientation.
   b. Repeat in 30–60 minutes if cyanosis or severe symptoms persist.
   c. The total dose of methylene blue should not exceed 7 mg/kg.
   d. Continue Oxygen for at least 2 hours following methylene blue administration.

   WARNING: Methylene blue is itself toxic, and may produce disorientation, elevated BP, nausea, diarrhea, and delayed hemolytic anemia.
9. Once the patient is stable, rule out other causes for methemoglobinemia (drug use, G-6-PD deficiency, hemoglobinopathies).

NOTE: Methylene blue will cause urine to turn blue-green.

PESTICIDES—CARBAMATES

FORMS:

Liquid (usually in solution with xylene or other organic solvent), solid (wettable power). May be inhaled in an aerosol form or as a component of smoke.

BACKGROUND:

Carbamate pesticides are widely used in home gardening and commercial agriculture. Like organophosphates, they inhibit the enzyme cholinesterase, resulting in buildup of excessive acetylcholine. Unlike organophosphates, the inhibition of cholinesterase is transient and self-limited. Symptoms and signs include hypersalivation, sweating, bronchospasm, abdominal cramps, diarrhea, muscle weakness, small pupils, twitching and seizures. Death is due to respiratory muscle paralysis. Nonspecific symptoms such as upper airway irritation, dizziness, nausea and headache after inhalation exposure may be due to the solvent vehicle (e.g., xylene) and not due to cholinesterase inhibition. Potential toxicity of the solvent vehicle should always be considered.

POTENTIAL FOR SECONDARY CONTAMINATION:

Many carbamates are well-absorbed through intact skin, and thus may pose a serious hazard to rescuers or health care personnel. Simple water washing may be insufficient to remove oily compounds.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol, unless decontaminated before arrival. Hospital personnel should don gloves, gowns, and occasionally goggles, until decontamination has been completed.
2. Evaluate and support ABC's (airway, breathing, and circulation). Intubation is occasionally required.
3. Provide O_2 by mask or endotracheal tube. Obtain and follow blood gases if respiratory distress is present.
4. If not decontaminated before arrival, remove clothing; wash skin with soap and water. Shampoo hair and scalp, clean under nails, and in ears.

5. Monitor cardiac rhythm; watch for either bradycardia or tachycardia.
6. Laboratory Tests: RBC and plasma cholinesterase. Levels should be sent.
7. Treat with atropine when appropriate.
   (DO NOT TREAT IF ASYMPTOMATIC!)
   a. In general, atropine is needed only if at least one or more of the following are present:
      - Alteration in mental status, confusion, seizures.
      - Nausea, vomiting, diarrhea, or abdominal cramps.
      - Pupillary constriction.
      - Salivation.
      - Diaphoresis.
      - Respiratory distress, wheezing, pulmonary edema.
      - Significant arrhythmia (particularly bradycardia).

   b. Atropine treatment:
      (1) Adults: Give atropine sulfate 0.5 to 1.0 mg initially, followed by 2 to 4 mg IV; repeat 2 to 4 mg every 3-10 minutes as needed until signs of parasympathetic (muscarinic) toxicity are controlled, the mouth is dry, and airway is clear. At this point, the pupils will generally be dilated, although not invariably, and the skin will be warm and dry).
      (2) Children: Atropine sulfate, .02–.05 mg/kg IV, as above, with a minimum dose of .1 mg.

   c. **Pralidoxime (2-PAM) is not recommended for carbamate poisoning.**
8. If ingestion is suspected, initiate gastric lavage and administer activated charcoal.
9. Other general treatment guidelines:
   a. Watch for signs of atropine toxicity. Note that disorientation, uncooperative behavior, hallucinations, blurred vision, tachycardia, fever, and convulsions may be due to atropine itself.
   b. Respiratory depression, due in part to respiratory muscle paralysis, is the usual cause of death, and is not completely prevented by atropine.
   c. Remove pulmonary secretions by suction if necessary.
   d. If seizures are not responsive to atropine, treat with diazepam 5 to 10 mg by slow IV push, Phenobarbital, or phenytoin, may be used.

10. Significant poisoning does not occur unless cholinesterase levels are depressed at least 30% below the individual's baseline levels, although this level may be within the laboratory range for normal values. In severe poisoning, levels are depressed 90% or more. It may be necessary to recheck cholinesterase levels in a few days to determine the individual's normal baseline cholinesterase levels. Note that some other conditions, including chronic cocaine use, can depress cholinesterase levels. Cholinesterase levels are helpful in documenting exposure to carbamate pesticides, although they may be less helpful for emergency management.

PESTICIDES—ORGANOPHOSPHATES

FORMS:

Liquid (usually solution with xylene or other organic solvent), solid (wettable power). May be inhaled in an aerosol form or as a component of smoke.

BACKGROUND:

Organophosphate pesticides are widely used in home gardening and commercial agriculture. A variety of products are available, with widely varying potencies. They inhibit the enzyme cholinesterase, resulting in buildup of excessive acetylcholine. Symptoms and signs include hypersalivation, sweating, bronchospasm, abdominal cramps, diarrhea, muscle weakness, small pupils, twitching and seizures. Death is due to respiratory muscle paralysis. If the victim survives the acute poisoning, they may develop delayed onset peripheral neuropathy. Nonspecific symptoms such as upper respiratory irritation, dizziness, nausea and headache after inhalation exposure may be due to the solvent vehicle (e.g., xylene) and not due to cholinesterase inhibition. Potential toxicity of the solvent vehicle should always be considered.

POTENTIAL FOR SECONDARY CONTAMINATION:

Many organophosphates are well-absorbed through intact skin, and thus may pose a serious hazard to rescuers or health care personnel. Simple water washing may be insufficient to remove oily compounds.

MANAGEMENT IN THE HOSPITAL:

1. Activate decontamination protocol, unless decontaminated before arrival. Medical personnel should don gown, gloves, and goggles. Respiratory protection will be needed if dust or high vapor levels are present.
2. Evaluate and support ABC's (airway, breathing, and circulation).
3. Administer O_2 by mask or endotracheal tube.
4. If not decontaminated before arrival, remove and double-bag clothing; wash skin with soap and water. Mucous membranes may require vigorous lavage. Wash ear canals and under fingernails.

5. Monitor cardiac rhythm; watch for either bradycardia or tachycardia, or for ventricular ectopy.
6. Laboratory Tests: RBC and plasma cholinesterase levels. Follow arterial blood gases if the patient has respiratory distress or altered mental status.
7. Treat with atropine when appropriate.
   (DO NOT TREAT IF ASYMPTOMATIC!)
   a. In general, atropine is needed if one or more of the following are present:
      - Altered mental status or seizures.
      - Nausea, vomiting, diarrhea, or abdominal cramps.
      - Pupillary constriction.
      - Salivation.
      - Diaphoresis.
      - Respiratory distress, wheezing, pulmonary edema.
      - Significant arrhythmia (particularly bradycardia).
      - Other medical conditions may cause these symptoms and should be ruled out.

   b. Atropine treatment:
      (1) Adults: Atropine sulfate, 0.5 to 1.0 mg initially, followed by 2 to 4 mg IV; repeat 2 to 4 mg every 3-10 minutes as needed until signs of parasympathetic (muscarinic) toxicity are controlled, the mouth is dry, and airway is clear. At this point, the pupils will generally be dilated, although not invariably, and the skin will be warm and dry).
      (2) Children: Atropine sulfate, .02–.05 mg/kg IV as above, minimum dose of .1 mg.

   c. Pralidoxime (2-PAM, or Protopam) is best given early (but may be of value in the first few days) and will reactive some cholinesterase activity. Treatment with pralidoxime is most helpful for control of nicotinic symptoms, particularly generalized muscle weakness or fasiculations which may contribute to respiratory paralysis. Dose is 1 gm for adults and 25-50 mg/kg for children, given IV over 5-10 minutes. In severe cases this may be repeated in 1 hour. Repeat treatment may be needed (can give 1-3 gm IV every 6 to 8 hours), especially for agents with prolonged effects like fenethion.
8. If symptoms have not appeared and ingestion is suspected, initiate gastric lavage and administer activated charcoal.
9. Other general treatment guidelines:
   a. Watch for signs of atropine toxicity. Note that disorientation, uncooperative behavior, hallucinations, blurred vision, tachycardia, fever, and convulsions may be due to atropine itself.

b. Respiratory depression, due in part to respiratory muscle paralysis, is the usual cause of death, and is not completely prevented by atropine.

c. Remove pulmonary secretions by suction if necessary.

d. If seizures are not responsive to atropine, treat with diazepam, 5 to 10 mg by slow IV push. Phenobarbital may also be used.

10. Significant poisoning does not occur unless cholinesterase levels are depressed at least 30% below the individual's baseline levels, although this level may be within the laboratory range for normal values. In severe poisoning, levels are depressed 90% or more. It may be necessary to recheck cholinesterase levels in 3 to 6 weeks to determine the individual's normal baseline cholinesterase levels. Note that some other conditions, including chronic cocaine use, can depress cholinesterase levels. Cholinesterase levels are helpful in documenting exposure to organophosphate pesticides, although they may be less helpful for emergency management

PHOSPINE

FORMS:

Gas. Extremely flammable, may ignite spontaneously in air or explode on contact with flame.

BACKGROUND:

Phosphine (PH_3) is an extremely toxic gas with a nauseating odor, used in the electronics industry, as an insect fumigant, and occasionally occurring as a by-product in manufacturing. Its toxicology is not well understood, but it appears to affect the central nervous system, the heart, lungs, and liver. Symptoms following low to moderate exposure include nausea, vomiting, headache, cough, dizziness, diarrhea, myalgias, fever, and chills. Severe exposure may produce syncope, stupor, coma, pulmonary edema and death. Unlike arsine, phosphine does not produce hemolysis.

POTENTIAL FOR SECONDARY CONTAMINATION:

Very small amounts of phosphine can be trapped in a victim's clothing after an overwhelming exposure, but are not usually sufficient to create a hazard for health care personnel away from the scene.

MANAGEMENT IN THE HOSPITAL:

1. Evaluate and support ABC's (airway, breathing, and circulation).
2. Administer O_2 by mask, if the patient has respiratory distress.
3. Monitor cardiac rhythm; obtain 12-lead EKG. Following severe exposures, rule out myocardial infarction.
4. Laboratory Tests: Hct, electrolytes, BUN and/or creatinine, liver enzymes, Ca, Mg, and blood gases. Other laboratory tests should be requested.
5. Treat pulmonary edema. Symptoms may not develop for 72 hours.
6. Liver damage may become evident 2-3 days later.

TREATMENT PROTOCOLS

FOR THE CHEMICAL AGENT CONTAMINATED PATIENT

Known Chemical Warfare Agents

NERVE AGENTS

Tabun (GA)—cholinesterase inhibitor

Sarin (GB)—cholinesterase inhibitor

Soman (GD)—cholinesterase inhibitor

GP—cholinesterase inhibitor

Thickened Soman (GD or VR-55)—cholinesterase inhibitor (U.S.S.R.)

Thickened Soman (VX)—cholinesterase inhibitor (U.S.)

Yellow Rain—Unknown compound that causes bleeding and rapid death. May include mycotoxins produced by the genus Fusarium fungi.

Black Rain—Unknown compound that causes instant death; used by U.S.S.R. in Afghanistan.

Novichok—Recently developed choline sterase inhibitor (U.S.S.R.). May affect human genes and thus damage could be genetically transmitted to offspring.

BLISTER AGENTS

Ethyldichlorarsine (ED)—blister agent

Lewisite (L)—irritates nasal passages, causes skin and membrane burns, poisonous

Mustard (H, HID, HS)—causes skin and membrane inflammation, blindness

Phosgene Oxime (CX)—destroys skin and membrane tissue

BLOOD AGENTS

A blood agent is absorbed into the body through the lungs where it is then picked up by the blood and carried to the rest of the body.

Arsine Trihydride (SA)—causes gasping and choking, asphyxiation

Cyangen Chloride (CK)—causes convulsions, asphyxiation

Hydrogen Cyanide (AC)—causes convulsions, gasping, choking, asphyxiation

Hydrogen Cyanide (PB) Penetrates current issue U.S. military gas masks. Allegedly used against U.S. forces by Iraq during Persian Gulf War. Causes convulsions, gasping, choking, asphyxiation

CHOKING AGENTS

Chlorpicrin (PS)—causes severe coughing, lung edema, choking, asphyxiation

Chlorine (CL)—causes severe coughing, choking, skin and membrane burns, asphyxiation

Phosgene (CG)—causes severe coughing, choking, asphyxiation

TEAR GASES

Tear gases cause eyes to smart and tear and irritate nerves in mucous membranes, including nose, mouth, throat and airway.

Brombenzylcyanide (CA)—long acting

Chloracetophenone (CN)—short acting

Chloracetophenone in Chlorpicrin (CS)

Dibenz (CR)

NAUSEA GASES

Adamsite (DM)—arsenic compound, causes sneezing, nausea and depression

Diphenylchlorarsine (DA)—causes sneezing, nausea and depression

OTHER

Buzz (13Z)—Hallucinogenic LSD derivative (U.S.)

Blue X—Unknown composition. Incapacitating variously estimated for 1-2 and 8-12 hours (U.S.S.R.)

CHEMICAL WARFARE AGENTS

Nerve Agents

Lethal substances that disable enzymes responsible for the transmission of nerve impulses.

Name/Symbol	Means of Exposure	Lethal Dosage[2]	Rate of Action[3]	Effects	Antidotes/Methods of Treatment
Tabun (GA)	Skin contact and/or inhalation	Via inhalation: 400 LCt_{50} Via skin exposure: 1,000 LD_{50}	Very rapid Incapacitating effects occur within 1 to 10 minutes; lethal effects occur within 10 to 15 minutes	Effects seen in eyes (contraction of pupils, pain, dim or blurred vision), nose (runny nose), and airways (chest tightness) Nausea and vomiting also possible Twitching/convulsions result when skeletal muscle reached	4 steps to management of exposure to nerve agents: • decontamination • ventilation • antidotes • supportive therapy Therapeutic drug options: • Atropine and Pralidoxime Chloride (autoinjectors packaged together in kits provided to military personnel) • Diazepam (anticonvulsant drug) Pretreatment option: • Pyridostigmine (can increase the lethal dose threshold significantly if ingested prior to exposure and if paired with traditional therapeutic options)
Sarin (GB)	Skin contact and/or inhalation	Via inhalation: 100 LCt_{50} Via skin exposure: 1,700 LD_{50}	Very rapid Incapacitating effects occur within 1 to 10 minutes; lethal effects occur within 2 to 15 minutes		
Soman (GD)	Skin contact and/or inhalation	Via inhalation: 70 LCt_{50} Via skin exposure: 50 LD_{50}	Very rapid Incapacitating effects occur within 1 to 10 minutes; lethal effects occur within 1 to 15 minutes	Fluctuations in heart rate Loss of consciousness and seizure activity can occur within one minute of exposure in cases of exposure to high concentration of agent	
VX	Skin contact and/or inhalation	Via inhalation: 50 LCt_{50} Via skin exposure: 10 LD_{50}	Rapid Incapacitating effects occur within 1 to 10 minutes; lethal effects occur within 4 to 42 hours	Eventual paralysis, death	
Novichok[5] agents		*Novichok 5* estimated to exceed effectiveness of VX by 5 to 8 times *Novichok 7* estimated to exceed effectiveness of soman by 10 times	Very rapid	Assumed to be similar to the effects of other nerve agents listed above	Assumed to be similar to treatment methods for other nerve agents listed above

Blister Agents

Agents that cause blisters on skin and damage the respiratory tract, mucous membranes, and eyes.

Name/Symbol	Means of Exposure	Lethal Dosage[2]	Rate of Action[3]	Effects	Antidotes/Methods of Treatment
Sulfur Mustard (HD)	Skin contact and/or inhalation	Via inhalation: 1,500 LCt_{50} Via skin exposure: 4,500 LD_{50}	Delayed (tissue damage occurs within minutes of contact, but clinical effects are not immediately evident) Effects manifested 2 to 24 hours after exposure	Pain is not immediate. Topical effects occur on the skin (blisters), in airways (coughing lesions, in rare cases resulting in respiratory failure) and in the eyes (itchiness, burning sensation, possible cornea damage) Nausea and vomiting can also result	
Lewisite (L)	Skin contact and/or inhalation	Via inhalation: 1,300 LCt_{50} Via skin exposure: greater than 4,500 LD_{50}	Rapid Pain and irritation occur immediately	Effects are similar to mustard: skin blistering, burning/watery/swollen eyes, upper airway irritation, systemic blood poisoning	Thorough decontamination using water Prevention of infection using antibiotics Application of lotions/ointments to soothe blisters
Nitrogen Mustard (HN-3)[4]	Skin contact and/or inhalation	Via inhalation: 1,500 LCt_{50} Via skin exposure: 4,500 LD_{50}	Rapid Rash occurs within one hour; blistering occurs between 6 to 12 hours after exposure	Skin blistering, respiratory tract damage	Mustard has no known antidote British-Anti-Lewisite can mitigate some systemic effects of lewisite, though it can itself cause some toxicity.
Mustard-Lewisite	Skin contact and/or inhalation	Via inhalation: 1,500 LCt_{50} Via skin exposure: 10,00 LCt_{50}	Rapid Stinging sensation occurs immediately; blisters follow hours later	Skin blistering, burning in the eyes, inflamation of respiratory tract	
Phosgene-oxime (CX)	Skin contact and/or inhalation	Via inhalation: 3,200 LCt_{50} Via skin exposure: 25 LD_{50}	Rapid	Extremely irritating to eyes, skin, and upper respiratory system	

Blood Agents

Agents that interfere with the absorption of oxygen into the bloodstream.

Name/Symbol	Means of Exposure	Lethal Dosage[2]	Rate of Action[3]	Effects	Antidotes/Methods of Treatment
Hydrogen Cyanide (AC)	Inhalation	2,000 to 5,00 LCt_{50}	Rapid Exposure to low concentrations causes symptoms in 1 or more hours Exposure to high concentrations causes sudden unconsciousness	Agents inhibit cell respiration; heart and central nervous system are susceptible Cyanogen Chloride also greatly irritates eyes and lungs In moderate cases: • vomiting • dizziness • deeper, more rapid breathing In severe cases: • convulsions • respiratory/ failure • sudden loss of consciousness leading to death	Agents are highly volatile; flush eyes with water; remove contaminated clothing; rinse exposed skin with water Antidotes: intravenous administration of sodium nitrite and sodium thiosulfate for detoxification purposes (i.e., to assist body's ability to excrete cyanide from system) Pretreatment under development in the United Kingdom
Cyanogen Chloride (CK)	Inhalation	11,000 LCt_{50}	Rapid Lethal concentration produces effects within 15 seconds of exposure; death following within 6 to 8 minutes		

Choking Agents

Substances that damage respiratory tract, causing extensive fluid build-up in the lungs..

Name/Symbol	Means of Exposure	Lethal Dosage[2]	Rate of Action[3]	Effects	Antidotes/Methods of Treatment
Chlorine	Inhalation	3,000 LCt_{50}	Rapid Lethal effects manifest 30 minutes after exposure	Shortness of breath irritation of mucous membranes; coughing; tightness of chest Culminates in fluid build-up in lungs leading to fatal choking	No antidote once exposed Individual should don gas masks and other protective gear to prevent inhalation
Phosgene (CG)	Inhalation	3,200 LCt_{50}	Delayed Asymptomatic period can last up to 24 hours		Medical responses include: • Relocation to decontamined environment • Enforced rest • Management of sections in airways • Oxygen therapy • Prevention/treatment of pulmonary edema
Diphosgene (DP)	Inhalation	3,200 LCt_{50}	Delayed Incapacitating and lethal effects felt after 3 or more hours		
Chloropicrin (PS)	Inhalation	20,000 LCt_{50}	Variable Produces tears in seconds; lethal effects felt after 10 minutes	Vomiting, fluid build-up in lungs	

NERVE AGENTS
GA GB GD GF VX

BACKGROUND:

Nerve agents are organophosphorous cholinesterase inhibitors. They inhibit the butyryl-cholinesterase in the plasma, the acetylcholinesterase on the red cell, and the acetyl-cholinesterase at cholinergic receptor sites in tissue.

After a nerve agent inhibits the tissue enzyme, the enzyme cannot hydrolyze acetylcholine, the neurotransmitter at cholinergic receptor sites. Acetylcholine accumulates and continues to stimulate the affected organ. The clinical effects from nerve agent exposure are caused by excess acetylcholine.

The attachment of the agent to the enzyme is permanent (unless removed by therapy). Erythrocyte enzyme activity returns at the rate of erythrocyte turnover, about 1% per day. Tissue and plasma enzyme activities return with synthesis of new enzymes. The rate of return of the tissue and plasma enzymes is not the same, nor is the rate the same for all tissue enzymes. However, the agent can be removed from the enzyme and the enzyme "reactivated" by several types of compounds, the most useful of which are the oximes. If the agent-enzyme complex has not "aged," oximes are useful therapeutically. Aging is a biochemical process by which the agent-enzyme complex becomes refractory to oxime reactivation of the enzyme. For most nerve agents the aging time is longer than the time within which acute casualties will be seen. However, the aging time of the GD-enzyme complex is about two minutes, and the usefulness of oximes in GD poisoning is greatly decreased after this period.

Organs with cholinergic receptor sites include the smooth muscles, the skeletal muscles, the central nervous system, and most exocrine glands. In addition, cranial efferents and ganglionic afferents are cholinergic nerves.

Muscarine will stimulate some of the cholinergic sites, and these are known as muscarinic sites. Organs with these sites include the smooth muscles and glands. Nicotine will stimulate other cholinergic sites, known as nicotinic sites, which are those in skeletal muscle and ganglia. The central nervous system (CNS) contains both types of receptors, but the pharmacology in the CNS is more complex and less well understood. Atropine and similar compounds block the effects of excess acetylcholine more effectively at muscarinic sites than at nicotinic sites.

Some commonly used pesticides (for example, the organophosphate (OP) Malathion and the carbamate Sevin) and some common therapeutic drugs (the carbamates pyridostigmine [Mestinon] and physostigmine [Antilirium]) also inhibit acetylcholinesterase and can be considered "nerve agents."

However, while the OP pesticides cause the same biological effects as nerve agents, there are some important differences in the duration of biological activity and response to therapy.

CLINICAL FEATURES:

The initial effects of exposure to a nerve agent depend on the dose and on the route of exposure. The initial effects from a sublethal amount of agent by vapor exposure are different from the initial effects from a similar amount of liquid agent on the skin.

Toxicities: The large amounts of GA and GB required to produce effects after skin application reflect the volatility of these agents. They evaporate rather than penetrate the skin. However, if these agents are occluded and prevented from evaporating they penetrate the skin very well.

GB, the agent studied most thoroughly in man, will cause miosis, rhinorrhea, and a feeling of tightness in the throat or chest at a Ct of 3 to 5 mg-min/m^3.

Effects: Exposure to a small amount of nerve agent vapor causes effects in the eyes, nose, and airways. These effects are from local contact of the vapor with the organ and do not indicate systemic absorption of the agent. In this circumstance, the erythrocyte-ChE may be normal or depressed. A small amount of liquid agent on the skin causes systemic effects initially in the gastrointestinal (GI) tract. Lethal amounts of vapor or liquid cause a rapid cascade of events culminating within a minute or two with loss of consciousness and convulsive activity followed by apnea and muscular flaccidity within several more minutes.

Eye: Miosis is a characteristic sign of exposure to nerve agent vapor. It occurs as a result of direct contact of vapor with the eye. Liquid agent on the skin will not cause miosis if the amount of liquid is small; a moderate amount of liquid may or may not cause miosis; and a lethal or near-lethal amount of agent usually causes miosis. A droplet of liquid in or near the eye will also cause miosis. Miosis will begin within seconds or minutes after the onset of exposure to agent vapor, but it may not be complete for many minutes if the vapor concentration is low. Miosis is bilateral in an unprotected individual, but occasionally may be unilateral in a masked person with a leak in his mask eyepiece.

Miosis is often accompanied by complaints of pain, dim vision, blurred vision, conjunctival injection, nausea, and occasionally vomiting. The pain may be sharp or dull in or around the eyeball, but more often is a dull ache in the frontal part of the head. Dim vision is due in part to the small pupil, and cholinergic mechanisms in the visual pathways also contribute. The complaint of blurred vision is less easily explained, as objective testing usually indicates an improvement in visual acuity because of the "pin-hole" effect. Conjunctival injection may be mild or severe, and occasionally subconjunctival hemorrhage is present. Nausea (and sometimes vomiting) are part of a generalized complaint of not feeling well. Miosis, pain, dim vision, and nausea can be relieved by topical homatropine or atropine in the eye.

Nose: Rhinorrhea may be the first indication of nerve agent vapor exposure. Its severity is dose dependent.

Airways: Nerve agent vapor causes bronchoconstriction and increased secretions of the glands in the airways in a dose-related manner. The exposed person may feel a slight tightness in his chest after a small amount of agent and may be in severe distress after a large amount of agent. Cessation of respiration occurs within minutes after the onset of effects from exposure to a large amount of nerve agent. This apnea is probably mediated through the CNS, although peripheral factors (skeletal muscle weakness, e.g., the intercostal muscles, and bronchoconstriction) may contribute.

Gastrointestinal tract: After they are absorbed, nerve agents cause an increase in the motility of the GI tract and an increase in secretions by the glands in the wall of the GI tract. Nausea and vomiting are early signs of liquid exposure on the skin. Diarrhea may occur with large amounts of agent.

Glands: Nerve agent vapor causes increases in secretions from the glands it contacts, such as the lacrimal, nasal, salivary, and bronchial glands. Localized sweating around site of liquid agent on the skin is common, and generalized sweating after a large liquid or vapor exposure is common. Increased secretions of the glands of the GI tract occur after systemic absorption of the agent by either route.

Skeletal Muscle: The first effect of nerve agents on skeletal muscle is stimulation producing muscular fasciculations and twitching. After a large amount of agent, fatigue and weakness of muscles are rapidly followed by muscular flaccidity.

Fasciculations are sometimes seen early at the site of a droplet of liquid agent on the skin, and generalized fasciculations are common after a large exposure. These may remain long after most of the other acute signs decrease.

Central Nervous System: The acute of CNS signs of exposure to a large amount of nerve agent are loss of consciousness, seizure activity, and apnea. These begin within a minute after exposure to a large amount of agent vapor and may be preceded by an asymptomatic period of one to 30 minutes after contact of liquid with the skin.

After exposure to smaller amounts of nerve agents, CNS effects vary and are nonspecific. They may include forgetfulness, an inability to concentrate fully, insomnia, bad dreams, irritability, impaired judgement, and depression. They do not include frank confusion and misperceptions (i.e., hallucinations). These may occur in the absence of physical signs or other symptoms of exposure. After a severe exposure these symptoms occur upon recovery from the acute severe effects. In either case they may persist for as long as four to six weeks.

Cardiovascular: The heart rate may be decreased because of stimulation by the vagus nerve, but it is often increased because of other factors, such as fright, hypoxia, and the influence of adrenergic stimulation secondary to ganglionic stimulation. Thus, the heart rate may be high, low, or in the normal range. Bradyarrhythmias, such as first-, second-, or third-degree heart block may occur. The blood pressure may be elevated from adrenergic factors, but is generally normal until the terminal decline.

DIAGNOSIS:

Physical findings depend on the amount and route of exposure. After exposure to small to moderate amounts of vapor, there are usually miosis and conjunctival injection, rhinorrhea, and pulmonary signs, although the latter may be absent even in the face of mild to moderate pulmonary complaints. In addition to these signs, an exposure to a high Ct may precipitate copious secretions from the nose and mouth, generalized muscular fasciculations, twitching or seizure activity, loss of consciousness, and apnea. Cyanosis, hypotension, and bradycardia may be present just before death.

Exposure to a small droplet of liquid on the skin may produce few physical findings. Sweating, blanching, and occasionally fasciculations at the site may be present soon after exposure, but may no longer be present at the onset of GI effects. After a large exposure, the signs are the same as after vapor exposure.

Miosis is a useful sign of exposure to vapor, but does not occur after a liquid exposure unless the amount of exposure is large or the exposure is in or close to the eye.

Time Course of Effects

Effects from nerve agent vapor begin within seconds to several minutes after exposure. Loss of consciousness and onset of seizure activity have occurred within a minute of exposure to a high CT. After exposure to a very low Ct, miosis and other effects may not begin for several mintues, and miosis may not be complete for 15 to 30 minutes after removal from the vapor. There is no latent period or delay in onset from vapor exposure. Effects may continue to progress for a period of time, but maximal effects usually occur within minutes after exposure stops.

A large amount of liquid on the skin causes effects within minutes. Commonly there is an asymptomatic period of one to 30 minutes, and then the sudden onset of an overwhelming cascade of events, including loss of consciousness, seizure activity, apnea, and muscular flaccidity. After small amounts of liquid agent on the skin the onset of effects has been delayed for as long as 18 hours after contact. These effects are initially gastrointestinal and are usually not life threatening. Generally, the longer the interval the less severe are the effects.

Differential Diagnosis

The effects caused by a mild vapor exposure, namely rhinorrhea and a tightness in the chest, may easily be confused with an upper respiratory malady or an allergy. Miosis, if present, will help to distinguish these, but the eyes must be examined in very dim light to detect this. Similarly, GI symptoms from another illness may be confused with those from nerve agent effects, and in this instance there will be no useful physical signs. History of possible exposure will be helpful, and laboratory evidence (decreased RBC-ChE activity), if available, will be useful to distinguish the two.

The diagnosis is easier in the severely intoxicated patient. The combination of miosis, copious secretions, and generalized muscular fasciculations in a gasping, cyanotic, and convulsing patient is characteristic.

Laboratory Findings

The cholinesterase activity of the blood components is inhibited by nerve agents, 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.

The amount of inhibition of this enzyme activity does not correlate well with the severity of local effects from mild to moderate vapor exposure. The enzyme activity may be from 0% to 100% of the individual's normal activity in the face of miosis, rhinorrhea, and/or airway symptoms. Normal or nearly normal erythrocyte acetylcholinesterase activity may be present with moderate effects in these organs. At the other extreme, the enzyme may be inhibited 60% to 70% when miosis or rhinorrhea is the only sign of exposure. Several systemic effects generally indicate inhibition of the erythrocyte acetylcholinesterase by 70% to 80% or greater.

Other laboratory findings will relate to complications. For example, acidosis may occur after prolonged hypoxia.

MEDICAL MANAGEMENT:

Management of a casualty with nerve agent intoxication consists of decontamination, ventilation, administration of the antidotes, and supportive therapy. The condition of the patient dictates the need for each of these and the order in which they are done.

Decontamination is described elsewhere in this manual. Skin decontamination is not necessary after exposure to vapor alone, but clothing should be removed because it may contain "trapped" vapor.

The need for **ventilation** will be obvious, and the means of ventilation will depend on available equipment. Airway resistance is high (50-70 cm of water) because of bronchoconstriction and secretions, and initial ventilation is difficult. The resistance decreases after atropine administration, after which ventilation will be easier. The copious secretions, which may be thickened by atropine, also impede ventilatory efforts and require frequent suctioning. In reported cases of severe nerve agent exposure, ventilation has been required from 0.5 to 3 hours.

Three drugs are used to treat nerve agent exposure, and another is used as pretreatment for potential nerve agent exposure. The three therapeutic drugs are atropine, pralidoxime chloride, and diazepam. The use of the pretreatment drug, pyridostigmine bromide, is discussed later in this chapter.

Atropine is a cholinergic blocking, or anticholinergic, compound. It is extremely effective in blocking the effects of excess acetylcholine at peripheral muscarinic sites. Under experimental conditions, very large amounts may block some cholinergic effects at nicotinic sites, but these antinicotinic effects are not evident even at high clinical doses. When small amounts (2 mg) are given to normal individuals without nerve agent intoxication, atropine causes mydriasis, a decrease in secretions (including a decrease in sweating), mild sedation, a decrease in GI motility, and tachycardia. The amount in three MARK I kits may cause adverse effects on military performance in a normal person. In people not exposed to nerve agents, amounts of 10 mg or higher may cause delirium. Potentially, the most hazardous effect of inadvertent use of atropine (2 mg, i.m.) in a young person not exposed to a cholinesterase inhibiting compound in a warm or hot atmosphere is inhibition of sweating, which may lead to heat injury. In the military, atropine is packaged in autoinjectors, each containing 2 mg.

Pralidoxime chloride (Protopam chloride; 2-PAMCl) is an oxime. Oximes attach to the nerve agent that is inhibiting the cholinesterase and break the agent-enzyme bond to restore the normal activity of the enzyme. Clinically, this is noticable in those organs with nicotinic receptors. Abnormal activity in skeletal muscles decreases, and normal strength returns. The effects of an oxime are not apparent in organs with muscarinic receptors; oximes do not cause a decrease in secretions, for example. They also are less useful after aging occurs, but with the exception of GD (soman) intoxicated individuals, casualties will be treated before significant aging occurs. Pralidoxime chloride (600 mg) is an autoinjector for self-use along with the atropine injector. These atropine and pralidoxime chloride autoinjectors are packaged together in a MARK I kit. Each military person is issued three MARK I kits. MARK I kits are now being carried by some EMS agencies and kept in hospitals.

Diazepam is an anticonvulsant drug used to decrease convulsive activity and to reduce the brain damage caused by prolonged seizure activity. Without the use of pyridostigmine pretreatment, experimental animals died quickly after superlethal doses of nerve agents despite conventional therapy. With pyridostigmine pretreatment (followed by conventional therapy) animals survived superlethal doses of soman, but had prolonged periods of seizure activity before recovery. They later had performance decrements and anatomic lesions in their brains. The administration of diazepam with other standard therapy to soman-poisoned animals pretreated with pyridostig-

mine reduced the seizure activity and its sequelae. Current military doctrine is to administer diazepam with other therapy (three MARK I's) at the onset of severe effects from a nerve agent, whether or not seizure activity is among those effects. Each military person carries one autoinjector containing 10 mg of diazepam for his buddy to administer to him (if he could self-administer it, he would not need it). **Diazepam should be administered with the three MARK I's when the casualty's condition warrants the use of three MARK I's at the same time.** Medical personnel can administer more diazepam to a casualty if necessary. The medical corpsman carries extra diazepam injectors and is authorized to administer two additional injectors at 10 minute intervals to a convulsing casualty.

The doctrine for **self-aid** for nerve agent intoxication states that if an individual has effects from the agent he/she should self-administer one MARK I. If there is no improvement in 10 minutes, he/she should seek out a buddy to assist in the evaluation of his/her condition before further MARK I's are given. If a buddy finds an individual severely intoxicated (e.g., gasping respirations, twitching, etc.) so that the individual can not self-administer a MARK I, the buddy should administer three MARK I's and diazepam immediately. The discussion below is advice for medical assistance.

The appropriate number of MARK I kits to administer initially to a casualty from nerve agent vapor depends on the severity of the effects. Systemic atropine will not reverse miosis (unless administered in very large amounts), and miosis alone is not an indication for a MARK I. If the eye or head pain and nausea associated with the miosis are severe, topical application of atropine (or homatropine) in the eye will bring relief. Topical atropine should not be used without good reason (severe pain), because it causes blurred vision for a day or longer. A casualty with miosis and rhinorrhea should be given one MARK I only if the rhinorrhea is severe and troublesome (he can not keep his mask on because of fluid). A casualty with mild to moderate dyspnea should be given one or two MARK I's, depending on the severity of his distress and the time between exposure and therapy. Some of the respiratory distress from a mild exposure will spontaneously decrease within 15 to 30 minutes after termination of exposure, so if the casualty is not severely uncomfortable only one MARK I should be used initially. Atropine is quite effective, and care should be taken not to give too much in a casualty who does not need it.

