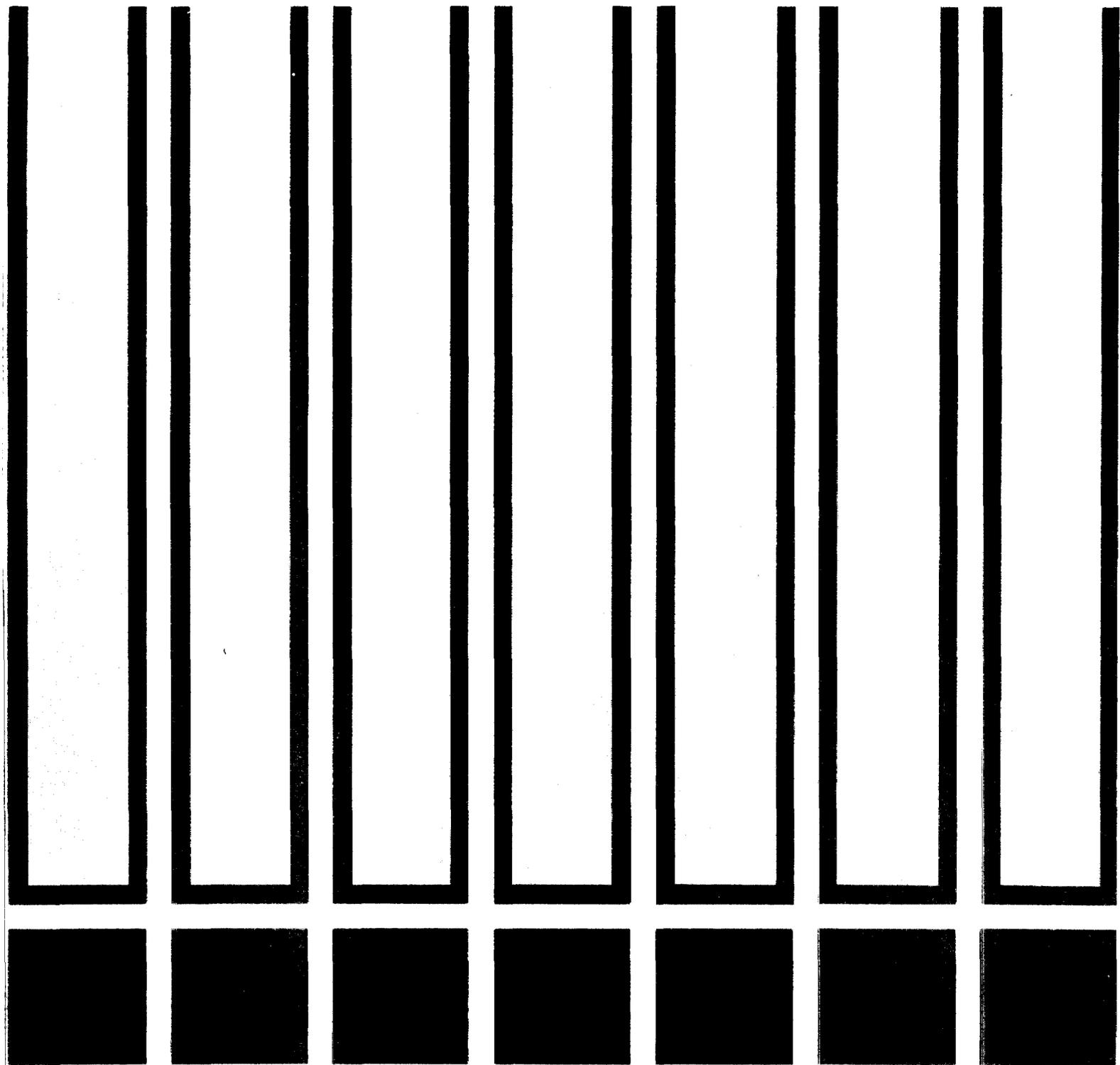


criteria for a recommended standard . . . .  
occupational exposure to

# CHLORINE



**criteria for a recommended standard....**

**OCCUPATIONAL EXPOSURE  
TO  
CHLORINE**



**U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE**

**Public Health Service**

**Center for Disease Control**

**National Institute for Occupational Safety and Health**

**MAY 1976**

**HEW Publication No. (NIOSH) 76-170**

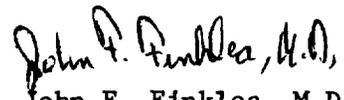
## PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. The National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices, to provide relevant data from which valid criteria for effective standards can be derived. Recommended standards for occupational exposure, which are the result of this work, are based on the health effects of exposure. The Secretary of Labor will weigh these recommendations along with other considerations such as feasibility and means of implementation in developing regulatory standards.

It is intended to present successive reports as research and epidemiologic studies are completed and as sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on chlorine by members of my staff and the valuable constructive comments by the Review Consultants on Chlorine and the ad hoc committees of the American Occupational Medical Association and the American Academy of Industrial Hygiene. The NIOSH recommendations for standards are not necessarily a consensus of the consultants and professional societies that

reviewed this criteria document on chlorine. Lists of the NIOSH Review Committee members and of the Review Consultants appear on the following pages.

A handwritten signature in black ink that reads "John F. Finklea, M.D.". The signature is written in a cursive style with a large initial 'J'.

John F. Finklea, M.D.  
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The Division of Criteria Documentation and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for development of the criteria and recommended standard for chlorine. The Division review staff for this document consisted of J. Henry Wills, Ph.D., Frank L. Mitchell, D.O., and Herbert E. Christensen, D.Sc., with Robert H. Duguid, M.D. (consultant) and Robert B. O'Connor, M.D. (consultant). The University of Washington School of Public Health and Community Medicine developed the basic information for consideration by NIOSH staff and consultants under contract No. HSM-99-73-36. Patricia G. Heitman had NIOSH program responsibility and served as criteria manager.

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CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN  
OCCUPATIONAL EXPOSURE STANDARD FOR CHLORINE

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## I. RECOMMENDATIONS FOR A CHLORINE STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to chlorine be controlled by requiring compliance with the following sections. The standard is designed to protect the health and safety of workers for up to a 10-hour workday, 40-hour workweek, over a working lifetime. Compliance with all sections of the standard should prevent adverse effects of exposure to gaseous or liquid chlorine in the workplace. The proposed environmental limit is measurable by techniques that are valid, reproducible, and available to industry and government agencies. Sufficient technology exists to permit compliance with the recommended standard. The standard will be subject to review and revision as necessary.

"Chlorine" is defined as liquid or gaseous molecular chlorine. Occupational exposure to chlorine is defined as exposure to airborne concentrations of chlorine at or above one-half of the recommended workplace environmental limit. Adherence only to sections 3, 4(a) (1,3, and 4), 4(b) (6, 7, 9, 10), 5, 6, and 7 is required when workplace environmental concentrations of chlorine are less than one-half of the recommended workplace environmental limit.

### Section 1 - Environmental (Workplace Air)

#### (a) Concentration

Exposure to chlorine shall be controlled so that no worker is exposed to chlorine at an airborne concentration greater than 0.5 parts of chlorine

per million parts of air (0.5 ppm) for any 15-minute sampling period. This shall be designated as a ceiling concentration.

(b) Sampling and analysis

Procedures for sampling, calibration of equipment, and analysis of chlorine samples shall be as provided in Appendices I and II, or by any method shown to be equivalent in precision, accuracy, and sensitivity to the methods specified.

Section 2 - Medical

Medical surveillance shall be made available as specified below for all workers subject to occupational exposure to chlorine.

(a) Preplacement examinations shall include as a minimum:

(1) Medical and occupational histories in sufficient detail to document the occurrence of cardiac disease as well as bronchitis, tuberculosis, pulmonary abscess, and other chronic respiratory diseases.

(2) A medical examination including but not limited to, simple tests of olfactory deficiency.

(3) A chest X-ray, 14 x 17 (posterior-anterior).

(4) An evaluation of the worker's physical capability to use respirators as defined in 29 CFR 1910.134.

(b) Pulmonary function studies and other objective indicators of normalcy or lack thereof may be performed at the discretion of the examining physician.

(c) A worker with evidence of respiratory impairment shall be evaluated by a physician and, if appropriate, counseled on the possibility

of an increased health risk resulting from exposure to chlorine.

(d) Periodic Examinations

(1) Periodic examinations shall be made available on an annual basis or at an interval to be determined by the responsible physician.

(2) If it is suspected that a worker has been exposed to high concentrations of chlorine and if he exhibits signs or symptoms of respiratory tract irritation, he shall be referred to a physician.

(e) Medical Records

All pertinent medical records shall be maintained at least 5 years after the individual's employment is terminated. These records shall be available to the medical representatives of the Secretary of Labor, of the Secretary of Health, Education, and Welfare, of the employer, and of the employee or former employee at his request.

Section 3 - Labeling (Posting)

(a) All shipping containers of chlorine shall bear the following label in addition to, or in combination with, labels required by other statutes, regulations, or ordinances:

CHLORINE  
DANGER! HAZARDOUS GAS OR LIQUID UNDER PRESSURE  
EXTREMELY IRRITATING  
MAY BE FATAL IF INHALED  
CAUSES BURNS  
SEVERE EYE HAZARD

Do not breathe gas; use only with adequate ventilation. In case of inhalation, remove to uncontaminated atmosphere, get medical attention immediately. If breathing has stopped, start artificial respiration.

Do not get in eyes, on skin, or on clothing. In case of contact, immediately flush skin or eyes with plenty of water for at least 15 minutes, and get medical attention immediately.

OPEN CONTAINERS WITH CARE AFTER SECURING THE CONNECTION TO THE DISTRIBUTION LINE INTO WHICH THE GAS IS TO PASS.  
HAVE SUPPLIED AIR RESPIRATORS OR SELF-CONTAINED BREATHING APPARATUS AVAILABLE FOR EMERGENCY. DO NOT HEAT CONTAINERS. AVOID CONTACT WITH OTHER MATERIALS.  
STORE ONLY IN AUTHORIZED AREAS.

(b) The following warning sign shall be affixed in a readily visible location at or near entrances to areas in which chlorine is present in containers or systems. This sign shall be printed both in English and in the predominant language of non-English-speaking workers. All employees shall be trained and informed of the hazardous areas, with special instruction given to illiterate workers.

CAUTION!  
CHLORINE HAZARD AREA  
UNAUTHORIZED PERSONS KEEP OUT  
CAUSES BURNS, SEVERE EYE HAZARD  
MAY BE FATAL IF INHALED  
PROTECTIVE MASKS FOR CHLORINE LOCATED AT \_\_\_\_\_  
(specific locations to be supplied by employer)

(c) All chlorine piping systems shall be plainly marked for positive identification in accordance with American National Standard

A13.1-1975. Associated vessels and critical shut-off valves shall be conspicuously labeled. Chlorine containers in use shall be plainly marked "in use" to distinguish them from those not in use. No container shall ever be presumed to be empty and therefore nonhazardous.

#### Section 4 - Personal Protective Equipment

##### (a) Protective Clothing

(1) Personnel working with, or exposed to, liquid or gaseous chlorine containers or systems where chlorine contact with the eyes can occur shall have eye protection. Unless eye protection is afforded by a respirator hood or facepiece, chemical goggles and face shields shall be worn.

(2) In addition to wearing the respiratory protective devices specified in Table I-1, personnel performing nonroutine operations where escape of liquid chlorine occurs or emergency operations involving escaping liquid chlorine should wear 1-piece suits which are impervious to chlorine and sealed at the ankles, wrists, and around the face. The suits shall be ventilated with supplied air, or stay time in the work area shall be limited with due consideration of the heat stress factors involved. Impervious gloves and boots should also be worn. Such protective clothing shall be kept readily available for emergencies.

(3) Impervious gloves shall be worn by persons connecting or disconnecting cylinders of chlorine.

(4) The employer shall supply and maintain all protective

clothing in a clean, sanitary, and usable condition.

(b) Respiratory Protection

Engineering controls shall be used wherever feasible to maintain airborne chlorine concentrations at or below the environmental limit recommended in Section 1 of this document. Compliance with the permissible exposure limit by the use of respirators is only allowed when airborne chlorine concentrations are in excess of the workplace environmental limit while required engineering controls are being installed or tested, when nonroutine maintenance or repair is being accomplished, or during emergencies. When a respirator is thus permitted, it shall be selected and used in accordance with the following requirements:

(1) For the purpose of determining the type of respirator to be used, the employer shall measure, when possible, the airborne concentration of chlorine in the workplace initially and thereafter whenever process, worksite, climate, or control changes occur which are likely to increase the airborne concentration of chlorine.