A severe casualty from nerve agent vapor has miosis, copious secretions from the nose and mouth, severe difficulty breathing or apnea, possibly some degree of cyanosis, muscular fasciculations, and twitching or convulsive activity, and is unconscious. He should be given three MARK I's and diazepam immediately. Ventilation will be needed and should be done via an endotracheal airway if possible. Suctioning of the excessive airway secretions will be necessary to enhance air exchange and will make ventilatory efforts easier. Atropine, 2 mg, should be repeated at three- to five-minute intervals and should be titrated to a reduction of secretions and to reduction of ventilatory resistance. When the intravenous preparation is available, the preferred route of atropine administration is via the intravenous route, but this route should be avoided until hypoxia is corrected, because intravenously administered atropine in hypoxic animals has produced ventricular fibrillation. In a hypotensive patient or a patient with poor veins, atropine might be

given intratracheally, either via the endotracheal tube or directly into the trachea, for more rapid absorption via the peribronchial vessels.

The medical care provider might err in giving too much atropine to a mild to moderate casualty. More importantly, the care provider might err by giving too little atropine to a severe casualty. In a severe casualty, atropine should be pushed at frequent intervals until secretions are dry (or nearly dry) and until ventilation can be accomplished with ease. In reported cases this has required 10 to 20 mg of atropine within the first several hours. A conscious, less-severely exposed casualty should receive atropine until he is breathing comfortably, and he will be able to communicate this. Dry secretions need not be an end point in mild to moderate casualties.

The casualty with skin exposure to liquid is more difficult to evaluate and manage than is a casualty from vapor exposure. Agent on the surface of the skin can be decontaminated, but agent absorbed into the skin cannot be removed. The initial effects from absorbed liquid agent can start two to three hours after thorough decontamination of agent droplets on the skin. A casualty from liquid exposure on the skin may continue to worsen because of continued absorption of the agent from the skin depot.

The first effects of a liquid droplet on the skin are sweating with or without blanching and occasionally with muscular fasciculations at the site. Gastrointestinal effects (nausea, vomiting, and sometimes diarrhea) are the first systemic effects, and these may start from 0.5 to 18 hours after contact with the agent. If these effects occur within the first several hours after exposure, they may portend more severe effects, and initial therapy should be two MARK I's. If effects begin later, initial therapy should be one MARK I.

A large amount of liquid agent on the skin will cause effects 1 to 30 minutes after contact, whether or not decontamination was done. Nevertheless, early decontamination may lessen the magnitude of the effects. After a one- to thirty-minute latent or asymptomatic period, the casualty will suddenly lose consciousness and begin seizure activity. The condition of the casualty and management are the same as described for a severe casualty from vapor or exposure.

Further care of the severe casualty consists of atropine administration to minimize secretions and of ventilation until spontaneous respiration resumes. Oxime administration should be repeated at hourly intervals for two or three additional doses. The preferred method of administration of the oxime is by intravenous drip of 1 gram over 20 to 30 minutes (more rapid administration will cause hypertension), but three additional oxime autoinjectors (total dose of 1.8 grams) may be given if the intravenous route cannot be used. The need for ventilation may continue for 0.5 to 3 hours. Unless prolonged hypoxia or other complications have occurred, the casualty will eventually begin having spontaneous muscular activity and make sporadic attempts to breathe. Muscles will become stronger and breathing more regular, and the casualty will have intermittent episodes of conscious behavior. Within an hour or two he will be breathing, moving, and conscious, although he will be weak and intermittently obtunded.

SUMMARY:

SIGNS AND SYMPTOMS:

Vapor: *Small exposure*—Miosis, rhinorrhea, mild difficulty breathing. *Large exposure*–Sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions, miosis.

Liquid on skin: *Small to moderate exposure*–Localized sweating; nausea, vomiting, feeling of weakness. *Large exposure*–Sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions.

DIAGNOSIS:

Effects of Nerve Agent Vapor

- Small Amount:

  Eyes: small pupils, red conjunctiva, dim/blurred vision, pain, nausea/vomiting

  Nose: runny nose

  Mouth: increased salivation

  Airways: tightness in chest, shortness of breath, cough

- Large Amount:

  Loss of consciousness

  Convulsions

  Flaccid paralysis

  Breathing stops

  Heart stops

Effects begin within seconds to a minute.

Effects of Nerve Agent Liquid on the Skin

- Very small drop: sweating, twitching at site
- Small drop: nausea, vomiting, diarrhea
- Drop: Loss of consciousness, convulsions, breathing stops, flaccid paralysis

Effects begin within 30 minutes (large amount) to 18 hours (small amount).

TREATMENT:

Administration of MARK I's (atropine and pralidoxime chloride); diazepam in addition if casualty is severe; ventilation and suction of airways for respiratory distress.

PROPHYLAXIS:

The U.S. military fielded pyridostigmine bromide as a pretreatment for nerve agent exposure. Each individual received a blister pack containing 21 30-mg tablets. The dose regimen is one 30-mg tablet every eight hours.

ISOLATION AND DECONTAMINATION:

Hypochlorite; large amounts of water. Protect yourself by wearing a mask (SCBA), gloves and a protective suit until the casualty is decontaminated.

NERVE AGENT EFFECTS

Nerve Agents—How They Work

- Nerve Agents interfere with transmission of the message from nerve to organ.
- The nerve is normal; the transmission to the organ (muscle, gland) is faulty.
- The organ (muscle, gland) gets the wrong message, and does the wrong thing.
- This causes too much activity in muscles,

Vapor Exposure

Mild

Eyes	Miosis
	Dim vision
	Headache
Nose	Rhinorrhea
Mouth	Salivation
Lungs	Dyspnea ("tightness in the chest")

Time of onset: Seconds to minutes after exposure

Self-aide: 1 MARK 1

Buddy-aid: Stand by

Severe

All the above, plus

Severe breathing difficulty or cessation of respiration

Generalized muscular twitching, weakness or paralysis

Convulsions

Loss of consciousness

Loss of bladder, bowel control

Time of onset: Seconds to minutes after exposure

Self-aid: None. Victim will be unable to help self.

Buddy-aid: 3 MARK I's and diazepam immediately

Liquid on skin

Mild/moderate

Muscle twitching at site of exposure

Sweating at site of exposure

Nausea, vomiting

Feeling of weakness

Time of onset: 10 minutes to 18 hours after exposure

Self-aid: 1-2 MARK I's, depending on severity of symptoms

Buddy-aid: Stand by

Severe

All the above, plus

Severe breathing difficulty or cessation of breathing

Generalized muscular twitching, weakness, or paralysis

Convulsions

Loss of consciousness

Loss of bladder and bowel control

Time of onset: Minutes to an hour after exposure

Self-aid: None. Victim will be unable to help himself

Buddy-aid: 3 MARK I's and diazepam immediately

BLISTER AGENTS (MUSTARD) HD H

BACKGROUND:

Vesicant agents, specifically sulfur mustard (H; HD), have been major military threat agents since their introduction in World War I. They constitute both a vapor and a liquid threat to all exposed skin and mucous membranes. Mustard's effects are delayed, appearing hours after exposure. Organs most commonly affected are the skin (with erythema and vesicles), eyes (with mild conjunctivitis to severe eye damage), and airways (with mild irritation of the upper respiratory tract to severe bronchiolar damage leading to necrosis and hemorrhage of the airway mucosa and musculature). Following exposure to large quantities of mustard, precursor cells of the bone marrow are damaged, leading to pancytopenia and increased susceptibility to infection. The gastrointestinal tract may be damaged, and there are sometimes central nervous system signs. There is no specific antidote, and management is symptomatic therapy. Immediate decontamination is the only way to reduce damage.

Mustard is an oily liquid with a color ranging from a light yellow to brown. Its odor is that of garlic, onion, or mustard (hence its name), but because of accomodation of the sense of smell, odor should not be relied on for detection. Under temperate conditions mustard evaporates slowly and is primarily a liquid hazard, but its vapor hazard increases with increasing temperature. At 100°F or above, it is a definite vapor hazard. Mustard freezes at 57°F and, since a solid is difficult to disperse, it is often mixed with substances with a lower freezing point, e.g., Lewisite (the mixture is HL), or agent T, a closely related vesicant (the mixture is HT) so that the mixture will remain liquid at lower temperatures.

After absorption into the body, mustard rapidly cyclizes (seconds to minutes) in extracellular water. This cyclic compound is extremely reactive and quickly binds to intra- and extra-cellular enzymes, proteins, and other substances. Mustard has many biological actions, but the exact mechanism by which it produces tissue injury is not known. According to one prominent hypothesis, biological damage from mustard results from DNA alkylation and crosslinking in rapidly dividing cells, such as basal keratinocytes, mucosal epithelium, and bone marrow precursor cells. This leads to cellular death and inflammatory reaction, and, in the skin, protease digestion of anchoring filaments at the epidermal-dermal junction and the formation of blisters.

Mustard possesses mild cholinergic activity, which may be responsible for effects such as early gastrointestinal symptoms and miosis.

Mustard reacts with tissue within minutes of entering the body and is no longer an intact molecule. Blood, tissue, and blister fluid do not contain mustard, and one cannot become exposed to mustard by contact with body fluids or tissues.

CLINICAL FEATURES:

Topical effects of mustard occur in the eye, airways, and skin. Systemically absorbed mustard may produce effects in the bone marrow, the gastrointestinal tract, and the central nervous system. Direct injury to the GI tract may also occur following ingestion of the compound.

Skin: Erythema is the mildest and earliest form of skin injury after exposure to mustard. It resembles sunburn, and is associated with pruritis or burning, stinging pain. Erythema begins to appear in 2 to 24 hours after vapor exposure with time of onset dependent on Ct, ambient temperature and humidity, and skin site exposed. The skin sites most sensitive are the warm, moist locations with thinner skin, such as the perineum, external genitalia, axillae, antecubital fossae, and neck.

Within the erythematous areas, small vesicles can develop, which may later coalesce to form bullae. The typical bulla, or blister, is large, dome-shaped, thin-walled, translucent, yellowish, and surrounded by erythema. The blister fluid is clear, at first thin and straw-colored, but later yellowish and tending to coagulate. The fluid does not contain mustard and is not a vesicant.

At extremely high doses, such as those from liquid exposure, lesions may develop a central zone of coagulation necrosis with blister formation at the periphery. These lesions take longer to heal and are more prone to secondary infection that the uncomplicated lesions seen at lower exposure levels.

Pulmonary: The primary airway lesion from mustard is necrosis of the mucosa with later damage to the musculature of the airways if the amount of agent is large. The damage begins in the upper airways and descends to the lower airways in a dose-dependent manner. Usually, the terminal airways and alveoli are affected only as a terminal event. Pulmonary edema is not usually present unless the damage is very severe and then it usually is hemorrhagic.

The earliest effects from mustard—perhaps the only effects from a low Ct—involve the nose, the sinuses, and the pharynx. There may be irritation or burning of the nares, epistaxis, sinus pain or irritation, and irritation or soreness of the pharynx. As the Ct increases other effects occur: laryngitis with voice changes and a nonproductive cough. Damage to the trachea and upper bronchi leads to a cough productive of sputum. Lower airway involvement causes dyspnea and an increasingly severe cough with increased quantities of sputum. Terminally, there may be necrosis of the smaller airways with hemorrhagic edema into surrounding alveoli. This hemorrhagic pulmonary edema is rarely a feature.

Necrosis of the airway mucosa with resulting inflammation can cause pseudomembrane formation, and pseudomembranes may occur from the most proximal parts of the airways to the most distal portions. These membranes may cause local airway obstruction at the sites of formation, and detachment may lead to obstruction of lower airways.

The cause of death in mustard poisoning is commonly respiratory failure. Mechanical obstruction by pseudomembranes may be a cause, but more commonly deaths occurring from the third to the sixth day after exposure result from secondary bacterial pneumonia caused by bacterial invasion of denuded respiratory mucosa and necrotic debris. Agent-induced bone marrow suppression is a contributory factor in later, septic deaths from pneumonia.

Ocular: The eyes are the organs most sensitive to mustard vapor injury. The latent period is shorter for eye injury than for skin injury and is also Ct dependent.

After low-dose vapor exposure, irritation, evidenced by reddening of the eyes, may be the only effect. As the dose increases, the spectrum of injury includes progressively more severe conjunctivitis, photophobia, blepharospasm, pain, and corneal damage.

Blisters do not normally form in the eyes. Instead, swelling and loosening of corneal epithelial cells leads to corneal edema and clouding with leukocytes (which affects vision). Corneal vascularization with secondary edema may last for weeks. Severe effects may be followed by scarring between the iris and lens; this scarring may restrict pupillary movements and may predispose victims to glaucoma.

The most severe damage is caused by liquid mustard from airborne droplets or by self-contamination. After extensive eye exposure, severe corneal damage with possible perforation of the cornea and loss of the eye can occur. Eye loss also results from panophthalmitis if appropriate therapy is not instituted.

Gastrointestinal tract: The mucosa of the gastrointestinal (GI) tract is very susceptible to mustard damage, either from systemic absorption or ingestion of the agent.

Mustard exposure, even exposure to a small amount, will often cause nausea with or without vomiting lasting 24 hours or less. The nausea and vomiting appear not to be a direct effect of the agent on the gastrointestinal tract, but rather they are from a stress reaction, a nonspecific reaction to the odor, or cholinergic stimulation by mustard. Diarrhea has been reported; constipation is equally common. Diarrhea (rarely bloody) and vomiting beginning days after a high-dose exposure imply a poor prognosis.

Central nervous system: The CNS effects of mustard remain poorly defined. Animal work demonstrated that mustards (particularly the nitrogen mustards) are convulsants, and there are several human case reports describing people who were exposed to very large amounts and who had neurological effects within several hours after exposure just prior to death.

Time Course of Effects

Mustard binds irreversibly to tissue within several minutes after contact. If decontamination is not done immediately after exposure there is no way to prevent injury, although later decontamination might prevent a more severe lesion.

The clinical effects of mustard are delayed. Signs and symptoms may appear as early as two hours after a high-dose exposure, whereas following a low-dose vapor exposure the latent or asymptomatic period may extend to 24 hours. There are several reports of individuals exposed to very large amounts who died within hours; this type of occurrence is extremely rare. The typical onset time is between four and eight hours. The concentration (C) of the mustard vapor, the time (t) of exposure, the ambient weather, and the body site exposed are factors in the onset time.

It must be emphasized that **mustard causes tissue damage within several minutes after contact without causing any concomitant clinical effects**, e.g., burning or erythema. Because of the lack of immediate effects, the contaminated person is often unaware of the exposure and does not decontaminate. **To prevent injury, decontamination must be done immediately after contact.** Later decontamination may prevent further damage, absorption, or spread of the agent.

DIAGNOSIS:

Of the three vesicant agents, mustard is the only one that does not cause immediate pain. The casualty is asymptomatic until the lesion becomes apparent hours later.

In contrast, Lewisite and phosgene oxime in either liquid or vapor form cause immediate pain or irritation to the eye, skin, or respiratory tract. This is sufficient stimulus to decontaminate immediately or to mask. Because of this, lesions from these agents may not be as severe as those from mustard.

Isolated small blisters or a small group of blisters suggest possible exposure to mustard, to plants such as poison ivy or poison oak, to drugs, or to other substances. The physical characteristics of the lesion are not distinctive, therefore the history of exposure is invaluable.

Although the blisters of mustard and Lewisite are slightly different (there is less erythema around the Lewisite blister) this information is of little value in individual cases.

Laboratory Findings

There are no available clinical laboratory tests for mustard exposure. Leukocytosis occurs during the first day and the magnitude of increase in leukocytes during the subsequent days correlates roughly with the amount of tissue injury, primarily to skin or pulmonary tissue. If systemic absorp-

tion is large, leukocytes in the peripheral blood will decrease beginning on day three to day five; this decrease indicates damage to precursor cells in the blood-forming organs. The fall may be precipitate, e.g., a decrease of 5,000 to 10,000 cells/day. If the marrow damage is severe, erythrocytes and platelets may later decrease, but the victim usually recovers or dies before this is apparent. A leukocyte count of 500 or fewer is a sign of an unfavorable prognosis.

Signs of a chemical pneumonitis may appear within the first 2 to 3 days after inhalational exposure. Leukocytosis, fever, and sputum production suggest a bacterial process, but within this time period sputum cultures are usually negative for pathogens. Organisms commonly invade the damaged airway tissue at days three to five, and a change in the fever pattern, an increase in leukocytosis, and a change in the character of the sputum in this time period suggest a bacterial process. Sputum Gram stain and culture should be done for identification of the specific organism.

Damaged skin should be cultured routinely, particularly if there is an increase in the exudate or an increase in the inflammatory reaction.

Although gastrointestinal bleeding is unusual, declining hematocrit values should prompt serial analyses of stool for occult blood.

There is no clinical laboratory test for mustard in blood or tissue, nor is one expected as mustard is biotransformed and bound to tissues within minutes after absorption. A method for analysis of urine for thiodiglycol, a metabolite of mustard, is in the investigational stage.

MEDICAL MANAGEMENT:

The management of a patient exposed to mustard may be simple, as in the provision of symptomatic care for a sunburn-like erythema, or extremely complex as providing total management for a severely ill patient with burns, immunosuppression, and multi-system involvement. The following are suggested therapeutic measures for each organ system. Guidelines for general patient care are not intended to take the place of sound clinical judgment, especially in the management of complicated cases.

Skin: Erythema should be treated with calamine or other soothing lotion or cream (e.g., 0.25% camphor and menthol, calamine) to reduce burning and itching. Small blisters (under 1-2 cm) should be left intact, but because larger ones will eventually break (the blister fluid does not contain mustard) they should be carefully unroofed. Denuded areas should be irrigated 3-4 times daily with saline, another sterile solution, or soapy water and then liberally covered with a topical antibiotic such as silver sulfadiazine or mafenide acetate to a thickness of 1-2 mm. If an antibiotic cream is not available, sterile petrolatum will be useful. Modified Dakins solution (sodium hypochlorite) was used in WWI and in Iranian casualties for irrigation and as an antiseptic.

Multiple or large areas of vesication suggest the need for hospitalization and whirlpool bath irrigation.

Systemic analgesics should be used liberally, particularly before manipulation of the patient or irrigation of the burn areas. Systemic antipruritics such as trimeprazine should be tried if needed. Monitoring of fluids and electrolytes is important in any sick patient, but it must be recognized that **fluid loss is not of the magnitude seen with thermal burns**. Clinicians accustomed to treating patients with thermal burns must resist the temptation to overhydrate a mustard casualty with a similar amount of burned body surface.

Eyes: Conjunctival irritation from a low Ct will respond to any of a number of available ophthalmic solutions after the eyes are thoroughly irrigated. Regular application of homatropine (or other anticholinergic drug) ophthalmic ointment will reduce or prevent future synechiae formation, and a topical antibiotic applied several times a day will reduce the incidence and severity of infection. Vaseline or a similar substance should be applied to the edges of the lids regularly to prevent them from sticking together. This prevents adhesions and later scarring during healing and also permits drainage of any underlying infection. Topical analgesics may be useful initially if blepharospasm is too severe to permit an adequate examination, but topical analgesics should otherwise be avoided, and systemic analgesics should be given for eye pain. Topical steroids are not of proven value, but their use during the first day or two might reduce inflammation. Further use should be relegated to an ophthalmologist. Sunglasses may reduce discomfort from photophobia.

The patient should be constantly reassured that complete healing and restoration of vision will be the outcome.

Pulmonary: Upper airway symptoms (sore throat, non-productive cough, hoarseness) may respond to steam inhalation and cough suppressants. Although a productive cough and dyspnea accompanied by fever and leukocytosis occurring 12 to 24 hours after exposure may suggest a bacterial process to the clinician, he must resist the urge to use antibiotics for this process, which in fact is a sterile bronchitis or pneumonitis. Infection often occurs on about the third day and its presence is signaled by an increased fever, an increase in the pulmonary infiltrate by x-ray, and an increase in sputum production and a change in sputum character to purulent. Appropriate antibiotic therapy should await confirmation of the clinical impression by positive sputum studies (Gram stain and culture).

Intubation should be performed early before laryngeal spasm or edema makes it difficult or impossible. Intubation permits better ventilation and facilitates suction of the necrotic and inflammatory debris. Oxygen may be needed, and early use of PEEP or CPAP may be of benefit. If there is a suggestion of pseudomembrane formation, bronchoscopy should be done to permit suctioning of the necrotic debris by direct vision.

Bronchodilators may be of benefit for bronchospasm. If they fail, steriods may be tried. There is little evidence that the routine use of steroids is beneficial. The need for continuous use of assisted or controlled ventilation suggests a poor prognosis.

Death often occurs between the fifth and tenth day after exposure because of pulmonary insufficiency and infection complicated by a compromised immune response from agent-induced bone marrow damage.

Gastrointestinal: Atropine (0.4-0.6 mg, i.m. or i.v.), another anticholinergic drug, or antiemetic should control the early nausea and vomiting. Prolonged vomiting or voluminous diarrhea beginning days after exposure suggests direct involvement of the gastrointestinal tract by severe systemic poisoning, a poor prognostic sign.

Bone marrow: Sterilization of the gut by non-absorbable antibiotics should be considered to reduce the possibility of sepsis from enteric organisms. Cellular replacement (bone marrow transplants or transfusions) may be successful as intact mustard does not persist beyond the few minutes following absorption and would not damage the new cells.

General: A patient severely ill from mustard poisoning requires the general supportive care provided for any severely ill patient as well as the specific care given to a burn patient. Liberal use of systemic analgesics and antipruritics, as needed, maintenance of fluid and electrolyte balance, and other supportive measures are necessary. Parenteral food supplements including vitamins may also be helpful.

Other: Sulfur donors such as sodium thiosulfate decreased systemic effects and elevated the LD_{50} when given before exposure or within 20 minutes after exposure in experimental animals. Activated charcoal given orally to casualties was of no value. Hemodialysis was not only ineffective, but was harmful in several casualties. The rapid biotransformation of the mustard molecule suggests that none of these measures would be beneficial hours or days after exposure.

SUMMARY:

SIGNS AND SYMPTOMS:

Asymptomatic latent period (hours). Erythema and blisters on the **skin**; irritation, conjunctivitis and corneal opacity and damage in the **eyes**; mild upper **respiratory** signs to marked **airway** damage; also gastrointestinal effects and bone marrow stem cell suppression.

DIAGNOSIS:

Redness of the skin, blisters. Irritation of eyes. Cough, shortness or breath.

TREATMENT:

Immediate Decontamination After Exposure is the only way to prevent damage. Symptomatic management of lesions.

PROPHYLAXIS:

None

ISOLATION AND DECONTAMINATION:

Protect yourself by wearing a mask, gloves and a protective suit until the patient is decontaminated.

Remove patient from contamination and contamination from patient. Get the patient away from the source, such as by moving him upwind or out of a contaminated building. If it is absolutely certain that exposure was to vapor only, remove outer clothing. If there is a possibility of liquid contamination, all clothing must be removed and the patient must be showered or washed with soap and water, dilute hypochlorite or water.

MUSTARD VAPOR EFFECTS

Mustard—How It Works

- Mustard quickly penetrates the skin, mucous membranes (eye, airways).
- It changes to another substance, and reacts with enzymes, proteins, DNA.
- It causes cell death.
- Mustard effects are like radiation ("radiomimetic").
- Mustard causes damage within minutes.

Organ	Severity	Effects	Onset of first effect
Eye	Mild	Tearing	4-12 hours
		Itchy	
		Burning	
		Gritty feeling	
	Moderate	Above, plus	3-6 hours
		Reddening	
		Swelling of lids	
		Moderate pain	
	Severe	Marked swelling of lids	1-2 hours
		Possible cornea damage	
		Severe pain	
Airways	Mild	Runny nose	12-24 hours
		Sneezing	
		Nosebleed	
		Hoarseness	
		Hacking cough	
	Severe	Above, plus	2-4 hours
		Severe productive cough	
		Shortness of breath mild to severe	
Skin		Erythema (redness)	
		Blisters	2-24 hours

BLISTER AGENTS (LEWISITE) L

BACKGROUND:

Lewisite is a vesicant that damages the eyes, skin, and airways by direct contact. After absorption, it causes an increase in capillary permeability to produce hypovolemia, shock, and organ damage. Exposure to Lewisite causes immediate pain or irritation, although lesions require hours to become full-blown. Management of a Lewisite casualty is similar to management of a mustard casualty, although a specific antidote, British-Anti-Lewisite (BAL; dimercaprol) will alleviate some effects.

Lewisite is an oily, colorless liquid with the odor of geraniums. It is more volatile than mustard.

Although Lewisite contains trivalent arsenic and combines with thiol groups in many enzymes, its exact mechanism of biological activity is unknown.

CLINICAL FEATURES:

Toxicities: Lewisite casues nasal irritation at a Ct of about 8 mg min/m^3, and its odor is noted at a Ct of about 20 mg min/m^3. Lewisite causes vesication and death from inhalation at the same Cts as mustard. Liquid Lewisite causes vesication at about 14µg, and the LD_{50} is about 2.8 grams on the skin.

Organ Systems: Unlike mustard, Lewisite vapor or liquid causes immediate pain or irritation. A person with a droplet of Lewisite on his skin will note the burning and will immediately take steps to try to remove it. The vapor is so irritating that a person will seek to mask or to leave the contaminated area if possible. Because this warning causes the person exposed to take immediate steps to decontaminate, the Lewisite lesion will probably not be as severe as the lesion from mustard, as exposure to mustard is often undetected and decontamination is not done.

There are almost no data on humans exposed to Lewisite, and the following is based on animal investigations.

Skin: Within about five minutes after contact liquid Lewisite will produce a grayish area of dead epithelium. Erythema and blister formation follow more rapidly than in a similar lesion from mustard, although the full lesion does not develop for 12 to 18 hours. The lesion has more tissue necrosis and tissue sloughing than does a mustard lesion.

Eye: Lewisite causes pain and blepharospasm on contact. Edema of the conjunctiva and lids follows, and the eyes may be swollen shut within an hour. Iritis and corneal damage may follow if the dose is high. Liquid Lewisite causes severe eye damage within minutes of contact.

Respiratory: The extreme irritancy of Lewisite to the nasal area and upper airways causes the person to mask or exit the area. Scanty data indicate that Lewisite causes the same airway signs and symptoms as does mustard. The airway mucosa is the primary target and damage progresses down the airways in a dose-dependent manner. Pseudomembrane formation is prominent. Pulmonary edema, which occurs rarely and usually only to a minimal degree after mustard exposure, may complicate exposure to Lewisite.

Other: Available data suggest that Lewisite causes an increase in permeability of systemic capillaries with resulting intravascular fluid loss, hypovolemia, shock, and organ congestion. This may lead to hepatic or renal necrosis with more prominent gastrointestinal effects (including vomiting and diarrhea) than after mustard.

Physical Findings: The findings are similar to those caused by mustard. As noted, the tissue damage at the site of the skin lesion may be more severe.

Time Course of Effects

Pain and irritation from either liquid or vapor Lewisite are immediate. Early tissue destruction is more obvious than after mustard, but the lesion is not full-blown for 12 hours or longer.

DIAGNOSIS:

Although differences have been reported between the skin lesions from mustard and Lewisite (less surrounding erythema and more tissue destruction characterize Lewisite blisters), these are of little diagnostic assistance in a single patient. The history of immediate pain on contact is absent after mustard exposure and present after Lewisite or phosgene oxime exposures.

Other substances cause erythema and blisters, and often the history of exposure is the most helpful tool in diagnosis.

LABORATORY FINDINGS:

There is no specific diagnostic test for Lewisite. Leukocytosis, fever, and other signs of tissue destruction will occur.

MEDICAL MANAGEMENT:

Early decontamination is the only way of preventing or lessening Lewisite damage. Since this must be accomplished within minutes after exposure, this is self-aid rather than medical management.

The guidelines for the management of a mustard casualty will be useful. Lewisite does not cause damage to hematopoietic organs as mustard does. However, fluid loss from the capillaries necessitates careful attention to fluid balance.

British-Anti-Lewisite (BAL; dimercaprol) was developed as an antidote for Lewisite and is used in medicine as a chelating agent for heavy metals. There is evidence that BAL in oil, given intramuscularly, will reduce the systemic effects of Lewisite. However, BAL itself causes some toxicity, and the user should read the package insert carefully. BAL skin ointment and BAL ophthalmic ointment decrease the severity of skin and eye lesions when applied immediately after early decontamination. However, neither is currently manufactured.

SUMMARY:

SIGNS AND SYMPTOMS:

Lewisite causes immediate pain or irritation of skin and mucous membranes. Erythema and blisters on the skin and eye and airway damage similar to those seen after mustard exposure develop later.

DIAGNOSIS:

Lewisite and phosgene oxime, in both their liquid and vapor forms, cause moderate to severe pain on contact with skin or the mucous membranes and produce visible grayish tissue damage within several minutes of contact. It also causes leakage of systemic capillaries, and hypovolemia and hypotension may result.

TREATMENT:

Immediate decontamination; symptomatic management of lesions the same as for mustard lesions; a specific antidote (BAL) will decrease systemic effects.

PROPHYLAXIS:

None

ISOLATION AND DECONTAMINATION:

Protect yourself by wearing a mask, gloves and a protective suit until the patient is decontaminated.

Remove patient from contamination and contamination from patient. Get the patient away from the source, such as by moving him upwind or out of a contaminated building. If it is absolutely certain that exposure was to vapor only, remove outer clothing. If there is a possibility of liquid contamination, all clothing must be removed and the patient must be showered or washed with soap and water, dilute hypochlorite or water.

BLISTER AGENTS (PHOSGENE OXIME) CX

BACKGROUND:

Phosgene oxime is an urticant or nettle agent that causes a corrosive type of skin and tissue lesion. It is not a true vesicant, since it does not cause blisters. The vapor is extremely irritating, and both the vapor and liquid cause almost immediate tissue damage upon contact. There is very scanty information on phosgene oxime.

CX is a solid at temperatures below 95°F, but the vapor pressure of the solid is high enough to produce symptoms. Traces of many metals cause it to decompose. However, it corrodes most metals.

The mechanism by which phosgene oxime causes biological effects is unknown.

CLINICAL FEATURES:

Toxicities: The estimated LCt_{50} by inhalation is 1500-2000 mg /min/m^3. The LD_{50} for skin exposure has been estimated as 25 mg/kg.

Skin: Phosgene oxime liquid or vapor causes pain on contact which is followed in turn by blanching with an erythematous ring in 30 seconds, a wheal in 30 minutes, and necrosis later. The extreme pain may persist for days.

Eyes: Phosgene oxime is extremely painful to the eyes. The damage is probably similar to that caused by Lewisite.

Pulmonary: Phosgene oxime is very irritating to the upper airways. This agent causes pulmonary edema after inhalation and after skin application.

Other: Some animal data suggest that phosgene oxime may cause hemorrhagic inflammatory changes in the gastrointestinal tract.

Time Course of Effects

Phosgene oxime causes immediate pain and irritation to all exposed skin and mucous membranes. The time course of damage to other tissue probably parallels that of damage to the skin.

DIAGNOSIS:

Other causes of urticaria and skin necrosis must be considered. Common urticants do not cause the extreme pain that phosgene oxime does.

Laboratory Findings

There are no distinctive laboratory findings.

MEDICAL MANAGEMENT:

Management is supportive. The skin lesion should be managed in the same way that a necrotic ulcerated lesion from another cause would be managed.

SUMMARY:

SIGNS AND SYMPTOMS:

Immediate burning and irritation followed by wheal-like skin lesions and eye and airway damage.

DIAGNOSIS:

Phosgene oxime, in both its liquid and vapor forms, cause moderate to severe pain on contact with skin or the mucous membranes of the eyes, nose, mouth and airways. It also produces visible grayish tissue damage within several minutes of contact. Later, severe damage of the skin, eyes and airways may appear.

TREATMENT:

Immediate decontamination; symptomatic management of lesions.

PROPHYLAXIS:

None

ISOLATION AND DECONTAMINATION:

Protect yourself by wearing a mask, gloves and a protective suit until the patient is decontaminated.

Remove patient from contamination and contamination from patient. Get the patient away from the source, such as by moving him upwind or out of a contaminated building. If it is absolutely certain that exposure was to vapor only, remove outer clothing. If there is a possibility of liquid contamination, all clothing must be removed and the patient must be showered or washed with soap and water, dilute hypochlorite or water.

BLOOD AGENTS (CYANIDE) AC—HYDROCYANIC ACID CK—CYANOGEN CHLORIDE

BACKGROUND:

Cyanide is a rapidly acting lethal agent that is limited in its military usefulness by its high LCt_{50} and high volatility. Death occurs in 6 to 8 minutes after inhalation of a high Ct. Sodium nitrite and sodium thiosulfate are effective antidotes.

Materials of interest as chemical agents are the cyanide **hydrogen cyanide (hydrocyanic acid; AC)** and the simple cyanogen, **cyanogen chloride (CK)**. Cyanogen bromide was used briefly in World War I, but is of no present interest.

CLINICAL FEATURES:

Toxicities: Cyanide is the least toxic of the "lethal" chemical agents. The **LCt_{50}s** of **AC** and **CK** by inhalation have been estimated to be **2500-5000 mg·min/m³** for **AC** and about **11,000 mg·min/m³** for **CK**. **LD_{50}s** for hydrogen cyanide have been estimated to be 1.1 mg/kg for intravenous administration and 100 mg/kg after skin exposure. The **oral LD_{50}s** for sodium and potassium cyanide are about 100 and 200 mg/kg respectively.

Cyanide is unique among military chemical agents because it is detoxified at a rate that is of practical importance, about 17 µg/kg. min. As a result the LCt_{50} is greater for a long exposure (e.g., 60 min) than for a short exposure (e.g., 2 min).

Effects: The organs most susceptible to cyanide are the central nervous system (CNS) and the heart. Most clinical effects are of CNS origin and are nonspecific.

About 15 seconds after inhalation of a high concentration of cyanide vapor concentration there is a transient hyperpnea followed in 15-30 seconds by the onset of convulsions. Respiratory activity stops two to three minutes later, and cardiac activity ceases several minutes later still, or at about six to eight minutes after exposure.

The onset and progression of signs and symptoms after ingestion of cyanide or after inhalation of a lower concentration of vapor are slower. The first effects may not occur until several minutes after exposure, and the time course of these effects depends on the amount absorbed and the

rate of absorption. The initial transient hyperpnea may be followed by feelings of anxiety or apprehension, agitation, vertigo, a feeling of weakness, nausea with or without vomiting, and muscular trembling. Later, consciousness is lost, respiration decreases in rate and depth, and convulsions, apnea, and cardiac dysrhythmias and standstill follow. Because this cascade of events is prolonged, diagnosis and successful treatment are possible.

The effects of cyanogen chloride include those described for hydrogen cyanide. Cyanogen chloride is also similar to the riot control agents in causing irritation to the eyes, nose, and airways as well as marked lacrimation, rhinorrhea, and bronchosecretions.

Physical Findings: Physical findings are few and non-specific. The two that are said to be characteristic are in fact not always observed. The first is severe respiratory distress in an acyanotic individual. When seen, "cherry-red" skin suggests either circulating carboxyhemoglobin from carbon monoxide poisoning or a high venous oxygen content from failure of extraction of oxygen by tissues poisoned by cyanide or hydrogen sulfide. However, cyanide victims may have normal appearing skin and may even be cyanotic, although cyanosis is not classically associated with cyanide poisoning.

The second classic sign is the odor of bitter almonds. However, about 50% of the population is genetically unable to detect the odor of cyanide.

The casualty may be diaphoretic with normal sized or large pupils. An initial hypertension and compensatory bradycardia are followed by a declining blood pressure and tachycardia. Terminal hypotension is accompanied by bradyarrhythmias before asystole.

Time Course of Effects

Effects begin in 15 seconds following inhalation of a lethal Ct; death ensues in six to eight minutes. The onset of effects following inhalation of lower Cts may be as early as minutes after the beginning of the exposure. After exposure is terminated by evacuation to fresh air or by masking, there is little danger of delayed onset of effects.

DIAGNOSIS:

Inhalational exposure to either cyanide or a nerve agent may precipitate the sudden onset of loss of consciousness followed by convulsions and apnea. The nerve agent victim has miosis (until shortly before death), copious oral and nasal secretions, and muscular fasciculations. The cyanide victim has normal sized or dilated pupils, few secretions, and muscular twitching but no fasciculations. In addition, the nerve agent victim may be cyanotic, and the cyanide victim usually is not cyanotic.

Laboratory Findings

1. An elevated blood cyanide concentration: Mild effects may be apparent at concentrations of 0.5-1.0 µg/mL, and concentrations of 2.5 µg/mL and higher are associated with coma, convulsions and death.

2. Acidosis: Metabolic acidosis with a high concentration of lactic acid (lactic acidosis), or a metabolic acidosis with an unexplained high anion gap (if the means to measure lactic acid are not available) may be present. Because oxygen cannot be utilized, anaerobic metabolism with the production of lactic acid replaces aerobic metabolism. Lactic acidosis, however, may reflect other disease states and is not specific for cyanide poisoning.

3. Oxygen content of venous blood greater than normal. This also is because of poisoning of the intramitochondrial respiratory chain and the resulting failure of cells to extract oxygen from arterial blood. This finding is also not specific for cyanide poisoning.

MEDICAL MANAGEMENT:

The primary goal in therapy is to remove the cyanide from the enzyme cytochrome a_3 in the cytochrome oxidase complex. A complicating factor is the rapidity with which cyanide, particularly inhaled cyanide, causes death.

A secondary goal is to detoxify or bind the cyanide so that it can not reenter the cell to reinhibit the enzyme. A closely associated goal is supportive management.

Methemoglobin has a high affinity for cyanide, and cyanide will preferentially bind to methemoglobin rather than to the cytochrome. Most methemoglobin formers have clinically significant side effects. The nitrites, which were first used to antagonize the effects of cyanide over a century ago, cause orthostatic hypotension, but this is relatively insignificant in a supine patient. Amyl nitrite, historically the first nitrite used, is a volatile substance formulated in a perle that is crushed or broken for the victim to inhale. In an apneic patient a means of ventilation is necessary.

Another methemoglobin former, sodium nitrite, is formulated for intravenous use. The standard ampule contains 300 mg of the drug in 10 mL of diluent, and this is injected intravenously over a two- to four-minute period.

Detoxification (metabolism) of cyanide is accomplished by the administration of a sulfur-containing compound that combines with cyanide to produce thiocyanate, a relatively non-toxic substance which is rapidly excreted via the kidneys. The hepatic enzyme rhodanese catalyzes the one-way reaction of cyanide and a sulfane to thiocyanate. Sodium thiosulfate is packaged in a 50-mL ampule containing 12.5 grams of the drug. Intravenous injection of all 12.5 grams follow successful completion of the intravenous injection of sodium nitrite. Half of the original dosage of each drug may be repeated if symptoms persist.

A commercially available Cyanide Antidote Kit, containing amyl nitrite, sodium nitrite, and sodium thiosulfate, is available to chemical depot medical facilities, military medical centers, and civilian facilities.

Supportive care consists of providing oxygen and correcting the metabolic acidosis. Although in theory oxygen should not help (because hemoglobin is saturated and the intracellular pathway for oxygen utilization is blocked), in both experimental studies and in actual patient management normobaric oxygen has provided some benefit. There is no firm evidence to support the use of hyperbaric oxygen. Correction of the acidosis has helped cyanide-poisoned patients in whom the etiology was not recognized and to whom the antidote was not given.

Other countries use different compounds. Germany uses the dimethylaminophenol (DMAP), a rapid methemoglobin former developed for intramuscular use. However, muscle necrosis at the site of injection occurs, and only the intravenous route of administration is recommended.

Certain cobalt compounds directly chelate cyanide to reduce its toxicity. Because cobalt compounds do not form the intermediate, methemoglobin, their antidotal activity may be faster than that of the methemoglobin-formers. Great Britain and France use cobalt edetate (Kelocyanor), but its clear superiority to the methemoglobin formers has not been demonstrated, and it occasionally causes severe side effects, particularly if the patient has only a mild exposure. The other cobalt compound sometimes used in Europe is hydroxycobalamin (vitamin B_{12a}), which complexes with cyanide on a molar basis; because of its high molecular weight with a large dose is required.

SUMMARY:

SIGNS AND SYMPTOMS:

Effects of Cyanide

- Small amount: no effects
- Medium amount: dizziness, nausea, feeling of weakness
- Large amount:

  Loss of consciousness

  Convulsions

  Breathing stops

  Death

  First effect: seconds

Effects of Cyanogen Chloride

- Small amount: irritation; giddiness, nausea, feeling of weakness
- Large amount: unconsciousness, convulsions

DIAGNOSIS:

People convulsing or who have convulsed from cyanide usually have **normal-sized to large pupils, usually do not have excessive secretions and do not have muscular fasciculations** (ripples under the skin)—**all of which are seen in nerve agent casualties**. On the other hand, those poisoned with cyanide often have skin that is redder than normal because of the reddish blood going through their veins. The odor of bitter almonds may be present.

TREATMENT:

Antidote: Intravenous sodium nitrite and sodium thiosulfate. **Supportive:** Oxygen; correct acidosis.

PROPHYLAXIS:

None

ISOLATION AND DECONTAMINATION:

Protect yourself by wearing a mask, gloves and a protective suit until the patient is decontaminated.

Remove patient from contamination and contamination from patient. Get the patient away from the source, such as by moving him upwind or out of a contaminated building. If it is absolutely certain that exposure was to vapor only, remove outer clothing. If there is a possibility of liquid contamination, all clothing must be removed and the patient must be showered or washed with soap and water, dilute hypochlorite or water.

ABC's (Airways, Breathing, Circulation)

CYANIDE VAPOR EFFECTS

Cyanide—How It Works

- Cyanide poisons cells (stops use of oxygen).
- The cell cannot use oxygen and it dies.
- Oxygen remains in the blood (blood stays red).

AC—Hydrocyanic Acid
CK—Cyanogen chloride

Moderate, from low concentration

Transient increase in rate and depth of breathing

Dizziness

Nausea, vomiting

Headache

These may progress to severe effects if exposure continues

The time of onset of these effects depends on the concentration, but is often within minutes after start of exposure

Severe, from high concentration

Transient increase in rate and depth of breathing—15 seconds

Convulsions—30 seconds

Cessation of respiration—2-4 minutes

Cessation of heartbeat—4-8 minutes

In addition to the above, CK causes intense irritation of the eyes, nose, and airways.

CHOKING (PULMONARY) AGENTS (PHOSGENE) CG

BACKGROUND:

Inhalation of selected organohalides, oxides of nitrogen (NO_x), and other compounds can result in varying degrees of pulmonary edema, usually after a symptom-free period that varies in duration with the amount inhaled. Chemically induced acute lung injury by these groups of agents involves a permeability defect in the blood-air-barrier (the alveolar-capillary membrane); however, the precise mechanisms of toxicity remain an enigma.

Phosgene is transported as a liquid. It spontaneously converted to a colorless, low-lying (density 4 x air) gas. Because of its relatively low boiling point (7.5°C), phosgene was often mixed with other substances. It has a characteristic odor of sweet, newly mown hay.

Mechanism of Toxicity

Phosgene is only slightly soluble in water and aqueous solutions. However, once dissolved it rapidly hydrolyzes to form carbon dioxide and hydrochloric acid. The early-onset ocular, nasal, and central airway irritation from high concentrations of phosgene is caused by hydrochloric acid released during phosgene hydrolysis; however, the carbonyl group (C=O) readily participates in acylation reactions with amino ($–NH_2$), hydroxyl (–OH), or sulfhydryl (–SH) groups and these reactions account for the major pathophysiological effects of phosgene. These acylations occur at alveolar-capillary membranes and lead to leakage of fluid from those capillaries into the interstitial portions of the lung. This effect is from direct contact of phosgene with these membranes; phosgene exposure by other routes, e.g., by intravenous administration, does not cause this damage.

Phosgene-induced leakage of fluid from capillaries into the pulmonary interstitium is normally opposed by lymphatic drainage from the parenchyma, but as the fluid leakage increases, normal drainage mechanisms become progressively overwhelmed. After a 20 minute to 24-hour long asymptomatic or latent period, fluid eventually reaches alveoli and peripheral airways, leading to increasingly severe dyspnea and clinically evident pulmonary edema.

CLINICAL FEATURES:

Toxicities: The odor threshold for phosgene is about 1.5 mg/m^3, and phosgene irritates the mucous membranes at 4 mg/m^3. The LCt_{50} is about 3200 mg•min/m^3.

Effects: Phosgene produces pulmonary edema following a clinical latent period of variable length that depends primarily on the intensity of exposure (i.e., the Ct) but also partly on the physical activity of the exposed individual. After the latent period, the patient experiences worsening respiratory distress that at first is unaccompanied by objectively verifiable signs of pulmonary damage but that may progress relentlessly to pulmonary edema and death.

During the time preceding the appearance of shortness of breath, individuals exposed to particularly high concentrations of organohalides may report symptoms associated with mucous membrane irritation. Exposure to large quantities of phosgene may irritate moist mucous membranes, presumably because of the generation of hydrochloric acid from the hydrolysis of phosgene. Transient burning sensation in the eyes with lacrimation and chemical conjunctivitis may coexist with mild, early-onset cough and a substernal ache with a sensation of pressure. Irritation of the larynx by very large concentrations of the agent may lead to sudden laryngeal spasm and death.

A clinical latent period during which the patient is asymptomatic may follow low Ct exposure or may follow the transient irritation associated with substantial phosgene exposure. This asymptomatic period may persist up to 24 hours after organohalide inhalation. The duration of this latent period is shorter following high Ct's and is shortened by physical exertion following exposure.

The most prominent symptom following the clinical latent period is dyspnea, perceived as shortness of breath with or without chest tightness. These sensations reflect hypoxemia, increased ventilatory drive, and decreased lung compliance, all of which result from the accumulation of fluid in the pulmonary interstitium and peripheral airways. Fine crackles appear at the lung bases, but these may not be clearly audible unless auscultation is conducted after a forced expiration. Later, auscultation reveals coarse crackles and râles in all lung fields, and increasing quantities of thin, watery secretions are noted. The buildup of fluid in the lungs has two clinically pertinent effects: First, developing pulmonary edema interferes with oxygen delivery to alveolar capillaries and may lead to hypoxemia, and if a sufficient percentage of hemoglobin is unoxygenated cyanosis will become apparent. Secondly, the sequestration of plasma-derived fluid (up to one liter per hour) in the lungs may lead to a hypovolemia and hypotension, interfering with oxygen delivery to the brain, kidneys, and other crucial organs. Death results from respiratory failure, hypoxemia, hypovolemia, or a combination of these factors. Hypoxia and hypotension may progress particularly rapidly and suggest a poor prognosis. The development of symptoms and signs of pulmonary edema within four hours of exposure is an especially accurate indicator of a poor prognosis; in the absence of immediately available intensive medical support, such patients are at high risk of a fatal outcome. Complications include infection of damaged lungs and delayed deaths following such respiratory infections.

DIAGNOSIS:

Phosgene is distinguished by its odor, its generalized mucous-membrane irritation in high concentrations, dyspnea, and **pulmonary edema of delayed onset**.

Riot control agents produce a burning sensation predominantly in the eyes and upper airways. This irritation is typically more intense than that caused by phosgene and is unaccompanied by the distinctive odor of phosgene.

Nerve agents induce the production of watery secretions as well as respiratory distress. However, their other characteristic effects distinguish nerve agent toxicity from organohalide inhalational injury.

The respiratory toxicity associated with vesicants is usually delayed but predominantly affects the central rather than the peripheral airways. Vesicant inhalation severe enough to cause dyspnea typically causes signs of airway necrosis, often with pseudomembrane formation and partial or complete upper airway obstruction. Finally, pulmonary parenchymal damage following vesicant exposure usually manifests itself as hemorrhage rather than pulmonary edema.

Laboratory Findings

No commonly available laboratory tests exist for the specific identification or quantitation of phosgene inhalation; however, an increase in the hematocrit may reflect the hemoconcentration induced by transudation of fluid into the pulmonary parenchyma. Arterial blood gases may show a low PaO_2 or $PaCO_2$, which are early nonspecific warnings of increased interstitial fluid in the lung.

Peak expiratory flow rate may decrease early after a massive phosgene exposure. This nonspecific test helps to assess the degree of airway damage and the effect of bronchodilator therapy. Decreased lung compliance and carbon monoxide diffusing capacity are particularly sensitive indicators of interstitial fluid volume in the lung, but are complex tests for hospital use only.

Early findings on chest x-ray are hyperinflation followed later by pulmonary edema without cardiovascular changes of redistribution or cardiomegaly. V/Q scanning is very sensitive but nonspecific and for hospital use only.

MEDICAL MANAGEMENT:

Terminate exposure as a vital first measure. This may be accomplished by physically removing the casualty from the contaminated environment or by isolating him from surrounding contamination by supplying a properly fitting mask. Decontamination of liquid agent on clothing or skin terminates exposure from that source.

Execute the ABCs of resuscitation as required. Establishing a patent airway is especially crucial in a patient exhibiting hoarseness or stridor; such individuals may face impending laryngeal spasm and require intubation. Establishing a clear airway also aids in interpretation of auscultatory findings. Steps to minimize the work of breathing must be taken. Because of the always present danger of hypotension induced by pulmonary edema or by positive airway pressure, accurate determination of the casualty's circulatory status is vital not just initially but also at regularly repeated intervals and whenever indicated by the clinical situation.

Enforce rest. Even minimal physical exertion may shorten the clinical latent period and increase the severity of respiratory symptoms and signs in an organohalide casualty, and physical activity in a symptomatic patient may precipitate acute clinical deterioration and even death. Strict limitation of activity (i.e., forced bed rest) and litter evacuation are mandatory for patients suspected of having inhaled any of the edemagenic agents. This is true whether or not the patient has respiratory symptoms and whether or not objective evidence of pulmonary edema is present.

Prepare to **manage airway secretions and prevent/treat bronchospasm.** Unless superinfection is present, secretions present in the airways of phosgene casualties are usually copious and watery; they may serve as an index to the degree of pulmonary edema and do not require specific therapy apart from suctioning and drainage. Antibiotics should be reserved for those patients with an infectious process documented by sputum gram staining and culture. Bronchospasm may occur in individuals with reactive airways, and these patients should receive theophylline, or β-adrenergic bronchodilators. Steroid therapy is also indicated for bronchospasm as long as parenteral administration is chosen over topical therapy, which may result in inadequate distribution to damaged airways. Methylprednisolone 700-1000 mg or its equivalent may be given in divided doses (i.v.) during the first day and then tapered during the duration of the clinical illness. The increased susceptibility to bacterial infection during steroid therapy mandates careful surveillance of the patient. No human studies have shown any benefit from steroids, and steroids are thus not recommended in individuals without evidence of overt or latent reactive airway disease.

Prevent/treat pulmonary edema. Positive airway pressure provides some control over the clinical complications of pulmonary edema. Early use of a positive pressure mask may be beneficial. Positive airway pressure may exacerbate hypotension by decreasing thoracic venous return, necessitating intravenous fluid administration and perhaps judicious use of the pneumatic antishock garment.

Prevent/treat hypoxia. Oxygen therapy is definitely indicated and may require supplemental positive airway pressure administered via one of the several available devices for generating intermittent or continuous positive pressure. Intubation with or without ventilatory assistance may be required, and positive pressure may need to be applied during at least the end-expiratory phase of the ventilator cycle.

Prevent/treat hypotension. Sequestration of plasma-derived fluid in the lungs may cause hypotension, which may be exacerbated by positive airway pressure. Urgent intravenous administration of either crystalloid or colloid (which in this situation appear equally effective) may need to be supplemented by the judicious application of the pneumatic anti-shock garment. The use of vasopressors is a temporizing measure until fluids can be replaced.

SUMMARY:

SIGNS AND SYMPTOMS:

Eye and airway irritation, dyspnea, chest tightness, and **delayed** pulmonary edema.

DIAGNOSIS:

The casualty will appear short of breath and may be coughing up clear frothy fluid. On auscultation there will be crackles and rales, initially at the bases and later throughout the lung fields.

Effects begin 2 to 24 hours after exposure.

TREATMENT:

Termination of exposure, ABCs of resuscitation, enforced rest and observation, oxygen with or without positive airway pressure for signs of respiratory distress, other supportive therapy as needed.

PROPHYLAXIS:

None

ISOLATION AND DECONTAMINATION:

Protect yourself by wearing a mask, gloves and a protective suit until the patient is decontaminated.

Remove patient from contamination and contamination from patient. Get the patient away from the source, such as by moving him upwind or out of a contaminated building. If it is absolutely certain that exposure was to vapor only, remove outer clothing. If there is a possibility of liquid contamination, all clothing must be removed and the patient must be showered or washed with soap and water, dilute hypochlorite or water.

ABC's: oxygen with or without assisted ventilation. Suction secretions.

INCAPACITATING AGENTS
CN = CHLORACETOPHENONE (MACE)
CS = ORTHO-CHLOROBENZYLMALONINITRILE
OC = OLEORESIN CAPSICUM (PEPPER-SPRAY)

BACKGROUND:

Riot control agents, also called irritants, lacrimators, and tear gas, produce transient discomfort and eye closure to render the recipient temporarily incapable of fighting or resisting. Law enforcement agencies use them for riot control and military forces use them for training and in combat. They have a high LCT_{50} and a low effective Ct_{50}, and therefore have a high safety ratio. Their major activity is to cause pain, burning, or discomfort on exposed mucous membranes and skin; these effects occur within seconds of exposure, but seldom persist more than a few minutes after exposure has ended.

Today CN is in commercially available devices for self-protection (Mace®), but CS is the agent otherwise used.

Unlike most agents, which are liquids under temperate conditions, riot control agents are solids with low vapor pressures and are dispersed as fine particles or in solution. Dispersion devices include small hand held spray cans, large spray tanks, grenades, and larger weapons.

The mechanism of biological activity is less well characterized for riot control agents than for most other agents; fortunately a detailed knowledge of the mechanism of action is not a prerequisite for appropriate medical management.

CS and CN are SN_2 alkylating agents (mustard, in contrast, is an SN_1 alkylator) and react readily at nucleophilic sites. Prime targets include sulfhydryl-containing enzymes, such as lactic dehydrogenase. In particular, CS reacts rapidly with the disulfhydryl form of lipoic acid, a coenzyme in the pyruvate decarboxylase system. It has been suggested that tissue injury may be related to inactivation of certain of these enzyme systems.

Pain can occur without tissue injury and may be bradykinin mediated. CS causes bradykinin release in vivo and in vitro, and elimination of bradykininogen in vivo abolishes the systemic response to CS.

The initial response to aerosolized CS is an increase in blood pressure and irregular respiration, suggestive of the Sherrington pseudoaffective response. By-passing the pain receptors of the nose and upper airway by endotracheal administration of CS leads to the same decrease in blood pressure and in respiration seen after intravenous injection and suggests that the initial pressor effect and irregular respiration are responses to a noxious stimulus rather than pharmacologic effects of CS.

CLINICAL FEATURES:

The main effects of riot control agents are pain, burning, and irritation of exposed mucous membranes and skin. These effects do not differ appreciably from one agent to another except in the case of DM (ADAMSITE), which will be discussed in a separate section.

Eye: The eye is the most sensitive organ to riot control agents. Contact with agent produces a sensation of conjunctival and corneal burning and leads to tearing, blepharospasm, and conjunctival injection. The severe blepharospasm causes the lids to close tightly and produces transient "blindness," an effect that could inhibit the recipient's ability to fight or resist. However, if the recipient opens his eyes, his vision is near normal even if a significant concentration of the agent persists.

Because these compounds are solids it is possible for a particle or clump to become embedded in the cornea or conjunctiva to cause tissue damage. With the caveat noted below, there is no evidence that this complication has ever occurred. However, a recipient seeking medical care for eye pain after exposure should have his eyes thoroughly decontaminated and undergo a thorough ophthalmic examination. It could be necessary to pick out the particles of agent from tissue.

Reviewers examined the evidence for permanent eye damage from riot control agents. In each instance, the damage was from a weapon fired from close range (about 50% were self-inflicted). The reviewers concluded that the blast force driving the agent deep into tissue (with or without the wadding of the weapon) was major cause of the permanent injuries. This should not happen under normal use.

Nose and mouth: Contact with the delicate mucous membranes of the nose produces a burning sensation, rhinorrhea, and sneezing; a similar burning sensation accompanied by increased salivation occurs after contact with the mouth.

Airways: Inhalation causes burning and irritation of the airways with bronchorrhea, coughing, and a perception of a "tight chest" or an inability to breathe. However, pulmonary function studies done immediately after exposure have shown minimal alterations.

An inhaled irritating compound might be expected to exacerbate a chronic pulmonary disease such as asthma, emphysema, or bronchitis, but this appears not to happen after CS or CN even though these agents have been used widely in mixed populations. The medical care provider should nevertheless anticipate airway problems in individuals with lung disease, particularly if they are exposed to higher than the average field use concentrations.

There is no evidence that CS causes permanent lung damage after one or several exposures to field concentrations. Following inhalation of lethal amounts animals died from severe airway damage 12-24 hours post-exposure, but survivors from large exposures had minimal or no pulmonary abnormalities. After multiple (50 or more) daily exposures to smaller amounts animals developed laryngitis and tracheitis.

Skin: Contact with skin causes a tingling or burning sensation and may cause erythema, particularly if the skin is raw or freshly abraded (e.g., shortly after shaving). The erythema begins several minutes after exposure and generally subsides 45-60 minutes after termination of exposure.

Under conditions of high temperature, high humidity, and high concentration of agent there may be more severe dermatitis starting with erythema hours after exposure and followed by vesication. Generally these are second-degree burns not unlike, but more severe than, sunburn. Firemen who entered contaminated buildings after summer riots several decades ago developed these lesions. After stirring up the contaminating particles, they later developed erythema and blisters on their exposed skin.

Hypersensitivity may develop. In one instance, an individual developed generalized vesication and high fever after an uneventful exposure to CS more than 20 years after his only and equally uneventful previous exposure.

Gastrointestinal tract: Gastrointestinal effects usually do not occur with most riot control agents (DM is an exception), although there may be retching or vomiting if the agent concentration is high, if the exposure is prolonged, or if the individual is sensitive.

Cardiovascular: A transient increase in heart rate and blood pressure has occurred in people immediately prior to an exposure to a riot control agent or immediately after onset of exposure. The heart rate and blood pressure returned essentially to pre-test ranges while exposure continued and may have been caused by the anxiety or the initial pain rather than to a pharmacological effect of these agents. This "alarm reaction" may cause adverse effects in one with preexistent cardiovascular disease.

Oral ingestion: Children occasionally eat CS and several adults have swallowed CS pellets. Aside from bouts of diarrhea and abdominal cramps (which might have been from the cathartics and antacids used as therapy) their courses have been uneventful. In animals, the LD_{50} is about 200 mg/kg (which is about 14 grams/70-kg person), an amount unlikely to be ingested even deliberately. A few animals fed lethal amounts (or greater) had gastric irritation or erosions, and several had signs of intestinal perforation. Recommended therapy after ingestion consists of cathartics, antacids, and surgical observation.

Lethality: CN, occasionally in combination with DM, has caused deaths in people who refused to exit a confined space. In each case the agent was used in excess. Death generally occurred hours after initial exposure, and post-mortem findings were those of severe airway damage similar to that seen in animals.

Metabolism: Animals given lethal amounts of CS by intravenous or intraperitoneal administration developed increased blood thiocyanate concentrations hours later, indicating that the malononitrile portion of CS had been metabolized to cyanide. Cyanide was not a factor in causing death (lung damage was). A significant increase in blood concentration of thiocyanate has not been noted after aerosol administration of CS. Several popular data bases mention this cyanogenic potential of CS and suggest that treatment of a CS casualty might require therapy for cyanide poisoning (this recommendation is apparently based on the i.v. or i.p. administration data). After receiving lethal amounts of CS by inhalation animals died 12-24 hours later from severe airway damage; cyanide was not implicated in their deaths.

DM

The effects of usual field concentrations of DM (Adamsite) are similar to those of the other riot control agents, except that DM has little irritancy to the skin. However, at higher concentrations, DM causes nausea, vomiting, and a feeling of generalized malaise. For this reason it is called a vomiting agent.

Time Course of Effects

Except for those produced by DM, the biological effects from these agents begin seconds after exposure and continue for 15 minutes or so after one exits the contamination to fresh, clean air. The effects from DM begin 3-4 minutes after the onset of exposure and may last an hour or two. (This is advantageous militarily as an individual, unaware of the agent, will continue to inhale it for several minutes and absorb a larger dose. He may then vomit, requiring mask removal, which leads to continued inhalation of agent.)

DIAGNOSIS:

Usually the circumstances of exposure make the diagnosis obvious. The history and the few physical signs (conjunctival injection with normal pupils, tearing, etc.) are usually adequate. The sudden onset of burning pain and irritation might lead one to consider Lewisite or phosgene oxime exposure, but the signs and symptoms of riot control agents gradually recede, whereas those from the vesicants worsen.

Laboratory Findings

There are no specific laboratory tests that will confirm the diagnosis. Complications, e.g., infection of a skin lesion, will produce the laboratory findings characteristic of the complication.

MEDICAL MANAGEMENT:

The effects of exposure to these agents under the usual field conditions usually are self limiting and require no specific therapy. Most will disappear in 15-30 minutes, although erythema may persist for an hour or longer.

The following section discusses potential complications occurring only under exceptional circumstances, such as exposure to a very large amount of agent (as in an enclosed space), exposure in adverse weather, or experimental studies in humans or animals. They are not to be expected with normal use of these agents.

Fewer than 1% of exposed people will have effects severe or prolonged enough to cause them to seek medical care. Those who do probably will have eye, airway, or skin complaints. Because there is no antidote for these agents, treatment consists of symptomatic management.

Eye: The eye should be carefully flushed with water or saline and impacted particles should be sought. General care consists of a topical solution (many are available) to relieve the irritation and topical antibiotics. An ophthalmalogist should be consulted for further evaluation and care.

Pulmonary: These agents may exacerbate chronic disease or unmask latent disease (although there is little evidence of this). Bronchospasm with wheezing and mild distress hours continuing after exposure may occur in a latent asthmatic and more severe effects and respiratory distress may occur in one with chronic bronchitis or emphysema. Management includes oxygen administration (with assisted ventilation, if necessary), bronchodilators if bronchospasm is present, and specific antibiotics dictated by the results of sputum studies (Gram stains of smears followed by culture). A specialist skilled in the treatment of inhalational injury should be consulted early. Animal studies and very limited human data indicate that maximal effects occur 12 hours after exposure.

Skin: The early erythema requires reassurance, but no specific therapy unless severe and prolonged more than an hour or two. The later-onset erythema, preciptated by a larger exposure in a hot and humid atmosphere, is usually more severe and less likely to resolve quickly; it may require the use of soothing compounds such as calamine, camphor, and mentholated creams. Small vesicles should be left intact, but larger ones will ultimately break and should be drained. Irrigation of denuded areas several times a day should be followed by the application of a topical antibiotic. Large oozing areas have responded to compresses containing substances such as colloidal oatmeal, Burow's solution, and other dermatologic preparations.

SUMMARY:

SIGNS AND SYMPTOMS:

Burning and pain on exposed mucous membranes and skin, eye pain and tearing, burning in the nostrils, respiratory discomfort, and tingling of the exposed skin. OC will cause stomach, abdominal and skin irritation along with constriction of the lungs bronchial tubes. OC—medical effects usually develop 60 seconds following contact. Remains about 45 minutes. CS and CN—signs and symptoms last about 15-30 minutes.

Effects start seconds after exposure.

DIAGNOSIS:

Burning in the eyes is usually accompanied by tearing, redness and closure of the eyes. If an individual inhales these substances, there is discomfort in the airways and a feeling of difficulty breathing or of a tight chest. They may also irritate or burn the skin, particularly if the temperature is warm and the skin is moist.

TREATMENT:

Immediate management: Usually none is necessary; effects are self limiting.

PROPHYLAXIS:

None

ISOLATION AND DECONTAMINATION:

Eyes: Thoroughly flush with water, saline, or similar substance. Skin: Flush with copious amounts of water, alkaline soap and water, or a mildly alkaline solution (sodium bicarbonate or sodium carbonate). Generally, decontamination is not needed if the wind is brisk. Hypochlorite exacerbates the skin lesion and should not be used.

TREATMENT PROTOCOLS

FOR THE BIOLOGICAL AGENT CONTAMINATED PATIENT

BIOLOGICAL AGENTS

- Biological agents are non-volatile and will be disseminated as either liquid or solid aerosols, where the biological materials will be subjected to the environment. Many biological agents are living organisms and adverse temperature and humidity will affect them. Sunlight, in particular ultraviolet rays, will kill many of them. In this environment, most will only last a few hours or days. Because of this, use of biological agents is more likely at night or in enclosed areas.

- By weight, biological agents are generally more toxic than chemical agents. For example, Ricin, one of the toxins, is 2 to 3 times more toxic than VX and Botulinium, another toxin, is 5,000 to 10,000 times more toxic than VX.

- Are invisible to our senses. We cannot see, taste or smell them.

- Unfortunately, there are limited methods to detect them in the field. This means that we may not know when we are under biological attack.

- As we look at biological agents, you will see some similarities with what we discussed earlier with chemical agents, but you will also note some significant differences.

- From a responder's point of view, the biggest difference is time. Unlike chemical agents, most of which have an immediate effect, most biological agents all have a delayed effect ranging from several hours to days, and in some cases weeks. Therefore, when you respond to a biological incident, there may be no casualties and nothing significant unless you or someone else happens to witness the actual release or some type of suspected dissemination device has been located.

- Are not dermally active: Unlike the nerve and blister agents, biological agents cannot penetrate healthy unbroken skin. (An exception is T-2 Mycotoxin, which causes skin damage). To cause disease, most biological agents must be inhaled or ingested. Our skin provides a good barrier to most agents, in contrast to some chemical agents which can cause toxic reactions and symptoms if placed on the skin.

- Since biological agents are particulate in form, they are usually disseminated as an aerosol (suspension of particles in air). In this Form the agent can most readily get into the lungs.

- Range of effects: Biological agents have a variety of effects, depending on the organism and how it affects us, the dose we receive and the route of entry. This range can run from skin irritation through death.

- Obtained from nature: Each of the biological agents has a natural host. In some instances, with little training or equipment, a small amount of culture or material can be "grown" into larger quantities which are then placed in a dissemination device.

- They can enter the body through inhalation or ingestion, through a break in the skin, or through other body openings or orifices. In a deliberate use, inhalation through the lungs is usually the targeted portal of entry.

- Some bacteria and viruses can cause epidemics, by being transmitted from one infected individual to another. This is true of only a few of the agents such as pneumonic plague (bacteria), smallpox and viral hemorrhagic fevers (viruses).

BACTERIAL AGENTS

Bacteria are unicellular organisms. They vary in shape and size from spherical cells—cocci—with a diameter of 0.5-1.0 μ m (micrometer), to long rod-shaped organisms—bacill— which may be from 1-5 μ m in size. Chains of bacilli may exceed 50 μ m. The shape of the bacterial cell is determined by the rigid cell wall. The interior of the cell contains the nuclear material (DNA), cytoplasm, and cell membrane, that are necessary for the life of the bacterium. Many bacteria also have glycoproteins on their outer surfaces which aid in bacterial attachment to surface receptors on cells and are of special importance in their ability to cause disease. Under special circumstances some types of bacteria can transform into spores. The spore of the bacterial cell is more resistant to cold, heat, drying, chemicals and radiation than the bacterium itself. Spores are a dormant form of the bacterium and, like the seeds of plants, they can germinate when conditions are favorable.

Bacteria can cause diseases in human beings and animals by means of two mechanisms which differ in principle: in one case by invading the tissues, in the other by producing poisons (toxins). In many cases pathogenic bacteria possess both properties. The diseases they produce often respond to specific therapy with antibiotics. This manual will cover several of the bacteria or rickettsia considered to be potential BW threat agents: *Bacillus anthracis* (Anthrax), *Brucella spp.* (Brucellosis), *Vibrio cholerae*(Cholera), *Burkholderia mallei*(Glanders), *Yersinia pestis* (Plague), *Francisella tularensis* (Tularemia), and *Coxiella burnetii*(Q Fever).

Biological Agent Quick Reference Guide

BIOLOGICAL AGENTS

AGENT	INCUBATION	LETHALITY	PERSISTANCE	DISSEMINATION
Bacteria				
Anthrax	1-5 days	3-5 days fatal	Very stable	Aerosol
Cholera	12 hours-6 days	Low with treatment High without treatment	Unstable Stable in saltwater	Aerosol Sabotage of water
Plague	1-3 days	1-6 days fatal	Extremely stable	Aerosol
Tularemia	1-10 days	2 weeks moderate	Very stable	Aerosol
Q fever	14-26 days	Weeks?	Stable	Aerosol Sabotage
Viruses				
Smallpox	10-12 days	High	Very stable	Aerosol
Venezuelan Equine Encephalitis	1-6 days	Low	Unstable Vectors	Aerosol
Ebola	4-6 days	7-16 days fatal	Unstable	Aerosol Direct contact
Biological Toxins				
Botulinum toxins	Hours to days	High without treatment	Stable	Aerosol Sabotage
Staphylococcal enterotoxin B	1-6 hours	Low	Stable	Aerosol Sabotage
Ricin	Hours to days	10-12 days fatal	Stable	Aerosol Sabotage
Tricothecene mycotoxins (T2)	2-4 hours	Moderate	Extremely stable	Aerosol Sabotage

BIO AGENTS
STANDARD PRECAUTIONS
HEALTHCARE PROVIDERS

	BACTERIAL AGENTS	Anthrax	Brucellosis	Cholera	Glanders (rarely seen)	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 Precaution																			
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			X								X			X					
Airborne Precautions					X						X								
Use of **N95** mask by **all** individuals entering the room											X								
Droplet Precautions							X					X							
Wash hands with antimicrobial soap			X	X							X			X					
Patient Placement																			
No restrictions		X						X								X	X	X	X
Cohort 'like' patients when private room unavailable				X		X	X		X				X						
Private Room			X	X	X	X	X				X	X		X					
Negative Pressure											X								
Door closed at all times					X						X								
Patient Transport																			
No restrictions		X						X	X				X			X	X	X	X
Limit movement to essential medical purposes only			X	X	X	X	X				X	X		X					
Place mask on patient to minimize dispersal of droplets					X		X				X	X							
Cleaning,Disinfection of Equipment																			
Routine terminal cleaning of room with hospital-approved disinfectant upon discharge				X	X			X	X		X	X	X			X	X	X	X
Disinfect surfaces with bleach/water sol.1:9 (10% sol.)		X	X			X	X							X					
Dedicated equipment disinfected prior to leaving room			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	X
Routine medical waste handled per internal policy		X	X	X	X	X	X	X	X		X	X	X	X		X	X	X	X
Discharge Management																			
No special discharge instruction necessary		X		X	X			X	X			X	X			X	X	X	X
Home care providers should be taught principles of **Standard Precautions**		X	X			X	X							X					
Patient not discharged from hospital until determined to be no longer infectious							X				X			X					
Patient generally not discharged until 72 hours of antibiotics completed							X												
Post-morten Care																			
Follow principles of **Standard Precautions**		X	X	X	X	X	X	X	X		X	X	X	X		X	X	X	X
Droplet Precautions							X												
Airborne Precautions											X								
Use of **N95** mask by **all** individuals entering the room											X								
Negative Pressure											X								
Contact Precautions											X			X					
Routine terminal cleaning of room with hospital-approved disinfectant upon autopsy			X	X	X			X	X		X	X	X			X	X	X	X
Disinfect surfaces with bleach/water sol.1:9 (10% sol.)		X				X	X							X					

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: **Handwashing,gloves** when contact with above, **mask/eye protection/face shield** while performing procedures.