(2) The employer shall ensure that no worker is overexposed to chlorine because of improper respirator selection, fit, use, or maintenance.

(3) A respiratory protection program meeting the requirements of 29 CFR 1910.134 which incorporates the American National Standard Practices for Respiratory Protection Z88.2-1969 shall be established and enforced by the employer.

(4) The employer shall provide respirators in accordance with Table I-1 and shall ensure that the employee uses the respirator provided.

(5) Respiratory protective devices described in Table I-1

TABLE I-1  
RESPIRATOR SELECTION GUIDE

---

Chlorine Concentration	Respirator Type
Less than or equal to 25 ppm	(1) Chemical cartridge respirator with full facepiece and cartridge(s) and filter(s) providing protection against chlorine (2) Full-face gas mask, chest- or back-mounted type, with industrial-size chlorine canister (3) Any supplied-air respirator with a full facepiece, hood, or helmet with shroud (4) Any self-contained breathing apparatus with a full facepiece
Greater than 25 ppm and Emergencies	(1) Self-contained breathing apparatus with full facepiece, pressure-demand or other positive pressure type (2) Combination respirator which includes a Type C supplied-air respirator with a full facepiece operated in pressure-demand or other positive pressure or continuous-flow mode, and an auxiliary self-contained breathing apparatus, pressure-demand or other positive pressure type
Evacuation or Escape	(1) Self-contained breathing apparatus with full facepiece (2) Full-face gas mask with industrial-size chlorine canister

---

shall be those approved under the provisions of 30 CFR 11.

(6) The employer shall ensure that employees are instructed on the use of respirators assigned to them and on how to test for leakage.

(7) Each indoor area required to be posted in accordance with Section 3(b) shall have emergency respiratory protective devices readily available in nearby locations which do not require entry into a contaminated atmosphere for access. Certain outdoor locations may be exempted from this requirement depending upon such factors as chlorine capacity, accessibility to facility, nearness to other occupied locations, and ease of evacuation. A decision regarding an exemption shall be made by an OSHA compliance officer. Respiratory protective devices provided shall consist of at least two self-contained breathing apparatus as described in Table I-1.

(8) Respirators specified for use in atmospheres of higher concentrations of chlorine may be used in atmospheres of lower concentrations.

(9) The employer shall ensure that respirators are cleaned, maintained, and stored in accordance with 29 CFR 1910.134.

(10) Canisters shall be discarded after use or whenever an odor or taste is detected, and replaced with fresh canisters. Unused canisters shall be discarded and replaced when the seal is broken or when the shelf life recommended by the manufacturer ends.

#### Section 5 - Informing Employees of Hazards from Chlorine

At the beginning of employment, workers whose jobs may involve

exposure to chlorine at concentrations greater than one-half of the environmental limit, or who will work in areas required to be posted in accordance with Section 3(b), shall be informed of the hazards, signs, symptoms, and effects of overexposure, emergency procedures, and precautions to take to ensure safe use of chlorine and to minimize exposure to chlorine. Information pertaining to first-aid procedures shall be included. The information shall be posted in the workplace and kept on file, readily accessible to workers at all places of employment where chlorine is involved in unit processes and operations, or is released as a product, byproduct, or contaminant.

A continuing educational program, conducted by a person or persons qualified by reason of experience or special training, shall be instituted to ensure that all workers have current knowledge of job hazards, first-aid procedures, maintenance procedures, and cleanup methods, and that they know how to use respiratory protective equipment and protective clothing. Retraining shall be repeated at least annually.

In addition, members of emergency teams and employees who work adjacent to chlorine systems or containers where a potential for emergencies due to chlorine exists shall be subjected to periodic drills simulating emergency situations appropriate to the work situation. These shall be held at intervals not exceeding 6 months. Drills should cover, but should not be limited to, the following:

Evacuation procedures.

Handling of spills and leaks, including decontamination  
and use of emergency leak-repair kits.

Location and use of emergency firefighting equipment, and handling of chlorine systems and containers in case of fire.

First-aid and rescue procedures, including procedures for obtaining emergency medical care.

Location, use, and care of protective clothing and respiratory protective equipment.

Location and use of shut-off valves.

Location, reason for, and use of safety showers, eyewash fountains, and other sources of water for emergency use.

Operating procedures.

Entry procedures for confined spaces.

Emergency phone numbers.

Deficiencies noted during the drill shall form the basis for a continuing educational program to ensure that all workers have current knowledge. Records of drills and training conducted shall be made available for inspection by authorized personnel as required.

Information as required shall be recorded on the "Material Safety Data Sheet" shown in Appendix IV or on a similar form approved by the Occupational Safety and Health Administration, US Department of Labor.

#### Section 6 - Work Practices

##### (a) Emergency Procedures

For all work areas in which there is a potential for emergencies,

procedures specified below, as well as any other procedures appropriate for a specific operation or process, shall be formulated in advance and employees shall be instructed and drilled in their implementation.

(1) Procedures shall include assignment of individual or team responsibilities and prearranged plans for:

(A) Immediate evacuation of workers with signs or symptoms of adverse effects resulting from exposure to chlorine.

(B) Transportation of injured persons to medical facilities.

(C) Any necessary calls to alert medical facilities of the impending arrival of injured persons or to people who have been identified as being able to provide assistance.

(D) Designation of medical receiving facilities and names of physicians trained in chlorine emergency procedures.

(2) Nonessential employees shall be evacuated from exposure areas during emergencies. Perimeters of areas of hazardous exposure shall be delineated, posted, and secured.

(3) Personnel who cannot be evacuated shall keep upwind of spills or leaks, if possible. Personnel who have appropriate training in the procedures and who are adequately protected against the attendant hazards shall take appropriate control action, eg, leak isolation and repair, cleanup of spills, etc.

(4) In case of fire, chlorine containers shall be removed to a safe place or cooled with water if leaks do not exist. Fusible plugs in chlorine containers melt at 70-74 C (158-165 F). Every effort shall be

made to prevent containers from reaching this temperature.

(5) Water may not be used on chlorine leaks because accelerated corrosion, resulting from the formation of hydrochloric acid when water is present, may quickly make the leak worse. Water spray or fog may, however, be used to help suppress the size of a chlorine cloud near the leak.

(6) Containers leaking liquid chlorine should be oriented so that gaseous chlorine is discharged through the leak until it is controlled.

(7) If possible, in emergency situations chlorine should be discharged to the industrial process through the regular chlorination equipment or by running a line directly to the consuming equipment or other control vessel or equipment.

(8) If the process cannot handle chlorine under emergency conditions, a standby alkali absorption system shall be made available for emergency use.

(9) Chlorine in contact with skin or eyes shall be removed by immediate washing with copious quantities of water, and immediate medical attention shall be obtained. Remove contaminated clothing immediately. If chlorine is inhaled, remove victim to uncontaminated atmosphere, give artificial respiration if required, and get immediate medical attention in accordance with Section 6(a)(1).

(b) General Work Practices

(1) Control of Airborne Chlorine

Engineering controls shall be used to maintain chlorine

concentrations within the limits of the recommended environmental limit. The use of completely enclosed processes is the preferred method of control for chlorine. Local exhaust ventilation may also be effective, either when used alone or in combination with process enclosure. Ventilation systems shall be designed to maintain airborne chlorine concentrations within the limits of the recommended environmental limit to prevent accumulation of chlorine in the workroom, and to remove chlorine from the breathing zones of workmen. Ventilation systems shall be subject to regular preventive maintenance and cleaning to ensure maximum effectiveness. This effectiveness shall be verified by periodic airflow measurements.

(2) Storage

(A) Chlorine shall be stored in adequately ventilated unoccupied rooms or outdoors shielded from the direct rays of the sun, unless the container is properly insulated and designed for unshaded outdoor storage. Indoor storage areas shall be cool and dry.

(B) At least two exits, remote from each other and opening outward of the building, should be provided for all chlorine storage rooms.

(C) Chlorine storage enclosures shall be provided with an inspection window to permit viewing of the interior without entry.

(D) Chlorine storage enclosures shall be completely isolated from work areas. If separated from a work area by a common wall, all holes, ducts, doors, and passthroughs which could allow chlorine to enter other parts of the plant shall be secured and sealed. Central cooling and heating ducts may not extend to chlorine storage areas, but

such areas may be cooled by terminal ducts with one-way flap or other appropriate valves to prevent significant reflux of air from the storage area into the duct system. If an enclosed storage area is cooled in this way, the pressure within the enclosure shall be maintained slightly below the atmospheric pressure by forced exhaust to the outside of the area.

(E) Ventilation switches and emergency respiratory protection shall be located outside storage areas in readily accessible locations which will be free of chlorine in an emergency. Fan switches shall be equipped with indicator lights.

(F) Containers shall be secured so they will not fall, upset, or roll.

(G) Chlorine containers shall be protected from flame, heat, corrosion, and mechanical damage.

(H) Incompatible materials which may react violently with chlorine such as hydrogen, ammonia, acetylene, fuel gases, ether, turpentine, most hydrocarbons, finely divided metals, and organic matter, may not be stored immediately adjacent to chlorine. The degree of separation required will be dictated by quantities stored and the type of storage facility (outdoor vs indoor, concrete walls vs wood, etc).

(I) Storage areas should not have low spots in which chlorine could accumulate in case of a leak, unless such places have been designed and constructed for such a purpose.

(J) Containers of chlorine shall be used on a first-in-first-out (FIFO) basis.

(K) Full and empty shipping containers shall be so marked, and containers in use shall be plainly marked "in use" to distinguish them from those not in use.

(3) Handling

(A) Areas containing chlorine containers and systems shall be checked daily for leaks. All newly made connections shall be checked for leaks immediately after chlorine is admitted. Required repairs and adjustments shall be promptly made. No water shall be applied to the source of leaking chlorine.

(B) Steel and iron in contact with chlorine may not be heated, welded, or flame-cut. Steel and iron will ignite and burn in an atmosphere of chlorine at about 251 C (484 F).

(C) Written operating instructions shall be formulated, posted, and up-dated periodically where chlorine is handled or used.

(D) Every precaution shall be taken to keep chlorine and chlorine equipment free of moisture. Piping, valves, and containers shall be capped or closed when not in use to keep atmospheric moisture out of the system.

(E) Transportation and use of chlorine shall comply with all applicable federal, state, and local regulations.

(F) When moving chlorine containers, valve protection hoods shall be in place. Containers shall be moved only with the proper equipment (eg, lifting bars and hand trucks) and shall be secured to prevent dropping or loss of control while moving. No slings or

magnetic devices shall be used to move chlorine containers.

(G) Containers and valves may not be modified, altered, or repaired except as normally intended by the supplier.

(H) Discharge rates may not be increased by use of hot water, radiant heat, or application of flames or heated objects to the containers. Air circulated around the containers at workroom temperature may be used. Properly designed chlorine vaporizing equipment (as distinct from storage and shipping containers) may be heated.

(I) The amount of chlorine used shall be determined by a positive method, eg, weighing the container.

(J) New gaskets shall be used each time chlorine system connections are made.

(K) Cylinder and ton-container valves may not be opened more than one complete turn. Wrenches longer than 8" shall not be used.

(L) Piping systems for chlorine shall be properly designed and manufactured from approved materials meeting or exceeding the provisions of American National Standard B31.1 1973, and shall be equipped with appropriate expansion chambers or pressure relief valves or rupture discs discharging to a receiver or safe area. All precautions shall be taken to prevent hydrostatic rupture of chlorine systems and containers.

(M) Before chlorine is admitted to a new or repaired system, the system shall be thoroughly cleaned, dried, and pressure-tested, using approved procedures. Pressure testing of cylinders designed for portable use shall be repeated at not longer than 5-year intervals.

(N) Materials for handling moist chlorine shall be selected with great care, considering the enhanced corrosiveness of the chlorine, and the requirements for strength.

(O) A vacuum placed on a chlorine line shall be broken with dry air or nitrogen rather than with chlorine to prevent rendering expansion chambers ineffective.

(P) No liquid chlorine containers shall be directly connected to containers of other liquids unless backflow is prevented by suitable check valves, traps, or vacuum breakers. Suckback may cause a violent reaction or explosion.

(Q) No personnel shall work alone when chlorine is first admitted to a newly designed or installed system, or while repairing leaks. Replenishment of chlorine to a previously properly functioning system is permitted.

(R) Any odor of chlorine from a normally closed system shall be reported without delayed to responsible supervisory personnel.

(S) Containers and systems shall be handled and opened with care. Approved skin, eye, and respiratory protection shall be worn while opening, connecting, or disconnecting chlorine containers and systems. When opening containers or systems, adequate ventilation shall be available to remove inadvertent discharges of chlorine.