ANTHRAX

BACKGROUND:

Bacillus anthracis, the causative agent of Anthrax, is a rod-shaped, gram-positive, sporulating organism with the spores constituting the usual infective form. Anthrax is primarily a zoonotic disease of herbivores, with cattle, sheep and horses being the usual domesticated animal hosts, but other animals may be infected. Human disease may be contracted by handling contaminated hair, wool, hides, flesh, blood and excreta of infected animals and from manufactured products such as bone meal, as well as by purposeful dissemination of spores. Infection is introduced through scratches or abrasions of the skin, wounds, inhalation of spores, eating insufficiently cooked infected meat, or by flies. All human populations are susceptible. Recovery from an attack of the disease may be followed by immunity. The spores are very stable and may remain viable for many years in soil and water. They will resist sunlight for varying periods.

POTENTIAL FOR SECONDARY CONTAMINATION:

Standard precautions for healthcare workers. After an invasive procedure or autopsy is performed, the instruments and area used should be thoroughly disinfected with a sporicidal agent (chlorine).

CLINICAL FEATURES:

Anthrax presents as three distinct clinical syndromes in man: cutaneous, inhalational, and gastrointestinal disease. The cutaneous form (also referred to as malignant pustule) occurs most frequently on the hands and forearms of persons working with infected livestock. It begins with a papule followed by formation of a blister-like fluid-filled vesicle. The vesicle typically dries and forms a coal-black scab, hence the term anthrax (Greek for coal). Sometimes this local infection will develop into a systemic infection which is often fatal. Endemic inhalational anthrax, known as Woolsorters' disease, is a rare infection contracted by inhalation of the spores. It occurs mainly among workers handling infected hides, wool, and furs. The intestinal form, which is also very rare in man, is contracted by the ingestion of insufficiently cooked meat from infected animals. In man, the mortality of untreated cutaneous anthrax ranges up to 25 percent; in inhalational and intestinal cases, the case fatality rate is almost 100 percent.

DIAGNOSIS:

After an incubation period of 1-6 days, presumably dependent upon the dose and strain of inhaled organisms, the onset of inhalation anthrax is gradual and nonspecific. Fever, malaise, and fatigue may be present, sometimes in association with a nonproductive cough and mild chest discomfort. These initial symptoms are often followed by a short period of improvement (hours to 2-3 days), followed by the abrupt development of severe respiratory distress with dyspnea, diaphoresis, stridor, and cyanosis. Shock and death usually follow within 24-36 hours after the onset of respiratory distress. Physical findings are typically non-specific. The chest X-ray may reveal a widened mediastinum ± pleural effusions late in the disease in about 55% of the cases, but typically is without infiltrates. *Bacillus anthracis* will be detectable by Gram stain of the blood and by blood culture with routine media, but often not until late in the course of the illness. Only vegetative encapsulated bacilli are present during infection. Spores are not found within the body unless it is open to ambient air. Studies of inhalation anthrax in non-human primates (rhesus monkey) showed that bacilli and toxin appear in the blood late in the course of illness.

MEDICAL MANAGEMENT IN HOSPITAL:

Almost all inhalational anthrax cases in which treatment was begun after patients were significantly symptomatic have been fatal, regardless of treatment. Penicillin has been regarded as the treatment of choice, with 2 million units given intravenously every 2 hours. Tetracyclines and erythromycin have been recommended in penicillin allergic patients. The vast majority of naturally-occurring anthrax strains are sensitive *in vitro* to penicillin. However, penicillin-resistant strains exist naturally, and one has been recovered from a fatal human case. Moreover, it might not be difficult for an adversary to induce resistance to penicillin, tetracyclines, erythromycin, and many other antibiotics through laboratory manipulation of organisms. All naturally occurring strains tested to date have been sensitive to erythromycin, chloramphenicol, gentamicin, and ciprofloxacin. In the absence of information concerning antibiotic sensitivity, treatment should be instituted at the earliest signs of disease with intravenous ciprofloxacin (400 mg q 8-12 hrs) or intravenous doxycycline (200 mg initially, followed by 100 mg q 12 hrs). Supportive therapy for shock, fluid volume deficit, and adequacy of airway may all be needed.

Standard Precautions should be practiced. After an invasive procedure or autopsy, the instruments and area used should be thoroughly disinfected with a sporicidal agent. Iodine can be used, but must be used at disinfectant strengths, as antiseptic-strength iodophors are not usually sporicidal. Chlorine, in the form of sodium or calcium hypochlorite, can also be used, but with the caution that the activity of hypochlorites is greatly reduced in the presence of organic material.

SUMMARY:

Signs and Symptoms: Incubation period is 1-6 days. Fever, malaise, fatigue, cough and mild chest discomfort is followed by severe respiratory distress with dyspnea, diaphoresis, stridor, and cyanosis. Shock and death occurs within 24-36 hours after onset of severe symptoms.

Diagnosis: Physical findings are non-specific. A widened mediastinum may be seen on CXR. Detectable by Gram stain of the blood and by blood culture late in the course of illness.

Treatment: Although effectiveness may be limited after symptoms are present, high dose antibiotic treatment with penicillin, ciprofloxacin, or doxycycline should be undertaken and supportive therapy maintained.

CHOLERA

BACKGROUND:

*Vibrio cholerae*is a short, curved, motile, gram-negative, non-sporulating rod. There are two serogroups, 01 and 0139, that have been associated with cholera in humans. The 01 serotype exists as 2 biotypes, classical and El Tor. The organisms are facultative anaerobes, growing best at a pH of 7.0, but able to tolerate an alkaline environment. They do not invade the intestinal mucosa, but rather "adhere" to it. Cholera is the prototype toxigenic diarrhea, which is secretory in nature. All strains elaborate the same enterotoxin, a protein molecule with a molecular weight of 84,000 daltons. The entire clinical syndrome is caused by the action of the toxin on the intestinal epithelial cell. Fluid loss in cholera originates in the small intestine with the colon being relatively insensitive to the toxin. The large volume of fluid produced in the upper intestine overwhelms the capacity of the lower intestine to absorb. Transmission is made through direct or indirect fecal contamination of water or foods, and by heavily soiled hands or utensils. All populations are susceptible, while natural resistance to infection is variable. Recovery from an attack is followed by a temporary immunity which may furnish some protection for years. The organism is easily killed by drying. It is not viable in pure water, but will survive up to 24 hours in sewage, and as long as 6 weeks in certain types of relatively impure water containing organic matter. It can withstand freezing for 3 to 4 days. It is readily killed by dry heat at 117° C, by steam and boiling, by short exposure to ordinary disinfectants, and by chlorination of water.

Standard Precautions for healthcare workers. Personal contact rarely causes infection; however, enteric precautions and careful hand-washing should by employed. Bactericidal solutions (hypochlorite) would provide adequate decontamination.

CLINICAL FEATURES:

Cholera is an acute infectious disease, characterized by sudden onset with nausea, vomiting, profuse watery diarrhea with 'rice water' appearance, the rapid loss of body fluids, toxemia, and frequent collapse. Mortality can range as high as 50 percent in untreated cases.

DIAGNOSIS:

After an incubation period varying from 4 hours to 5 days (average 2-3 days), presumably dependent upon the dose of ingested organisms, onset is usually rather sudden, although the clinical manifestations range from an asymptomatic carrier state to severe illness. Initially the disease presents with intestinal cramping and painless diarrhea. Vomiting, malaise and headache often accompany the diarrhea, especially early in the illness. If fever is present, it is usually low grade. Diarrhea may be mild or profuse and watery, with fluid losses exceeding 5 to 10 liters or more per day. Electrolyte loss can explain almost all clinical signs and symptoms. Without treatment, death may result from severe dehydration, hypovolemia and shock.

On microscopic examination of stool samples there are few or no red cells or white cells and almost no protein. The absence of inflammatory cells and erythrocytes reflects the non-invasive character of *V. cholerae* infection of the intestinal lumen. The organism can be identified in liquid stool or enrichment broths by darkfield or phase contrast microscopy, and by identifying darting motile vibrio. The organism must be transported using Cary-Blair medium and then streaked for isolation onto TCBS (Thiosulfate Citrate Bile Salt Sucrose) medium. Bacteriologic identification is not necessary to treat cholera, as it can be diagnosed clinically.

MEDICAL MANAGEMENT IN HOSPITAL:

Treatment of cholera depends primarily on replacement of fluid and electrolyte losses. This is best accomplished using oral rehydration therapy with the World Health Organization solution (3.5 g NaCl, 2.5 g $NaHCO_3$, 1.5 g KCl and 20 g of glucose per liter). Intravenous fluid replacement is occasionally needed in patients with persistent vomiting or high rates of stool loss (>10ml/kg/hr). Antibiotics will shorten the duration of diarrhea and thereby reduce fluid losses. Tetracycline (500 mg every 6 hours for 3 days) or doxycycline (300 mg once or 100 mg every 12 hours for 3 days) is generally adequate. However, due to widespread tetracycline resistance, ciprofloxacin (500 mg every 12 hours for 3 days) or erythromycin (500 mg every 6 hours for 3 days) should be considered. For pediatric treatment, tetracycline (50 mg/kg/d divided into 4 doses $\times$ 3 days) can be used, as dental staining has only occurred after >6 courses of treatment lasting 6 or more days. Alternates are erythromycin (40 mg/kg/d divided into 4 doses $\times$ 3 days), trimethoprim 8 mg and sulfamethoxazole 40 mg/kg day divided into 2 doses $\times$ 3 days, and furazolidone (5 mg/kg/d divided into 4 doses $\times$ 3 days or 7 mg/kg $\times$ one dose).

SUMMARY:

Signs and Symptoms: Incubation period 4 hours to 5 days; average 2-3 days. Asymptomatic to severe with sudden onset. Vomiting, headache, intestinal cramping with little or no fever followed rapidly by painless, voluminous diarrhea. Fluid losses may exceed 5 to 10 liters per day. Without treatment, death may result from severe dehydration, hypovolemia and shock.

Diagnosis: Clinical diagnosis. 'Rice water' diarrhea and dehydration. Microscopic exam of stool samples reveals few or no red or white cells. Can be identified by darkfield or phase contrast microscopy, and by direct visualization of darting motile vibrio.

Treatment: Fluid and electrolyte replacement. Antibiotics (tetracycline, ciprofloxacin or erythromycin) may shorten the duration of diarrhea and, more importantly, reduce shedding of the organism.

Prophylaxis: A licensed, killed vaccine is available but provides only about 50 percent protection that lasts for no more than 6 months. Vaccination schedule is at 0 and 4 weeks, with booster doses every 6 months.

BRUCELLOSIS

BACKGROUND:

The Brucellae are a group of gram-negative cocco-baccillary organisms, of which four species are pathogenic in humans. Abattoir and laboratory worker infections suggest that *Brucella* spp. are highly infectious via the aerosol route. It is estimated that inhalation of only 10 to 100 bacteria is sufficient to cause disease in man. The relatively long and variable incubation period (5-60 days) and the fact that many infections are asymptomatic under natural conditions has made it a less desirable agent for weaponization, although large aerosol doses may shorten the incubation period and increase the clinical attack rate. Brucellosis infection has a low mortality rate (5% of untreated cases) with most deaths caused by endocarditis or meningitis. It is an incapacitating and disabling disease in its natural form.

CLINICAL FEATURES:

Brucellosis may present as a nonspecific febrile illness which resembles influenza. Fever, headache, myalgia, arthralgia, back pain, sweats, chills, and generalized weakness and malaise are common complaints. Cough and pleuritic chest pain may occur in up to twenty percent of cases, but these are usually not associated with acute pneumonitis. Pulmonary symptoms may not correlate with radiographic findings. The chest x-ray may be normal, or show lung abscesses, single or miliary nodules, bronchopneumonia, enlarged hilar lymph nodes, and pleural effusions. Gastrointestinal symptoms occur in up to 70 percent of adult cases, and less frequently in children. These include anorexia, nausea, vomiting, diarrhea and constipation Ileitis, colitis and granulomatous or a mononuclear infiltrative hepatitis may occur. Lumbar pain and tenderness can occur in up to 60% of cases and is due to various osteoarticular infections of the axial skeletal system. Paravertebral abscesses may occur and can be imaged by CT scan or MRI. CT scans often show vertebral sclerosis. Vertebral and disc space destruction may occur in chronic cases. One or, less frequently, both sacroiliac joints may be infected causing low back and buttock pain that is intensified by stressing the sacroiliac joints on physical exam. Hepatomegaly and splenomegaly can occur in up to 45-63 percent of cases. Peripheral joint involvement may vary from pain on range of motion testing to joint immobility and effusion. Peripheral joint effusions usually show a mononuclear cell predominance and organisms can be isolated in up to 50% of cases. The hip joints are the most commonly involved peripheral joints, but ankle, knee, and sternoclavicular joint infection may occur. Plain radiographs of involved sacroiliac joints usually show blurring of articular margins and widening of the joint space. Technetium or Gallium-67 bone scans are 90% sensitive for detecting sacroileitis and will also detect other sites of bone and joint involvement; they are also useful for differentiating sacroiliac from hip joint involvement.

Meningitis occurs in less than 5% of cases and may be an acute presenting illness of a chronic syndrome occurring late in the course of a persistent infection. The cerebrospinal fluid contains an increased number of lymphocytes and a low to normal glucose. Culture of the CSF has sensitivity of 50%, and specific brucella antibodies can be detected in the fluid in a higher percentage of cases. Encephalitis, peripheral neuropathy, radiculoneuropathy and meningovascular syndromes have also been observed in rare cases. Behavioral disturbances in children and psychoses may occur in the meningoencephalitic form of the disease. Epididymo-orchitis may occur in men as the most frequent genitourinary form of brucellosis. Rashes occur in less than 5% of cases and include macules, papules, ulcers, purpura, petechiae, and erythema nodosum.

DIAGNOSIS:

The leukocyte count is usually normal but may be low. Anemia and thrombocytopenia may occur. Blood and bone marrow culture during the acute febrile phase of the illness will yield a positivity rate of 15-70% and 92% respectively. A biphasic culture method for blood (Castaneda bottle) may increase the number of isolates. The serum agglutination test (SAT) will detect both IgM and IgG antibodies. A titer of 1:160 or greater is indicative of active disease. The IgM titer can be measured by adding a reduced agent such as 2-mercaptoethanol to the serum. This will destroy the agglutinability of IgM allowing the IgM titer to be measured by subtracting the now lower titer from the total serum agglutinin titer. A dot-ELISA using an autoclaved extract of *B. abortus* has been found to be a sensitive and specific screening test for detection of *Brucella* antibodies under field conditions. ELISA tests for antibody detection require standardization using a specific antigen before they will be widely available. Antigen detection on DNA extracted from blood mononuclear cells has been accomplished using PCR analysis of a target sequence on the 31-kilodalton *B. abortus* protein BCSP 31. This test has been proven to be rapid and specific and may replace blood culture in the future, since the latter may require incubation for up to 6 weeks. PCR for *Brucella* species is not available at this time except in research laboratories, but shows promise for future use.

MEDICAL MANAGEMENT IN THE HOSPITAL:

Isolation is not required other than contact isolation for draining lesions. Person to person transmission is possible via contact with such lesions. Biosafety level 3 practices should be used for suspected brucella cultures in the laboratory because of the danger of inhalation infection. Antibiotic therapy is recommended as the sole therapy unless there are surgical indications for the treatment of localized diseases (e.g., valve replacement for endocarditis).

The treatment recommended by the World Health Organization for acute brucellosis in adults is doxycycline 200 mg/day p.o. plus rifampin 600-900 mg/day for a minimum of six weeks. The previously established regimen of intramuscular streptomycin along with an oral tetracycline may give fewer relapses but is no longer the primary recommendation. Ofloxacin 400 mg/day and rifampin 600 mg/day p.o. is also an effective combination. Combination therapy with rifampin, a tetracycline, and an aminoglycoside is indicated for infections with complications such as meningoencephalitis or endocarditis. Doxycycline clearance is increased in the presence of rifampin and plasma levels are lower than when streptomycin is used instead of rifampin.

SUMMARY:

Signs and Symptoms: Incubation period from 5-60 days; average of 1-2 months. Highly variable. Acute and subacute brucellosis are non-specific. Irregular fever, headache, profound weakness and fatigue, chills, sweating, arthralgias, myalgias. Depression and mental status changes. Osteoarticular findings (i.e., sacroiliitis, vertebral osteomyleitis). Fatalities are uncommon.

Diagnosis: Blood cultures require a prolonged period of incubation in the acute phase. Bone marrow cultures produce a higher yield. Confirmation requires phage-typing, oxidative metabolism, or genotyping procedures. ELISA's followed by Western blotting are used.

Treatment: Doxycycline and rifampin for a minimum of six weeks. Olfloxacin + rifampin is also effective. Therapy with rifampin, a tetracycline, and an aminoglycoside is indicated for infections with complications such as endocarditis or meningoencephalitis.

Prophylaxis: No approved human vaccine is available. Avoid consumption of unpasteurized milk and cheese.

Isolation and Decontamination: Standard precautions for healthcare workers. Person-to-person transmission via tissue transplantation and sexual contact have been reported but are insignificant. Environmental decontamination can be accomplished with a 0.5% hypochlorite solution.

GLANDERS

BACKGROUND:

The causative agent of Glanders is *Burkholderia*(formerly Pseudomonas) *mallei*, a gram-negative bacillus primarily noted for producing disease in horses, mules, and donkeys. In the past man has seldom been infected, despite frequent and often close contact with infected animals. This may be due to exposure to low concentrations of organisms from infected sites in sick animals and the fact that strains virulent for equids are often less virulent for man. There are four basic forms of disease in horses and man. The acute forms are more common in mules and donkeys and death typically follows in 3 to 4 weeks. The chronic form of the disease is more common in horses and causes generalized lymphadenopathy, multiple skin nodules that ulcerate and drain, and induration, enlargement and nodularity of regional lymphatics on the extremities and in other areas. The lymphatic thickening and induration has been called farcy. Human cases have occurred primarily in veterinarians, horse and donkey caretakers, and abattoir workers. The organism spreads to man by invading the nasal, oral, and conjunctival mucous membranes, by inhalation into the lungs, and by invading abraded or lacerated skin. Aerosols from cultures have been observed to be highly infectious to laboratory workers. Work with this organism in the laboratory requires biosafety level 3 containment practices. Despite the rarity of contagion to man from infected horses and donkeys, the attack rates caused by laboratory aerosols have been as high as 46% and cases have been severe. Since aerosol spread is efficient, and there is no available vaccine or really dependable therapy, *B. mallei* has been viewed as a potential BW agent. The disease in Equidae in its natural form poses a minimal threat to military personnel.

CLINICAL FEATURES:

Glanders may occur in an acute localized form, as a septicemic rapidly fatal illness, or as an acute pulmonary infection. Combinations of these syndromes commonly occur in human cases. A chronic cutaneous form with lymphangitis and regional adenopathy is also frequent.

Aerosol infection produced by a BW weapon containing *B. mallei* could produce any of these syndromes. The incubation period ranges from 10-14 days, depending on the inhaled dose and agent virulence. The septicemic form begins suddenly with fever, rigors, sweats, myalgia, pleuritic chest pain, photophobia, lacrimation, and diarrhea. Physical examination may reveal fever, tachycardia, cervical adenopathy and mild splenomegaly. Blood cultures are usually negative until the patient is moribund. Mild leukocytosis with a shift to the left or leukopenia may occur.

The pulmonary form may follow inhalation or arise by hematogenous spread. Systemic symptoms as described for the septicemic form occur. Chest radiographs may show miliary nodules (0.5-1.0 cm) and/or a bilateral bronchopneumonia, segmental, or lobar pneumonia and necrotizing nodular lesions.

Acute infections of the oral, nasal and/or conjunctival mucosa can cause mucopurulent, blood streaked discharge from the nose, associated with septal and turbinate nodules and ulcerations. If systemic invasion occurs from mucosal or cutaneous lesions then a papular and/or pustular rash may occur that can be mistaken for smallpox (another possible BW agent).

The chronic form is unlikely to be present within 14 days after a BW aerosol attack. It is characterized by cutaneous and intramuscular abscesses on the legs and arms. These lesions are associated with enlargement and induration of the regional lymph channels and nodes. Rare cases develop osteomyelitis, brain abscess, and meningitis. Recovery from chronic glanders may occur or the disease may erupt into an acute septicemic illness. Nasal discharge and ulceration are present in 50% of chronic cases.

DIAGNOSIS:

Gram stain of lesion exudates reveals small gram negative bacteria. These stain irregularly with methylene blue. *B. mallei* grows slowly on ordinary nutrient agar, but growth is accelerated with addition of 1-5% glucose and or 5% glycerol. Primary isolation requires 48 hous at 37.5 °C. Growth is also rapid on most meat infusion nutrient media. Agglutination tests are not positive for 7-10 days, and a high background titer in normal sera (1:320 to 1:640) makes interpretation difficult. Complement fixation tests are more specific and are considered positive if the titer is equal to, or exceeds 1:20. Cultures of autopsy nodules in septicemic cases will usually establish the presence of *B. mallei*. Occurrence in the absence of animal contact and/or in a human epidemic form is presumptive evidence of a BW attack. Mortality will be high despite antibiotic use. In the hamster 1 to 10 organisms administered by aerosol is lethal. “Resistant species” such as albino mouse can be infected with higher inhalation doses.

MEDICAL MANAGEMENT IN HOSPITAL:

Standard Precautions should be used to prevent person-to-person transmission in proven or suspected cases. Sulfadiazine 100 mg/kg per day in divided doses for 3 weeks has been found to be effective in experimental animals and in humans. Other antibiotics that have been effective in experimental infection in hamsters include doxycycline, rifampin, trimethoprim-sulfamethoxazole, and ciprofloxacin. The limited number of infections in humans has precluded therapeutic evaluation of most of the antibiotic agents, therefore, most antibiotic sensitivities are based on animal *in vitro* studies. Various isolates have markedly different antibiotic sensitivities, so that each isolate should be tested for its own individual resistance pattern.

SUMMARY:

Signs and Symptoms: Incubation period ranges from 10-14 days after inhalation. Inhalational exposure produces fever, rigors, sweats, myalgia, headache, pleuritic chest pain, cervical adenopathy, splenomegaly, and generalized papular/pustular eruptions. Almost always fatal without treatment.

Diagnosis: Methylene blue stain of exudates may reveal scant small bacilli. CXR may show miliary lesions, small multiple lung abscesses, or bronchopneumonia. *B. mallei* can be cultured from infected secretions using meat nutrients.

Treatment: Few antibiotics have been evaluated *in vivo*. Sulfadiazine may be effective in some cases. Ciprofloxacin, doxycycline, and rifampin have in vitro efficacy. Extrapolating from melioidosis guidelines, a combination of TMP-SMX + ceftazidime ± gentamicin might be considered.

Prophylaxis: No human or veterinary vaccine. Post-exposure prophylaxis may be tried with TMP-SMX.

Isolation and Decontamination: Standard Precautions for healthcare workers. Person-to-person airborne transmission is unlikely, although secondary cases may occur through improper handling of infected secretions. Environmental decontamination using a 0.5% hypochlorite solution is effective.

PLAGUE

BACKGROUND:

Yersinia pestis, a rod-shaped, non-motile, non-sporulating, gram-negative, bipolar staining, facultative anaerobic bacterium. It causes plague, normally a zoonotic disease of rodents (e.g., rats, mice, ground squirrels). Fleas which live on the rodents can sometimes pass the bacteria to human beings, who then suffer from the bubonic form of plague. The pneumonic form of the disease would be seen as the primary form after purposeful aerosol dissemination of the organisms. The bubonic form would be seen after purposeful dissemination through the release of infected fleas. All human populations are susceptible. Recovery from the disease may be followed by temporary immunity. The organism will probably remain viable in water and moist meals and grains for several weeks. At near freezing temperatures, it will remain alive from months to years but is killed by 15 minutes exposure to 72° C. It also remains viable for some time in dry sputum, flea feces, and buried bodies but is killed within several hours of exposure to sunlight.

CLINICAL FEATURES:

Plague normally appears in three forms in man; bubonic, primary septicemic, and pneumonic. The buboes in the bubonic form are normally seen in the inguinal lymph nodes as the legs are the most commonly "flea-bitten" part of the human body. Septicemia is common, as greater than 80 percent of blood cultures are positive for the organism in bubonic plague, although primary septicemia may occur without lymphadenopathy. The pneumonic form is an infection of the lungs due either to inhalation of the organisms (primary pneumonic plague), or spread to the lungs from septicemia (secondary pneumonic plague). In man, the mortality of untreated bubonic plague is approximately 50 percent, whereas in pneumonic plague the mortality rate is 100 percent.

DIAGNOSIS:

After an incubation period varying from 2-3 days for primary pneumonic plague, onset is acute and often fulminant. The presentation is one of malaise, high fever, chills, headache, myalgia, cough with production of a bloody sputum, and toxemia. The chest x-ray reveals a patchy or consolidated bronchopneumonia. The pneumonia progresses rapidly, resulting in dyspnea, stridor, and cyanosis. The terminal event is one of respiratory failure, circulatory collapse, and a bleeding diathesis. In bubonic plague the incubation period ranges from 2 to 10 days with the onset also being acute and often fulminant. The presentation is one of malaise, high fever, and one or more tender lymph nodes. The liver and spleen are often tender and palpable. One quarter of patients will have various types of skin lesions. Occasionally a pustule, vesicle, eschar or papule containing leukocytes and bacteria will be apparent in the bubo distribution and presumably represents the site of the inoculating flea bite. Bubonic plague may progress spontaneously to the septicemic form with organisms spreading to the central nervous system, lungs, and elsewhere. Black necrotic and purpuric lesions caused by endotoxemia are also often present.

Laboratory findings include a leukocytosis, with a total WBC count up to 20,000 cells with increased bands, and greater than 80 percent polymorphonuclear cells. One also often finds increased fibrin split products in the blood indicative of a low-grade DIC, and the ALT, AST, and bilirubin are also elevated.

A presumptive diagnosis can be made microscopically by identification of the gram-negative coccobacillus with safety-pin bipolar staining in Gram or Wayson's stained smears from a lymph node needle aspirate, sputum, or cerebrospinal fluid sample. When available, immunofluorescent staining is very useful. A definitive diagnosis can be readily made by culturing the organism from blood, sputum, and bubo aspirates. The organism grows slowly at normal incubation temperatures, and may be misidentified by automated systems because of delayed biochemical reactions. It may be cultured on blood agar, MacConkey agar or infusion broth. Most naturally occurring strains of *Y pestis* produce an F1-antigen *in vivo*, which can be detected in serum samples by immunoassay. A four-fold rise in antibody titer in patient serum is also diagnostic.

MEDICAL MANAGEMENT IN HOSPITAL:

Use Standard Precautions for healthcare workers exposed to bubonic plague and Droplet Precautions for healthcare workers exposed to pneumonic plague until the patient has been on antibiotic therapy for at least 48 hours and there has been a favorable clinical response to treatment. Streptomycin, tetracycline, chloramphenicol, and gentamicin are highly effective, especially if begun early (within 24 hours of onset of symptoms). Plague pneumonia is almost always fatal if treatment is not initiated within 24 hours of the onset of symptoms. Streptomycin remains the drug of choice and is given 30 mg/kg/day (IM) in two divided doses for ten days. Gentamicin is acceptable if streptomycin is unavailable. While the patient is typically afebrile after 3 days, the extra week of therapy prevents relapses. Intravenous doxycycline 200 mg initially, followed by 100 mg every 12 hours for 10-14 days is also effective. Results obtained from laboratory animal, but not human, experience, indicate that quinolone antibiotics, such as ofloxacin and ciprofloxacin, may also be effective. The addition of chloramphenicol (1 gm IV QID x 10-14 days) is required for the treatment of plague meningitis.

Usual supportive therapy required includes IV crystalloids and hemodynamic monitoring. Although low-grade DIC may occur, clinically significant hemorrhage is uncommon as is the need to treat with heparin. Finally, buboes rarely require incision and drainage or any form of local care, but instead recede with systemic antibiotic therapy. In fact, incision and drainage may pose a risk to others in contact with the patient.

SUMMARY:

Signs and Symptoms: Pneumonic plague incubates 2-3 days. High fever, chills, headache, hemoptysis, and toxemia, progressing rapidly to dyspnea, stridor, and cyanosis. Death from respiratory failure, circulatory collapse, and a bleeding diathesis. Bubonic plague incubates 2-10 days. Malaise, high fever, and tender lymph nodes (buboes); may progress spontaneously to the septicemic form, with spread to the CNS, lungs, etc.

Diagnosis: Presumptive diagnosis can be made by Gram or Wayson stain of lymph node aspirates, sputum, or CSF. Plague bacilli may also be cultured on standard media.

Treatment: Early administration of antibiotics is very effective. Supportive therapy is required.

Prophylaxis: A licensed, killed vaccine is available. Primary series of an initial dose followed by a second smaller dose 1-3 months later, and a third dose 5-6 months after the second dose. Give 3 booster doses at 6 month intervals following dose 3 of the primary series then every 1-2 years. This vaccine is effective against bubonic plague, but probably not against aerosol exposure.

Isolation and Decontamination: Standard Precautions for healthcare workers exposed to bubonic plague. Droplet Precautions for healthcare workers exposed to pneumonic plague. Heat, disinfectants (2-5% hypochlorite) and exposure to sunlight renders bacteria harmless.

TULAREMIA

BACKGROUND:

Francisella tularensis, the causative agent of tularemia, is a small aerobic non-motile, gram-negative cocco-bacillus. Tularemia (also known as rabbit fever and deer fly fever) is a zoonotic disease which humans typically acquire after contact of their skin or mucous membranes with tissues or body fluids of infected animals, or from bites of infected deerflies, mosquitoes, or ticks. Less commonly, inhalation of contaminated dusts or ingestion of contaminated foods or water may produce clinical disease. Respiratory exposure by aerosol would cause typhoidal or pneumonic tularemia. *F. tularensis* can remain viable for weeks in water, soil, carcasses, and hides, and for years in frozen rabbit meat. It is resistant for months to temperatures of freezing and below. It is rather easily killed by heat and disinfectants.

CLINICAL FEATURES:

After an incubation period varying from 1-21 days (average 3-5 days), presumably dependent upon the dose of organisms, onset is usually acute. Tularemia may appear in several forms in man depending upon the route of inoculation: ulceroglandular, glandular, typhoidal, oculoglandular, pharyngeal, and pneumonic tularemia. In humans, as few as 10 to 50 organisms will cause disease if inhaled or injected intradermally, whereas approximately 10^8 organisms are required with oral challenge.

Ulceroglandular tularemia (75-85 percent of cases) is most often acquired through inoculation of the skin or mucous membranes with blood or tissue fluids of infected animals. It is characterized by fever, chills, headache, and malaise, an ulcerated skin lesion and painful regional lymphadenopathy. The skin lesion is usually located on the fingers or hand.

Glandular tularemia (5-10 percent of cases) results in fever and tender lymphadenopathy but no skin ulcer.

Typhoidal tularemia accounts for 5-15 percent of naturally occurring cases and occurs mainly after inhalation of infectious aerosols, but can occur after intradermal or gastrointestinal challenge. It manifests as fever, prostration, and weight loss but without lymphadenopathy. Pneumonia may be associated with any form but is most common in typhoidal tularemia. Diagnosis of primary typhoidal tularemia is difficult, as signs and symptoms are non-specific and there frequently is no suggestive exposure history. Respiratory symptoms, substernal discomfort, and a non-productive cough may also be present. Radiologic evidence of pneumonia or mediastinal lymphadenopathy is most common with typhoidal disease but may or may not be present in all other forms of tularemia.

Oculoglandular tularemia (1-2 percent of cases) occurs after inoculation of the conjunctivae with infectious material. Patients have unilateral, painful, purulent conjunctivitis with preauricular or cervical lymphadenopathy. Chemosis, periorbital edema, and small nodular lesions or ulcerations of the palpebral conjunctiva are noted in some patients.

Oropharyngeal tularemia refers to primary ulceroglandular disease confined to the throat. It produces an acute exudative or membranous pharyngotonsillitis with cervical lymphadenopathy.

Pneumonic tularemia is an illness characterized primarily by pneumonia. Pneumonia is common in tularemia. It is seen in 30-80 percent of the typhoidal cases and in 10-15 percent of the ulceroglandular cases. The case fatality rate without treatment is approximately 5 percent for the ulceroglandular form and 35 percent for the typhoidal form. All ages are susceptible, and recovery is generally followed by permanent immunity.

DIAGNOSIS:

Identification of organisms by staining ulcer or sputum is generally not helpful. Routine culture is difficult, due to unusual growth requirements and/or overgrowth of commensal bacteria. Isolation represents a clear hazard to laboratory personnel and should only be attempted in BL-3 laboratory. The diagnosis can be established retrospectively serologically. A fourfold rise in the tularemia tube agglutination or microagglutination titer is diagnostic of infection. A single convalescent titer of 1:160 or greater is diagnostic of past or current infection. Titers are usually negative the first week of infection, positive the second week in 50-70 percent of cases and reach a maximum in 4-8 weeks.

MEDICAL MANAGEMENT IN HOSPITAL:

Standard Precautions are recommended for healthcare workers. Streptomycin (1 gm every 12 hours IM for 10-14 days) is the treatment of choice. Gentamicin 3-5 mg/kg/day divided TID parenterally for 10-14 days is also effective. Tetracycline and chloramphenicol treatment are effective as well, but are associated with significant relapse rates. Although laboratory related infections with this organism are very common, person-to-person spread is unusual and respiratory isolation is not required.

SUMMARY:

Signs and Symptoms: Ulceroglandular tularemia presents with a local ulcer and regional lymphadenopathy, fever, chills, headache and malaise. Typhoidal tularemia presents with fever, headache, malaise, substernal discomfort, prostration, weight loss and a non-productive cough.

Diagnosis: Clinical diagnosis. Physical findings are usually non-specific. Chest x-ray may reveal a pneumonic process, mediasternal lymphadenopathy or pleural effusion. Routine culture is possible but difficult. The diagnosis can be established restrospectively by serology.

Treatment: Administration of antibiotics (streptomycin or gentamicin) with early treatment is very effective.

Prophylaxis: A live, attenuated vaccine is available as an investigational new drug. It is administered once by scarification. A two week course of tetracycline is effective as prophylaxis when given after exposure.

Isolation and Decontamination: Standard Precautions for healthcare workers. Organisms are relatively easy to render harmless by mild heat (55 degrees Celsius for 10 minutes) and standard disinfectants.

Q FEVER

BACKGROUND:

The endemic form of Q fever is a zoonotic disease caused by a rickettsia, *Coxiella burnetii* Its natural reservoirs are sheep, cattle and goats, and grows to especially high concentrations in placental tissues. Exposure to infected animals at parturition is an important risk factor for endemic disease. The organisms are also excreted in animal milk, urine, and feces. Humans acquire the disease by inhalation of aerosols contaminated with the organisms. Farmers and abattoir workers are at greatest risk occupationally. A biological warfare attack with Q fever would cause a disease similar to that occurring naturally. Q fever is also a significant hazard in laboratory personnel who are working with the organism.

CLINICAL FEATURES:

Following the usual incubation period of 10-40 days, Q fever generally occurs as a self-limiting febrile illness lasting 2 days to 2 weeks. The incubation period varies according to the numbers of organisms inhaled, with longer periods between exposure and illness with lower numbers of inhaled organisms (up to forty days in some cases). The disease generally presents as an acute nondifferentiated febrile illness, with headaches, fatigue, and myalgias as prominent symptoms. Pneumonia manifested by an abnormal chest X-ray occurs in half of all patients, but only half of these, or 25 percent of patients, will have a cough (usually non-productive) or rales. Pleuritic chest pain occurs in about one-fourth of patients with Q fever pneumonia. Chest radiograph abnormalities, when present, are patchy infiltrates that may resemble viral or mycoplasma pneumonia. Rounded opacities and adenopathy have also been described.