(T) Inadvertent entry of chlorine into disconnected containers and systems while work is in progress shall be prevented by blanking off chlorine supply lines. Repairs of leaks may not be

accomplished on chlorine systems while the systems are in service, except when a chlorine repair kit utilized by trained and protected emergency team personnel can be used with reasonable safety.

(4) Work Areas

(A) Where chlorine is handled or used, eyewash fountains and safety showers shall be located immediately outside the chlorine work area. They shall be kept readily accessible and shall be inspected frequently and kept in good working order.

(B) Enclosed chlorine work areas shall be equipped with at least two exits, remote from each other, to allow escape into uncontaminated areas in case of emergency. Doors shall open outward.

(C) No unauthorized personnel shall be permitted to enter areas where chlorine is handled or used.

(D) No nonessential combustible or flammable materials shall be stored or processed in areas where chlorine is handled or used. All elements of chlorine systems shall be protected from fire hazards.

(E) At least two self-contained breathing apparatus shall be located outside of each facility handling or using chlorine. In case of emergency, they shall be readily accessible without entering contaminated atmospheres. Employees shall be trained and drilled in their use.

(F) Critical isolation valves shall be conspicuously marked, and employees shall be familiarized with their use. Access to shutoff valves shall be kept unobstructed.

(G) Work areas and means of egress shall be kept clean and orderly.

(5) Waste Disposal

(A) Disposal of waste chlorine shall conform to all applicable local, state, and federal regulations.

(B) Discharges of chlorine into the atmosphere shall first be rendered neutral or harmless, or shall be prevented by proper absorbing devices.

(C) Discharges of chlorine solutions shall be neutral or mildly alkaline, or adequately diluted.

(D) No discharges shall be allowed which will be harmful to humans, vegetation, animals, materials, or sewerage systems.

(6) Confined Spaces

(A) Entry into confined spaces such as tanks, pits, tank cars, barges, process vessels, tunnels, and sewers shall be controlled by a permit system. Permits shall be signed by an authorized employer representative certifying that preparation of the confined space, precautionary measures, and personal protective equipment are adequate, and that predetermined procedures will be followed.

(B) Confined spaces which have contained chlorine shall be thoroughly cleaned, tested for oxygen deficiency and the presence of chlorine, and inspected prior to entry.

(C) Inadvertent entry of chlorine into a confined space while work is in progress shall be prevented by disconnecting and blanking off chlorine supply lines.

(D) Confined spaces shall be ventilated while work is in progress to keep any chlorine concentration below the environmental limit and to prevent oxygen deficiency.

(E) Personnel entering confined spaces where they may be exposed to chlorine shall be equipped with the necessary personal protective equipment and a lifeline tended by another worker outside the space who shall be trained and equipped to perform rescue.

(7) Enclosed Spaces

Enclosed spaces (rooms, buildings, etc) which are not constantly occupied and which are ordinarily safe to enter, but because of the failure of a system inside could contain hazardous concentrations of chlorine, should have a continuous automatic monitor set to sound an alarm outside the enclosed space if chlorine concentrations exceed the recommended standard. If such areas are not monitored in this way, the enclosed space shall be entered only if the worker is under observation by a coworker and if the worker has in his possession a respirator suitable for escape.

(8) Miscellaneous

No hypochlorite solutions shall be mixed with acidic materials, such as toilet-bowl cleaners or vinegar, because chlorine will be generated with the potential for hazardous exposure. Custodial and maintenance personnel shall be alerted to this potential chlorine exposure.

Chlorinators in use at public swimming pool facilities should be located away from entrance and egress areas.

## Section 7 - Sanitation Practices

(a) Plant sanitation shall meet the requirements of 29 CFR 1910.141.

(b) Appropriate locker rooms shall be available for changing into required protective clothing in accordance with 29 CFR 1910.141(e).

(c) Good personal hygiene shall be enforced. Hands, arms, and face shall be thoroughly washed prior to eating and at the end of the shift. Facilities shall be provided for this purpose in conformance with 29 CFR 1910.141(d).

(d) No food shall be stored, prepared, dispensed (even from vending machines), or eaten in areas where occupational exposure to chlorine may occur. Drinking, smoking, and chewing tobacco or gum shall be prohibited in chlorine exposure areas. The employer shall furnish an uncontaminated area for these purposes in conformance with 29 CFR 1910.141(g). A source of drinking water protected from contamination may be provided in hot environments.

(e) After each use, protective clothing shall be neutralized, washed, dried, and inspected before reissue.

## Section 8 - Monitoring and Recordkeeping Requirements

Workers will not be considered to have occupational exposure to chlorine if environmental concentrations, as determined on the basis of a professional industrial hygiene survey conducted within 6 months of the promulgation of this standard, do not exceed one-half of the recommended ceiling concentration (ie, action level). Surveys shall be repeated at

least once every 3 years and within 30 days after any process change likely to result in increases of airborne concentrations of chlorine. Records of these surveys, including the basis for concluding that airborne concentrations of chlorine are at or below the action level, shall be maintained. If the survey indicates that airborne concentrations of chlorine exceed the action level, then the following requirements apply:

(a) Personal Monitoring

(1) A program of personal monitoring shall be instituted to identify and measure or permit calculation of the exposure of all employees who are occupationally exposed to chlorine. Monitoring of employee exposure to airborne concentrations of chlorine shall be conducted at least every 6 months. If monitoring of an employee's exposure to chlorine reveals that he is exposed at concentrations in excess of the recommended environmental limit, the exposure of that employee shall be measured at least once every 30 days, control measures shall be initiated, and the employee shall be notified of his exposure and the control measures being implemented to correct the situation. Such monitoring shall continue until two consecutive samplings, at least a week apart, indicate that employee exposure no longer exceeds the environmental limit in Section 1(a). Semiannual monitoring may then be resumed.

(2) In all personal monitoring, samples of airborne chlorine that, when analyzed, will provide an accurate representation of the concentration of chlorine in the air breathed by the worker shall be collected. Procedures for sampling, calibration of equipment, and analysis of chlorine in samples shall be as provided in Appendices I and II, or by

any method shown to be equivalent in precision, accuracy, and sensitivity to the methods specified.

(3) For each ceiling determination, a sufficiently large number of samples shall be taken to characterize every employee's peak exposure during each workshift. Variations in work and production schedules shall be considered in deciding when samples are to be collected. The number of representative ceiling determinations for an operation or process shall be based on the variations in location and job functions of employees in relation to that operation or process.

(b) Recordkeeping Procedures

Records shall be maintained and shall include sampling and analytical methods, types of respiratory protective devices used, and ceiling concentrations found. Each employee shall have access to data on his own environmental exposures and records of such data shall be included in his medical records. Pertinent records of required medical examinations, including records of occupational accidents and environmental exposures within the workplace, shall be maintained for 5 years after the worker's employment has ended and shall be available to the designated medical representatives of the Secretary of Labor, of the Secretary of Health, Education and Welfare, of the employer, and of the employee or former employee.

## II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon which were prepared to meet the need for preventing occupational disease arising from exposure to chlorine. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to "...develop criteria dealing with toxic materials and harmful physical agents and substances which will describe...exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents. It should be pointed out that any criteria for a recommended standard should enable management and labor to develop better engineering controls, resulting in more healthful work environments. Mere compliance with the recommended standard should not be used as a final goal.

Chlorine is a pungent, gaseous (at normal temperatures and pressures) element produced primarily by electrolysis of common salt. Its bleaching power and disinfecting action as well as its reactivity and its ability to form highly reactive organic compounds lead to wide use in the synthesis of solvents, plastics, and resins, in the pulp and paper and textile

industries, and in the treatment of sewage and water. The irritating properties of chlorine make it a serious respiratory hazard, as well as a skin and eye irritant. Its distinctive odor provides an indication of its presence.

These criteria for a standard for chlorine are part of a continuing series of criteria developed by NIOSH. The proposed standard applies only to the processing, manufacture, use, and handling of chlorine as applicable under the Occupational Safety and Health Act of 1970.

The standard was not designed for the population-at-large, and any extrapolation beyond occupational exposures is not necessarily warranted. It is intended to (1) protect against injury from chlorine, (2) be measurable by techniques that are valid, reproducible, and available to industry and official agencies, and (3) be attainable with existing technology.

Although the effects of massive exposure to airborne chlorine have been documented, and a limited number of experimental and epidemiologic studies have been conducted to determine the relationship between airborne chlorine concentrations and resulting effects, at the present time insufficient data exists to present a definitive correlation between chlorine exposure concentrations and acute and chronic effects observed in humans and in animals. Further research is needed to determine this correlation as well as to assess the possibility of chlorine tolerance at low concentrations, and to precisely define exposure symptoms.

### III. BIOLOGIC EFFECTS OF EXPOSURE

#### Extent of Exposure

At workplace temperatures and pressures, chlorine is a yellowish green gas with a distinctive, irritating odor. Chemical and physical properties of chlorine are presented in Table XIII-1. Because of its reactivity, it is not found in the uncombined state in nature, but commonly occurs in salt (NaCl), sylvite (KCl), and carnallite (KMgCl<sub>3</sub>·6H<sub>2</sub>O). [1] Chlorine is produced commercially by electrolysis of brine, electrolysis of fused sodium chloride, or by oxidation of chlorides using chemical methods. [1] By far the most important production method is the electrolysis of brine using diaphragm cells or mercury cells. [1] In 1973, [2] chlorine was produced by 33 companies in 65 operating plants, including 6 pulp mill plants producing their own chlorine. Of these 65 plants, 29 were diaphragm cell plants, 23 were mercury cell plants, 5 were combined mercury cell and diaphragm cell plants, 4 were fused salt plants, 1 was a diaphragm and fused salt cell plant, 1 a hydrogen chloride electrolysis plant, 1 a nonelectrolytic plant, and 1 a diaphragm and magnesium cell plant.

US chlorine production increased from 24,754 short tons in 1909 to 10,753,109 short tons in 1974. [2] The production of chlorine increased at a compound annual rate of 8.1% between 1948 and 1968. [1] Preliminary estimates place the 1976 production at 11,000,000 short tons. [3]

In 1968, the pulp and paper industry used chlorine primarily in the elemental form for bleaching pulp and paper. [1] Chlorine is used in the production of plastic and resins which are ultimately used in the

manufacture of upholstery fabrics, floor coverings, food packaging, films, bottles, utensils, hose and tubing, and electrical insulation. Chlorinated solvents are used as drycleaning agents, paint thinners, metal degreasing agents, and machinery cleaners. The production of automotive fluids, such as ethylene glycol antifreeze and ethylene chloride (used in antiknock additives), uses chlorine as an intermediate. Chlorine is also used in textile and household bleaches, refrigerants, pharmaceuticals, cosmetics, in the beneficiating of ores, and in metal extraction. [1] Exposure to chlorine can occur in any of these operations.

In addition, exposure to chlorine can occur when hypochlorites are mixed with materials such as toilet bowl cleaners [4] or vinegar [5], and when chlorinated hydrocarbons are decomposed thermally [6] or by actinic rays from welding operations. [7,8]

Some occupations with potential exposure to chlorine are listed in Table XIII-2. In 1968, it was estimated that 26,000 persons were employed in the chlor-alkali manufacturing industry. [1] NIOSH estimated in 1973 that 15,000 workers had potential occupational exposure to chlorine.

### Historical Reports

Interest was focused on the toxic effects of chlorine by its use during World War I as a war gas. Four reports [9-12] centered on the health effects of acute exposure to chlorine as a war gas and the possibility of residual effects from acute chlorine overexposure. Meakins [9] in 1919 reviewed the after-effects of chlorine war-gas poisoning by following 700 consecutive cases in the admission and discharge books of the

Canadian field ambulances serving at Ypres, France, for several weeks in the spring of 1915. Of these, 222 (31.7%) had no further details of clinical conditions ascribed to gas poisoning in their records; 478 were evacuated to the base. At the base, 146 (20.8%) were treated at the hospital: 6 patients died and the rest returned to duty. The remainder, 332 (47.4%), were evacuated to the United Kingdom for further treatment. Later, 80 returned to France and resumed duty. Forty had had irritable heart (cardiac neurasthenia), 10 bronchitis, and 4 gastric symptoms while in the hospital. [9] Of 204 invalided to Canada, 118 had symptoms of irritable heart, 30 symptoms of bronchitis and pneumonia, 4 symptoms of hemoptysis, 22 symptoms of asthma, and 20 symptoms of neuroses. The remaining 30 cases were grouped in an "indefinite symptoms" category. The average duration of hospitalization before personnel were invalided to Canada was 17 weeks. Four years after exposure to chlorine, 188 of the men invalided to Canada were studied. Seventy-eight of the men had irritable heart, 18 had neuroses, 8 had asthma, 18 had "bronchitis, etc," 14 could not be traced, and 54 were reported to have no appreciable disease.