Uncommon complications include chronic hepatitis, culture-negative endocarditis, aseptic meningitis, encephalitis and osteomyelitis. Most patients who develop endocarditis have pre-existing valvular heart disease.

DIAGNOSIS:

Routine Laboratory Findings: The white blood cell count is elevated in one third of patients. Most patients with Q fever have a mild elevation of hepatic transaminase levels.

Differential Diagnosis: As Q fever usually presents as an undifferentiated febrile illness, or a primary atypical pneumonia, it may be difficult to distinguish from viral illnesses and must be differentiated from pneumonia caused by *Mycoplasma pneumoniae, Legionella pneumophila, Chlamydia psittaci*, and *Chlamydia pneumoniae*(TWAR). More rapidly progressive forms of Q fever pneumonia may look like bacterial pneumonias such as tularemia or plague. Significant numbers of soldiers (from the same geographic area) presenting over a one to two week period with a nonspecific febrile illness, with associated pneumonic symptoms in about half of cases, should trigger the possibility of an attack with aerosolized Q fever in the minds of the treating physicians. The diagnosis will often rest on the clinical and epidemiologic picture in the setting of a possible biowarfare attack.

Specific Laboratory Diagnosis: Identification of organisms by examination of the sputum is not helpful. Isolation of the organism is impractical, as the organism is difficult to culture and a significant hazard to laboratory workers. Serological tests for Q fever include identification of antibody to *C. burnetii* by indirect fluorescent antibody (IFA), enzyme-linked immunosorbent assay (ELISA), and complement fixation. Specific IgM antibodies may be detectable as early as the second week after onset of illness. ELISA testing is available at USAMRIID. A single serum specimen can be used to reliably diagnose acute Q fever with this test as early as 1 1/2-2 weeks into the illness. The most commonly available serologic test is the complement fixation test (CF) which is relatively insensitive and may not be useful if sera have intrinsic anti-complement activity.

MEDICAL MANAGEMENT IN HOSPITAL:

Standard Precautions are recommended for healthcare workers. Most cases of acute Q fever will eventually resolve without antibiotic treatment. Tetracycline 500 mg every 6 hr or doxycycline 100 mg every 12 hr for 5-7 days will shorten the duration of illness, and fever usually disappears within one to two days after treatment is begun. Successful treatment of Q fever endocarditis is much more difficult. Tetracycline or doxycycline given in combination with trimethoprim-sulfamethoxazole (TMP-SMX) or rifampin for 12 months or longer has been successful in some cases. However, valve replacement is often required to achieve a cure.

SUMMARY:

Signs and Symptoms: Fever, cough, and pleuritic chest pain may occur as early as ten days after exposure. Patients are not generally critically ill, and the illness lasts from 2 days to 2 weeks.

Diagnosis: Q fever is not a clinically distinct illness and may resemble a viral illness or other types of atypical pneumonia. The diagnosis is confirmed serologically.

Treatment: Q fever is generally a self-limited illness even without treatment. Tetracycline or doxycycline are the treatments of choice and are given orally for 5 to 7 days. Q fever endocarditis (rare) is much more difficult to treat.

Prophylaxis: Treatment with tetracycline during the incubation period may delay but not prevent the onset of symptoms. An inactivated whole cell IND vaccine is effective in eliciting protection against exposure, but severe local reactions to this vaccine may be seen in those who already possess immunity.

Isolation and Decontamination: Standard Precautions are recommended for healthcare workers. Person-to-person transmission is rare. Patients exposed to Q fever by aerosol do not present a risk for secondary contamination or a re-aerosolization of the organism. Decontamination is accomplished with soap and water or after a 30 minute contact time with 5% microchem plus (quaternary ammonium compound) or 70% ethyl alcohol.

VIRAL AGENTS

Viruses are the simplest type of microorganism and consist of a nucleocapsid protein coat containing genetic material, either RNA or DNA. In some cases the virus particle is also surrounded by an outer layer of lipids. Viruses are much smaller than bacteria and vary in size from 0.02 µm to 0.2 µm (1 µm = 1/1000 mm). Viruses lack a system for their own metabolism and are therefore dependent on the synthetic machinery of their host cells: viruses are thus intracellular parasites. This also means that the virus, unlike the bacterium, cannot be cultivated in synthetic nutritive solutions but requires living cells in order to multiply. The host cells can be from human beings, animals, plants, or bacteria. Every virus needs its own special type of host cell because a complicated interaction is required between the cell and virus if the virus is to be able to multiply. Many virus-specific host cells can be cultivated in synthetic nutrient solutions and afterwards can be infected with the virus in question. Another usual way of cultivating viruses is to let them grown on chorioallantoic membranes (from fertilized eggs). The cultivation of viruses is costly, demanding, and time-consuming. A virus normally brings about changes in the host cell such that the cell dies. This handbook will cover a virus considered by some to be the most likely viral agent that would be used in a BW attack, the alpha virus that causes Venezuelan equine encephalitis, known as VEE. We also discuss smallpox and homorrhagic fever viruses which could potentially be employed as BW agents.

SMALLPOX

BACKGROUND:

Variola virus causes smallpox. It is an Orthopox virus and occurs in at least two strains, variola major and the milder disease, variola minor. Despite the global eradication of smallpox and continued availability of a vaccine, the potential weaponization of variola continues to pose a military threat. This threat can be attributed to the aerosol infectivity of the virus, the relative ease of large-scale production, and an increasingly *Orthopoxvirus*-naive populace. Although the fully-developed cutaneous eruption of smallpox is unique, earlier stages of the rash could be mistaken for varicella. Secondary spread of infection constitutes a nosocomial hazard from the time of onset of a smallpox patient's exanthem until scabs have separated. Quarantine with respiratory isolation should be applied to secondary contacts for 17 days post-exposure. Vaccinia vaccination and vaccinia immune globulin each possess some efficacy in post-exposure prophylaxis.

Control and Prevention (CDC) in Atlanta and the Institute for Viral Preparations in Moscow, the extent of clandestine stockpiles in other parts of the world remains unknown. In January 1996, WHO's governing board recommended that all stocks of smallpox be destroyed by 30 June, 1999.

The United States stopped vaccinating its military population in 1989 and civilians in the early 1980s. These populations are now susceptible to variola major, although recruits immunized in 1989 may retain some degree of immunity. Variola may have been used by the British Army against native Americans by giving them contaminated blankets from the beds of smallpox victims during the eighteenth century. Japan considered the use of smallpox as a BW weapon in World War II and it has been considered as a possible threat agent against US forces for many years.

CLINICAL FEATURES:

The incubation period of smallpox averaged 12 days, and contacts were quarantined for a minimum of 16-17 days following exposure. Clinical manifestations began acutely with malise, fever, rigors, vomiting, headache, and backache; 15% of patients developed delirium. Approximately 10% of light-skinned patients exhibited an erythematous rash during this phase. Two to three days later, an enanthem appeared concomitantly with a discrete rash about the face, hands and forearms.

Following eruptions on the lower extremities, the rash spread centrally to the trunk over the next week. Lesions quickly progressed from macules to papules, and eventually to pustular vesicles. Lesions were more abundant on the extremities and face, and this centrifugal distribution is an important diagnostic feature. In distinct contrast to varicella, lesions on various segments of the body remained generally synchronous in their stage of development. From 8 to 14 days after onset, the pustules formed scabs which leave depressed depigmented scars upon healing. Although variola concentrations in the throat, conjunctiva, and urine diminished with time, virus could readily be recovered from scabs throughout convalescence. Therefore, patients should be isolated and considered infectious until all scabs separate.

For the past century, two distinct types of smallpox were recognized. Variola minor was distinguished by milder systemic toxicity and more diminutive pox lesions, and caused 1% mortality in unvaccinated victims. However, the prototypical disease variola major caused mortality of 3% and 30% in the vaccinated and unvaccinated, respectively. Other clinical forms associated with variola major, flat-type and hemorrhagic-type smallpox, were notable for severe mortality. A naturally occurring relative of variola, monkeypox, occurs in Africa, and is clinically indistinguishable from smallpox with the exception of notable enlargement of cervical and inguinal lymph nodes.

DIAGNOSIS:

Smallpox must be distinguished from other vesicular exanthems, such as chickenpox, erythema multiforme with bullae, or allergic contact dermatitis. Particularly problematic to infection control measures would be the failure to recognize relatively mild cases of smallpox in persons with partial immunity. An additional threat to effective quarantine is the fact that exposed persons may shed virus from the oropharynx without ever manifesting disease. Therefore, quarantine and initiation of medical countermeasures should be promptly followed by an accurate diagnosis so as to avert panic.

The usual method of diagnosis is demonstration of characteristic virions on electron microscopy of vesicular scrapings. Under light microscopy, aggregations of variola virus particles, called Guarnieri bodies, are found. Another rapid but relatively insensitive test for Guarnieri bodies in vesicular scrapings is Gispen's modified silver stain, in which cytoplasmic inclusions appear black.

None of the above laboratory tests are capable of discriminating variola from vaccinia, monkeypox or cowpox. This differentiation classically required isolation of the virus and characterization of its growth on chorioallantoic membrane. The development of polymerase chain reaction diagnostic techniques promises a more accurate and less cumbersome method of discriminating between variola and other *Orthopoxviruses*

MEDICAL MANAGEMENT IN HOSPITAL:

Medical personnel must be prepared to recognize a vesicular exanthem in possible biowarfare theaters as potentially variola, and to initiate appropriate countermeasures. Any confirmed case of smallpox should be considered an international emergency with immediate report made to public health authorities. Droplet and Airborne Precautions for a minimum of 16-17 days following exposure for *all* persons in direct contact with the index case, especially the unvaccinated. Patients should be considered infectious until all scabs separate. Immediate vaccination or revaccination should also be undertaken for all personnel exposed to either weaponized variola virus or a clinical case of smallpox.

The potential for airborne spread to other than close contacts is controversial. In general, close person-to-person proximity is required for transmission to reliably occur. Nevertheless, variola's potential in low relative humidity for airborne dissemination was alarming in two hospital outbreaks. Smallpox patients were infectious from the time of onset of their eruptive exanthem, most commonly from days 3-6 after onset of fever. Infectivity was markedly enhanced if the patient manifested a cough. Indirect transmission via contaminated bedding or other fomites was infrequent. Some close contacts harbored virus in their throats without developing disease, and hence might have served as a means of secondary transmission.

Vaccination with a verified clinical "take" (vesicle with scar formation) within the past 3 years is considered to render a person immune to smallpox. However, given the difficulties and uncertainties under wartime conditions of verifying the adequacy of troops' prior vaccination, routine revaccination of all potentially exposed personnel would seem prudent if there existed a significant prospect of smallpox exposure.

Antivirals for use against smallpox are under investigation. Cidofovir has been shown to have significant *in vitro* and *in vivo* activity in experimental animals.

SUMMARY:

Signs and Symptoms: Clinical manifestations begin acutely with malaise, fever, rigors, vomiting, headache, and backache. 2-3 days later lesions appear which quickly progress from macules to papules, and eventually to pustular vesicles. They are more abundant on the extremities and face, and develop synchronously.

Diagnosis: Electron and light microscopy are not capable of discriminating variola from vaccinia, monkeypox or cowpox. The new PCR diagnostic techniques may be more accurate in discriminating between variola and other *Orthopoxviruses*

Treatment: At present there is no effective chemotherapy, and treatment of a clinical case remains supportive.

Prophylaxis: Immediate vaccination or revaccination should be undertaken for all personnel exposed. Vaccinia immune globulin (VIG) is of value in post-exposure prophylaxis of smallpox when given within the first week following exposure.

Isolation and Decontamination: Droplet and Airborne Precautions for a minimum of 16-17 days following exposure for *all* contacts. Patients should be considered infectious until all scabs separate.

VENEZUELAN EQUINE ENCEPHALITIS

BACKGROUND:

Venezuelan equine encephalitis (VEE) virus is an arthropod-borne alphavirus that is endemic in northern South America, Trinidad, Central America, Mexico, and Florida. Eight serologically distinct viruses belonging to the VEE complex have been associated with human disease; the two most important of these pathogens are designated subtype I, variants A/B, and C. These agents also cause severe disease in horses, mules, burros and donkeys (Equidae). Natural infections are acquired by the bites of a wide variety of mosquitoes. Equidae serve as amplifying hosts and source of mosquito infection. In natural human epidemics, severe and often fatal encephalitis in Equidae always precedes disease in humans. The virus is rather easily killed by heat and disinfectants.

CLINICAL FEATURES:

VEE is characterized by inflammation of the meninges of the brain and of the brain itself, thus accounting for the predominance of CNS symptoms in the small percentage of infections that develop encephalitis. The disease is usually acute, prostrating and of short duration. The case fatality rate is less than 1 percent, although is somewhat higher in the very young or aged. Nearly 100 percent of those infected suffer an overt illness. Recovery from an infection results in excellent short-term and long-term immunity.

DIAGNOSIS:

After an incubation period varying from 2-6 days, onset is usually sudden. It is manifested by generalized malaise, spiking fever, rigors, severe headache, photophobia, and myalgias in the legs and lumbosacral area. Nausea, vomiting, cough, sore throat, and diarrhea may follow. This acute phase lasts 24-72 hours. A prolonged period of asthenia and lethargy may follow, with full health and activity regained after 1-2 weeks. Approximately 4 percent of children during natural epidemics develop signs of central nervous system infection, with meningismus, convulsions, coma, and paralysis. Adults rarely develop neurologic complications. In children manifesting severe encephalitis, the fatality rate may reach 20 percent. Permanent neurologic sequelae are reported in survivors. Experimental aerosol challenges in animals suggest that the incidence of CNS disease and associated morbidity and mortality would be high after a BW attack, as the VEE virus would infect the olfactory nerve and spread directly to the CNS. A VEE infection during pregnancy may cause encephalitis in the fetus, placental damage, abortion, or severe congenital neuroanatomical anomalies.

The white blood cell count shows a striking leukopenia and lymphopenia. In cases with encephalitis, the cerebrospinal fluid may be under increased pressure and contain up to 1,000 white cells/mm^3 (predominantly mononuclear cells) and mildly elevated protein concentration. Viremia during the acute phase of the illness (but not during encephalitis) is generally high enough to allow detection by antigen-capture enzyme immunoassay. Virus isolation may be made from serum, and in some cases throat swab specimens, by inoculation of cell cultures or suckling mice. A variety of serological tests are applicable, including the IgM ELISA indirect FA, hemagglutination inhibition, complement-fixation, and neutralization. For persons without prior exposure to VEE complex viruses, a presumptive diagnosis may be made by finding IgM antibody in a single serum sample taken 5 to 7 days after onset of illness.

MEDICAL MANAGEMENT IN HOSPITAL:

Standard Precautions are recommended for healthcare workers. Person-to-person transmission may *theoretically* occur by means of respiratory droplet infection. There is no specific therapy. Patients with uncomplicated VEE infection may be treated with analgesics to relieve headache and myalgia. Patients who develop encephalitis may require anticonvulsants and intensive supportive care to maintain fluid and electrolyte balance, ensure adequate ventilation, and avoid complicating secondary bacterial infections. Patients should be treated in a screened room or in quarters treated with a residual insecticide for at least 5 days after onset, or until afebrile, as human cases may be infectious for mosquitoes for at least 72 hours. The virus can be destroyed by heat and disinfectants.

SUMMARY:

Signs and Symptoms: Sudden onset of illness with generalized malaise, spiking fevers, rigors, severe headache, photophobia, and myalgias. Nausea, vomiting, cough, sore throat, and diarrhea may follow. Full recovery takes 1-2 weeks.

Diagnosis: Clinical diagnosis. Physical findings are usually non-specific. The white blood cell count often shows a striking leukopenia and lymphopenia. Virus isolation may be made from serum, and in some cases throat swab specimens. Both neutralizing or IgG antibody in paired sera or VEE specific IgM present in a single serum sample indicate recent infection.

Treatment: Supportive only.

Prophylaxis: A live, attenuated vaccine is available as an investigational new drug. A second, formalin-inactivated, killed vaccine is available for boosting antibody titers in those initially receiving the live vaccine.

Isolation and Decontamination: Standard Precautions for healthcare workers. Human cases are infectious for mosquitoes for at least 72 hours. The virus can be destroyed by heat (80 degrees centrigrade for 30 minutes) and standard disinfectants.

VIRAL HEMORRHAGIC FEVERS

BACKGROUND:

The viral hemorrhagic fevers are a diverse group of human illnesses that are due to RNA viruses from several different viral families: the *Filoviridae*, which consists of Ebola and Marburg viruses; the *Arenaviridae*, including Lassa fever, Argentine and Bolivian hemorrhagic fever viruses; the *Bunyaviridae*, including various members from the Hantavirus genus, Congo-Crimean hemorrhagic fever virus from the Nairovirus genus, and Rift Valley fever from the *Phlebovirus* genus; and *Flaviviridae*, such as Yellow fever virus, Dengue hemorrhagic fever virus, and others. The viruses may be spread in a variety of ways, and for some there is a possibility that humans could be infected through a respiratory portal of entry. Although evidence for weaponization does not exist for many of these viruses, many are included in this handbook because of their *potential* for aerosol dissemination or weaponization, or likelihood for confusion with similar agents which might be weaponized.

CLINICAL FEATURES:

The clinical syndrome which these viruses may cause in humans is generally referred to as viral hemorrhagic fever or VHF. Not all infected patients develop VHF; there is both divergence and uncertainty about which host factors and virus strain differences might be responsible for clinically manifesting hemorrhagic disease. For instance, an immunopathogenic mechanism has been identified for dengue hemorrhagic fever, which is seen only in patients previously infected with heterologous dengue serotype. The target organ in the VHF syndrome is the vascular bed; correspondingly, the dominant clinical features are usually a consequence of microvascular damage and changes in vascular permeability. Common presenting complaints are fever, myalgia, and prostration; clinical examination may reveal only conjunctival injection, mild hypotension, flushing, and petechial hemorrhages. Full-blown VHF typically evolves to shock and generalized mucous membrane hemorrhage and often is accompanied by evidence of neurologic, hematopoietic, or pulmonary involvement. Apart from epidemiologic and intelligence information, some distinctive clinical features may suggest a specific etiologic agent: high AST elevation correlates with severity of illness from Lassa fever, and jaundice is a poor prognostic sign in yellow fever. Hepatic involvement is common among the VHFs, but a clinical picture dominated by jaundice and other evidence of hepatic failure is only seen in some cases of Rift Valley fever, Congo-Crimean HF, Marburg HF, Ebola HF, and yellow fever. Neurologic symptoms and thrombocytopenia are common in Argentine and Bolivian hemorrhagic fever. Kyanasur Forest disease and Omsk hemorrhagic fever are notable for concomitant pulmonary involvement, and a biphasic illness with subsequent CNS manifestations. With regard to the Bunyaviruses, copious hemorrhage and nosocomial transmission are typical for Congo-Crimean HF, and retinitis is commonly seen in Rift Valley fever. Renal insufficiency is proportional to cardiovascular compromise, except in

hemorrhagic fever with renal syndrome (HFRS) due to hantaviruses, where renal azotemia is an integral part of the disease process. Mortality may be substantial, ranging from 5 to 20 percent or higher in recognized cases. Ebola outbreaks in Africa have been notable for the extreme prostration and toxicity of the victims, as well as frighteningly high case fatality rates ranging from 50 to 90 percent. This particularly virulent virus could conceivably be chosen by an adversary as a biological warfare agent due to its probable aerosol infectivity and high mortality.

DIAGNOSIS:

A detailed travel history and a high index of suspicion are essential in making the diagnosis of VHF. Patients with arenaviral or hantaviral infections often recall having seen rodents during the presumed incubation period, but, since the viruses are spread to man by aerosolized excreta or environmental contamination, actual contact with the reservoir is not necessary. Large mosquito populations are common during Rift Valley fever or flaviviral transmission, but a history of mosquito bite is sufficiently common to be of little assistance, whereas tick bites or nosocomial exposure are of some significance in suspecting Congo-Crimean hemorrhagic fever. Large numbers of military personnel presenting with VHF manifestations in the same geographic area over a short time period should lead treating medical care providers to suspect either a natural outbreak if in an endemic setting, or possibly a biowarfare attack, particularly if this type of disease does not occur naturally in the local area where troops are deployed.

VHF should be suspected in any patient presenting with a severe febrile illness and evidence of vascular involvement (subnormal blood pressure, postural hypotension, petechiae, easy bleeding, flushing of face and chest, non-dependent edema) who has traveled to an area where the virus is known to occur, or where intelligence information suggests a biological warfare threat. Signs and symptoms suggesting additional organ system involvement are common (headache, photophobia, pharyngitis, cough, nausea or vomiting, diarrhea, constipation, abdominal pain, hyperesthesia, dizziness, confusion, tremor), but usually do not dominate the picture with the exceptions listed above under “Clinical Features.”

For much of the world, the major differential diagnosis is malaria. It must be borne in mind that parasitemia in patients partially immune to malaria does not prove that symptoms are due to malaria. Typhoid fever, rickettsial, and leptospiral diseases are major confounding infections, with nontyphoidal salmonellosis, shigellosis, relapsing fever, fulminant hepatitis, and meningococcemia being some of the other important diagnoses to exclude. Any condition leading to disseminated intravascular coagulation could present in a confusing fashion, as well as diseases such as acute leukemia, lupus erythematosus, idiopathic or thrombotic thrombocytopenic purpura, and hemolytic uremic syndrome.

Because of recent recognition of their worldwide occurrence, additional consideration should be given to infection with hantavirus. Classic HFRS (also referred to as Korean hemorrhagic fever or epidemic hemorrhagic fever) has a severe course which progresses sequentially from fever through hemorrhage, shock, renal failure, and polyuria. This clinical form of HFRS is widely distributed in China, the Korean peninsula, and the Far Eastern USSR. Severe disease also is found in some Balkan states, including Bosnia/Serbia and Greece. However, the Scandinavian and most European virus strains carried by bank voles usually produce a milder disease (referred to as nephropathia epidemica) with prominent fever, myalgia, abdominal pain, and oliguria, but without shock or severe hemorrhagic manifestations. Hantavirus Pulmonary Syndrome, recently recognized in the Americas and probably worldwide, lacks hemorrhagic manifestations, but nevertheless carries a very high mortality due to its rapidly progressive and severe pulmonary capillary leak which presents as ARDS.

The clinical laboratory can be very helpful. Thrombocytopenia (exception: Lassa) and leukopenia (exception: Lassa, Hantaan, and some severe CCHF cases) are the rule. Proteinuria and/or hematuria are common, and their presence is the rule for Argentine HF, Bolivian HF, and HFRS. A positive tourniquet test has been particularly useful in Dengue hemorrhagic fever, but should be sought in other hemorrhagic fevers as well.

Definitive diagnosis in an individual case rests on specific virologic diagnosis. Most patients have readily detectable viremia at presentation (exception: hantaviral infections). Rapid enzyme immunoassays can detect viral antigens in acute sera from patients with Lassa, Argentine HF, Rift Valley fever, Congo-Crimean HF, yellow fever and specific IgM antibodies in early convalescence. Lassa- and Hantaan-specific IgM often are detectable during the acute illness. Diagnosis by virus cultivation and identification will require 3 to 10 days or longer. With the exception of dengue, specialized microbiologic containment is required for safe handling of these viruses. Appropriate precautions should be observed in collection, handling, shipping, and processing of diagnostic samples. Both the Centers for Disease Control and Prevention (CDC, Atlanta, Georgia) and the U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID), Frederick, Maryland) have diagnostic laboratories functioning at the highest (BL-4 or P-4) containment level.

MEDICAL MANAGEMENT IN HOSPITAL:

Contact Precautions required for healthcare workers. General principles of supportive care apply to hemodynamic, hematologic, pulmonary, and neurologic manifestations of VHF, regardless of the specific etiologic agent concerned. Patients generally are either moribund or recovering by the second week of illness, but only intensive care will save the most severely ill patients. Health care providers employing vigorous fluid resuscitation of patients with hemodynamic compromise must be mindful of the propensity of some VHF cases (e.g., hantaviral) for pulmonary capillary leak. Pressor agents are frequently required. Invasive hemodynamic monitoring should be used where normal indications warrant, but extra caution should be exercised with regard to sharp objects and their potential for nosocomial transmission of a viral agent (see below). Intramuscular injections, aspirin and other anticoagulant drugs should be avoided. Restlessness, confusion, myalgia, and hyperesthesia should be managed by conservative measures and judicious use of sedative, pain-relieving, and amnestic medications. Secondary infections may occur as with any patient undergoing intensive care and invasive procedures, such as intravenous lines and indwelling catheters.

The management of clinical bleeding should follow the same principles as for any patient with a systemic coagulopathy, assisted by coagulation studies. DIC has been implicated specifically in Rift Valley fever and Marburg/Ebola infections, but in most VHF the etiology of the coagulopathy is multifactorial (e.g., hepatic damage, consumptive coagulopathy, and primary marrow injury to megakaryocytes). Dengue HF is a notable case where antibody-mediated enhancement of dengue virus infection of monocytes and cytotoxic T-cell responses to these presented viral antigens precipitates vascular injury and permeability, complement activation, and a systemic coagulopathy.

The investigational antiviral drug ribavirin is available via compassionate use protocols for therapy of Lassa fever hemorrhagic fever with renal syndrome (HFRS), Congo-Crimean hemorrhagic fever, and Rift Valley fever. Separate Phase III efficacy trials have indicated that parenteral ribavirin reduces morbidity in both HFRS and Lassa fever, in addition to lowering mortality in the latter disease. In the human field trial with HFRS, treatment was effective if begun within the first 4 days of fever, and was continued for 7 days total. For Lassa fever patients, a compassionate use protocol utilizing intravenous ribavirin as a treatment is sponsored by the CDC. Dosages used were slightly different, and continued for 10 days total; treatment is most effective if begun within 7 days of onset. The only significant side effect of ribavirin is a modest anemia related to reversible block in erythropoiesis and mild hemolysis. Although ribavirin has demonstrated teratogenicity in animal studies, its use in a pregnant woman with grave illness from one of these VHFs must be weighed against potential benefit. Safety in infants and children has not been established. A similar dose of ribavirin begun within 4 days of disease may be effective in HFRS patients. It is important to note that ribavirin has poor *in vitro* and *in vivo* activity against either the filoviruses (Marburg and Ebola) or the flaviviruses (Dengue, Yellow Fever, Omsk HF and Kyanasur Forest Disease).

Argentine HF responds to therapy with 2 or more units of convalescent plasma containing adequate amounts of neutralizing antibody and given within 8 days of onset.

SUMMARY:

Signs and Symptoms: VHFs are febrile illnesses which can be complicated by easy bleeding, petechiae, hypotension and even shock, flushing of the face and chest, and edema. Constitutional symptoms such as malaise, myalgias, headache, vomiting, and diarrhea may occur in any of the hemorrhagic fevers.

Diagnosis: Definitive diagnosis rests on specific virologic techniques. Significant numbers of military personnel with a hemorrhagic fever syndrome should suggest the diagnosis of viral hemorrhagic fever.

Treatment: Intensive supportive care may be required. Antiviral therapy with ribavirin may be useful in several of these infections. Convalescent plasma may be effective in Argentine hemorrhagic fever.

Prophylaxis: The only licensed VHF vaccine is yellow fever vaccine. Prophylactic ribavirin may be effective for Lassa fever, Rift Valley fever, CCHF, and possibly HFRS.

Isolation and Decontamination: Contact Precautions for healthcare workers. Decontamination is accomplished with hypochlorite or phenolic disinfectants. Isolation measures and barrier nursing procedures are indicated.

Isolation and Containment: It should be noted that strict adherence to Contact Precautions has halted secondary transmission in the vast majority of circumstances. With the exception of dengue (virus present, but no secondary infection hazard) and hanta viruses (infectious virus not present in blood or excreta at the time of diagnosis), VHF patients generally have significant quantities of virus in blood and often other secretions. Special caution must be exercised in handling sharps, needles, and other potential sources of parenteral exposure. Clinical laboratory personnel are also at risk for exposure, and should employ a biosafety cabinet (if available) and barrier precautions when handling specimens.

Caution should be exercised in evaluating and treating the patient with a suspected VHF. Overreaction on the part of health care providers is inappropriate and detrimental to both patient and staff, but it is prudent to provide as rigorous isolation measures as feasible. These should include: isolation of the patient; stringent adherence to barrier nursing practices; mask, gown, glove, and needle precautions; decontamination of the outside of double-bagged specimens proceeding from the patient's room; autoclaving or liberal application of hypochlorite or phenolic disinfectants to excreta and other contaminated materials; and biosafety cabinet containment of laboratory specimens undergoing analysis.

Experience has shown that Marburg, Ebola, Lassa, and Congo-Crimean HF viruses may be particularly prone to aerosol nosocomial spread. Well-documented secondary infections among contacts and medical personnel who were not parenterally exposed have occurred. Sometimes this occurred when the acute hemorrhagic disease (as seen in CCHF) mimicked a surgical emergency such as a bleeding gastric ulcer, with subsequent exposure and secondary spread among emergency and operating room personnel. Therefore, when a significant suspicion of one of these diseases exists, additional management measures should include: an anteroom adjoining the patient's isolation room to facilitate putting on and removing protective barriers and storage of supplies; use of a negative pressure room for patient care if available; minimal handling of the body should the patient die, with sealing of the corpse in leak-proof material for prompt burial or cremation.

No carrier state has ever been observed with any VHF, but excretion of virus in urine (e.g., hantaviruses) or semen (e.g., Argentine hemorrhagic fever) may occur in convalescence.

BIOLOGICAL TOXINS

Toxins are defined as any toxic substance of natural origin produced by an animal, plant, or microbe. They are different from chemical agents such as VX, cyanide, or mustard in that they are not man-made. They are non-volatile, are usually not dermally active (mycotoxins are an exception), and tend to be more toxic per weight than many chemical agents. Their lack of volatility also distinguishes them from many of the chemical threat agents, and is very important in that they would not be either a persistent battlefield threat or be likely to produce secondary or person to person exposures. Many of the toxins, such as low molecular weight toxins and some peptides, are quite stable, as where the stability of the larger protein bacterial toxins is more variable. The bacterial toxins, such as botulinum toxins or shiga toxin, tend to be the most toxic in terms of dose required for lethality (Appendix C), whereas the mycotoxins tend to be among the least toxic compounds, thousands of times less toxic than the botulinum toxins. Some toxins are more toxic by the aerosol route than when delivered orally or parenterally (ricin, saxitoxin, and T2 mycotoxins are examples), whereas botulinum toxins have lower toxicity when delivered by the aerosol route than when ingested. However, botulinum is so toxic inherently that this characteristic does not limit its potential as a biological warfare agent. The utility of many toxins as military weapons is potentially limited by their inherent low toxicity (too much toxin would be required), or by the fact that some, such as saxitoxin, can only feasibly be produced in minute quantities. The relationship between aerosol toxicity and the quantity of toxin required to provide an effective open-air exposure is shown in Appendix D. The lower the lethal dose for fifty percent of those exposed (LD_{50}), in micrograms per kilogram, the less agent would be required to cover a large battlefield sized area. The converse is also true, and means that for some agents such as ricin, very large quantities (tons) would be needed for an effective open-air attack.

Where toxins are concerned, incapacitation as well as lethality must be considered. Several toxins cause significant illness at levels much lower than the level required for lethality, and are thus militarily significant in their ability to incapacitate soldiers.

This manual will cover four toxins considered to be among the most likely toxins which could be used: botulinum toxins, staphylococcal enterotoxin B (SEB), ricin, and T-2 mycotoxins.

BOTULINUM

BACKGROUND:

The botulinum toxins are a group of seven related neurotoxins produced by the bacillus *Clostridium botulinum*These toxins, types A through G, could be delivered by aerosol over concentrations of troops. When inhaled, these toxins produce a clinical picture very similar to foodborne intoxication, although the time to onset of paralytic symptoms may actually be longer than for foodborne cases, and may vary by type and dose of toxin. The clinical syndrome produced by one or more of these toxins is known as "botulism."

Botulinum toxins are proteins of approximately 150 kD molecular weight which can be produced from the anaerobic bacterium *Clostridium botulinum*As noted above, there are seven distinct but related neurotoxins, A through G, produced by different strains of the clostridial bacillus. All seven types act by similar mechanisms. The toxins produce similar effects when inhaled or ingested, although the time course may vary depending on the route of exposure and the dose received. Although an aerosol attack is by far the most likely scenario for the use of botulinum toxins, theoretically the agent could be used to sabotage food supplies; enemy special forces or terrorists might use this method in certain scenarios to produce foodborne botulism in those so targated.

MECHANISM OF TOXICITY:

The botulinum toxins as a group are among the most toxic compounds known to man. Appendix C shows the comparative lethality of selected toxins and chemical agents in laboratory mice. Botulinum toxin is the most toxic compound per weight of agent, requiring only 0.001 microgram per kilogram of body weight to kill 50 percent of the animals studied. As a group, bacterial toxins such as botulinum tend to be the most lethal of all toxins. Note that botulinum toxin type A is 15,000 times more toxic than VX and 100,000 times more toxic than Sarin, two of the well known organophosphate nerve agents.

Botulinum toxins act by binding to the presynaptic nerve terminal at the neuromuscular junction and at cholinergic autonomic sites. These toxins then act to prevent the release of acetylcholine presynaptically, and thus block neurotransmission. This interruption of neurotransmission causes both bulbar palsies and the skeletal muscle weakness seen in clinical botulism.

Unlike the situation with nerve agent intoxication, where there is too much acetylcholine due to inhibition of acetylcholinesterase, the problem in botulism is lack of the neurotransmitter in the synapse. Thus, pharmacologic measures such as atropine are not indicated in botulism and would likely exacerbate symptoms.

CLINICAL FEATURES:

The onset of symptoms of inhalation botulism may vary from 24 to 36 hours, to several days following exposure. Recent primate studies indicate that the signs and symptoms may in fact not appear for several days when a low dose of the toxin is inhaled versus a shorter time period following ingestion of toxin or inhalation of higher doses. Bulbar palsies are prominent early, with eye symptoms such as blurred vision due to mydriasis, diplopia, ptosis, and photophobia, in addition to other bulbar signs such as dysarthria, dysphonia, and dysphagia. Skeletal muscle paralysis follows, with a symmetrical, descending, and progressive weakness which may culminate abruptly in respiratory failure. Progression from onset of symptoms to respiratory failure has occurred in as little as 24 hours in cases of foodborne botulims.

Physical examination usually reveals an alert and oriented patient without fever. Postural hypotension may be present. Mucous membranes may be dry and crusted and the patient may complain of dry mouth or even sore throat. There may be difficulty with speaking and with swallowing. gag reflex may be absent. Pupils may be dilated and even fixed. Ptosis and extraocular muscle palsies may also be observed. Variable degrees of skeletal muscle weakness may be observed depending on the degree of progression in an individual patient. Deep tendon reflexes may be present or absent. With severe respiratory muscle paralysis, the patient may become cyanotic or exhibit narcosis from CO2 retention.

DIAGNOSIS:

The occurrence of an epidemic of cases of a descending and progressive bulbar and skeletal paralysis in afebrile patients points to the diagnosis of botulinum intoxication. Foodborne outbreaks tend to occur in small clusters.

Individual cases might be confused clinically with other neuromuscular disorders such as Guillain-Barre syndrome, myasthenia gravis, or tick paralysis. The edrophonium or Tensilon® test may be transiently positive in botulism, so it may not distinguish botulinum intoxication from myasthenia. The cerebrospinal fluid in botulism is normal and the paralysis is generally symmetrical, which distinguishes it from enteroviral myelitis. Mental status changes generally seen in viral encephalitis should not occur with botulinum intoxication.

It may become necessary to distinguish nerve agent and/or atropine poisoning from botulinum intoxication. Nerve agent poisoning produces copious respiratory secretions and miotic pupils, whereas there is if anything a decrease in secretions in botulinum intoxication. Atropine overdose is distinguished from botulism by its central nervous system excitation (hallucinations and delirium) even though the mucous membranes are dry and mydriasis is present. The clinical differences between botulinum intoxication and nerve agent poisoning are depicted in Appendix E.