In 1919, Berghoff [10] observed a total of 520 soldiers who, 3 - 4 months earlier, had been exposed to chlorine during warfare. Clinical examinations revealed instances of bronchitis and emphysema, but the author did not distinguish between physical findings unique to those soldiers exposed to chlorine and those findings resulting from exposures to other war gases.

As reported by Gilchrist and Matz [11] in 1933, US War Department statistics showed that 1,843 casualties out of a total of 70,742 casualties

caused by gas poisoning were the result of exposure to chlorine. A study was made of 838 of these 1,843 casualties for the purpose of ascertaining the sequelae; of the 838, 28 had died. Four of the deaths were attributed to "later effects" of chlorine gassing: bronchopneumonia, lobar pneumonia, purulent pleurisy, and tubercular meningitis. Nine of the 838 were discharged because of disabilities attributable to gassing. These disabilities included pulmonary tuberculosis, bronchitis, pleurisy, neurocirculatory asthenia, tachycardia, dyspnea, and nephritis. Of the 838, 39 were disabled at the time of discharge from conditions attributed to chlorine gassing: bronchitis, pleurisy, laryngitis, valvular heart disease, keratitis, and conjunctivitis. Of the 838, 96 were reexamined clinically and by X-ray at the time of the study. The authors concluded that 9 of the 96 men showed definite asymptomatic or symptomatic residual effects which could be attributed to chlorine gassing. The relationship of disabilities to chlorine gassing was questionable in seven instances. In 80 patients, the disabilities found at the time of the study were concluded to be in no way related to chlorine gassing incurred during the service. Of the nine men showing definite residual effects [11], five had pulmonary tuberculosis, with a coexisting emphysema in three. Three of the nine men showed evidence of chronic bronchitis; of these, one had a coexisting emphysema, one had chronic conjunctivitis, and one was free of coexisting conditions. One of the nine men had chronic adhesive pleurisy. In analyzing the five cases of pulmonary tuberculosis, the authors concluded that it was probable that gassing led to reactivation of previously quiescent tuberculous foci.

Seven men who showed disabilities that were questionably related to chlorine gassing had a history of intercurrent respiratory disease or a history of respiratory disease for which the claimants were treated just prior to, or immediately after, the gassing. In these cases, it was not possible to determine the role played by chlorine in the causation of the disabilities which appeared subsequently.

Pearce [12] in 1919 studied one person who was gassed with chlorine during the war. The man, who first received treatment some 12 months after he was gassed, failed to exhibit on medical examination any impairment of his heart and lungs, except for bronchitis. The respiratory quotient, minute volume of air, depth and rate of respiration, and tension of carbon dioxide in the alveolar air were determined at rest, while walking, and while running at a "dog trot" for a short distance, and were compared with those of the author. At rest, practically "normal" values were obtained. At exercise, the patient's minute volume of air was greater than expected from the work done, as measured by the oxygen consumption. His breathing was labored and rapid, and he felt faint. The disability in this case was interpreted as being due to a discrepancy between the ability of the blood to obtain oxygen and to rid itself of carbon dioxide. The patient was considered to be able to excrete his carbon dioxide without difficulty but to be unable to get enough oxygen. This condition was thought to be caused by the presence in many of the alveoli of bubbles of foam which prevented a free exchange of air. No definite improvement was found when the man worked while breathing oxygen at high pressures, however. He was kept under observation for about a year. He gradually developed a more severe

bronchitis, together with asthma and emphysema. No information on his smoking habits or any other significant exposure was given.

### Effects on Humans

#### (a) Odor Perception

The effect of chlorine on the sense of smell was studied in 1957 by Styazhkin [13] who conducted 144 tests on 12 persons ranging in age from 17 to 28 years. They were exposed to chlorine at low concentrations and asked if they detected the gas. Subjects inhaled through the nose from two tanks, one with clean air and one with chlorine, and were asked to designate the one containing chlorine. The threshold of chlorine odor perception occurred at 0.7 mg/cu m (about 0.2 ppm). Leonardos et al [14] determined odor threshold under controlled laboratory conditions. The odorants were presented to a trained odor panel in a static air system using a low odor background air as the diluting medium. The odor threshold was defined as the first concentration at which all four panel members could detect the odor. The odor threshold for chlorine was reported as 0.314 ppm. Ryazanov [15] reported that the odor threshold of a group of volunteers ranged from 0.80 to 1.30 mg/cu m (0.3-0.4 ppm).

Rupp and Henschler [16] determined the olfactory thresholds for chlorine in 20 healthy subjects; they were exposed for 30 minutes to chlorine. Odor was first perceived at concentrations of 0.012-0.92 ppm. Seven of 14 persons detected the smell of chlorine at concentrations which averaged 0.02 ppm. All 20 test subjects detected the odor of chlorine at a

concentration averaging 0.452 ppm, and at concentrations averaging 0.72 ppm, all subjects correctly identified it as chlorine.

The authors qualified the results of these tests by stating that the concentration of irritant gas in the test room before the test subjects entered dropped considerably after they entered. The drop amounted to 26-57% when four test subjects were exposed, and it varied from 17 to 40% for two persons. The higher the concentration set beforehand, the less the drop. In another series of experiments, chlorine was slowly introduced so that the concentration increased from 0 to 1.3 ppm during a 50-minute period. The odor of chlorine was first detected at 0.06 ppm, and by 0.2 ppm all persons exposed (number unspecified) could smell the gas.

Odor perception also was studied by Rupp and Henschler. [16] They found that the ability to perceive chlorine did not remain constant. There was a positive correlation between the length of time the odor impression remained and the chlorine concentration. At concentrations averaging 0.022 ppm, the impression disappeared for most observers within the first 5 minutes after exposure. At concentrations averaging 0.027 ppm, the impression disappeared between 5 and 15 minutes. At concentrations averaging 0.058 ppm, only a few persons still perceived the odor after 20 minutes. Starting with concentrations averaging 0.12 ppm, the test subjects in increasing numbers still had an impression of odor until the end of exposure (30 minutes), and concentrations averaging 0.72 ppm were clearly perceived by all until the end of exposure. There was no comment on the mechanism of this tolerance.

Beck [17] exposed four subjects to chlorine at concentrations increasing from 0 to 1.8 ppm. The first perception of chlorine appeared for the individuals after 31 minutes at 0.3 ppm, 35 minutes at 0.32 ppm, 44 minutes at 0.4 ppm, and 48 minutes at 0.46 ppm. When Beck exposed 10 subjects to chlorine at 0.044 ppm, 4 perceived an odor which "became increasingly weak and after 1-24 minutes could no longer be objectified." When the concentration was raised to 0.09 ppm, 7 of the 10 noticed an odor and recognized the gas, but for 6 of the 7 the odor disappeared after 1-25 minutes (average: 9 minutes). At 0.2 ppm, 13 subjects all noticed an odor, and the duration of the perception was longer by an average of 13 minutes than that for lower concentrations.

Laciak and Sipa [18] studied olfaction in 173 randomly selected workers; 17 came in contact with chlorine. The 173 workers were asked to identify eugenol, coumarin, iodoform, dinitrobenzene, and methyl salicylate in increasing olfactory dilutions of 1,5,10,20,50,100, and 200. The results were measured in "olfacties," not further described, such that a slight olfactory deficiency meant an average loss of 20 olfacties; a moderate one, 20-100; and a severe deficiency, 100 olfacties to complete anosmia. Four workers had been exposed to chlorine for 1 year or less; of these four, olfactory deficiency was slight in two, moderate in one, and severe in one. Of the 13 workers exposed to chlorine for 2-5 years, 1 suffered slight deficiency, 1 moderate, and 11 severe. The significance of the relationship between chlorine exposure and olfactory deficiency was not discussed.

According to CB Kramer (written communication, June 1974), Dow Chemical Company collected information on odor thresholds for chlorine. In 65 tests, individuals who were industrial hygienists with the company perceived no odor when exposed to chlorine at concentrations ranging from 0.08 to 2.9 ppm; in 16 tests, the odor was described as minimal at an exposure concentration of 1.1-2.7 ppm. Data illustrated individual variation. Furthermore, it was noted that odor perceptions by the same individual made late in the day, after previous exposure, were frequently less discerning than those made earlier the same day.

(b) Case Reports

(1) Severe Exposures

The dramatic response to substantial exposure is well documented in a number of accidents involving chlorine. Romcke and Evensen [19] in 1940 reported an accident in Norway that released 7-8 tons of chlorine. The number of those exposed was not given, but 85 were hospitalized and 3 died. The authors commented that some victims had latent periods as long as several hours before they developed symptoms of pulmonary congestion disturbing enough for them to seek medical attention. The authors also commented that the most severe symptoms of pulmonary edema developed most rapidly in those subjected to physical exertions. In the milder cases, the pulmonary symptoms disappeared in 2-3 days; 54 of the hospitalized patients were discharged in 3 days. In other hospitalized patients, the bronchitic sounds lasted for 8-10 days. Signs of pulmonary edema occurred in 6 patients. Autopsies of two victims revealed intense tracheobronchitis, hyperemia of the brain, and intensely edematous lungs

weighing 2,300 and 2,500 g that almost completely covered the heart.

Stout [20] recounted the occurrence of oral burns from an unusual exposure to chlorine. As a prank, a laboratory student who had filled a bottle with chlorine gas poked it under the nose of a second student. The second student recoiled and gasped for air through his mouth, but inhaled some chlorine instead. The pain in his throat increased during the first day, and he became unable to swallow. Although the inflammation gradually subsided, an unproductive cough continued for several months after the incident.

Monto and Woodall [21] reported the case of a 20-year-old man exposed to chlorine gas at approximately 0.05 ppm for several minutes because of a poorly fitting gas mask. At the time of the exposure, there was no unusual burning of the eyes, throat, or nasal passages, or any difficulty in breathing. Several hours later, he was admitted to a hospital and treated for mediastinal emphysema. His convalescence was uneventful, and all signs of disorder had disappeared by the sixth hospital day. He had had a questionable asthmatic attack 5 years before. At that time, the patient had been told that he was sensitive to dust, but diagnostic tests were not made. Since then, he had been free of asthma. In this case, chlorine gas probably produced irritation in the terminal bronchioles, causing their occlusion and resultant trapping of the contained air. It then found its way into the interstitial tissue of the lungs, probably as a result of coughing and previous chemical injury to the cells lining the alveoli.

Chasis et al [22] reported a chlorine accident which occurred in 1944 in Brooklyn, New York, and involved at least 418 persons. During

transport, one of several cylinders containing approximately 100 pounds of chlorine leaked liquid chlorine through a 1/8-inch hole for about 17 minutes. Most of the chlorine contaminated the air in a nearby subway; the chlorine created a visible cloud. No other estimates of the actual concentration or duration of exposure were made.

Of 418 persons exposed and examined, [22] 208 were hospitalized; 133 were in one hospital under the care of the authors. Of these, 33 exhibited evidence of moderate-to-severe chlorine intoxication and remained in the hospital 1-2 weeks; 35 others had milder symptoms, and the rest left to seek care elsewhere. The records of the 140 admitted to other hospitals (75 directly and 65 by transfer from the first hospital) were reviewed and, where possible, the attending physicians were interviewed. When first exposed, most persons were overcome by choking, nausea, vomiting, anxiety, and syncope.

The 33 who remained in the first hospital [22] appeared acutely ill on admission and were in moderate-to-marked respiratory distress. Twenty-eight of the 33 had a slight fever. Approximately half were cyanotic. Adventitious pulmonary sounds were present in all: 28 had dry rales on admission, whereas the rest of the patients, with one exception, developed them shortly thereafter. Subsequent moist rales developed in all but two patients. Pulmonary edema was seen in 23 of 30 patients; the others were not observed in the early postexposure period. Respiratory distress subsided, for the great majority, within 72 hours. [22] However, in five patients, it ceased within 6 days; only one patient had prolonged dyspnea, a symptom to which preexisting heart disease was presumed to have

contributed. Substernal pain generally subsided in the first 3 days, leaving a soreness attributed to tracheobronchitis. A dry cough was present initially in every patient, but promptly became quiescent with administration of oxygen and codeine, only to return in most patients after 2-5 days with the production of tenacious mucopurulent sputum, blood-tinged when first produced. Dry rales cleared by the 10th day; moist rales were still present in 20 patients during the second week. The febrile period lasted 2-13 days.

The following summarizes the clinical test data: chest X-rays showed mottling, patches of irregular densities, and differences in the degree of aeration in both lung fields. X-ray changes in most patients were not remarkable, and it was felt that readings of single roentgenograms could easily have been judged to be normal. In 3, a transient unequal aeration was noted, consistent with obstructive emphysema. In 14, serial changes permitted the diagnosis of pneumonia, basilar in 13. At the time of discharge, all chlorine-related abnormalities visible on chest X-rays were clearing or had cleared. Arterial oxygen saturation was measured 7-8 hours after exposure in eight patients selected for examination because of cyanosis and extensive pulmonary involvement. The values, ranging from 88.1 to 91.2%, were lower than normal (reported as approximately 96%) in six. Serial ECG tracings on 12 patients showed either no abnormality or a preexisting heart disease. For eight patients, vital capacity determined 48 hours after exposure gave values ranging from 16 to 57% of the predicted normal.

A special follow-up clinic [22] was established and attended by 29 of these 33 patients, usually for 16 months after exposure. Eleven had no abnormal symptoms or signs. One patient had cough and sputum for 6 months, with medium moist rales at the base of the left lung for 3 months. Upon death 10 months after exposure, a post-mortem examination showed a pulmonary embolus, but otherwise normal lungs and bronchi. A second patient, who had marked congenital kyphoscoliosis with pulmonary fibrosis (there was no comment as to its etiology), had periodic episodes of cough and dyspnea, each lasting a few days to a few weeks. Sixteen patients had what were considered anxiety reactions with phobias, hysterical phenomena, and psychosomatic dysfunctions for 1-16 months: anorexia, nausea, vomiting, weakness, nervousness, dizziness, palpitation, a sense of suffocation, and the odor and taste of chlorine. Two intrauterine pregnancies were reported to be unaffected by the exposure, but no details were given. There was no correlation between severity of symptoms during the hospital stay and the continuance of symptoms thereafter. No pulmonary function studies were reported from the special follow-up.

Baader [23] described a freak nighttime industrial accident in which there was a release of "enormous" amounts of "chlorine anhydride". Fortunately, only 190 of the 900 workers of the mill were at work, but the wind carried the cloud of gas to the town. Reportedly, some 240 people were taken to clinics, 4 workers died, and another 42 persons were in very serious condition. The signs and symptoms present in 46 patients examined by the author were as follows, in order of decreasing frequency: fever, moist rales in some pulmonary fields, dyspnea, blood in sputum,

tachycardia, vomiting or nausea, reduced arterial pressure, cyanosis, blood in urine, coated tongue, headache, severe diarrhea, "sticky sweat", fainting, infrasternal pains, constipation, pains below the costal ridge, heart pains, bradycardia, and arrhythmia. One patient who fainted from the exposure developed glucosuria. Three autopsies were performed; aside from pulmonary edema, emphysema, and the presence of bronchopneumonic condensation foci in the lungs, the most striking findings were small hemorrhages in the white matter of the cortex, corpus callosum, internal capsule, and cerebellum.

Hoveid [24] described a railcar accident in Norway which released 14 tons of chlorine. The exposure resulted in the hospitalization of 85 people. No information was presented about any others exposed. Three of the 85 died and the others were discharged following treatment as in-patients. Information on 75 was secured by mail questionnaires; 4 had died since discharge, and 3 could not be located. The questionnaire asked about "difficulties of any type...caused by this gas exposure," the use of physician services in this regard, and the incidence of recurrences. How long after the incident the questionnaires were mailed was not given, but the spill occurred in 1940 and the article was published in 1956. No difficulties were ascertained in 48 of those who responded, 16 reported difficulties "believed to be a reasonable consequence of the accident," while 11 had a "possible, but somewhat doubtful consequence." The "reasonable consequences" included dyspnea (1 person with dyspnea had pulmonary tuberculosis), bronchitis, "tightness under the chest," and "lacing under the chest." "Possible consequences" included coughing,

spontaneous pneumothorax, asthma, emphysema (6 years after exposure), bronchitis (beginning 4 years after exposure), loss of memory, "bad throat," "legs and the strength failing," "poor heart, high blood pressure," and claustrophobia. Half of those with dyspnea did not consult a physician. Eight of 16 with "reasonable consequences" had received oxygen, while 5 of 11 with "possible consequences" and 11 of 48 without difficulties received this therapy; the differences were not statistically significant.

In 1962, Joyner and Durel [25] reported a spill of about 36 tons of liquid chlorine in Louisiana. Three hours later, chlorine at an airborne concentration of 10 ppm was found in the fringes of the contaminated area; 7 hours after the spill, levels of 400 ppm were recorded 75 yards from the spill, and this was felt not to represent maximal values even at that time. Approximately 100 persons were treated for exposure to chlorine of various degrees. Of the 65 casualties handled in one hospital, 15 were admitted. Three children and one adult were unconscious on admission; an 11-month-old infant died. Ten of the hospitalized patients developed frank and unmistakable pulmonary edema. All heavily exposed victims experienced severe dyspnea, coughing, vomiting, and retching. Most of these patients complained of burning of the eyes and had acute conjunctival injection with profuse tearing and photophobia. Some victims had minor first-degree skin burns, principally of the face. The authors stated that these burns resulted from gas exposure rather than from splashes. Examination of the chest in all heavily exposed patients revealed diffuse, moist, crackling rales throughout both lung fields which were loud both on inspiration and

expiration. Harsh, sibilant rales were also audible in one patient. Sputum in bedside containers was copious, thin, and very frothy; in one patient, sputum was faintly tinged with blood on the second day after exposure. Chest X-rays made on hospitalized patients on the third and fourth days after exposure revealed striking changes: fine miliary mottling was distributed bilaterally and symmetrically throughout both lung fields. With therapy, these clinical findings slowly cleared and all hospitalized patients were discharged by the sixteenth day.

In 1969, Weill et al [26] reviewed the case histories of 12 of those who had been exposed in the spill reported above by Joyner and Durel. [25] In general, these 12 patients were the ones most severely affected in the community. Three of the 12 were studied 3 years after exposure; all 12 were studied again 7 years after exposure. The 12 study subjects included 11 of the 16 surviving hospitalized patients and the spouse of one subject, an individual who had had prominent symptoms after exposure. Observed values for total lung capacity (TLC), vital capacity (VC), residual volume (RV), and forced expiratory volume at 1 second (FEV 1) were all within two standard deviations of predicted values. [26] (A complete listing of pulmonary function abbreviations used here and subsequently is given in Appendix V.) The subjects were essentially asymptomatic from a respiratory standpoint. Chest X-rays were normal in all cases. Minor abnormalities in lung volumes were accounted for by factors other than chlorine exposure. No definite change in respiratory function was found in the three subjects who were studied both 3 and 7 years after exposure.

Gervais et al [27] studied a worker accidentally exposed to chlorine in 1965. There was no estimate of the degree of exposure except that the worker was unable to leave the area by his own efforts. The patient had rales in both lung fields but the chest X-ray was normal. The ECG showed a transient right heart block. The authors did not clearly indicate that they considered the transient heart block to be of any clinical significance or associated specifically with the exposure. The patient recovered uneventfully.

In 1967, Kowitz et al [28] presented details of an accidental chlorine exposure of at least 156 workmen during cargo unloading. No estimates of chlorine concentrations or durations of exposure were reported. Most men experienced acute symptoms. All were taken rapidly to 3 local hospitals, and 37 of the 156 were admitted. Several men returned to the hospital within 48 hours and were admitted at that time. There were no recorded deaths. Of the 17 subjects admitted to the first hospital, 11 were studied serially. All 11 had shown respiratory distress on admission; it was judged to be severe in 7. One developed bacterial pneumonia. Other clinical findings included hemoptysis, rales, wheezes or rhonchi, or both, and edema of the lungs. Within 1-3 weeks, all findings had disappeared except for symptoms of exertional dyspnea, easy fatigability, and cough. Two months after exposure, all 11 appeared clinically recovered, despite the findings of reduced lung volumes, reduced arterial oxygen partial pressures at rest which were significantly lowered upon mild exercise, and hyperventilation at rest and upon exercise. This symptomatology is consistent with acute alveolo-capillary injury (Table III-1).

Six months later, mean total lung capacity was still reduced, mean vital capacity was further reduced, and mean airway resistance had significantly increased. There was arterial hypoxemia at rest and after exercise, and a decrease in the degree of hyperventilation. At the time of the last two studies lung volumes were returning to normal, although they were still low for up to 3 years after the incident, while airway resistance remained

TABLE III-1

ARITHMETIC MEANS AND STANDARD DEVIATIONS OF THE MEANS OF SELECTED  
RESPIRATORY FUNCTION TESTS IN MAN OBTAINED SERIALY  
FOLLOWING CHLORINE EXPOSURE

Test***	Before Cl Exposure	Time of Testing After Exposure, in Months			
		2	6	14	19-35
TLC (liters)	6.31 (+0.76)	5.56 (+1.06)	5.23* (+1.00)	5.44 (+0.88)	5.74 (+0.89)
RV (liters)	1.94 (+0.4)	1.62 (+0.61)	1.91 (+0.49)	1.80 (+0.55)	1.69 (+0.57)
FRC (liters)	3.40 (+0.50)	2.51** (+0.61)	2.78* (+0.58)	2.79** (+0.55)	2.91 (+0.62)
VC (liters)	4.37 (+0.47)	3.94 (+0.78)	3.33** (+0.83)	3.64** (+0.68)	4.05 (+0.67)
Raw (cm H <sub>2</sub> O) liter/sec	1.50 (+0.20)	1.36 (+0.78)	1.95 (+0.69)	2.11 (+0.81)	2.13 (+1.08)
Glaw (liters/sec/cm H <sub>2</sub> O) liter	0.900	-----	0.966 (+0.361)	0.966 (+0.386)	0.883 (+0.581)

TABLE III-1 (Continued)

ARITHMETIC MEANS AND STANDARD DEVIATIONS OF THE MEANS OF SELECTED  
RESPIRATORY FUNCTION TESTS IN MAN OBTAINED SERIALY  
FOLLOWING CHLORINE EXPOSURE

Test***	Before Cl Exposure	Time of Testing After Exposure, in Months			
		2	6	14	19-35
PaO2 rest	>90.0 (mmHg)	71.3 (+24.0)	68.0* (+15.2)	75.5* (+12.3)	81.8 (+12.1)
PaO2 exercise	>90.0 (mmHg)	63.7* (+18.5)	70.8* (+8.9)	80.3 (+17.2)	87.3 (+10.7)
Blood pH rest	7.38-7.42	7.481 (+0.066)	7.443 (+0.042)	7.419 (+0.042)	7.427 (+0.029)
Blood pH exercise	7.38-7.42	7.476 (+0.060)	7.424 (+0.021)	7.423 (+0.037)	7.421 (+0.043)

\* Comparison of test results with predicted values when  $p < 0.05$

\*\* Comparison of test results with predicted values when  $p < 0.01$

\*\*\*Refer to Appendix V for explanation of test abbreviations

From reference 28

elevated. Carbon dioxide partial pressure and blood pH returned to normal levels, although hyperventilation was still apparent 14 months after the study. Arterial oxygen partial pressure at rest improved and, by the fourth study, definitely increased upon exercise. The authors concluded that these serial studies suggested the presence of permanent lung damage with prior attempts at repair.

All of the men involved in the foregoing accident [28] were asked to participate in a respiratory disease study approximately 18-20 months after

the accident; 73 of the 156 were evaluated. The authors commented that it was likely that the majority of those who refused to participate considered themselves well so that those studied were not a representative sample of all exposed. Of the 73, 12 were excluded because of conditions other than chlorine exposure that might have altered pulmonary function, and studies of 2 were incomplete, leaving 59 for analysis. These 59 included the original 17 admitted to the first hospital. All but 2 of the 59 subjects [28] were black, with an average age of 51.3 years. At the time of follow-up examination, the authors judged 16 of the 59 to have moderate-to-severe dyspnea on the basis of subjective complaints. Other signs and symptoms described at the time of follow-up, in order of decreasing frequency, were: cough, nonspecific chest pain, oropharyngeal membrane irritation, decreased stamina, and muscular weakness. Gross abnormalities on follow-up physical examination of the chest were the exception. Abnormal findings consisted of diminution of chest expansion, decreased breath sounds, and prolongation of the expiratory phase. Wheezing or rhonchi appeared infrequently.