Laboratory testing is generally not helpful in the diagnosis of botulism. Survivors do not usually develop an antibody response due to the very small amount of toxin necessary to produce clinical symptoms. Detection of toxin in serum or gastric contents is possible, and mouse neutralization (bioassay) remains the most sensitive test. Other assays include gel hydralization or ELISA. Serum specimens should be drawn from suspected cases and held for testing at a facility cabable of performing these tests.

MEDICAL MANAGEMENT IN HOSPITAL:

Respiratory failure secondary to paralysis of respiratory muscles is the most serious complication and, generally, the cause of death. Reported cases of botulism prior to 1950 had a mortality of 60%. With tracheotomy or endotracheal intubation and ventilatory assistance, fatalities should be less than five percent. Intensive and prolonged nursing care may be required for recovery which may take several weeks or even months.

Antitoxin: In isolated cases of food-borne botulism, circulating toxin is present, perhaps due to continued absorption through the gut wall. Botulinum antitoxin (equine origin) has been used in those circumstances, and is thought to be helpful. Animal experiments show that after aerosol exposure, botulinum antitoxin can be very effective if given before the onset of clinical signs. Administration of antitoxin is reasonable if disease has not progressed to a stable state.

A trivalent equine antitoxin has been available from the Centers for Disease Control and Prevention for cases of foodborne botulism. This product has all the disadvantages of a horse serum product, including the risks of anaphylaxis and serum sickness. A “despeciated” equine heptavalent antitoxin against types A, B, C, D, E, F, and G has been prepared by cleaving the Fc fragments from horse IgG molecules, leaving $F(ab)_2$ fragments. This product is under advanced development, and is currently available under IND status. Its efficacy is inferred from its performance in animal studies. Disadvantages include a reduced, but theoretical risk of serum sickness.

Use of the antitoxin requires skin testing for horse serum sensitivity prior to administration. Skin testing is performed by injecting 0.1 ml of a 1:10 dilution (in sterile physiological saline) of antitoxin intradermally in the patient's forearm with a 26 or 27 gauge needle. Monitor the injection site and observe the patient for allergic reaction for 20 minutes. The skin test is positive if any of these allergic reactions occur: hyperemic areola at the site of the injection >0.5 cm; fever or chills; hypotension with decrease of blood pressure >20 mm Hg for systolic and diastolic pressures; skin rash; respiratory difficulty; nausea or vomiting; generalized itching. Do NOT administer botulinum $F(ab')_2$ Antitoxin, Heptavalent (equine derived) if the skin test is positive. If no allergic symptoms are observed, the antitoxin is administered intravenously in a normal saline solution, 10 mls over 20 minutes.

With a positive skin test, desensitization is carried out by administering 0.01-0.1 ml of antitoxin subcutaneously, doubling the previous dose every 20 minutes until 1.0-2.0 ml can be sustained without any marked reaction.

SUMMARY:

Signs and Symptoms: Ptosis, generalized weakness, dizziness, dry mouth and throat, blurred vision and diplopia, dysarthria, dysphonia, and dysphagia followed by symmetrical descending flaccid paralysis and development of respiratory failure. Symptoms begin as early as 24-36 hours but may take several days after inhalation of toxin.

Diagnosis: Clinical diagnosis. No routine laboratory findings. Biowarfare attack should be suspected if multiple casualties simultaneously present with progressive descending bulbar, muscular, and respiratory weakness.

Treatment: Intubation and ventilatory assistance for respiratory failure. Tracheostomy may be required for long term management. Administration of heptavalent botulinum antitoxin (IND product) may prevent or decrease progression to respiratory failure and hasten recovery.

Prophylaxis: Pentavalent toxoid vaccine (types A, B, C, D, and E) is available as an IND product for those at high risk of exposure.

Isolation and Decontamination: Standard Precautions for healthcare workers. Toxin is not dermally active and secondary aerosols are not a hazard from patients. Hypochlorite (0.5% for 10-15 minutes) and/or soap and water.

STAPHYLOCOCCAL ENTEROTOXIN B

BACKGROUND:

*Staphylococcus aureus*produces a number of exotoxins, one of which is Staphylococcal enterotoxin B, or SEB. Such toxins are referred to as exotoxins since they are excreted from the organism; however, they normally exert their effects on the intestines and thereby are called enterotoxins. SEB is one of the pyrogenic toxins that commonly causes food poisoning in humans after the toxin is produced in improperly handled foodstuffs and subsequently ingested. SEB has a very broad spectrum of biological activity. This toxin causes a markedly different clinical syndrome when inhaled than it characteristically produces when ingested. Significant morbidity is produced in individuals who are exposed to SEB by either portal of entry to the body.

Staphylococcal enterotoxins are extracellular products produced by coagulase-positive staphylocci. They are produced in culture media and also in foods when there is overgrowth of the staph organisms. At least five antigenically distinct enterotoxins have been identified, SEB being one of them. These toxins are heat stable. SEB causes symptoms when inhaled at very low doses in humans; a dose of several logs lower than the lethal dose by the inhaled route would be sufficient to incapacitate 50 percent of those persons so exposed. This toxin could also be used (theoretically) in a special forces or terrorist mode to sabotage food or small volume water supplies.

MECHANISM OF TOXICITY:

Staphylococcal enterotoxins produce a variety of toxic effects. Inhalation of SEB can induce extensive pathophysiological changes to include widespread systemic damage and even septic shock. Many of the effects of staphylococcal enterotoxins are mediated by interactions with the host's own immune system. The mechanisms of toxicity are complex, but are related to toxin binding directly to the major histocompatibility complex that subsequently stimulates the proliferation of large numbers of T cell lymphocytes. Because these exotoxins are extremely potent activators of T cells, they are commonly referred to as bacterial superantigens. These superantigens stimulate the production and secretion of various cytokines, such as tumor necrosis factor, interferon, interleukin-1 and interleukin-2, from immune system cells. Released cytokines are thought to mediate many of the toxic effects of SEB.

CLINICAL FEATURES:

Relevant exposures to SEB are projected to cause primarily clinical illness and incapacitation. However, higher exposure levels can presumably lead to septic shock and death. Intoxication with SEB begins 3 to 12 hours after inhalation of the toxin. Victims may experience the sudden onset of fever, headache, chills, myalgias, and a nonproductive cough. More severe cases may develop dyspnea and retrosternal chest pain. Nausea, vomiting, and diarrhea will also occur in many patients due to inadvertently swallowed toxin, and fluid losses can be marked. The fever may last up to five days and range from 103 to 106 degrees F, with variable degrees of chills and prostration. The cough may persist up to four weeks, and patients may not be able to return to duty for two weeks.

Physical examination in patients with SEB intoxication is often unremarkable. Conjunctival injection may be present, and postural hypotension may develop due to fluid losses. Chest examination is unremarkable except in the unusual case where pulmonary edema develops. The chest X-ray is also generally normal, but in severe cases increased interstitial markings, atelectasis, and possibly overt pulmonary edema or an ARDS picture may develop.

DIAGNOSIS:

As is the case with botulinum toxins, intoxication due to SEB inhalation is a clinical and epidemiologic diagnosis. Because the symptoms of SEB intoxication may be similar to several respiratory pathogens such as influenza, adenovirus, and mycoplasma, the diagnosis may initially be unclear. All of these might present with fever, nonproductive cough, myalgia, and headache. SEB attack would cause cases to present in large numbers over a very short period of time, probably within a single 24 hour period. Naturally occurring pneumonias or influenza would involve patients presenting over a more prolonged interval of time. Naturally occurring staphylococcal food poisoning cases would not present with pulmonary symptoms.

SEB intoxication tends to progress rapidly to a fairly stable clinical state, whereas pulmonary anthrax, tularemia pneumonia, or pneumonic plague would all progress if left untreated. Tularemia and plague, as well as Q fever, would be associated with infiltrates on chest radiographs. Nerve agent intoxication would cause fasciculations and copious secretions, and mustard would cause skin lesions in addition to pulmonary findings; SEB inhalation would not be characterized by these findings. The dyspnea associated with botulinum intoxication is associated with obvious signs of muscular paralysis, bulbar palsies, lack of fever, and a dry pulmonary tree due to cholinergic blockade; respiratory difficulties occur late rather than early as with SEB inhalation.

Laboratory findings are not very helpful in the diagnosis of SEB intoxication. A nonspecific neutrophilic leukocytosis and an elevated erythrocyte sedimentation rate may be seen, but these abnormalities are present in many illnesses. Toxin is very difficult to detect in the serum by the tie symptoms occur; however, a serum specimen should be drawn as early as possible after exposure. Data from rabbit studies clearly show that SEB in the serum is transient; however, it accumulates in the urine and can be detected for several hours post exposure. Therefore, urine samples should be obtained and tested for SEB. Because most patients will develop a significant antibody response to the toxin, acute and convalescent serum should be drawn which may be helpful retrospectively in the diagnosis.

MEDICAL MANAGEMENT IN HOSPITAL:

Currently, therapy is limited to supportive care. Close attention to oxygenation and hydration are important, and in severe cases with pulmonary edema, ventilation with positive and expiratory pressure and diuretics might be necessary. Acetaminophen for fever, and cough suppressants may make the patient more comfortable. The value of steroids is unknown. Most patients would be expected to do quite well after the initial acute phase of their illness, but most would generally be unfit for duty for one to two weeks.

SUMMARY:

Signs and Symptoms: From 3-12 hours after aerosol exposure, sudden onset of fever, chills, headache, myalgia, and nonproductive cough. Some patients may develop shortness of breath and retrosternal chest pain. Fever may last 2 to 5 days, and cough may persist for up to 4 weeks. Patients may also present with nausea, vomiting, and diarrhea if they swallow toxin. Presumably, higher exposure can lead to septic shock and death.

Diagnosis: Diagnosis is clinical. Patients present with a febrile respiratory syndrome without CXR abnormalities. Large numbers of soldiers presenting with typical symptoms and signs of SEB pulmonary exposure would suggest an intentional attack with this toxin.

Treatment: Treatment is limited to supportive care. Artificial ventilation might be needed for very severe cases, and attention to fluid management is important.

Prophylaxis: Use of protective mask. There is currently no human vaccine available to prevent SEB intoxication.

Isolation and Decontamination: Standard Precautions for healthcare workers. Hypochlorite (0.5% for 10-15 minutes) and/or soap and water. Destroy any food that may have been contaminated.

RICIN

BACKGROUND:

Ricin is a potent protein toxin derived from the beans of the castor plant (*Ricinus communis*). Castor beans are ubiquitous worldwide, and the toxin is fairly easily produced. Ricin is therefore a potentially widely available toxin. When inhaled as a small particle aerosol, this toxin may produce pathologic changes within 8 hours and severe respiratory symptoms followed by acute hypoxic respiratory failure in 36-72 hours. When ingested, ricin causes severe gastrointestinal symptoms followed by vascular collapse and death. This toxin may also cause disseminated intravascular coagulation, microcirculatory failure and multiple organ failure if given intravenously in laboratory animals.

Ricin is actually made up of two hemagglutinins and two toxins. The toxins, RCL III and RCL IV, are dimers of about 66,000 daltons molecular weight. The toxins are made up of two polypeptide chains, an A chain and a B chain, which are joined by a disulfide bond. Ricin can be produced relatively easily and inexpensively in large quantities in a fairly low technology setting. It is of marginal toxicity in terms of its LED_{50} in comparison to toxins such as botulinum and SEB (incapacitating dose), so an enemy would have to produce it in larger quantities to cover a significant area on the battlefield. This might limit large-scale use of ricin by an adversary. Ricin can be prepared in liquid or crystalline form, or it can be lyophilized to make it a dry powder. It could be disseminated by an enemy as an aerosol, or it could be used as a sabotage, assassination, or terrorist weapon.

MECHANISM OF TOXICITY:

Ricin is very toxic to cells. It acts by inhibiting protein synthesis. The B chain binds to cell surface receptors and the toxin-receptor complex is taken into the cell; the A chain has endonuclease activity and the extremely low concentrations will inhibit protein synthesis. In rodents, the histopathology of aerosol exposure is characterized by necrotizing airway lesions causing tracheitis, bronchitis, bronchiolitis, and interstitial pneumonia with perivascular and alveolar edema. There is a latent period of 8 hours post-inhalation exposure before histologic lesions are observed in animal models. In rodents, ricin is more toxic by the aerosol route than by other routes of exposure.

There is little toxicity data in humans. The exact cause of morbidity and mortality would be dependent upon the route of exposure. Aerosol exposure in man would be expected to cause acute lung injury, pulmonary edema secondary to increased capillary permeability, and eventual acute hypoxic respiratory failure.

CLINICAL FEATURES:

The clinical picture in intoxicated victims would depend on the route of exposure. After aerosol exposure, signs and symptoms would depend on the dose inhaled. Accidental sublethal aerosol exposures which occurred in humans in the 1940's were characterized by onset of the following symptoms in four to eight hours: fever, chest tightness, cough, dyspnea, nausea, and arthralgias. The onset of profuse sweating some hours later was commonly the sign of termination of most of the symptoms. Although lethal human aerosol exposures have not been described, the severe pathophysiologic changes seen in the animal respiratory tract, including necrosis and severe alveolar flooding, are probably sufficient to cause death if enough toxin is inhaled. Time to death in experimental animals is dose dependent, occurring 36-72 hours post inhalation exposure. Humans would be expected to develop severe lung inflammation with progressive cough, dyspnea, cyanosis and pulmonary edema.

By other routes of exposure, ricin is not a direct lung irritant; however, intravascular injection can cause minimal pulmonary perivascular edema due to vascular endothelial injury. Ingestion causes gastrointestinal hemorrhage with hepatic, splenic, and renal necrosis. Intramuscular administration causes severe local necrosis of muscle and regional lymph nodes with moderate visceral organ involvement.

DIAGNOSIS:

An attack with aerosolized ricin would be, as with many biological warfare agents, primarily diagnosed by the clinical and epidemiological setting. Acute lung injury affecting a large number of cases in a war zone (where a BW attack could occur) should raise suspicion of an attack with a pulmonary irritant such as ricin, although other pulmonary pathogens could present with similar signs and symptoms. Other biological threats, such as SEB, Q fever, tularemia, plague, and some chemical warfare agents like phosgene, need to be included in a differential diagnosis. Ricin intoxication would be expected to progress despite treatment with antibiotics, as opposed to an infectious process. There would be no mediastinitis as seen with inhalation anthrax. SEB would be different in that most patients would not progress to a life-threatening syndrome but would tend to plateau clinically. Phosgene-induced acute lung injury would progress much faster than that caused by ricin.

Additional supportive clinical or diagnostic features after aerosol exposure to ricin may include the following: bilateral infiltrates on chest radiographs, arterial hypoxemia, neutrophilic leukocytosis, and a bronchial aspirate rich in protein compared to plasma which is characteristic of high permeability pulmonary edema. Specific ELISA testing on serum or immunohistochemical techniques for direct tissue analysis may be used where available to confirm the diagnosis. Ricin is an extremely immunogenic toxin, and acute as well as convalescent sera should be obtained from survivors for measurement of antibody response.

MEDICAL MANAGEMENT IN HOSPITAL:

Management of ricin-intoxicated patients again depends on the route of exposure. Patients with pulmonary intoxication are managed by appropriate treatment for pulmonary edema and respiratory support as indicated. Gastrointestinal intoxication is best managed by vigorous gastric decontamination with superactivated charcoal, followed by use of cathartics such as magnesium citrate. Volume replacement of GI fluid losses is important. In percutaneous exposures, treatment would be primarily supportive.

SUMMARY:

Signs and Symptoms: Weakness, fever, cough and pulmonary edema occur 18-24 hours after inhalation exposure, followed by severe respiratory distress and death from hypoxemia in 36-72 hours.

Diagnosis: Signs and symptoms noted above in large numbers of geographically clustered patients could suggest an exposure to aerosolized ricin. The rapid time course to severe symptoms and death would be unusual for infectious agents. Laboratory findings are nonspecific but similar to other pulmonary irritants which cause pulmonary edema. Specific serum ELISA is available. Acute and convalescent sera should be collected.

Treatment: Management is supportive and should include treatment for pulmonary edema. Gastric decontamination measures should be used if ingested.

Prophylaxis: There is currently no vaccine or propohylactic antitoxin available for human use, although immunization appears promising in animal models. Use of the protective mask is currently the best protection against inhalation.

Isolation and Decontamination: Standard Precautions for healthcare workers. Secondary aerosols should generally not be a danger to healthcare providers. Weak hypochlorite solutions (0.1% sodium hypochlorite) and/or soap and water can decontaminate skin surfaces.

T-2 MYCOTOXINS

BACKGROUND:

The trichothecene mycotoxins are low molecular weight (250-500 daltons) nonvolatile compounds produced by filamentous fungi (molds) of the genera *Fusarium, Myrotecium, Trichoderma, Stachybotrys* and others. The structures of approximately 150 trichothecene derivatives have been described in the literature. These substances are relatively insoluble in water but are highly soluble in ethanol, methanol and propylene glycol. The trichothecenes are extremely stable to heat and ultraviolet light inactivation. Heating to 1500° F for 30 minutes is required for inactivation, while brief exposure to NaOCl destroys toxic activity. The potential for use as a BW toxin was demonstrated to the Russian military shortly after World War II when flour contaminated with species of *Fusarium* was unknowningly baked into bread that was ingested by civilians. Some developed a protracted lethal illness called alimentary toxic aleukia (ATA) characterized by initial symptoms of abdominal pain, diarrhea, vomiting, prostration, and within days fever, chills, myalgias and bone marrow depression with granulocytopenia and secondary sepsis. Survival beyond this point allowed the development of painful pharyngeal/laryngeal ulceration and diffuse bleeding into the skin (petechiae and ecchymoses), melena, bloody diarrhea, hematuria, hematemesis, epistaxis and vaginal bleeding. Pancytopenia, and gastrointestinal ulceration and erosion were secondary to the ability of these toxins to profoundly arrest bone marrow and mucosal protein synthesis and cell cycle progression through DNA replication.

CLINICAL FEATURES:

T-2 and other mycotoxins may enter the body through the skin and digestive or respiratory epithelium. They are fast acting potent inhibitors of protein and nucleic acid synthesis. Their main effects are on rapidly proliferating tissues such as the bone marrow, skin, mucosal epithelia, and germ cells. In a successful BW attack with trichothecene toxin (T-2), the toxin(s) can adhere to and penetrate the skin, be inhaled, or can be ingested. Clothing would be contaminated and serve as a reservoir for further toxin exposure. Early symptoms beginning within minutes of exposure include burning skin pain, redness, tenderness, blistering, and progression to skin necrosis with leathery blackening and sloughing of large areas of skin in lethal cases. Nasal contact is manifested by nasal itching and pain, sneezing, epistaxis and rhinorrhea; pulmonary/tracheobronchial toxicity by dyspnea, wheezing, and cough; and mouth and throat exposure by pain and blood tinged saliva and sputum. Anorexia, nausea, vomiting and water or bloody diarrhea with abdominal

crampy pain occurs with gastrointestinal toxicity. Eye pain, tearing, redness, foreign body sensation and blurred vision may follow entry of toxin into the eyes. Skin symptoms occur in minutes to hours and eye symptoms in minutes. Systemic toxicity is manifested by weakness, prostration, dizziness, ataxia, and loss of coordination. Tachycardia, hypothermia, and hypotension follow in fatal cases. Death may occur in minutes, hours or days. The most common symptoms are vomiting, diarrhea, skin involvement with burning pain, redness and pruritus, rash or blisters, bleeding, and dyspnea.

DIAGNOSIS:

Rapid onset of symptoms in minutes to hours supports a diagnosis of a chemical or toxin attack. Mustard agents must be considered but they have an odor, are visible, and can be rapidly detected by a field available chemical test. Symptoms from mustard toxicity are also delayed for several hours after which mustard can cause skin, eye and respiratory symptoms. Staphylococcal enterotoxin B delivered by an aerosol attack can cause fever, cough, dyspnea and wheezing but does not involve the skin and eyes. Nausea, vomiting, and diarrhea may follow swallowing of inhaled toxin. Ricin inhalation can cause severe respiratory distress, cough, nausea and arthralgias. Swallowed agent can cause vomiting, diarrhea, and gastrointestinal bleeding, but it spares the skin, nose and eyes. Specific diagnosis of T-2 mycotoxins in the form of a rapid diagnostic test is not presently available in the field. Removal of blood, tissue from fatal cases, and environmental samples for testing using a gas liquid chromatography-mass spectrometry technique will confirm the toxic exposure. This system can detect as little as 0.1-1.0 ppb of T-2. This degree of sensitivity is capable of measuring T-2 levels in the plasma of toxin victims.

MEDICAL MANAGEMENT IN HOSPITAL:

Use of a chemical protective mask and clothing prior to and during a mycotoxin aerosol attack will prevent illness. If a person is exposed during an attack the clothing should be removed and decontaminated by exposure to 5% hypochlorite for 6-10 hours. The skin should be thoroughly washed with soap and water if available. Superactivated charcoal can absorb swallowed T-2 and should be administered to victims of an unprotected aerosol attack. The eyes should be irrigated with normal saline or water to remove toxin. No specific antidote or therapeutic regimen is currently available. All therapy is supportive.

SUMMARY:

Signs and symptoms: Exposure causes skin pain, pruritus, redness, vesicles, necrosis and sloughing of epidermis. Effects on the airway include nose and throat pain, nasal discharge, itching and sneezing, cough, dyspnea, wheezing, chest pain and hemoptysis. Toxin also produces effects after ingestion or eye contact. Severe poisoning results in prostration, weakness, ataxia, collapse, shock, and death.

Diagnosis: Should be suspected if an aerosol attack occurs in the form of "yellow rain" with droplets of yellow fluid contaminating clothes and the environment. Confirmation requires testing of blood, tissue and environmental samples.

Treatment: There is no specific antidote. Superactivated charcoal should be given orally if the toxin is swallowed.

Prophylaxis: The only defense is to wear a protective mask and clothing during an attack. No specific immunotherapy or chemotherapy is available.

Isolation and Decontamination: Standard Precautions for healthcare workers. Outer clothing should be removed and exposed skin should be decontaminated with soap and water. Eye exposure should be treated with copious saline irrigation. Once decontamination is complete, isolation is not required. Environmental decontamination requires the use of a hypochlorite solution under alkaline conditions such as 1% sodium hypochlorite and 0.1M NAOH with 1 hour contact time.

SPECIMEN COLLECTION FOR SUSPECT BIOLOGICAL WARFARE AGENTS

EARLY POST-EXPOSURE	CLINICAL	CONVALESCENT/TERMINAL/ POSTMORTEM
ANTHRAX		
0 TO 24 HOURS. NASAL AND THROAT SWABS, AND INDUCED RESPIRATORY SECRETIONS FOR CULTURE, FA, AND PCR.	*24 TO 72 HOURS.* SERUM (TT OR RT) FOR TOXIN ASSAYS. BLOOD (E, C, H) FOR PCR. BLOOD (BC OR C) FOR CULTURES.	*3 TO 10 DAYS.* SERUM (TT OR RT) FOR TOXIN ASSAYS BLOOD (BC OR C) FOR CULTURE. PATHOLOGY SPECIMENS.
PLAGUE		
0 TO 24 HOURS. NASAL SWABS; SPUTUM, AND INDUCED RESPIRATORY SECRETIONS FOR CULTURE, FA, AND PCR.	*24 TO 72 HOURS.* BLOOD (BC AND C) FOR CULTURE AND BLOODY SPUTUM (C) FOR FA. SERUM (TT OR RT) FOR F-1 ANTIGEN ASSAYS. BLOOD (E, C, OR H) FOR PCR.	*>6 DAYS.* SERUM (TT OR RT) FOR IgM, LATER FOR IgG. PATHOLOGY SPECIMENS.
TULAREMIA		
0 TO 24 HOURS. NASAL SWABS, SPUTUM, AND INDUCED RESPIRATORY SECRETIONS FOR CULTURE, FA, AND PCR.	*24 TO 72 HOURS.* BLOOD (BC OR C) FOR CULTURE. BLOOD (E, C, OR H) FOR PCR. SPUTUM FOR FA AND PCR.	*>6 DAYS.* SERUM (TT OR RT) FOR IgM AND LATER IgG, AGGLUTINATION TITERS. PATHOLOGY SPECIMENS.
MELIOIDOSIS/GLANDERS		
0 TO 24 HOURS. NASAL SWABS, SPUTUM, AND INDUCED RESPIRATORY SECRETIONS FOR CULTURE, AND PCR.	*24 TO 72 HOURS.* BLOOD (BC OR C) FOR CULTURE. BLOOD (E, C, OR H) FOR PCR. SPUTUM AND DRAINAGE FROM SKIN LESIONS FOR PCR AND CULTURE.	*>6 DAYS.* BLOOD (BC OR C) AND TISSUE FOR CULTURE. SERUM (TT OR RT) FOR IMMUNOASSAYS. PATHOLOGY SPECIMENS.
BRUCELLOSIS		
0 TO 24 HOURS. NASAL SWABS, SPUTUM, AND INDUCED RESPIRATORY SECRETIONS FOR CULTURE AND PCR.	*24 TO 72 HOURS.* BLOOD (BC OR C) FOR CULTURE. BLOOD (E, C, AND H) FOR PCR.	*>6 DAYS.* BLOOD (BC OR C) AND TISSUE FOR CULTURE. SERUM (TT OR RT) FOR IMMUNOASSAYS. PATHOLOGY SPECIMENS.
Q FEVER		
0 TO 24 HOURS. NASAL SWABS, SPUTUM, AND INDUCED RESPIRATORY SECRETIONS FOR CULTURE AND PCR.	*2 TO 5 DAYS.* BLOOD (BC OR C) FOR CULTURE IN EGGS OR MOUSE INOCULATION. BLOOD (E, C, AND H) FOR PCR.	*>6 DAYS.* BLOOD (BC OR C) FOR CULTURE IN EGGS OR MOUSE INOCULATION. PATHOLOGY SPECIMENS.
BOTULISM		
0 TO 24 HOURS. NASAL SWABS AND INDUCED RESPIRATORY SECRETIONS FOR PCR (CONTAMINATING BACTERIAL DNA) AND TOXIN ASSAYS. SERUM (TT OR RT) FOR TOXIN ASSAYS.	*24 TO 72 HOURS.* NASAL SWABS AND RESPIRATORY SECRETIONS FOR PCR (CONTAMINATING BACTERIAL DNA) AND TOXIN ASSAYS.	*>6 DAYS.* USUALLY NO IgM OR IgG. PATHOLOGY SPECIMENS (LIVER AND SPLEEN FOR TOXIN DETECTION).

RICIN INTOXICATION		
0 TO 24 HOURS. NASAL SWABS AND INDUCED RESPIRATORY SECRETIONS FOR PCR (CONTAMINATING CASTOR BEAN DNA) AND TOXIN ASSAYS. SERUM (TT OR RT) FOR TOXIN ASSAYS.	*36 TO 48 HOURS.* SERUM (TT OR RT) FOR TOXIN ASSAY. TISSUE FOR IMMUNOHISTOLOGICAL STAINING. PATHOLOGY SPECIMENS.	*>6 DAYS.* SERUM (TT OR RT) FOR IgM AND IgG IN SURVIVORS.
STAPH ENTEROTOXICOSIS		
0 TO 3 HOURS. NASAL SWABS AND INDUCED RESPIRATORY SECRETIONS FOR PCR (CONTAMINATING BACTERIAL DNA) AND TOXIN ASSAYS. SERUM (TT OR RT) FOR TOXIN ASSAYS.	*2 TO 6 HOURS.* URINE FOR IMMUNOASSAYS. NASAL SWABS AND INDUCED RESPIRATORY SECRETIONS FOR PCR (CONTAMINATING BACTERIAL DNA) AND TOXIN ASSAYS. SERUM (TT OR RT) FOR TOXIN ASSAYS.	*>6 DAYS.* SERUM FOR IgM AND IgG.
T-2 TOXICOSIS		
0 TO 24 HOURS POST EXPOSURE. NASAL AND THROAT SWABS AND INDUCED RESPIRATORY SECRETIONS FOR IMMUNOASSAYS, HPLC/MASS SPECTROMETRY	*1 TO 5 DAYS.* SERUM (TT OR RT) AND TISSUE FOR TOXIN DETECTION.	*>6 DAYS POST EXPOSURE.* URINE FOR DETECTION OF TOXIN METABOLITES.
EQUINE ENCEPHALOMYELITIS		
(VEE, EEE, AND WEE VIRUSES) *0 TO 24 HOURS.* NASAL SWABS AND INDUCED RESPIRATORY SECRETIONS FOR RT-PCR AND VIRAL CULTURE.	*24 TO 72 HOURS.* SERUM (TT OR RT) AND THROAT FOR CULTURE. SERUM (E, C, H, TT, OR RT) FOR RT-PCR. THROAT SWABS UP TO 5 DAYS FOR CULTURE THEN CSF SERUM (TT OR RT) FOR ANTIGEN ELISA.	*>6 DAYS.* SERUM (TT OR RT) FOR IgM. PATHOLOGY SPECIMENS PLUS BRAIN.
POX		
(SMALL POX AND MONKEYPOX) *0 TO 24 HOURS.* NASAL SWABS AND INDUCED RESPIRATORY SECRETIONS FOR PCR AND VIRAL CULTURE.	*2 TO 5 DAYS.* SERUM (TT OR RT) FOR VIRAL CULTURE.	*>6 DAYS.* SERUM (TT OR RT) FOR VIRAL CULTURE. DRAINAGE FROM SKIN LESIONS/ SCRAPINGS FOR MICROSCOPY, EM, VIRAL CULTURE, AND PCR. PATHOLOGY SPECIMENS.
EBOLA		
0 TO 24 HOURS. NASAL SWABS AND INDUCED RESPIRATORY SECRETIONS FOR RT-PCR AND VIRAL CULTURE.	*2 TO 5 DAYS.* SERUM (TT OR RT) FOR VIRAL CULTURE.	*>6 DAYS.* SERUM (TT OR RT) FOR VIRAL CULTURE. PATHOLOGY SPECIMENS PLUS ADRENAL GLAND.

LEGEND:

BC	Blood Culture	H	Heparin
C	Citrated blood	HPLC	high-pressure liquid chromatography
CSF	cerebrospinal fluid	IgG	immunoglobulin class G
DNA	deoxyribonucleic acid	IgM	immunoglobulin class M
E	EDTA	PCR	polymerase chain reaction
EEE	eastern equine encephalitis	RT	Red Top, if TT is not available
ELISA	enzyme-linked immunosorbent assay	RT-PCR	reverse transcriptase/polymerase chain reaction
EM	electron microscopy	TT	Tiger top
F-1	fraction-1	VEE	Venezuelan equine encephalitis
FA	fluorescent antibody	WEE	western equine encephalitis

TREATMENT PROTOCOLS

FOR THE EXPLOSIVE AGENT/DEVICE INJURED PATIENT

1. Introduction

• Preparing and training to respond to a terrorist event presents many unique challenges to first responders. When studying the consequences of terrorism, the effects of nuclear, biological, and chemical (NBC) exposures must be analyzed individually to determine the impact on victims. Unfortunately, in many terrorist attacks, these types of injuries do not occur in isolation. Rather, multiple complicating factors occur simultaneously. For example, a terrorist may use a small conventional explosive device laced with radioactive materials. In this instance, first responders will be forced to deal with four situations simultaneously: bomb blast injuries, radioactive contamination, psychological injuries, and the threat of personal contamination. Numerous victims will need to be evaluated, triaged, decontaminated, treated, and transported to local hospitals. Since the incident area is now a crime scene, issues of evidence preservation become extremely important.

2. Objective

The purpose of this module is to present an overview of the challenges that face medical professionals responding to

- A mass casualty terrorist incident that involves an explosive device
- A mass casualty terrorist incident which may also contain a nuclear, biological, or chemical weapon of mass destruction (WMD)

3. Mass Casualty Incident

• In an MCI, available resources are taxed by an unusually high number of patients. Triage decisions must be made regarding treatment and disposition of these victims. The number of victims and the availability of personnel and equipment govern these decisions. This definition of an MCI will vary from community to community and from hospital to hospital, depending on the availability of resources.

4. Challenges

- A mass casualty event associated with a terrorist attack may pose special hazards to the responder. It is imperative that first responders take appropriate steps to routinely protect themselves from the possibility of exposure to NBC hazards, and injury from secondary explosive devices.

- Terrorist attacks using NBC weapons may produce large numbers of casualties. In past incidents, many of the victims who sought medical care were suffering from psychosomatic ailments produced by the stress of the incident. These psychogenic casualties can create major logistical problems for the healthcare system. These victims should be transported to a Casualty Collection Point (CCP), where they can be observed by medical personnel for worsening conditions and "defused" by crisis intervention teams. Consider using schools, parks, gymnasiums, etc., as patient collection points. Crisis teams should have sufficient personnel to aid in the assessment of these victims within emergency departments (EDs) as well.

5. Mechanics of an Explosion

- Bombs are composed of a variety of explosive materials. When these bombs are detonated, the reaction produces an instantaneous chain of events in which the explosive material is rapidly converted into a gas under extremely high pressure and temperature. This gaseous by-product is transmitted to the surrounding medium as a blast wave (or shock wave) that travels outward from the explosion.

- After the explosion occurs, a mass movement of air (blast wind) that was originally displaced by the explosive products follows the explosion at speeds that can reach hurricane proportions. This blast wind may be as damaging as the original explosion.

6. Injury Mechanisms

• This type of reaction occurs when high-energy explosives are used (such as plastic explosives, TNT, diesel fuel, and fertilizer. High-energy explosives detonate faster than the speed of sound. In low-energy explosives such as a pipe bomb, the pressure within the casing increases so rapidly that it explodes, releasing high velocity shrapnel as its most deadly byproduct. Low-energy explosives react slower than the speed of sound.

• If a solid structure such as a wall or building is present in the path of the explosion, the blast wave will rebound off this structure and generate a reflective force that is magnified almost nine times its original strength. As a result, victims caught between the blast and a building may suffer injuries two to three times greater than expected for the amount of explosive detonated and the distance from the explosion.

7. Mechanism of Injury

After the bomb explodes, the sudden change in pressure causes a variety of injuries that are divided into four main categories.

- **Primary blast injuries.**
  - These injuries occur when the blast wave travels through the body and damages organs and tissues that have air and fluid (blood) in contact with each other. This is most readily seen in the lungs, ears, bowel, heart, and brain. As the blast wave strikes these organs, the blood, which is a more dense and non-compressible tissue, is either thrown (spalling) or pulled into the less dense air containing tissue, resulting in injury. For example, when a blast wave strikes and begins to pass through the chest, the pressure of the blast wave forces the blood of the pulmonary vasculature into the less dense air cells of the lungs (alveoli). As the blast wave passes through, additional blood from the pulmonary vasculature is then "pulled" into the lung tissue. Both processes combine to cause hemorrhage.
  - In addition, when the blast wave passes through an organ containing pockets of air (that is, middle ear, lungs, and intestines), the pressure of the wave compresses the air within. Once the shock wave passes, the compressed air re-expands with a greater intensity causing miniature explosions called "implosions."
- **Secondary blast injuries.** These injuries occur from the rapid acceleration of small debris such as flying glass and shrapnel produced from the explosion. These small fragments may be accelerated to velocities capable of causing skin lacerations and body cavity penetrations. The energy from the shrapnel (related to mass and velocity) is transmitted directly and completely to the traumatized tissue, causing fractures to bones and massive soft tissue damage.

- **Tertiary blast injuries.** These injuries occur when a victim is thrown in the air from the force of the explosion (blast wind) and is pushed into a stationary object. If a 70-kilogram (kg) person is accelerated into a solid vertical object at 18 mph, 50 percent mortality can be expected.
- **Miscellaneous blast effects.** These include flash injuries from the thermal component of the explosion, burns from secondary fires started from the blast, and crush injuries resulting in kidney failure and sepsis. Inhalation of toxic fumes or exposure to NBC contaminants is also possible. Neuropsychiatric conditions such as amnesia, temporary blindness, or paresthesias are common.

8. Injury Patterns

• Most victims who survive a bomb blast will suffer from some degree of secondary and/or tertiary bomb injuries. Primary blast injuries, beyond injuries to the ear (such as eardrum rupture, nerve injury), are infrequently seen in survivors. Individuals who would suffer primary blast injuries are usually so close to the explosion that they are typically killed by the secondary and tertiary blast effects. They die from brain injuries, skull fractures, diffuse lung contusions, liver and spleen lacerations, or traumatic amputations.

• There are, however, exceptions to this general rule. For example, after a recent bus bombing in Israel, a number of survivors were found to have primary blast injuries to the lung and gut. From a number of terrorists bombing studies, only about 15 percent of survivors require hospital admission. Most of these individuals suffered multiple injuries, but their admission was related to one single cause, such as concussion, fracture, or burn. Most victims are treated and released from the ED.

9. Explosive Agent Triage

• Bombing casualties that can walk and talk, who are alert and oriented, and have intact hearing are triaged as **minimal**, but those who have experienced a decrease or loss of hearing may have suffered trauma from the blast and are placed in the **immediate** category. These patients should be observed closely for at least 6 to 12 hours after the incident because primary blast injuries may not always be present when the victim is first evaluated.

• In a study of victims after a bus bombing in Israel, two victims had serious gut injuries that were missed for 3 to 7 days after the explosion.

- The basic principles of trauma life support emphasize life-saving intervention (ABCs). Oxygen should be used liberally for those complaining of shortness of breath. Respiratory assistance (that is, bag-mask ventilation or intubation) should be provided with care, especially in those patients suspected to have primary blast injury to the lungs (i.e., short of breath and hypoxic). In these patients, the torn lung tissue and damaged blood vessels are in direct communication with each other, increasing the likelihood of air entering the vasculature and causing an air embolism. These patients will require high frequency/low pressure ventilation. In addition, the increased pressure generated from mechanical ventilation may cause air to leak out of the damaged alveoli and collect in the pleural space, resulting in a pneumothorax. If this were to occur, chest tube placement would be required (preceded by needle decompression in the case of a tension pneumothorax).

- Air embolism appears clinically as dyspnea, tachycardia, hypoxia, tachypnea, chest pain, altered mental status, anxiety, and syncope. Treatment of an air embolism initially requires the patient to lie on their left side with legs elevated (Trendelenburg position). Hyperbaric therapy is the preferred treatment and must be instituted quickly. Injured extremities should be splinted. Intravenous fluids should be used in a gentle manner to prevent further harm to the blast-injured pulmonary tissue.

- Wound management takes on great importance since the amount of tissue damaged from an explosion is typically severe. The bodily injuries from terrorist bombings are caused by high-velocity, irregularly shaped shrapnel and debris that result in extensive tissue destruction and contamination. For these reasons, adequate and extensive surgical debridement is essential and primary closure (sutures) should be delayed for at least 5 days.

10. Special Considerations

- In a terrorist bombing, the potential for secondary contamination with NBC agents should always be considered. If contaminants are found or suspected, victims should be decontaminated with soap and water. At a minimum, their clothing should be removed, double-bagged (paper bags for explosives, paper bags into plastic bags for chemicals and explosives), and their wounds irrigated with sterile water and covered with a sterile dressing prior to hospital transport. This is especially true in unstable, multiple trauma victims who are potentially contaminated with NBC agents. Contaminated foreign bodies that remain in the wound require emergency surgical intervention and removal.

- A bombing site should be secured and declared free of any additional explosive, chemical, or radioactive material before **unprotected** emergency responders are allowed to enter the scene. A second weapon has been frequently utilized by terrorists in Ireland and Israel, and most recently in the U.S. (Atlanta, Georgia in January 1997). The presence of radiological materials (alpha, beta, and gamma) can be quickly determined by using survey meters (such as an alpha meter and/or a Geiger Counter). This should be considered a routine practice at any bombing site. The responding Hazardous Materials Response Team (HMRT) should also undertake a routine survey for chemical contamination. If a biological weapon is suspected, routine protective gear worn by first responders (fire, EMS, law enforcement) will be adequate if it includes gloves and respiratory protection [such as high-efficiency air particulate (HEPA) filter-style mask or an air-purifying or atmosphere-supplied respirator].

11. Crush Syndrome

- Produced by prolonged and continuous pressure on extremities
- Skeletal muscle death releases cellular toxins
- Results in renal failure, lethal cardiac arrhythmias, and sudden death
- Clinical presentation depends upon length of time extremity has been crushed

TREATMENT PROTOCOLS

FOR THE RADIOLOGICAL AGENT INJURED PATIENT

1. Definitions

a. **Radiation:** In its simplest definition, radiation can be defined as either electromagnetic or particulate emissions of energy from the disintegration of the nucleus of an atom. This energy, when impacting on or passing through material, including us, can cause some form of reaction. This radiation is also referred to as ionizing radiation.

b. **Radioactive material:** Again, this is simply any material which is giving off some form of radiation.

2. Ionizing Radiation

a. When ionizing radiation is absorbed by our bodies, it can cause changes to our cells. Small amounts can be tolerated; larger amounts can be harmful.

For our purposes, this radiation can be classified as:

(1) Alpha particles

(2) Beta particles

(3) Gamma Radiation

Again, for our purposes, we're not so concerned with the mechanism of radiation as we are with the hazard, the detection of it and protection from it.

Types of Ionizing Radiation

Alpha particles are massive, charged particles (4 times the mass of a neutron). Because of their size, alpha particles cannot travel far and are fully stopped by the dead layers of the skin or by a uniform. Alpha particles are a negligible external hazard, but when they are emitted from an internalized radionuclide source, they can cause significant cellular damage in the region immediately adjacent to their physical location.

Beta particles are very light, charged particles that are found primarily in fallout radiation. These particles can travel a short distance in tissue; if large quantities are involved, they can produce damage to the basal stratum of the skin. The lesion produced, a "beta burn," can appear similar to a thermal burn.

Gamma rays, emitted during a nuclear detonation and in fallout, are uncharged radiation similar to x rays. They are highly energetic and pass through matter easily. Because of its high penetrability, gamma radiation can result in whole-body exposure.

Neutrons, like gamma rays, are uncharged, are only emitted during the nuclear detonation, and are not a fallout hazard. However, neutrons have significant mass and interact with the nuclei of atoms, severely disrupting atomic structures. Compared to gamma rays, they can cause 20 times more damage to tissue.

When radiation interacts with atoms, energy is deposited, resulting in ionization (electron excitation). This ionization may damage certain critical molecules or structures in a cell. Two modes of action in the cell are direct and indirect action. The radiation may directly hit a particularly sensitive atom or molecule in the cell. The damage from this is irreparable; the cell either dies or is caused to malfunction.

The radiation can also damage a cell indirectly by interacting with water molecules in the body. The energy deposited in the water leads to the creation of unstable, toxic hyperoxide molecules; these then damage sensitive molecules and afflict subcellular structures.

3. Units of Radiation

To quantify amounts of radiation, the term rem or millirem is used. It has a specific definition, but we're concerned with how many rather than a definition.

Note: rem = roentgen equivalent man

rem = rad 2 RBE

rad = radiation absorbed dose (deposition of 100 ergs of radiation energy per gram of absorbed material)

RBE = relative biological effectiveness

This charge will give you an idea of doses we receive through some normal activities. The threshold for any real consequences begins around 200 rem. The LD_{50} is around 450 rem.

Note: In 1975, the 15th General Conference on Weights and Measurements adopted the International System of Units (SI System).

Other terms you may see or encounter are:

a. **Gray (Gy): 1 rad = 1cGy or 1Gy = 100 rads**
b. **Sievert (Sv): 1 Sv = 100 rems**
c. **So again for our purposes, 1 Sv = 1 Gy**

4. Detection

Radiation cannot be detected by our senses, but each type can be detected and identified with instrumentation.

Most HAZMAT teams are already equipped with radiation detectors. These types of detectors will be covered in a later class.

Most of the radiation detection instruments will measure radiation in dose rates, or how much radiation is being absorbed per unit of time, i.e., 50 mrem/hr.
Because the threat exists, checking for the presence of radiation as part of a HAZMAT response is probably a good idea.

a. Symptoms of Radiation

In most instances, it takes considerable time before an individual begins to show symptoms of radiation. Of course, there are always exceptions. If one would pick up a very active material, he/she could receive radioactive burns on the skin which would show up in a matter of hours.

5. Health Hazards

Risk depends upon several factors:

a. The total of radiation received (dose)
b. The dose rate (how fast the dose is received)
c. The specific type of radiation

The dose rate can further be defined by the duration of exposure. Radiation effects are further defined or categorized as acute, where you begin to show symptoms within 24 hours; chronic, where one receives a lesser dose of radiation resulting in less noticeable symptoms; and delayed, where symptoms such as a tumor cancer may not show up until years later.

6. Health Risks During an Incident

The three concerns at an incident involve whole body exposure, ingestion of radioactive material (inhalation, ingestion) or contamination by radioactive material. Incidents involving either an explosion or fire will elevate the potential for the ingestion or contamination by the spreading of the radioactive material in the form of small fragments (dust) or smoke.

a. Terrorist Use of Radioactive Material

It is not inconceivable that a terrorist could obtain radioactive material from a medical facility or other activity and place it in a facility, more to cause an incident and scare a lot of people rather than actually create casualties. This exact scenario occurred in Russia in November 1995. A 30 pound package containing explosives and Cesium, a radioactive material, was placed in a Moscow park by Chechan Separatists. In this instance, the device was located and rendered safe before it detonated. If it had detonated, it would have created a significant cleanup problem; $Cesium^{137}$ has a half-life of about 30 years.

7. Protection

a. Time
b. Distance
c. Shielding

Looking at each of these, the amount of radiation you receive will depend on the type and strength of the radiation and the amount of time you are exposed.

a. Time

An example is as follows: you are exposed to radioactive source and are receiving 100 mrems per hour. If your are exposed for 15 minutes, you have received 25 mrems. Cutting down your time reduces your exposure.

b. Distance

Distance is also critical. Referring back to our forms of radiation, Alpha particles only travel a little over an inch in air. Beta particles will not travel over a few yards in air. However, gamma will travel extensive distances and this is the radiation we are the most concerned with. The farther you are from a source the better. With gamma, the intensity decreases by a factor of the square of the distance.

Note: There is a simple formula for computing the distance factor:

$$D=S/d^2$$

Suppose you are standing 1 meter (2 steps) from a source, and are being exposed to 100 mrem per hour. By moving back to 2 meters (4 steps), you reduce your exposure to 25 mrem per hour ($D=S/d^2$ or D=100/4 or 25). Conversely, if you move to within 1@ meter (1 step), your exposure jumps to 400 mrem per hour. ($D=S/d^2$ or D=100/.25 or 400 mrem per hour).

c. Shielding

Radiation can also be blocked or partially blocked by various materials: Alpha radiation is stopped by a sheet of paper, Beta radiation is stopped by aluminum foil or clothing, and Gamma rays are only reduced by dense materials such as lead or earth.

Note:

a. **Alpha travels approximately 1-1.5 inches in air and cannot penetrate unbroken skin or paper.**
b. **Beta travels approximately 10 feet in air and can penetrate a few millimeters of tissue. Can be stopped by light layers of clothing, aluminum foil or an average book (approx. 1-1.5 inches thick).**
c. **Gamma travels indefinitely in air, and can penetrate the human body. Intensity is reduced by heavy, dense materials such as steel, concrete, earth or lead.**

8. HEPA Filters

Here is an example of High Efficiency Particulate Absorbing P100 (HEPA) filter attached to a full-face Air Purifying Respirator (APR). There are numerous manufacturers.
Because of the ease of protecting from alpha and beta radiation, our main concern from these is inhalation or ingestion of actual radioactive material in the form of dust or contaminated food or water. This type of mask filter provides effective protection against inhalation of radionuclides. Gamma is more difficult to protect against and this is where time, distance and shielding are most important.

9. Decontamination

a. Wet—wetting down will tend to cause the radioactive material to adhere to clothing and skin, rather than re-aerosolizing, thus preventing it from being ingested.

b. Strip—remove contaminated clothing.

c. Flush—remove any contamination from exposed skin and hair.

d. Cover—for protection

Radiological decontamination is performed in an identical manner to doctrinal chemical decontamination. The main difference is in timing. Chemical decontamination is an emergency. Radiological decontamination is not.

Decontamination of casualties is an enormous task. The process requires dedication of both large numbers of personnel and large amounts of time. Even with appropriate planning and training, the requirement demands a significant contribution of resources.

Removal of outer clothing and rapid washing of exposed skin and hair removes 95% of contamination. The 0.5% hypochlorite solution used for chemicals will also remove radiological contaminants. Care must be taken to not irritate the skin. If the skin becomes erythematous, some radionuclides can be absorbed directly through the skin. Surgical irrigation solutions should be used in liberal amounts in wounds, the abdomen, and the chest. All such solutions should be removed by suction instead of sponging and wiping. Only copious amounts of water, normal saline, or eye solutions are recommended for the eye. Additional care of contaminated wounds is discussed below.

Radiological particulate transfer is a potential problem that can be resolved by a second deliberate decontamination. Decontamination at the medical treatment facility prevents spread of contamination to areas of the body previously uncontaminated, contamination of personnel assisting the patient, and contamination of the medical facility.

Wound Decontamination

All casualties entering a medical unit after experiencing a radiological attack are to be considered contaminated unless there is certification of noncontamination.

The initial management of a casualty contaminated by radiological agents is to perform all immediate life/limb-saving actions without regard to contamination. Removal of clothing and other exterior garments during the course of resuscitation will remove nearly all contamination except where the suit has been breached.

Initial Decontamination

During initial decontamination in the receiving areas, bandages are removed and the wounds are flushed; the bandages are replaced only if bleeding recurs.

General Considerations

Only high energetic gamma emitters present any immediate hazard in wound contamination. It is impossible for a living patient to be so contaminated as to pose a threat to medical providers. Local wound contamination is by particulate matter that should be removed if possible. Alpha and beta emitters left in the wound will cause extensive local damage and may be absorbed into the systemic circulation and redistributed as internal contaminants.

Aggressive surgery such as amputation or extensive exploration should not be undertaken to "eliminate radioactive contamination." The surgical damage will far exceed any potential decrease in lifetime radiological exposure risk.

Partial-thickness burns should be thoroughly irrigated and cleaned with mild solutions to minimize irritation of the burned skin. Blisters should be left closed; open blisters should be irrigated and treated in accordance with appropriate burn protocols. In full-thickness burns, radioactive contaminants will slough in the eschar. As there is no circulation in the burned tissue, contaminants will remain in the layers of dead tissue.

Excision of wounds is appropriate when surgically reasonable. Radioactive contaminants will be in the wound surfaces and will be removed with the tissue.

Decontamination of Equipment

In most cases of contamination of equipment and buildings, a mixture of normal housecleaning methods will remove the material. Vacuum cleaners that can handle wet material and have high-efficiency filters are particularly useful. Some surfaces may require repeated scrubbing and vacuuming before they are free of contamination.

Table for Medical Assay of the Radiological Patient

Test/location	Decon point	Medic. treat unit	Hospital	Tertiary care
Nasal swabs for Inhalation of contaminants	+			
External Contamination	+	+	+	
Urine and stool sample for internal contamination		Base-line sample	24-h sample	+
CBC*/platelets		Daily	Daily × 1 wk	Daily × 1wk
Absolute lymphocyte count		Every 12 h	Every 12 h × 3 d	
HLA† subtyping		Draw sample	Draw sample before lymphocyte count falls	Draw sample before lymphocyte count falls
Cytomegalovirus			+	+
Hemoglobin agglutinin			+	+
Human syncytial cell virus antibodies				+
Human immunovirus			+	+
Vesiculovirus				+
Lymphocyte cytogenetics		Draw sample and send forward	Draw sample before lymphocyte count falls	+

*CBC = complete blood count
†HLA = human leucocyte antigen

Table of Internal Contaminant Radionuclide

Element	Respiratory absorption, deposition	GI absorption, deposition	Skin wound absorption	Primary toxicity	Treatment
^{241}Am	75% absorbed 10% retained	Minimal, usually insoluble	Rapid in first few days	Skeletal deposition Marrow suppression Hepatic deposition	Chelation with DTPA or EDTA
^{137}-^{134}Ce	Completely absorbed Follows Potassium	Completely absorbed Follows potassium	Completely absorbed Follows potassium	Renal excretion Beta and gamma emissions	Ion exchange resins Prussian blue
^{60}Co	High absorption Limited retention	<5% absorption	Unknown	Gamma emitter	Gastric lavage Penicillamine in severe cases
^{131}I	High absorption Limited retention	High absorption Limited retention	High absorption Limited retention	Thyroid ablation	Iodine therapy
^{32}P	High absorption Limited retention	High absorption Limited retention	High absorption Limited retention	Bone, rapidly replicating cells	Lavage, Aluminum hydroxide Phosphates
$^{238\text{-}239}$Pu metal or salt	High absorption Limited retention	Minimal, usually insoluble	Limited absorption May form nodules	Lung, bone, liver	Chelation with DTPA or EDTA

$^{238-239}$Pu High-Fired oxides	Limited absorption High retention	Minimal, usually insoluble	Limited absorption May form nodules	Local effects from retention in lung	Chelation with DTPA or EDTA Pulmonary lavage*
^{210}Po	Moderate absorption Moderate retention	Minimal	Moderate absorption	Spleen, kidney	Lavage Dimer-caprol
^{226}Ra	Unknown absorption, but 95%	30% fecal excretion	Unknown deposition Marrow	Skeletal lavage Ammo-suppres-sion Sarcoma	MgSO4 nium chloride Calcium Alginates
^{90}Sr	Limited retention	Moderate absorption	Unknown	Bone-follows calcium	Strontium Calcium Ammo-nium chloride
Tritium (T or ^{3}H) Hydro-gen-3 Tritiated water = HTO	HT— minimal HTO– complete	HT— minimal HTO— complete	HTO— complete	Panmyelo-cytopenia	Dilution with controlled water intake, Diuresis
$^{238-235}$U fluorides UO3, sulfates, carbonates	High absorption High retention	High absorption	High absorption Skin irritant	Renal, urinary excretion	Chelation with DPTA* or EDTA $NaHCO^{3}$
$^{238-235}$U Some oxides, nitrates	Moderate absorption High retention	Moderate absorption	Unknown	Nephro-toxic Urinary excretion	Chelation with DTPA* or EDTA $NaHCO^{3}$

$^{238\text{-}235}U$ High oxides, hydrides, carbides salvage ash	Minimal absorption Retention based on particle size	Minimal absorption High excretion	Unknown	Nephro-toxic Urinary excretion	Chelation with DTPA* or EDTA $NaHCO^3$
^{228}U Depleted uranium metal	Retention based on particle size	Minimal absorption High excretion	Forms pseudo-cysts with urinary excretion	Nephro-toxic Deposits in bone, kidney, Limited absorption	Particulate removal when possible brain

*Treatment is not approved by the Food and Drug Administration. Clinical investigations have not begun in the United States.

APPENDICES

APPENDIX
MSDS Review

Material Safety Data Sheets (MSDS) are critical documents. Getting them or getting any information about a hazardous material that might be involved in an incident can sometimes be a problem.

Here are some possible solutions:

1. Get MSDS from the responding fire company with whom the MSDS should have been filed according to SARA Title III regulations or from the company whose product is involved.
2. Ask questions of any company personnel on site who may have manufacturing expertise.
3. Encourage local companies to place near their entrance a lock box with their MSDS inside.
4. Bring with you in the rig whatever MSDS books or hazardous materials guides you may have.

Using an MSDS

Materials Safety Data Sheets vary in format. Since there is no standard format, manufacturers can present their document information in their own way. The information you need to know, however, is always present—but you may have to hunt for it. Tip: Get MSDS from local companies and review their approach to communicating this kind of information.
In looking at an MSDS, search for:

1. the product name,
2. precautionary statements,
3. first aid information regarding routes of exposure, exposure limits, effects of exposure (target organs),

Look for this information under headings such as:

"BLS/First Aid Procedures"
"Primary Routes of Entry"
"Emergency and First Aid Procedures"
"Health Hazard Information"

4. personal protective clothing recommended.
5. Other chemical and physical characteristics such as fire, explosion and reactivity hazards.

6. The name of the manufacturer or MSDS preparer, address and emergency telephone number.
7. Safe handling procedures for spills or leaks.
8. An indication if the material is listed with National Toxicology Program, IARC or OSHA.

At times certain information on chemical contents will be withheld from the MSDS as "trade secrets." A treating physician can obtain this information on an emergency need to know basis from the manufacturer immediately. The physician will be required to follow up with a written request and sign a non-disclosure agreement.

Enclosed are **sections** from Materials Safety Data Sheets for your review. For each material, please discuss:

1. What should be your basic response to an incident involving this material?
2. What EMS protective clothing would be needed?
3. What equipment would be needed?
4. What disposable equipment could we use?*
5. What equipment could we commit that could be decontaminated later?

*Tip: Practice working with both surgical gloves and latex gloves on. Test what duties you can and cannot perform when wearing this protective combination.

MSDS NO. 1218-04
CAS NO. ————————-
DATE: 10/03/86

PRODUCT IDENTIFICATION

TRADE NAME:	**ACCURAC(r) 135 RETENTION AID**
SYNONYMS:	Cationic polyacrylamide in water-in-oil emulsion
CHEMICAL FAMILY:	Cationic polyacrylamide
MOLECULAR FORMULA:	Mixture
MOLECULAR WGT.:	Mixture

WARNING

DANGER! CAUSES SKIN BURNS
HARMFUL IF INHALED
MAY CAUSE EYE IRRITATION

HAZARDOUS INGREDIENTS

COMPONENT	CAS. NO.	%	TWA/CEILING	REFERENCE
Petroleum distillate	008002-05-9	26.5	500 ppm	OSHA

NFPA HAZARD RATING

Fire
1
Health 2 0 Reactivity
Special

FIRE: Material that must be preheated before ignition can occur.
HEALTH: Materials which on intense or continued exposure could cause temporary incapacitation or possible residual injury unless prompt medical treatment is given.
REACTIVITY: Materials which in themselves are normally stable, even under fire exposure conditions, and which are not reactive with water.

HEALTH HAZARD INFORMATION	EFFECTS OF OVEREXPOSURE	The acute oral (rat) and acute dermal (rabbit) LD_{50} values are both >10 ml/kg. Minimal eye irritation was produced during primary irritation testing in rabbits. When this product was tested for skin irritation under occlusive conditions, as would occur if the product was spilled into boots, irreversible skin damage was produced. However, when this product was tested under open conditions as would occur if the product was spilled on clothing, only mild skin irritation was produced after 24 hours of contact. Aspiration of the solvent, petroleum distillate, may cause chemical pneumonitis. Overexposure to vapor of petroleum distillates may cause dizziness, headache, nausea, and irritation of the respiratory tract.
	FIRST AID:	In case of skin contact, remove contaminated clothing without delay. Wear impervious gloves. Cleanse skin thoroughly with soap and water. Do not omit cleaning hair or under fingernails if contaminated. Do not reuse clothing without laundering. Do not reuse contaminated leatherware. In case of eye contact, immediately irrigate with plenty of water for 15 minutes.

PRODUCT NAME: Glutaraldehyde (25% by weight)

III. INGREDIENTS

MATERIAL	%	TLV (Units)	HAZARD
Glutaraldehyde CAS # 111-30-8	25	See Section V	See Section V
Water CAS # 7732-18-5	~75	None established	See Section V
Methanol CAS # 67-56-1	<0.05	See Section V	See Section V

IV. FIRE AND EXPLOSION HAZARD DATA

FLASH POINT (test method(s)):	None, Tag Closed Cup ASTM D 56 None, Cleveland Open Cup ASTM D 92	
FLAMMABLE LIMITS IN AIR, % by volume:	LOWER: UPPER:	Not determined (aqueous system) Not determined (aqueous system)
EXTINGUISHING MEDIA:	Non-Flammable (Aqueous System): After the water evaporates, the remaining material will burn. Use alcohol-type or all-purpose-type foam applied by manufacturer's recommended technique for large fires. Use CO2 or dry chemical media for small fires.	
SPECIAL FIRE FIGHTING PROCEDURES:	Use self-contained breathing apparatus and protective clothing.	
UNUSUAL FIRE AND EXPLOSION HAZARDS:	None	

PRODUCT NAME: Glutaraldehyde (25% by weight)

V. HEALTH HAZARD DATA

TLV AND SOURCE:	Glutaraldehyde—0.2 ppmv, ceiling OSHA & ACGIH 1988-89 Methanol—200 ppm, skin OSHA & ACGIH 1988-89
EFFECTS OF SINGLE OVEREXPOSURE: SWALLOWING:	Moderately toxic. May cause moderate to marked irritation or chemical burns of the mouth, throat, esophagus, and stomach. There will be discomfort or pain in the chest and abdomen, nausea, vomiting, diarrhea, dizziness, faintness, drowsiness, weakness, circulatory shock, collapse and coma.
SKIN ABSORPTION:	Toxicology studies indicate that prolonged or widespread contact could result in the absorption of potentially harmful amounts of material.
INHALATION:	Vapor is irritating and will cause stinging sensations in the nose and throat, coughing, chest discomfort and tightness, difficulty with breathing, and headache.
SKIN CONTACT:	Brief contact may result in mild to moderate local redness and possibly swelling. Prolonged contact may result in severe inflammation.
EYE CONTACT:	Liquid will cause severe conjunctivitis, seen as discharge with marked swelling and excess redness of the conjunctiva. Severe corneal injury may occur. Vapor will cause stinging sensations with excess lachrymation, but not injury.
EFFECTS OF REPEATED OVEREXPOSURE:	None known from currently available information.
MEDICAL CONDITIONS AGGRAVATED BY OVEREXPOSURE:	Because of its irritating properties, this material may aggravate an existing dermatitis.
SIGNIFICANT LABORATORY DATA WITH POSSIBLE RELEVANCE TO HUMAN HEALTH HAZARD EVALUATION:	Laboratory studies have shown that glutaraldehyde is not teratogenic, and several studies have shown the material not to be a mutagen.
OTHER EFFECTS OF OVEREXPOSURE:	May cause skin sensitization in a small proportion of individuals, and present as an allergic contact dermatitis.
EMERGENCY AND FIRST AID PROCEDURES: SWALLOWING:	Give at least two glasses of water. Do not induce vomiting. Seek medical assistance with urgency.
SKIN:	Wash contaminated skin with soap and water. If contact has been widespread and prolonged, or if irritation persists, seek medical advice. Contaminated clothing should be washed before reuse.
INHALATION:	Remove to fresh air. If breathing is difficult, administer oxygen. If symptoms persist, call a physician.
EYES:	Immediately flush eyes thoroughly with water and continue flushing for at least 15 minutes. See an ophthalmologist urgently.

NOTES TO PHYSICAN:

Aspiration may cause lung damage. Probable mucosal damage may contraindicate the use of gastric lavage; however, if gastric lavage is considered necessary, it should be undertaken with caution. Most of the adverse effects of glutaraldehyde are due to its intensely irritating properties. Because of this vomiting should not be induced in cases of poisoning by swallowing. There is no specific antidote. Treatment of overexposure should be directed at the control of symptoms and the clinical condition of the patient.

IX. SPECIAL PRECAUTIONS

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORAGE:

DANGER: CORROSIVE
CAUSES IRREVERSIBLE EYE DAMAGE.
CAUSES SKIN IRRITATION.
HARMFUL IF INHALED.
HARMFUL IF SWALLOWED.
HARMFUL IF ABSORBED THROUGH SKIN.
MAY CAUSE SKIN SENSITIZATION.
Do not get in eyes, on skin, on clothing.
Avoid breathing vapor.
Do not swallow.
Wear goggles, protective clothing, and rubber gloves.
Wash thoroughly with soap and water after handling.
Remove contaminated clothing and wash before reuse.
FOR INDUSTRY USE ONLY

OTHER PRECAUTIONS:

Laboratory studies, using an odor test panel, indicated glutaraldehyde vapors in air may be 'irritating' to humans at about 0.3 ppm in air; the TLV has been established as 0.2 ppm ceiling. Thus, if vapors are concentrated enough to be irritating, the TLV is probably being exceeded.

SECTION I PRODUCT IDENTIFICATION & EMERGENCY INFORMATION

PRODUCT NAME

ECA 10454

CHEMICAL FAMILY

Lube oil additive containing a zinc salt of dialkyl dithiophosphoric acid, borated polyisobutenyl succinic anhydride nitrogen functionalized dispersant, magnesium alkylaryl detergent, solvent extracted mineral oil, and other components judged not to affect the potential health or environmental impact of the product.

EMERGENCY TELEPHONE NUMBERS:

CHEMTREC 800-424-9300

SECTION II HAZARDOUS COMPONENTS OF MIXTURES

THE PRECISE COMPOSITION OF THIS MIXTURE IS PROPRIETARY INFORMATION. A MORE COMPLETE DISCLOSURE WILL BE PROVIDED TO A PHYSICIAN OR NURSE IN THE EVENT OF A MEDICAL EMERGENCY. THE FOLLOWING COMPONENTS ARE DEFINED HAZARDOUS IN ACCORDANCE WITH 29 CFR 1910, 1200:

OSHA HAZARD	COMPONENT
Eye irritant	Zinc salt of dialkyl dithiophosphoric acid

For additional information see Section X.

SECTION III HEALTH INFORMATION AND PROTECTION

FIRST AID & NATURE OF HAZARD

EYE CONTACT:

Flush eyes with large amounts of water until irritation subsides. If irritation persists, get medical attention.
Irritating, and may injure eye tissue if not removed promptly.

SKIN CONTACT:

Flush with large amounts of water; use soap if available.
Remove grossly contaminated clothing, including shoes, and launder before reuse.
Low order of toxicity.
Frequent or prolonged contact may irritate.

INHALATION:

Using proper respiratory protection, immediately remove the affected victim from exposure. Administer artificial respiration if breathing is stopped. Keep at rest. Call for prompt medical attention.
Negligible hazard at ambient (–18 to 38 Deg. C) or recommended blending temperature.
Warning if heated above 60 Deg. C (140 Deg. F) especially in the presence of water, hydrogen sulfide may be released; this can cause respiratory collapse, coma and death without necessarily any warning odor being sensed.
Avoid breathing vapors or mists.

INGESTION:

DO NOT induce vomiting. If individual is conscious, give milk or water to dilute stomach contents. Keep warm and quiet. Get prompt medical attention. DO NOT attempt to give anything by mouth to an unconscious person.
Minimal toxicity.

Toxicity information is often expressed as the dose of the compound that causes an effect in a percentage of the exposed subjects, which are mostly experimental animals. These dose-response terms are often found in Material Safety Data Sheets (MSDS) and other sources of health information. One dose-response term that is commonly used is the lethal dose 50 (LD_{50}), the dose which is lethal to 50% of an animal population from exposure by any route other than inhalation when given all in one dose. Another similar term is the lethal concentration 50 (LC_{50}), which is the concentration of a material in air that on the basis of respiratory exposure in laboratory tests is expected to kill 50% of a group of test animals when administered as a single exposure (usually 1 hour).

The LD_{50} values that appear in an MSDS or in the literature must be used with caution by emergency medical personnel. These values are an index of only one type of response and give no indication of the ability of the compound to cause nonlethal, adverse or chronic effects. Furthermore, LD_{50} values typically come from experimental animal studies. Because of the anatomical and physiological differences between animals and humans, it is difficult to compare the effects seen in experimental animal studies to the effects expected in humans exposed to hazardous materials in the field. Therefore, emergency medical personnel should remember that the LD_{50} and LC_{50} values are only useful for comparing the relative toxicity of compounds and should only be used to determine if one chemical is more toxic than another.

Responses to toxic chemicals may differ among individuals because of the physiological variability that is present in the human population. For example, an individual may be more likely to experience an adverse health effect after exposure to a toxic chemical because of a reduced ability to metabolize that compound. The presence of preexisting medical conditions can also increase one's susceptibility to toxic chemicals. Respiratory distress in patients or workers with asthma may be triggered by exposure to toxic chemicals at lower concentrations than might be expected to produce the same effect in individuals without respiratory disease. Factors such as age, personal habits (i.e., smoking, diet), previous exposure to toxic chemicals, and medications may also increase one's sensitivity to toxic chemicals. Therefore, exposure to concentrations of toxic compounds that would not be expected to result in the development of a toxic response in most individuals may cause an effect in susceptible individuals. Not all chemicals, however, have a threshold level. Some chemicals that produce cancer (carcinogens) may produce a response (tumors) at any dose level. Any exposure to these compounds may be associated with some risk of developing cancer. Thus, literature values for levels which are not likely to produce an effect do not guarantee that an effect will not occur.

Exposure Limits

The various occupational exposure limits found in the literature or in an MSDS are based primarily on time-weighted average limits, ceiling values, or ceiling concentration limits to which the worker can be exposed to without adverse effects.

Because the settings in which these values are appropriate are quite different than an uncontrolled spill site, it is difficult to interpret how these values should be used by emergency medical personnel dealing with a hazardous materials incident. At best, TLV, PEL, IDLH, and REL values can be used as a benchmark for determining relative toxicity, and perhaps assist in selecting appropriate levels of Personal Protective Equipment (PPE). Furthermore, these occupational exposure limits are only useful if the appropriate instrumentation is available for measuring the levels of toxic chemicals in the air at the chemical spill site. Of the above occupational exposure limit values, only the OSHA values are regulatory limits. The ACGIH values are for guidance only and are not regulatory limits. In addition, the ACGIH limits have certain caveats that may or may not affect the usefulness of the values. Some of these conditions are individual susceptibility or aggravation of a preexisting condition. Nevertheless, all emergency medical personnel responsible for the management of chemically contaminated patients should be familiar with these concepts because they will be encountered in various documents dealing with patient care or the selection of PPE.

This brief discussion highlights some fundamental concepts of toxicology. Emergency medical personnel responsible for managing chemically contaminated patients are encouraged to obtain further training in recognizing and treating health effects related to chemical exposures. Also, a list of general references in toxicology is provided at the end of this section that will allow emergency medical personnel to undertake a more in-depth examination of the principles of toxicology.

APPENDIX

SOME USEFUL WEBSITES

1. FEDERAL RESOURCES:

A. EPA/FEMA Websites:

EPA Homepage: http://www.epa.gov/
EPA News and Events, Laws and regulations, Offices, Publications and other resources available to access information about EPA.

Brownfields: http://www.epa.gov/brownfields/
This site provides information on all facets relating to Brownfields development. Information is provided on Brownfields Pilots, liability & cleanup, partnership & outreach, laws & regulations, publications, money matters and other resources.

Chemical Accident Prevention and Risk Management Planning (RMP):
http:/www.epa.gov/swercepp/acc-pre.html
Contains information on the Clean Air Act, Section 112(r) legislation, the Risk Management Program Rule, Fact Sheets, Basic Awareness brochures, training modules, Federal Register notices, press releases, technical guidance documents, model risk management program plans by industrial sector, downloadable computer software, downloadable RMP publications, and many other resource links.

Chemical Emergency Preparedness and Prevention Office (CEPPO):
http://www.epa.gov/swercepp/acc-pre.html
This site provides helpful information on Chemical Accident Prevention and Risk Management Planning. There are links to Fact Sheets, Laws & Regulations, Publications, Federal Register Notices, Press Releases, Technical Guidance Documents, and General Guidance for Risk Management Programs.

Chemical Fact Sheets: gopher://ecosys.drdr.Virginia.edu:70/11/library/gen/toxics
Summaries of information on over 300 chemicals including identifying characteristics, health hazards, ecological effects, and methods to reduce exposure to the chemical. Maintained by the University of Virginia.