An attempt was made by the authors to quantify the degree of association between the results of pulmonary function tests performed on the 59 patients and (a) antecedent history not related to chlorine exposure (cardiovascular disease, smoking, abnormalities of the chest) and (b) amount of chlorine exposure (patient's account of exposure, hospitalization, dyspnea, and reduced exercise tolerance). The profiles developed did not make a strong case for an effect resulting from chlorine exposure; however when the categories were considered individually, those with a history of more severe exposure, hospitalization, or persisting

decreased exercise tolerance had a lower diffusion capacity ( $p < 0.05$ ).

Dixon and Drew [29] reported a fatal case of chlorine poisoning. A chlorine cloud resulted when a valve was incompletely closed. For reasons which were not clear, a boiler plant operator, age 49, remained in the cloud for about 30 minutes without immediately putting on the canister mask which was available; it is not certain that he used the mask. When he reported for medical assistance, he began vomiting and complained of severe pains in the stomach and chest. There were signs of bronchial irritation and congestion, which were not further described. After an hour's observation, he was sent home; on the way, he became increasingly ill and died. The interval between initial exposure and death was 3-3.5 hours. Post-mortem examination revealed pulmonary edema as the cause of death, with coronary insufficiency due to atheroma also reported.

Beach et al [30] published the case history of a 44-year-old process worker exposed to chlorine gas at an unstated "high" concentration because of a leaking valve. He soon began to choke and then developed severe dyspnea, a persistent cough, and chest pain. His eyes "smarted" and his conjunctivae were markedly injected. Ten hours later, he was cyanotic and had rapid and shallow breathing; he coughed up pink frothy sputum. Numerous coarse crepitations were heard. He was given "continuous oxygen" for 9 days and prednisolone for 12 days. He remained critically ill for 48 hours and then gradually improved. His dyspnea at rest slowly abated and disappeared by the 10th day. The patient was discharged from the hospital after 13 days. Exercise dyspnea persisted for 5 weeks. Further followup data were not reported.

Uragoda [31] reported on a water purification plant worker who was exposed to leaking chlorine gas for a period of 20 minutes before he finally succeeded in controlling the leak. There was no further description of the exposure. He had immediate tightness of the chest, bouts of nonproductive coughing, and a severe headache. He sought medical treatment 4 days later because the cough persisted. Upon auscultation, there were scattered post-tussic rhonchi. His initial ECG showed ventricular extrasystoles every 3 beats with a pulse of 56; 27 days later, the ventricular extrasystoles occurred only occasionally. A slight cough was still present at that time. No further followup data were reported.

In 1970, Faure et al [32] analyzed 87 cases of chlorine exposure over approximately 10 years in 2 French industrial towns. Reported signs and symptoms included smarting of the eyes in 20%, burning of the nose, pharynx, and respiratory tract in 35%, a feeling of suffocation with a sensation of chest tightness in 45%, dyspnea in 45%, and cyanosis in 15%. Objective medical examination revealed signs of bronchitis with rhonchi and wheezing in 35%, indications of parenchymal disorders with crepitating rales in 20%, hemoptysis in < 3%, and lung edema in 7%. No data regarding chlorine exposure concentrations were provided. Neither the total number of workers nor the number of exposure-years in this group was given. One woman, age 40, had her first attack of asthma 6 months after excessive chlorine exposure. The authors made a strong point of the need for workers to have hazard information. Of 99 workers, 80% were ignorant of the dangers of chlorine. In a group of 55 workers supposedly better informed about hazards, with a total of 306 years of work, only 8 had exposure of "a

certain gravity." The authors felt that this paucity of serious exposures reflected the workers' better information about chlorine, presumably derived from their work experience.

Sessa et al [33] studied 12 workers who had been poisoned by chlorine. The authors made clinical observations at an unspecified time following exposures. The average age of the workers was 54 years, and the average period of employment was 28 years. Vital capacity was normal in 4, reduced in 2, and severely reduced in 6. The diffusing capacities of the lungs of chlorine workers, when averaged, were less than the normal value, but no value for range or variance was given. The timing of pulmonary function studies in relation to exposure, the criteria for these classifications, and the actual chlorine exposure concentrations were, unfortunately, not given.

Leube and Kreiter [34] examined 90 persons acutely poisoned when chlorine gas was blown by the wind across a factory site. These people were treated at a local hospital, 72 as inpatients and 18 as outpatients. There was no estimate of the degree of exposure. The following signs and symptoms were reported in 88 of the 90: coughing in 97%, dyspnea in 75%, headaches in 66%, retrosternal pain in 47%, nausea in 44%, vertigo in 33%, and vomiting in 11%. All inpatients had chest X-ray examinations between 5 and 8 hours after exposure; 10 showed early pulmonary edema. In the 48 who had ECG examinations, there were several instances of significant sinus tachycardia, isolated ventricular extrasystoles, and a repolarization disturbance of the left ventricle. Blood sedimentation rates were normal in the 30 patients who were checked. Two hours after exposure,

leukocytosis was marked -- in 60 of 68 inpatients so tested the number of white cells was above 10,000 /cu mm. Within 7 hours, 36 patients still had values over 10,000. On the following day, only six persons still showed white cell values over 10,000/cu mm; the average was once again within the norm. The activity of serum glutamic oxalic transaminase (SGOT) was abnormal in 15%, and serum glutamic pyruvate transaminase (SGPT) was abnormal in 40% of the inpatients (normal range: 12- >48 mU/ml). Sixty-six determinations of LDH (lactate-dehydrogenase) activity in serum yielded normal values. Liver biopsies were taken for two patients with exceptionally high SGPT values. In one case, some individual swollen liver epithelia, besides a nuclear perturbation was seen. No complications developed, even for the patients with heavy intoxication who were released from the hospital after 3-5 days. No further follow-up was reported.

Kaufman and Burkons [35] studied persons exposed to chlorine as a result of a leak in a liquid chlorine storage tank. Within 30 minutes of exposure, 27 exposed persons were examined in an emergency room: 5 were infants and children under 7 years of age who required hospitalization but who were not included in the study; 2 adults died of severe hemorrhagic pulmonary edema. Of the 20 survivors, 9 men and 9 women, ranging in age from 21 to 68 years, agreed to participate in the study. Thirteen of the participants were nearby-residents or passers-by at the time of the leak, while the other 5 were workers heavily exposed in the storage room. Eleven were studied within 48 hours of exposure and the rest within 48 hours to 7 days. Repeat studies of all were done at 1, 2, and 4 months following exposure. Only 12 subjects returned for examination 12-14 months after

exposure, but this number included those most heavily exposed.

Each member of the group received an exposure rating of from 1 to 4, based on the subject's description of the color and density of the chlorine gas and on the length of exposure time. A rating of 4 represented the most extensive exposure. This subjective estimate of exposure was then related to the signs and symptoms each subject displayed (Table III-2). All subjects were questioned in regard to their cardiopulmonary disease history. Pulmonary function tests measuring forced expiratory volume (FEV), FEV 1, maximum midexpiratory flowrate (MMF), RV, DLCO, VC, maximum voluntary ventilation (MVV), and partial pressure of oxygen (PO<sub>2</sub>) were performed.

Clinical results [35] revealed that exposure ratings above 2 were often associated with manifestations of pulmonary edema, although this condition was diagnosed in only one heavily exposed chlorine worker. In addition, rales, dyspnea, and cyanosis were seen in those most heavily exposed and cough was present in nearly all patients. At the time of the 30-day follow-up and subsequently, roentgenologic findings were all normal, and abnormal signs and symptoms were no longer present. Subnormal VC was observed in three patients initially, while reduction in FEV 1 was noted in four patients, three of whom were chlorine workers. Residual volume was above the predicted level in those persons most heavily exposed. Within 30-90 days, these abnormalities were less evident. Subnormal levels of MMF's were evident in six patients, all heavily exposed, but within 30 days after exposure, MMF values were normal except in one nonworker and in three chlorine workers; these three still showed low MMF's a year later. Low MVV

values returned to normal in two heavily exposed and in two mildly exposed nonworkers within 30 days. Persistently low MVV was seen in two chlorine workers up to a year after exposure. The DLCO remained persistently low

TABLE III-2  
ASSOCIATION OF CHLORINE EXPOSURE RATING  
WITH SIGNS AND SYMPTOMS

Patient	Sex	Age	Exposure Rating	X-Ray Abnormalities	Rales	Dyspnea	Cough	Cyanosis
Nonworkers								
1	F	28	4	+	+	+	+	+
2	F	30	4	+	+	+	+	+
3	F	29	3	+	0	+	+	0
4	M	21	3	+	+	+	0	0
5	M	25	3	0	+	+	+	0
6	M	33	3	0	0	0	+	0
7	F	68	3	0	0	0	+	0
8	F	30	3	0	0	0	+	0
9	F	47	3	0	0	0	+	0
10	F	65	2	0	0	0	0	0
11	F	53	2	0	0	+	+	0
12	F	48	1	0	0	0	+	0
13	M	46	1	0	0	0	+	0

TABLE III-2 (Continued)  
 ASSOCIATION OF CHLORINE EXPOSURE RATING  
 WITH SIGNS AND SYMPTOMS

Patient	Sex	Age	Exposure Rating	X-Ray Abnormalities	Rales	Dyspnea	Cough	Cyanosis
Workers								
14	M	55	4	+	+	+	+	0
15	M	56	4	0	+	+	+	+
16	M	49	4	0	+	0	0	0
17	M	32	4	0	+	0	0	0
18	M	22	4	0	0	0	+	0

Note: + indicates the presence of the sign or symptom  
 0 indicates the absence of the sign or symptom

From reference 35

throughout the study only in one mildly exposed nonworker. The P02 was subnormal initially in four patients, and 3 months later in two nonworkers who were over 65 and had no previous history of cardiopulmonary disease, and in three chlorine workers.

In summary, [35] the most heavily exposed residents and neighbors showed a pattern of airway obstruction and uneven ventilation which, for the most part, was transitory. Those moderately or lightly exposed had no physiologic disturbance except for that considered commensurate with age. Four of the five chlorine workers, with occupational exposure in a chlorine

environment for 5-30 years, showed persistent airway obstruction and mild hypoxemia. There was no comment as to their degree of exposure preceding or during the accident. Only one patient, not a worker, had continuously reduced DLCO, arterial hypoxia, and excessive ventilation, despite a mild chlorine exposure and lack of symptoms.

(2) Less Severe Exposures

Instead of having been exposed to massive amounts of chlorine because of accidents, many workers have been exposed for relatively long periods to chlorine at low airborne concentrations. Some reports [36,37] suggest a possible chronic effect from such exposures. McCord [36] reported on one worker who was employed in 1920 to shovel paper bleached with chlorine out of a cellar room, load it onto hand trucks, and transport it to another room. The worker said the odor of chlorine was always present and sometimes was stronger than at other times, but that he knew of no gross exposure. No measurements of airborne chlorine concentration or duration of exposure were reported. No smoking history or record of exposure to other contaminants was presented. In 1924, the worker first noticed the development of a slight cough, associated with sneezing and burning in the eyes. During this period, there were intervals of 1-2 weeks in which he noticed no discomfort; then the coughing and associated symptoms would return for a period of several days. The coughing became increasingly severe. By February 1925, pain was constant in the upper portions of his chest, particularly in his right lung. By August 1925, the patient became dyspneic, the pain in his lungs had increased, and his coughing and bronchitis were marked. During September 1925, after 5 years of digging

paper out of the cellar room, he was forced by his illness to discontinue work and remain at home. His chief complaints at that time were incessant coughing, severe pain in the chest, sore throat, hemoptysis, and the feeling of a "belt around the chest." Examination of his chest in March 1926, 7 months after his last exposure, showed hyperresonance of the left lung, diminished respiratory motility, harsh breath sounds, especially on expiration, distant breath sounds at the base, and occasional dry rales. [36] He was clinically diagnosed as having low-grade bronchitis with emphysema. X-ray examination showed an old tubercular lesion of the upper right lung, an old healed cavity in the upper right lung, and fibrosis in the upper left lung. The intracostal spaces were enlarged, and the density of the lung tissue was definitely diminished throughout the lower lobe of both lungs. The author stated that the patient may have had a decrease in pulmonary function because of chlorine exposure; however, the possibility of deterioration of the lung over time from other causes could not be excluded.

Bates and Christie [37] reported a chlorine exposure in a 59-year-old worker who had been engaged in the remelting of aluminum where liquid chlorine was used under pressure as a fluxing agent. He had been exposed to chlorine on at least five occasions from 1942 to 1960. With each exposure he reported having a temporary cough and shortness of breath, but chlorine concentrations and duration of exposure to chlorine were not given. After the fourth exposure, he developed severe, persistent dyspnea which was brought on by even mild exertion or talking. He did not have a cough. He had a history of diabetes mellitus and myocardial infarction.