Current Hazardous Waste Sites (CERCLIS):
http://www.epa.gov/superfund/oerr/impm/products/cursites/csitetoc.htm
A listing of sites on the CERCLIS list.

Glossary of Terms of the Environment: http://earth1.epa.gov:80/OCEPAterms/
An alphabetical listing of terms associated with the environment.

Headquarters Resources Center Internet Newsbrief Resources:
http://www.epa.gov/natlibra/hqire/inb.htm
A weekly service from EPA Headquarters Resources Center that provides a sampling of new and or useful internet resources for EPA staff or other Environmental Professionals.

Index of EPA Clearinghouses: http://www.epa.gov/epahome/clearing.htm
Includes a link to Air Risk Information Support Centers, Asbestos Management, Clean Air Technology, EPA Learning Institute, Indoor Air Quality, National Response Center, etc.

Integrated Risk Information System (IRIS): http://www.epa.gov/ngispgm3/iris/index.html
IRIS Homepage, database of human health effects that may result from exposure to various substances found in the environment.

Numbers (EPA): http://www.epa.gov/epanumbers.html
Lists the various programs and updated telephone #'s for EPA. The site is supposed to update when changes occur.

Office of Solid Waste and Emergency Response: http://www.epa.gov/swerrims/index.htm
Information on RCRA, Superfund Sites, Solid Waste, Underground Storage Tanks, Chemical Emergency Preparedness and Prevention, Oil Spill Program, etc.

Regional EPA Offices: http://www.epa.gov/swercepp/pubs/regions.html
A listing of all EPA Regional Offices.

Regional FEMA Offices: http://www.epa.gov/swercepp/pubs/fema.html
A listing of all the FEMA Regional Offices.

Whats Hot in EPA: http://www.epa.gov/epahome/hot.html
What is hot on the EPA server.

Whats New in EPA: http://www.epa.gov/docs/WhatsNew.html
Lists daily minutes, updates, documents, standards, etc. as they are released.

B. Federal General Environmental:

Centers for Disease Control and Prevention (CDC): http://www.cdc.gov
This site provides links to health information, travelers' health, subscriptions, publications and products, data & statistics, training & employment opportunities, and funding.

Federal Emergency Management Agency (FEMA): http://www.fema.gov:80/fema/
An index of links alphabetically listed to all FEMA related sites.

National Institute for Occupational Safety & Health (NIOSH):
http://www.cdc.gov/niosh/homepage.html
Information on NIOSH services, publications, documents, training, research, patterns and general information. There are also links to databases and health hazard evaluations.

Occupational Safety and Health Administration (OSHA):
http://www.OSHA.gov/index.html
Information regarding OSHA news releases, regulations, compliance, programs, statistics, training and a myriad of many other topics can be found here.

USGS Guide to Federal Environmental Laws and Regulations:
http://water.usgs.gov/public/eap/env guide/
Contains information on: Air Quality, Water Quality, Solid and Hazardous Substances, Lists of Statutes by Sections, etc.

C. Federal Hazmat:

DOT's Office of Hazardous Materials Safety: http://hazmat.dot.gov
Contains information on: Rules and Regulations, Exemptions and Approvals, Hazmat Enforcement, Spills, International Standards, COHMED, Emergency Response Guidebook, etc.

The National Clearinghouse for Worker Safety and Health Training for Hazardous Materials: http://www.niehs.nih.gov/wetp/clear.htm
Contains information on Safety and Health Resources.

D. Federal Other:

Federal Bureau of Investigation www.FBI.gov

Code of Federal Regulations (CFR): http://www.access.gpo.gov/nara/cfr/cfr-table-search.html
A listing of all available CFR tables available for internet access, search engine for CFR databases, browse capability for all CFR titles and Federal Registry access.

Federal Registry: http://www.access.gpo.gov/su docs/
Access to Federal Registry books, Private Act Issuances, Public laws, United States Government Manual and US Congress Information.

Keeping America Informed—U.S. Government Printing Office: http://www.gpo.gov/
This site provides access to the Code of Federal Regulations, the Federal Register, Public Laws, etc. This is probably todays best and fastest way to access the Federal Register and the CFRs.

National Archives and Records Administration, Code of Federal Regulations:
http://www.access.gpo.gov/nara/cfr/cfr-table-search.html#page1
Official Electronic Copy. Contains information on: Search CFR databases by keywords, Search CFR titles and/or volumes, Search the Federal Register for related documents, etc.

2. STATE RESOURCES:

A. New Jersey Department of Environmental Protection (NJDEP) Websites:

NJDEP Home Page: http://www.state.nj.us/dep/

Air Quality Permitting Program (AQPP): http://www.state.nj.us/dep/aqpp/
This site provides links to help sources on Minor Facilities & Preconstruction Permits, Major Facilities & Operating Permits, Stack Testing, CEMs, Modeling & Risk Assessment, Engineering, and RADIUS and electronic applications.

Bureau of Discharge Prevention:
http://www.state.nj.us/dep/enforcement/relprev/dpcc/fsdpcc.html

Compliance and Enforcement: http://www.state.nj.us/dep/enforcement/index.html

Division of Solid and Hazardous Waste: http://www.state.nj.us/dep/dshw/

Division of Water Quality: http://www.state.nj.us/dep/dwq/

Office of the Commissioner: http://www.state.nj.us/dep/commissioner/index.html

Radiation Protection Programs: http://www.state.nj.us/dep/rpp/
Radon Section: http://www.state.nj.us/dep/rpp/ber/radon/index.htm
This site provides useful information on radon testing and mitigation for home buyers and sellers, and testing for radon in your home. It also provides information on average radon levels and tier assessment in New Jersey. There is information on the Radon Certification Program as well as a list of Certified Radon Testing Businesses and Certified Radon Mitigation Businesses in New Jersey.

Site Remediation Program: http://www.state.nj.us/dep/srp
Contains information on: ISRA, Brownfields, Superfund, UST, Known Contaminated Sites, Regulations and Guidance, and Financial Assistance.

B. State & Local Resources:

State of New Jersey Homepage: http//www.state.nj.us
Access to state agencies, Governor's office, and Legislature.

LOIS, Electronic Law Library: http://www.pita.com
Regulations for Other States.

New Jersey Online (NJO): http://www.njo.com/
Lists communities, forums, news, sports, entertainment, businesses, living, classifieds/ads, user guides, etc.

New Jersey State Police: http://www.state.nj.us/lps/njsp/
Academy, Special School and EMS training, recruiting, current events and road and weather conditions can be found here.

State, County and Local Governments on the net:
http://www.piperinfo.com/piper/state/states.html
A site that gives you links to all state and local government sponsored websites. Also included are some Federal Resources, National Organizations and other miscellaneous links.

3. OTHER RESOURCES:

A. Miscellaneous Resources

Clay Net: http://www.clay.net/ep1.html
Good links to federal/state websites run by EPA, OSHA, DOD, NJDEP, PADEP, etc. It also contains industry sites and links to professional organizations and references.

The Weather Channel: http://www.weather.com/twc/homepage.twc

B. General Environmental:

Agency for Toxic Substances and Disease Registry (ATSDR):
http://atsdr1.atsdr.cdc.gov:8080/atsdrhome.html

All information about the ATSDR program can be found here: Announcements, Address and Phone numbers, Health Assessments and Consultations, Education and Communication, HazMat databases, health studies and many other topics are addressed at this website.

C. Organizations/Commissions:

Academy of Certified Hazardous Materials Managers (ACHMM): http://www.achmm.org
Access to state, federal and other hazardous materials management, safety and environmental links as well as job postings, resumes, and currect technical articles.

American Congress of Governmental Industrial Hygienists (ACGIH) Publications:
http://www.acgih.org/catalog/catfind.asp
Provides a catalog list of ACGIH publications, meeting and event information, and membership.

Chemical Manufacturers Association (CMA): http://www.cmahq.com
Provides links to information on Responsible Care®, publications, workshops/seminars, CHEMTREC, CHEMSTAR, ChemEcology, Health Research.

Joint Commission for Accreditation of Healthcare Organizations http://www.jcaho.org

N.J. Water Environment Association: http://www.njwea.org

Union/Middlesex County Hazardous Materials Advisory Council (HMAC):
http://www.hmac-inc.org
HMAC is a non-profit organization who's mission is to promote the responsible handling of hazardous materials. Committee projects support HMAC's goals: to contribute to a reduction in hazardous materials incidents; promote education to responders, industry, government and the public regarding hazardous materials and their proper handling; promote open communications among all types of residents in Union/Middlesex Counties; and to enhance preparedness, response and recovery capabilities in the event of incidents in Union/Middlesex Counties. Information on the committees and their projects can be found here along with available HMAC publications, training and seminar information and other resource links.

D. Chemcial Databases:

NJ DHSS: http//www.state.nj.us/health/eoh/odisweb/
Access to information from NJRTK (imel. FactSheets) and Occupational Health programs.

Chemfinder: http://chemfinder.camsoft.com/
Allows you to locate common types of chemical information by entering a chemical name, molecular weight or CAS registry number.

Chemical Abstracts Service: http://www.cas.org
Database includes approximately 14 million document records and more than 18 million substance records respectively. Includes databases of chemical reactions, commercially available chemicals and listed regulated chemicals.

Material Safety Data Sheet (MSDS) Search: http://www.msdssearch.com/
A forms-searchable database of MSDS entries providing FREE access to over 750,000 MSDSs.

RTK-Net, the Right to Know Network: http://rtk.net/www/rtknet/homepage.html
A network providing free access to numerous databases, text files and conferences on the environment, housing and sustainable development.

Toxic Release Inventory (TRI): http://www.epa.gov/opptintr/tri/
Contains information on the TRI Program. TRI data, chemicals, envirofacts, national and international programs and TRI contacts are some of the topics found on this page.

E. Bioterrorism Information:

Federal Bureau of Investigation: www.fbi.gov

Medical Management of Chemical and Biological Casualties: www.nbc-med.org

Anthrax Advisory: www.emergency.com

National Workshop on Domestic Preparedness: www.wmdnationalworkshop.com

Counter Terrorism Program Link: www.oep.dhhs.gov

Chemical and Biological Information Analysis Center: www.cbiac.apgea.armymil

Federal Emergency Management Agency www.fema.gov

Soldier Biological and Chemical Command: www.sbccom.gov

National Emergency Management Association: www.nemaweb.org

CDC Bioterrorism Preparedness & Response Network: www.bt.cdc.gov

US Army Medical Research Institute for Infectious Disease: www.dad.gov

APPENDIX

IMPORTANT TELEPHONE NUMBERS

EMERGENCY RESPONSE

NJ DEP HOTLINE . . . (EMERGENCY ACTION LINE) 1-877-WARNDEP
NEW JERSEY STATE POLICE NORMAL WORK HOURS .....609-882-2000 x. 6453
AFTER WORK HOURS
(Weeknights, Weekends) .....609-882-2000 x. 6311
CHEMTREC 800-424-9300
ASSOCIATION OF AMERICAN RAILROADS
BUREAU OF EXPLOSIVES 202-639-2222
EPA REGION II EMERGENCY RESPONSE 908-321-6657
NATIONAL RESPONSE CENTER (U.S.C.G.) 800-424-8802
NEW JERSEY POISON CONTROL CENTER.....800-962-1253
NATIONAL POISON CONTROL HOTLINE 800-764-7661

STATE AGENCIES

NEW JERSEY STATE POLICE:
OFFICE OF EMERGENCY MANAGEMENT .....609-882-2000 x. 6453
DOMESTIC PREPAREDNESS/HAZ MAT EMERGENCY
RESPONSE PLANNING UNIT .....609-882-2000 x. 6463
HAZ MAT TRANSPORTATION .....609-882-2000 x. 2586

DEPARTMENT OF ENVIRONMENTAL PROTECTION:
COMMUNITY RIGHT TO KNOW 609-292-6714
TOXIC CATASTROPHE PREVENTION ACT.....609-633-7289
COUNTY ENVIRONMENTAL HEALTH ACT.....609-292-6028
DISCHARGE PREVENTION CONTAINMENT &
COUNTERMEASURE 609-633-0610
RESPONSIBLE PARTY SITE REMEDIATION 609-633-2168
BUREAU OF HAZARDOUS WASTE ENFORCEMENT 609-984-5855

DEPARTMENT OF HEALTH:
WORKER RIGHT TO KNOW 609-984-2202
PUBLIC EMPLOYEES OSH SERVICE 609-984-1863

DEPARTMENT OF LABOR:

WORKER RIGHT TO KNOW 609-292-7036

DEPARTMENT OF PERSONNEL—HUMAN RESOURCE
DEVELOPMENT INSTITUTE (HRDI) 609-292-7115

FEDERAL AGENCIES

FEDERAL BUREAU OF INVESTIGATION 973-792-3000

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION

OSHA202-219-7162

OSHA REGION II OFFICE 212-337-2378

NEW JERSEY AREA OSHA OFFICES:

HASBROUCK HEIGHTS 201-288-1700

DOVER 201-263-1003

AVENEL 908-750-3270

CAMDEN 609-757-5181

NIOSH HOTLINE 800-356-4674

NIOSH HEADQUARTERS 404-639-3771

NIOSH REGION II OFFICE 212-264-4600

ENVIRONMENTAL PROTECTION AGENCY

EPA HOTLINE 202-382-3000

EPA SUPERFUND HOTLINE 800-424-9346

REGION II SARA TITLE III ASSISTANCE 908-906-6900

DEPARTMENT OF TRANSPORTATION

U.S. COAST GUARD THIRD DISTRICT 212-668-7152

ATLANTIC STRIKE TEAM .....

DAYTIME 609-724-0008

NIGHTTIME (ANSWERING MACHINE) 609-562-6730

DOT HOTLINE (CFR TITLE 49) 202-366-4488

DOT/FEMA HAZ MAT TRANSPORTATION HOTLINE 800-752-6367

DOT/REGIONAL EMERGENCY TRANSPORTATION 617-223-8480

OTHER FEDERAL AGENCIES

DEPARTMENT OF ENERGY 800-428-2525

TOXIC SUBSTANCES CONTROL ACT HOTLINE 202-554-1404

U.S. ARMY CORPS OF ENGINEERS 202-272-0001

U.S. AGENCY FOR TOXIC SUBSTANCES AND
DISEASE REGISTRY HOTLINE 404-639-0615

CENTERS FOR DISEASE CONTROL 404-639-3291

OTHER ASSOCIATIONS

AMERICAN CONFERENCE OF GOVERNMENTAL
INDUSTRIAL HYGIENISTS (ACGIH) 513-661-7881
AMERICAN INDUSTRIAL HYGIENE ASSOCIATION 216-873-2442
AMERICAN INSTITUTE OF CHEMICAL ENGINEERS 201-763-2877
AMERICAN SOCIETY OF SAFETY ENGINEERS 312-692-4121
AMERICAN TRUCKING ASSOCIATION 800-ATA-LINE
CHEMICAL INDUSTRY COUNCIL OF NJ 609-392-4214
CHEMICAL MANUFACTURERS ASSOCIATION (CMA) 202-887-1100
CMA CHEMICAL REFERRAL CENTER
(NON-EMERGENCY CHEMICAL INFORMATION) 800-CMA-8200
NATIONAL SAFETY COUNCIL (NSC) 312-527-4800
NATIONAL FIRE PROTECTION ASSOCIATION 617-770-3000
TEXAS TECH UNIVERSITY PESTICIDE HOTLINE 800-858-7378

GLOSSARY

-A-

29 CFR 1910.120—Hazardous Waste Operations and Emergency Response (OSHA)

A-310—(Public Law 1984, Ch. 210) Inter-Agency Notification

ABSORBANT MATERIAL—Loose or bagged material like commercial bagged clay, kitty litter, Zorbal, or "pigs" used to soak up liquid hazardous materials.

ACTIVE IMMUNIZATION—The administration of a vaccine to stimulate the host immune system to develop immunity (protection) against a specific pathogen or toxin.

ACGIH—**A**merican **C**onference of **G**overnmental **I**ndustrial **H**ygienists. Recommends upper limits (TLVs) for exposure to workplace chemicals.

AIRBORNE PRECAUTIONS—Standard Precautions plus: Placing the patient in a private room that has negative air pressure, at least six air changes/hour, and appropriate filtration of air before it is discharged from the room. Use of respiratory protection when entering the room. Limiting movement and transport of the patient. Using a mask on the patient if he needs to be moved.

AIR REACTIVE MATERIALS—Materials that will react with atmospheric moisture and rapidly decompose.

ANSI—**A**merican **N**ational **S**tandards **I**nstitute

APR—**A**ir **P**urifying **R**espirator

ASPHYXIANT—A substance that can cause unconsciousness or death by lowering the concen tration of oxygen in the air by out competing oxygen metabolically in the body.

ASYMPTOMATIC—Exposed persons who are not exhibiting signs/symptoms of exposure.

-B-

BACTERIAL AGENT—A live pathogenic organism that can cause disease, illness, or death.

BIOLOGICAL CONTAMINATION—The presence of an infectious agent on a body surface or on an environmental surface.

BIOLOGICAL WARFARE AGENT—A biological warfare agent is a pathogen (microorganism capable of causing disease) or toxin derived from a living organism that is deliberately used to produce disease or death in humans, animals, or plants.

BLEVE—**B**oiling, **L**iquid **E**xpanding **V**apor **E**xplosion

B-NICE—Pertaining to biological, nuclear, incendiary, chemical, or explosives.

BUNG—1) The cap or plug used to seal the small opening in the top of a drum or barrel. 2) The small opening in the top of a drum or barrel.

-C-

CARCINOGEN—A substance that causes cancer.

CAS—**C**hemical **A**bstract **S**ervice

CASUALTY COLLECTION POINT (CCP)—Predefined location at which patients are collected, triaged, and provided with initial medical care.

CEHA—**C**ounty **E**nvironmental **H**ealth **A**ct

CFR—**C**ode of **F**ederal **R**egulations

CGI—**C**ombustible **G**as **I**ndicator

CHEMOPROPHYLAXIS—The administration of an antibiotic to prevent an infection, or to prevent an incubating infection from progressing to disease, or to eliminate a carrier state to prevent transmission and disease in others.

CHEMTREC—**Chem**ical **Tr**ansportation **E**mergency **C**enter

COCARCINOGEN— (or promoter)—Not a carcinogen by itself, but promotes the effects of a carcinogen.

COLD (SUPPORT) ZONE—Clean area outside the inner perimeter where command and support functions take place. Special protective clothing is not required in this area.

COMBUSTIBLE SUBSTANCE—A solid, liquid, or gas that will burn.

COMMON NAME—Each of the agents has a complex chemical name based on its composition and formula. They also have a common name that you need to recognize.

CONFINED SPACE—A space which, by design, has limited openings for entry and exit, unfavorable natural ventilation which could contain or produce dangerous air contaminants, could contain a hazardous atmosphere and which is not intended for continuous employee occupancy. A confined space includes (but is not limited to) a tank, vessel, pit, ventilation duct work, vat, boiler, sewer, or underground utility vault. (NJAC 12:100-9.2).

CONTACT PRECAUTIONS—Standard Precautions plus: Placing the patient in a private room or with someone with the same infection, if possible. Using gloves when entering the room. Changing gloves after contact with infective material. Using gown when entering the room if contact with patient is anticipated or if the patient has diarrhea, or colostomy, or wound drainage not covered by a dressing. Limiting the movement or transport of the patient from the room. Ensuring that patient care items, bedside equipment, and frequently touched surfaces receive daily cleaning. Dedicating use of noncritical patient-care equipment to a single patient, or cohort of patients with the same pathogen. If not feasible, adequate disinfection between patients is necessary.

CRYOGENIC—Pertaining to materials at extreme low temperatures (below –90 degrees C or –130 degrees F).

-D-

DECOMPOSITION—The basic breakdown of a substance into different substances. Energy will be released by this reaction; in the case of highly reactive materials, the release may be sudden i.e. explosive.

DECONTAMINATION—The process of removing hazardous substances to prevent adverse health, safety, or environmental effects. Takes place at three levels based on exposure.

DEGRADATION—(applied to protective clothing) Chemical decomposition brought about by exposure to heat, sunlight, solvents, or oxidation.

DEP—**D**epartment of **E**nvironmental **P**rotection

DMAT—**D**isaster **M**edical **A**ssistance **T**eam

DMORT—**D**isaster **Mo**rtuary **R**esponse **T**eam

DOE—**D**epartment **O**f **E**nergy

DOH—**D**epartment **O**f **H**ealth

DOL—**D**epartment **O**f **L**abor

DOT—**D**epartment **O**f **T**ransportation

DOWNWIND—The area directly in the path of the wind from the incident site.

DROPLET PRECAUTIONS—Standard Precautions plus: Placing the patient in a private room or with someone with the same infection. If not feasible, maintaining at least 3 feet between patients. Using a mask when working within 3 feet of the patient. Limiting movement and transport of the patient. Using a mask on the patient if he needs to be moved.

-E-

EFFLUENT—Waste material (such as smoke, liquid industrial refuse, or sewage) discharge into the environment. It generally refers to water pollution.

EGRESS—Designated exit area.

EIS—**E**mergency **I**nformation **S**ystem

EMS—**E**mergency **M**edical **S**ervice

ENDEMIC—A disease that is present in a human population, or in an animal population that is transmittable to humans, but has a very low morbidity rate.

ENZOOTIC—A disease that is present in an animal population at all times, but has a low morbidity rate.

EOC—**E**mergency **O**perations **C**enter

EOD—**E**xplosive **O**rdinance **D**isposal

EPA—United States **E**nvironmental **P**rotection **A**gency

EPIDEMIC—A disease that is only present for a limited time in a human population or animal population that is transmittable to humans, and has a very high morbidity rate.

EPIZOOTIC—A disease that is only present in an animal population for limited periods, but has a high morbidity rate.

ERG—USDOT **E**mergency **R**esponse **G**uidebook

ERP—**E**mergency **R**esponse **P**lan

ETIOLOGIC—Cause of the disease/illness.

EXPLOSIVE LIMITS—The range of concentration of a gas or vapor (measured in percent by volume in air) that can explode upon ignition in a confined space. The highest and lowest concentration are called, respectively, the Upper Explosive Limit **(UEL)** and the Lower Explosive Limit **(LEL)**. At concentrations lower than the LEL, there is not enough product in the air to explode; the mixture is "too lean." At concentrations above the UEL, there is not enough oxygen to sustain an explosion; the mixture is "too rich."

EXPLOSIVE RANGE—The number (as a percentage) that results from subtracting the LEL of a substance from its UEL.

-F-

FEMA—**F**ederal **E**mergency **M**anagement **A**gency

FLAMMABLE SUBSTANCE—A solid, liquid, vapor, or gas that will ignite easily and burn rapidly.

FLASH POINT (FP)—The lowest temperature at which the vapor given off by a liquid within a test vessel forms an ignitable mixture with air. This is only a flash, not a sustained fire.

FR—**F**ederal **R**egister

FREEZING POINT—The freezing point or melting point of a substance is the temperature at which its crystal are at equilibrium with its liquid state. The terms melting point and freezing point are used interchangeably, depending on whether that temperature is approached by heating or cooling the substance.

FUMES—The particulate, smoke-like emanation from the surface of heated metals. Also, the vapor from concentrated acids, evaporating solvents, or as a result of combustion or other decomposition reaction.

-G-

GROSS DECONTAMINATION—Initial decontamination to remove large amounts of decontaminants.

-H-

HAZARDOUS MATERIAL—Any substance that, when released from its container, is a potential or actual threat to the safety of life or property when it touches or impinges upon them.

HAZARDOUS WASTE—Any substance that may pose an unreasonable risk to health, safety, or property when transported in commerce for the purpose of treatment, storage, or disposal as waste.

HAZMAT—**Haz**ardous **Mat**erials

HMT—**H**az**m**at **T**echnician

HMRT—**H**azardous **M**aterials **R**esponse **T**eam

HOT (EXCLUSION) ZONE—Area immediately around the incident where serious threat of harm exists. It should extend far enough to prevent adverse effects from B-NICE agents to personnel outside the zone. Entry into the hot zone requires appropriately trained personnel and use of proper personal protective equipment.

HSFS—**H**azardous **S**ubstance **F**act **S**heet (NJDOH publication)

HVAC—**H**eating, **V**entilating, and **A**ir **C**onditioning

-I-

IC—**I**ncident **C**ommander

ICS—**I**ncident **C**ommand **S**ystem

IDLH—**I**mmediately **D**angerous to **L**ife and **H**ealth

IGNITION TEMPERATURE (Ign. Temp.)—The minimum temperature required to initiate sustained self-combustion of a material or compound.

INNER PERIMETER—Secured inner area of operations.

INOCULUM—The amount of microorganisms introduced into a host.

-L-

LEL—**L**ower **E**xplosive **L**imit

LEPC—**L**ocal **E**mergency **P**lanning **C**ommittee

LINCS—**L**ocal **I**nformation **N**etwork **C**ommunication **S**ystem

-M-

MASS DECONTAMINATION—Decontamination process used on large number of contaminated victims.

MILITARY DESIGNATION/SYMBOL—Each of the agents have been given a symbol. This is not a chemical symbol or formula, but rather a shorthand way of designating the agent. You need to learn these symbols.

MISCIBILITY—The ability of a liquid or gas to dissolve completely and evenly in another liquid or gas at any concentration.

MMRS—**M**etropolitan **M**edical **R**esponse **S**ystem

MSDS—**M**aterial **S**afety **D**ata **S**heet

MUTAGEN—A substance that causes mutations. A mutation is a change in the genetic material in a body cell. Mutations can lead to birth defects, miscarriages, or cancer.

-N-

N.O.S.—**N**ot **O**therwise **S**pecified

NFPA—**N**ational **F**ire **P**rotection **A**ssociation

NIOSH—**N**ational **I**nstitute for **O**ccupational **S**afety and **H**ealth

NJAC—**N**ew **J**ersey **A**dministrative **C**ode

NJRTK—**N**ew **J**ersey **R**ight **T**o **K**now law (also called Worker and Community Right To Know)

NJSA—**N**ew **J**ersey **S**tatutes **A**nnotated

NMRT—**N**ational **M**edical **R**esponse **T**eam

-O-

OEM—**O**ffice of **E**mergency **M**anagement

OSHA—**O**ccupational **S**afety and **H**ealth **A**dministration

OSIC—**O**n **S**cene **I**ncident **C**ommander

OUTER PERIMETER—Outermost area from hazard that is secure.

OVERPACK—An enclosure used by a consignor to provide protection or convenience in handling a package or to consolidate two or more packages. It does not include a freight container.

-P-

PASSIVE IMMUNIZATION—The administration of pre-formed antibodies to confer immunity to a specific pathogen or toxin.

PATIENT STAGING AREA (PSA)—Area where patients may receive continued medical treatment.

PEL—**P**ermissible **E**xposure **L**imit

PENETRATION—1) Refers to chemicals physically passing through protective clothing by way of a tear, cut, or improperly sealed closure. 2) Introducing contaminants into the body by way of exposed cuts or injection by sharp materials (broken glass, metal shards, etc.).

PEOSHA—**P**ublic **E**mployee **O**ccupational **S**afety and **H**ealth **A**ct

PERMEATION—Refers to chemicals passing through protective clothing by absorption. All protective clothing is permeable to some extent.

PERSISTENT AGENT—An agent that upon release retains its casualty-producing effects for an extended period of time, usually anywhere from 30 minutes to several days. A persistent agent usually has a low evaporation rate and its vapor is heavier than air. Therefore, its vapor cloud tends to hug the ground. It is considered to be a long-term hazard. Although inhalation hazards are still a concern, take extreme caution to avoid skin contact as well.

PLUME—A vapor cloud formation which has shape and buoyancy.

POC—**P**oint **O**f **C**ontact

POINT SOURCE—Letter, package, or dispersal area of agent.

POISON—Any substance that is harmful to living tissue when applied in relatively small doses. (See toxin).

PPE—**P**ersonal **P**rotective **E**quipment

PROTECT IN PLACE—Method of protecting public by limiting exposure.

PULMONARY EDEMA—The condition of having fluid in the lungs. The condition may be fatal.

-R-

RALLY POINT—A predetermined location to which all persons evacuate in an emergency. In industry, facilities are evacuated and a rally point is usually predetermined. It is at this rally point that resources can regroup and a revised plan can be established.

RATE OF ACTION/ONSET TIME—The rate of action or onset time is the period of time that elapses before a victim begins to show or feel the symptoms of the particular agent. With some agents, this time will be just a few seconds; in other cases it could be minutes to hours. Knowing onset time is important because it tells you how much time you have to react.

REACTIVE SUBSTANCE—A solid, liquid, or gas that can cause an explosion under certain conditions or on contact with other specific substances.

ROUTE OF ENTRY—The route of entry is how the agent gets into your body. Most of the agents will enter through the respiratory tract, that is, through inhalation. Some of the agents can also attack through skin and eye.

RTK—**R**ight **T**o **K**now; May refer to State or Federal law

-S-

SAFE REFUGE AREA (SRA)—An area within the contamination reduction zone for assembling individuals who are witnesses to the incident. This assemblage will provide for the separation of contaminated persons from non-contaminated persons.

SAMPLE—Material collected from a source other than an animal or man for laboratory analysis (such as water sample or soil sample).

SCBA—**S**elf-**C**ontained **B**reathing **A**pparatus

SHIPPING PAPER—A shipping order, bill of lading, manifest or other document containing the information required by 172.202, 172.203 and 172.204.

SLUDGEM—Acronym for salivation, lacrimation, urination, defecation, gastric distress, emesis, and miosis.

SOLUBILITY—The ability or tendency of one substance to dissolve evenly in another.

SOLVENT—A substance capable of dissolving another substance (the solute) to form a uniformly dispersed mixture (the solution). Water, referred to as the "universal solvent," is a strongly polar solvent.

SOP—**S**tandard **O**perating **P**rocedure

SPECIMEN—Material collected from a man or animal for laboratory analysis (such as tissue or blood specimen).

SPONTANEOUSLY COMBUSTIBLE—The ignition of a substance from the rapid oxidation of its own constituents.

STANDARD PRECAUTIONS—Handwashing after patient contact. Using gloves when touching blood, body fluids, secretions, excretions, and contaminated items. Using mask, eye protection, and gown during procedures likely to generate splashes or sprays of blood, body fluids, secretions, or excretions. Handling contaminated patient-care equipment and linens in a manner that prevents the transfer of microorganisms to people or equipment. Practicing care when handling sharps and using a mouthpiece or other ventilation device as an alternative to mouth-to-mouth resuscitation, when practical. Placing the patient in a private room if they contaminate the environment, when feasible.

STEL—**S**hort **T**erm **E**xposure **L**imit

STLC—**S**hort **T**erm **L**ethal **C**oncentration

SYMPTOMATIC—Exhibiting signs/symptoms of exposure.

SYMPTOMS—Each of the agents will cause the victim to exhibit symptoms. In many cases these symptoms can be recognized and provide an indicator of the type of agent.

-T-

TERATOGEN—A substance that causes birth defects by damaging a fetus.

TIME, DISTANCE AND SHIELDING (TDS)—Three types of protective measures commonly associated with hazardous materials training.

TLV—**T**hreshold **L**imit **V**alue—recommended air concentration in which most persons can work for an 8-hour work day without ill effects. Set by the ACGIH.

TLV-C—**T**hreshold **L**imit **V**alue—**C**eiling—Exposure level to employees that shall not be exceeded during any part of the work day.

TLV-STEL—See STEL

TOXICITY—Toxicity is the term used to indicate how much of a substance (in this case, one of the agents), it takes to cause a specified effect such as incapacitation or death. The amount of agent it takes to cause an effect is also referred to as a dose.
Respiratory lethality or toxicity can be expressed in parts per million (ppm). PPM is an expression of concentration **(C)** or how many parts of a given substance are mixed in a million parts of air. If an individual is exposed to this concentration for a period of time **(t)**, usually expressed in one (1) minute, the he or she will receive a dosage. The exact ppm concentration required to cause lethality is a variable, depending on breathing rate, overall health, etc. Taking into consideration these variables, the term LCt_{50} is the expression used to indicate a given ppm concentration expected to be **L**ethal to 50 percent of those exposed for 1 minute. ICT_{50} would express the same, except **I** is the **I**ncapacitating dosage.
Skin lethality is expressed as LD_{50} and will normally be expressed in grams or milligrams per individual.

TOXIN—Anything harmful, destructive, or poisonous to the body (adj. Toxic). (See Poison.)

TOXIN AGENTS—Poisonous by-products of living organisms used to cause disease, illness or death in susceptible individuals.

TWA—**T**ime **W**eighted **A**verage—The calculated average concentration for an 8-hour work day, 10-hour work day or 40-hour work week to which workers may be exposed over their working career without ill effects. Set by the ACGIH.

TRACEM—The acronym used to identify the six types of harm one may encounter at a terrorist incident: thermal, radioactive, asphyxiation, chemical, etiological, and mechanical. Note: Some sources use the acronym TEAM CPR, which stands for thermal, etiological, asphyxiation, mechanical, chemical, psychological, and radioactive.

-U-

UNIFIED COMMAND—In ICS, Unified Command is a unified team effort which allows all agencies with responsibility for the incident to establish a common set of incident objectives and strategies. This is accomplished without losing or abdicating agency authority, responsibility or accountability.

UNSTABLE MATERIALS—Those which, in the pure state, will vigorously polymerize, decompose, condense, or become self-reactive, and undergo other violent chemical changes.

UPWIND—The direction from which the wind is coming.

-V-

VAPOR—An air dispersion of molecules of a substance that is liquid or solid in its normal state (room temperature).

VEE—**V**enezuelan **E**quine **E**ncephalitis

VIRAL AGENTS—A group of viruses that have been selected as BW agents because of their ability to produce disease, illness, and death in susceptible individuals.

VOC—**V**olatile **O**rganic **O**ompound

VOLATILITY—The tendency of a solid or liquid to pass into the gaseous state at a given temperature.

VOLATILITY/PERSISTENCY—Volatility is important because it gives you an indication of how rapidly an agent will evaporate. The more volatile an agent is, the more rapidly it will evaporate. Evaporation will cause the agent to become a true gas or vapor and reduce the liquid hazard. Temperature, wind speed and humidity at the incident site influence how rapidly an agent will evaporate.

This evaporation process is also referred to as persistency, or the amount of time an agent will remain a threat in the incident site. A non-persistent agent will not remain at the incident site as long as a persistent agent. Obviously, if an agent is released inside an enclosed space, weather will not play a role and the persistency will normally increase.

Most of the agents we will discuss will be disseminated as gases or vapors and are heavier than air.

A more definitive definition of Vapor Density pressure and Volatility is in the reference section.

-W-

WARM ZONE—In HazMat incidents, this zone is the contamination reduction zone where initial decontamination activities occur. This zone requires the use of proper personal protective equipment once contaminated people or equipment enter the zone.

WATER REACTIVE MATERIALS—Materials which will violently decompose and/or burn vigorously when they come in contact with water.

WATER SOLUBILITY—The degree to which a material, or its vapors, are soluble in water. Materials that are completely soluble in water are said to be **miscible**.

WEAPON OF MASS DESTRUCTION (WMD)—1) Any explosive, incendiary, poison gas, bomb, grenade, or rocket having a propellant charge of more than four ounces, missile having an explosive or incendiary charge of more than one-quarter ounce, or mine or device similar to the above. 2) Poison gas. 3) Any weapon involving a disease organism. 4) Any weapon designed to release radiation at a level dangerous to human life.

Telephone Information and Technical Support Resource Worksheet

Resource	Contact (fill in for future reference)	Services Provided (fill in for future reference)
Administrator (Hospital)		
Local Police Department		
Local Fire Department		
Local Office of Emergency Management		
Local/County HazMat Team		
NJ Poison Control Center **1-800-962-1253**		
N.J. Department of Health and Senior Services **1-800-792-9770 Hot Line**		
DEP **1-877-WARNDEP**		
Local Health Department		
County Office of Emergency Management		
Center for Disease Control **404-639-2191**		
New Jersey State Police **1-609-882-2000 x6311**		
CHEMTREC **1-800-424-9300**		