It was not stated whether the patient smoked or was exposed to other contaminants potentially damaging to the lungs. Clinical examination of the lungs revealed no abnormalities. A low-grade hypertrophic laryngitis was found, but whether the examination of the larynx was direct or indirect was not reported. With the exception of an increase in the anterior-posterior diameter indicating moderate overinflation and a slight mid-dorsal kyphosis, radiologic examination of the lungs showed no abnormalities. The results of pulmonary function tests showed a reduction in VC and an increase in RV. Airway resistance, which was increased in the patient, was measured unreliably because the patient did not fully cooperate. A low partial pressure of carbon dioxide in the arterial blood (PaCO<sub>2</sub>) and elevated pH suggested some hyperventilation.

In summary, the above reports [19-37] indicate that exposure to chlorine may cause severe irritation, in some cases resulting in death. Thirteen of the approximately 1,250 exposed persons died. Autopsy following fatalities that resulted from acute exposure to chlorine revealed inflamed bronchi, pulmonary edema, and small foci of bronchopneumonia in the lungs. [19,23]

Nonfatal doses resulted in severe signs and symptoms including dyspnea and cough, expectoration of bloody froth, sensation of tightness in the chest, cyanosis, conjunctival injection, severe headache, nausea and vomiting, and syncope. [22,23,25,27,30] In those persons severely affected, clinical examination and chest X-rays corroborated the presence of pulmonary edema [22,25] and oxygen desaturation. [22,35] One study [34] reported serum enzyme abnormalities in SGOT and SGPT but not lactic

dehydrogenase (LDH). The same study [34] reported sharp transient leukocytosis; less marked leukocytosis was observed in a second study. [22] The absence of any mention of damage to the skin from gaseous chlorine, except in one article, [25] suggests that exposures to chlorine at high concentrations are required for this effect. There were no case reports of exposure to liquid chlorine. The bulk of evidence suggests, albeit follow-up was generally very incomplete, that most persons recover completely and relatively rapidly after massive accidental exposures. [22,26,35] On the other hand, there was some evidence of chronic impairment of pulmonary function following acute exposure. [24,28,33] There is insufficient evidence to conclude that persons chronically exposed to chlorine developed chronic impairment.

All of the reports suffered from a lack of precise data regarding airborne concentrations and exposure durations. Follow-up data on those exposed was generally very limited.

(c) Human Exposure Studies

Ryazanov in 1962 [15] described a "sensory basis" for setting occupational health standards in the USSR. The odor threshold of a group of volunteers ranged from 0.80 to 1.30 mg/cu m (0.3-0.4 ppm) chlorine. One physiologic response that was measured was optical chronaxie, the time necessary for the appearance of a sensation of light when an electrical current of twice the threshold amperage was applied to the eyeball. When air containing chlorine at concentrations of 1.5 mg/cu m (0.5 ppm) was inhaled, the chronaxie was raised just significantly. This was taken as indicating a reflex inhibition of the visual cortex from the olfactory

cortex of excitation by nerve impulses originating in the retina. Another response that was measured was the change in sensitivity to light by the dark-adapted eye. At concentrations of 1.0 mg/cu m (0.33 ppm) chlorine, a detectable change occurred. These observations [15] used very fine alterations in physiology as indications of chlorine effect. The degree to which these minute physiologic alterations represent a significant alteration of the health of exposed workers is poorly understood.

Matt [38] subjected himself along with another human subject to chlorine at various airborne concentrations. Although the concentration of chlorine was given, the magnitude of possible error was not. At the beginning of an exposure to chlorine at 1.3 ppm, the odor of chlorine was hardly noticeable, but after 7 minutes unpleasant burning of the eyes and nose was observed in one subject. At 2.5 ppm, severe burning of the eyes, mouth, and throat was apparent in 5 minutes. Exposure to chlorine at a concentration of 3.5-4.0 ppm produced nasal congestion which could be tolerated for only 16 minutes, and a coughing stimulus that lasted some 18 hours. Although symptoms of irritation were reported at an exposure concentration of 1.3 ppm, Matt concluded that exposure to chlorine at concentrations of 1.0-2.0 ppm would not disturb work.

In 1921, Fieldner et al [39] listed the Chemical Warfare Service chlorine concentrations producing irritation in man. The minimum concentration of chlorine producing irritation in the throat was 15.1 ppm; the lowest concentration causing coughing was 30.2 ppm. The "least detectable odor" was listed at 3.5 ppm. The basis for the determination of these values was not given.

Rupp and Henschler [16] exposed human volunteers to chlorine at various concentrations in two series of experiments. Chlorine was bubbled through liquid paraffin in a flask until the paraffin was saturated. The flask and paraffin were then maintained in a thermostated bath at 21 C. Air was passed at a constant rate through a sintered glass bubbler into the solution. The chlorine-air mixture thus produced was further diluted with air in a mixing flask. Confirmation of the chlorine concentrations produced was obtained by the o-tolidine method of analysis. In the first experiment, 14-20 subjects were exposed to chlorine at fixed concentrations in a test chamber in order to determine the olfactory threshold for chlorine. Concomitant with the measurement of the threshold, certain other observations were made. The duration of exposure at the specified level before symptoms appeared was not given. The authors indicated that they had some difficulty in maintaining constant concentrations of chlorine within the test chamber. A decrease in value occurred between the time the chamber chlorine concentration was set and the time the test subjects entered. Tickling in the nose occurred at concentrations averaging 0.027 ppm and in the throat at concentrations averaging 0.058 ppm. Burning of the conjunctivae was reported at concentrations averaging 0.452 ppm; the response to chlorine at this concentration was clearly felt to be pain by a few subjects (numbers not given) after 15 minutes. The authors, in the second series of tests, exposed subjects to chlorine gas at concentrations slowly increasing from zero to 1.3 ppm over 50 minutes. The number of subjects used for testing with chlorine was not specifically given; however, analogous testing for bromine was done with three test subjects.

Itching in the nose was noted at 0.06 ppm by the first subject after approximately 4 minutes, and at 0.2 ppm by the last subject after approximately 20 minutes. Cough began after 25 minutes when the concentration had risen to 0.5 ppm. When the concentration had risen to 1.0 ppm, after 35 minutes, one subject had a headache. One person had a severe shortness of breath and cough with a violent headache at 1.0-1.3 ppm. Beyond 1 ppm, the stay was felt to be uncomfortable by all test subjects.

Beck [17] experimented with chlorine using chemistry students as subjects. In order to avoid subjective effects in the experimental volunteer subjects, the chlorine concentrations were not arranged in a series of increasing or decreasing steps, but rather selected randomly according to a predetermined plan. The air in the experimental chamber was renewed 22 times/hour. Because of this frequent air exchange, the concentrations decreased only about 5% due to adsorption of chlorine by body surfaces when the subjects entered the chamber. Two overlapping determinations of airborne chlorine concentrations were made as close together as possible.

Upon exposure to chlorine at 0.044 ppm, 5 out of 10 subjects [17] noticed no alteration, 1 was undecided about his perception, and 4 stated that there was an odor. Two out of the four persons who sensed an odor were able to recognize the gas. When the concentration of chlorine increased to 0.09 ppm, all 10 persons noticed an odor, and 7 recognized the gas. Four subjects reported irritation in the upper respiratory passages consisting of tickling and stinging in the nose, a weak cough (one

subject), and increased dryness in the throat.

In another series of tests, [17] an odor was noticed by all 13 subjects at 0.2 ppm and recognized as chlorine by 5. Reports of irritation increased both in number and in intensity as compared with those at 0.09 ppm. Seven subjects reported a slight tickling in the nose and throat; one subject developed a dry, scratchy throat causing a slight cough. Three persons observed slight sensations in the conjunctivae. At 1 ppm, the intensity of these signs and symptoms increased further. Of 10 subjects, only 3 were without signs or symptoms of irritation. Tickling and stinging in the nose were reported in six instances and scratchiness and dryness in the throat in four instances. One subject reported a dull sensation in the teeth and a slight metallic taste; another felt slight pressure in the head together with headache, burning in the conjunctivae, burning of the skin, a distinct taste, coughing, and constriction of breathing (expressed as the sensation of not being able to inhale deeply). Exposure to chlorine at 1 ppm was terminated after 20 minutes because it was judged to be unbearable. One subject complained in all the experiments with chlorine of increasing irritation of the conjunctivae. This was shown not to be caused by air flow drying the conjunctivae.

Additional experiments [17] were performed to determine the effect of humidity on the perception of chlorine. Within 50 minutes, the relative humidity was increased twice within a short period (12-13 minutes) from 56 to 72%, the chlorine concentration remaining unchanged. In one experiment, three subjects were exposed to chlorine at 0.18 ppm; in another, two subjects to chlorine at 0.38 ppm. The introduction of water vapor

increased the temperature 1.5-2 C in the experimental chamber simultaneously with the increase in moisture. The odor of chlorine at both concentrations was noticed by all subjects. At 0.18 ppm, the increase in moisture decreased the odor. When the humidity was then decreased, two subjects noticed an increase in odor, but no clear correlation could be obtained between the variations in humidity and the return of the chlorine odor. At 0.38 ppm, the odor of chlorine was perceived throughout the experiment. Changes in humidity did not have a clear effect on the perception of chlorine at this concentration.

In another experiment, [17] four subjects were exposed in a chamber to chlorine at continuously increasing concentrations from 0 to 1.8 ppm. From 0.3 ppm on, three subjects felt a stinging in the throat. By 0.36 ppm, one subject had a sensation of choking; chlorine at 1.4 ppm apparently caused slight neck pain, substernal pain, and conjunctival irritation in one subject; another subject experienced a slight headache at this level.

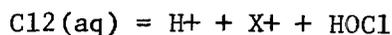
In summary, [17] in sensitive subjects, slight irritation in the nose and throat from chlorine appeared at or above 0.09 ppm; at 1 ppm, most subjects felt annoying symptoms, especially in the nose, but also in the throat and sometimes in the conjunctivae. Chlorine exposure at 1 ppm could not be withstood for longer than 20 minutes. With exposure to chlorine at concentrations less than 1 ppm, there appeared a slight adaptation to odor, but the irritation symptoms increased with increasing length of exposure to chlorine at these same concentrations. Changes in humidity did not appear to effect odor perception or symptoms of irritation.

The Dow Chemical Company (CB Kramer, written communication, June 1974) reported the results of subjective responses by their industrial hygienists when sampling workroom atmospheres for chlorine. During sampling periods of 10 minutes or more, the odor of chlorine was perceived by an unspecified number of industrial hygienists at concentrations which averaged 1.1-41.0 ppm. A respiratory response of "minimal," "easily noticed," or "strong" was experienced at concentrations which averaged 1.92-41.0 ppm. "Minimal" or "easily noticed" eye irritation was experienced at concentrations which averaged 7.7-41.0 ppm. It was noted that observations made by the same person late in the day after previous exposure were frequently less discerning than those observations made earlier in the day.

Table XIII-5 summarizes the above mentioned [16, 17, 38] exposure-effect data.

(d) Mutagenicity and Carcinogenicity

The widespread use of chlorination of potable water to kill bacteria [40] has lead to the study of the biochemical mechanism of chlorine-induced alteration of cells. [41-43] In an aqueous milieu such as that found in tissue, molecular chlorine disproportionates rapidly according to the following equation:



The equilibrium constant for this reaction in aqueous solutions is  $4.2 \times 10^{-4}$ . [44] Hypochlorous acid, which is formed as a result of this reaction, will react with ammonia and other amines. This reaction results in the introduction of the chlorine radical into the reaction products. [44]

Patton et al [41] have demonstrated that aqueous solutions of hypochlorous acid react with cytosine which is a constituent of the cellular genetic material, deoxyribonucleic acid (DNA). When one gram equivalent weight of hypochlorous acid was reacted with one gram equivalent weight of cytosine under physiologic conditions, a 76% yield of 4-N-chlorocytosine was obtained. When excess hypochlorous acid was reacted with cytosine, several more highly chlorinated derivatives of cytosine were formed. Under acidic conditions, 4-N-chlorocytosine was converted to the more stable 5-chlorocytosine. Prat et al, as cited by Patton et al, [41] isolated the latter compound from DNA treated with sodium hypochlorite.

Using transforming DNA of *Haemophilus influenza* pretreated with chlorine, Hsu [42] demonstrated that chlorine can interfere with the biologic activity of the macromolecule. Eisenstark, as cited by Shih and Lederberg [43] showed that *Bacillus subtilis* DNA has a decreased ability to transform cells after chloramine or hypochlorous acid treatment. Shih and Lederberg [43] studied the induction of breaks in the chromosome of *Bacillus subtilis* following treatment of the organism with chloramine. The number of observed DNA breaks increased monotonically as the dose of chloramine and the treatment time increased. The authors concluded that the DNA breaks induced in vivo were caused by the direct reaction of chloramine with DNA. However, they indicated that it was also possible that chloramine-induced alterations in the functional dynamics of the endonucleolytic DNA monitoring system caused the breaks. Shih and Lederberg [43] also noted that pretreatment of DNA either in vivo or in vitro reduced both the transforming ability of DNA and the cotransduction

of genetic characteristics known to be adjacent on the chromosome.

While observed changes in the cellular genetic material following treatment with chlorinating agents are a matter of grave concern, the available information does not provide any evidence concerning the magnitude of these effects in any higher organism or in humans.

No evidence has been found to indicate that chlorine is a carcinogen.

[45]

#### Epidemiologic Studies

Evans [46] in 1940 reported on chest X-rays taken in the chemical industry over 5 years. A random sample of those exposed to chlorine and hydrogen chloride (no total number given) resulted in a cohort of 35 men exposed for an average of 6.4 years. The substances were handled in a closed system. However, it was reported that low-level concentrations occurred throughout the workday and frequently there were breaks in pipelines and failures in equipment thereby allowing the liberation of unspecified quantities of chlorine and hydrochloric acid gases. Chlorine exposure concentrations were not reported. Short clinical histories of five of those exposed were presented. Three had experienced attacks of respiratory disease during their periods of employment. While employed in the area, one worker was found to have inactive tuberculosis, but it was not apparently affected by continued work in that area. For all 5, it was stated that no X-ray changes were observed.

In 1967, Ferris et al [47] compared the prevalence of chronic respiratory disease in workers exposed to sulfur dioxide, chlorine, and

chlorine dioxide in a pulp mill and a paper mill. The sample from the pulp mill consisted of 147 men who worked in either the chlorine plant, sulfite mill, Kraft plant, or in the chlorine dioxide plant. The sample from the paper mill consisted of 124 men who operated paper machines or were involved in maintenance. The study [47] was conducted over 2 months and included the taking of an occupational history, the use of a standard respiratory disease questionnaire, and the performance of pulmonary function tests for forced expiration (spirometry and peak flow measurement). Standard criteria were used for classification of these data.

Three industrial hygiene surveys [47] were done in the pulp mill between April 1958 and February 1963. Twenty-four samples indicated that chlorine concentrations ranged from trace to 64.0 ppm; only the first survey, accounting for 9 samples, showed any chlorine concentration above trace. No industrial hygiene data were given for the paper mill. In the pulp mill, 73 of the 147 workers were exposed to chlorine for an average of 20.4 years. Nine of the 124 paper-mill workers were exposed to chlorine for an average of 7.5 years. Expected rates of chronic nonspecific respiratory disease were calculated from the rates of the two mill populations pooled for the various categories of age and current smoking habits. The overall expected rates of respiratory disease were then compared with the observed rates to determine whether there was any significant difference between the two mills; the prevalence of chronic nonspecific respiratory disease was 32.5% in the pulp mill and 27.4% in the paper mill. This was not judged by the authors to be a significant

difference. Formulae based on age and height for predicting forced vital capacity (FVC), FEV, and peak expiratory flowrate (PEFR) were calculated for those in the pulp mill and the paper mill; an analysis of variance for testing the equality of regression coefficients (including the constant term) was done on the two equations for each group and, according to the authors, no significant difference was demonstrated.

No difference was noted in results of tests of pulmonary function of 118 pulp-mill workers exposed primarily to sulfur dioxide and the 73 exposed to chlorine or chlorine dioxide. However, when the responses to 12 questions about respiratory symptoms were compared, 3 were answered positively more often by men exposed to chlorine: "gassed at work" ( $p < 0.05$ ), "phlegm past 3 years" ( $p < 0.05$ ), and "shortness of breath grade 3 or more" ( $p < 0.01$ ).

There are problems in interpreting these results, some of which were pointed out by the authors. [47] The industrial hygiene surveys began in 1958; higher chlorine concentrations had probably existed in the past, possibly higher in one mill than in the other, but there were no records. It is also possible that higher levels occurred during the time surveyed, since the sampling was very limited. It is also not clear where sampling had taken place. The authors commented that many men transferred to the paper mill because they disliked the odors in the pulp mill. Because of this, men working in the paper mill may have been more sensitive to irritant gases. Finally, workers were not only exposed to chlorine, but also to sulfur dioxide and chlorine dioxide, although one usually predominated at any given location.

In 1967, Leduc [48] reported studies conducted at the request of 620 employees exposed to various irritant gases to determine effects of chronic exposure. There were 15 workers who were exposed to chlorine. The author questioned physicians in localities where workers were exposed to chlorine, specialists, and industrial physicians of factories with similar risks about their experiences with acute chlorine intoxication and any sequelae, and about their observations of ill effects from chronic exposure to chlorine. Private physicians reported treating 5 cases of acute chlorine intoxication; the author's implication was that all 5 were probably not among the 15 chlorine workers in the group requesting the investigation. The extent of exposure for the five was not quantified. Of the five, one had occasional bronchitis since exposure and one had a 5% disability granted because of bronchitis subsequent to exposure. There were no known sequelae for three; the extent of follow-up was not given. Responses from industrial physicians revealed reports on at least 301 workers; there were 2 fatalities and 2 cases of serious pulmonary edema attributed to chlorine exposure. After acute intoxication, one worker developed a serious allergic colitis which necessitated several months of hospitalization; it was not further characterized.

Capadoglio et al [49] examined all 52 workers employed in a plant for electrolytic production of chlorine and soda. Their average length of service was 10 years. With various frequency, each person experienced the irritating effect of chlorine at high concentrations. None suffered clinically significant incapacity, even temporarily. In 18 determinations of chlorine, the average concentration was 0.298 ppm (SD = 0.181). Another

group of 27 clinically healthy workers who had no current or previous exposure to chlorine, halogens, or other respiratory irritants, served as controls. The 52 exposed and the 27 controls were also classified according to smoking habits yielding 4 categories: exposed smokers, exposed nonsmokers, nonexposed smokers, and nonexposed nonsmokers. Those studied had similar ages and heights.

In comparing the four groups as to VC, FEV 1, RV, DLCO, and helium concentration gradient in a single breath during washout, only the results of the test of DLCO showed a significant difference between exposed and control workers. This value was significantly lower in exposed smokers than in nonexposed smokers ( $p < 0.02$ ), lower in exposed smokers than in exposed nonsmokers ( $p < 0.04$ ), and lower in exposed smokers than in nonexposed nonsmokers ( $p < 0.003$ ). Controlling for smoking, prior accidental exposure to chlorine was associated with a decreasing diffusion capacity. All values were corrected for height and age.

Tawast et al [50] studied 49 workers whose stays in a chlorine mill averaged 12 years. No exposure data were given. The average blood values for hemoglobin, red cell count, and leukocyte count and differential did not differ from those of 39 workers not exposed to chlorine.

During a 3-day study period, Chester et al in 1969 [51] examined all 139 men in a plant producing chlorine and sodium hydroxide by electrolysis of brine. Approximately 99% of the air samples taken in this and similar plants contained less than 1 ppm chlorine (number of samples not given). Fifty-five of the 139 workers had been accidentally exposed one or more times to chlorine at higher concentrations and had required oxygen therapy

at least once during their employment. Posterior-anterior chest films were abnormal in 56 of 138 men. The degree of exposure or length of employment of these 56 was not given. One man had a mottled infiltrate in the right apex most consistent with active tuberculosis. Extensive pleural reaction, pulmonary fibrosis, and a high-right diaphragm with plate-like atelectasis and discrete densities in the right lower lobe were separately noted in three other men. Only one subject had abnormal ventilatory function. All but 7 of the 56 revealed evidence of parenchymal or hilar calcifications that were considered to be consistent with old granulomatous disease. Evaluation of a standard respiratory questionnaire revealed that there was no significant difference between the prevalence of symptoms in those exposed to chlorine who smoked, and in those nonsmokers not exposed to chlorine. A significant difference in maximal midexpiratory flow was seen, however, when chlorine and smoking were considered as additive noxious agents (Table III-3). The authors stated that before chlorine could be indicted as a specific health hazard, a detailed study of the smoker-chlorine cohort would have to be made.

Accidental exposure was defined by the authors as one occurring at least once in the history of each worker and severe enough to require oxygen therapy. The prevalence of such exposure in smokers correlated positively with a decrease in MMF ( $p < 0.02$ ). Ages of smokers accidentally exposed averaged 42.5 years, while those with no exposure averaged 35.7 years. The authors felt that this age difference was insufficient to explain the difference in MMF.

TABLE III-3

MAXIMAL MIDEPIRATORY FLOW VALUES IN SMOKING AND NONSMOKING WORKERS  
WITH ACCIDENTAL CHLORINE EXPOSURE

Smoking	<u>Exposed</u>			<u>Nonexposed</u>		
	No.	Mean	SD	No.	Mean	SD
yes	46	3.57	1.03	56	4.13	1.33
no	12	4.10	0.76	25	4.36	1.18

From reference 51

During the course of this survey, [51] two subjects were exposed to chlorine and were treated with oxygen. One subject was observed 24 and 48 hours after this acute exposure; the other was observed immediately and then again 24 hours after exposure. The data for these two subjects revealed acute obstructive ventilatory defects with rapid clearing within 24 hours in the second subject and clearing in the first subject at 48 hours.

This prevalence study [51] indicated that workers accidentally exposed to chlorine suffered a greater reduction in pulmonary function than did those who were exposed only to chlorine at the levels usual for their work situation. Since the function studies were not expressed as percentages of normal or predicted values, no conclusions could be drawn about the effect of chlorine at lower airborne concentrations.

In 1970, Patil et al [52] studied 25 chlorine-producing plants in the United States and Canada. Air sampling at representative locations within each plant was done every 2 months throughout the study year. In addition

to the air analysis, each plant assigned its employees a job classification and described the work experience of each classification in relation to actual or potential chlorine exposure. TWA exposure data were calculated for each worker on an 8-hour basis.

A total of 600 male workers from diaphragm-cell plants constituted the total work force considered to have been exposed to chlorine throughout their employment in cell rooms. [52] Because of lack of exposure data on 268, the study population for determining dose-response relationships was 332 workers. The control group of 382 consisted of workers from many of the same plants who were not routinely exposed to chlorine. There were no other control groups. TWA exposures to chlorine ranged from 0.006 to 1.42 ppm, with a mean of 0.15 ppm. All but 6 of the 332 workers had TWA exposures less than 1 ppm and only 21 had TWA's above 0.52 ppm. The average number of chlorine-exposure years for all diaphragm-cell workers was 10.9. Employees with 10-14 years' experience constituted the single largest group, and this group also contained the most workers exposed to more than 0.52 ppm chlorine. There was no correlation between the chlorine concentration and the number of years a person was so exposed.

The exposed and control groups described above were well-matched with respect to age, ranging from 19 to 69 years; 60% of the workers were 30-49 years old. [52] The mean age of the two groups combined was 31.2 years. About 60% of the workers in both groups smoked at the time of the study. In order to determine whether a significant number of workers with occupational exposure to chlorine had retired due to causes related to chlorine exposure, health data were collected on workers not involved in

the study who had terminated employment. No patterns were evident, and it appeared that most workers had resigned or were reassigned for reasons unrelated to health.

The following observations [52] were reported. Chlorine workers showed a higher incidence of history of tooth decay ( $p = 0.025$ ) than controls, but there were far less actual observed abnormalities of teeth and gums of chlorine workers as seen by the examining physician than those reported: out of 332, 98 actually had abnormal teeth and gums. The authors reported no significant dose-response relationship.

Medical histories of the prevalence of frequent colds, dyspnea, palpitation, and chest pain showed no dose-response correlation; however, values were not given. [52] Chest X-rays were evaluated for 544 workers exposed to chlorine. Of these, 21.3% had abnormalities, compared with 26.8% among the controls. Most of these abnormalities, 75%, represented hilar or parenchymal calcifications. Pleural and diaphragmatic abnormalities accounted for 11.4%. No neoplasia or serious acute pulmonary diseases were reported. No significant dose-response correlation was found when chlorine exposure was related to VC, MVV, FEV, and forced expiratory volume at 3 seconds (FEV 3) values. There was, in summary, no evidence of permanent lung damage attributable to chlorine at the levels reported.

Of the 329 ECG's from 332 workers, 9.4% were abnormal as compared to 8.5% in controls; the number of ECG's taken in each group was not given. [52] The incidence of fatigue (undefined) was greater in workers exposed to chlorine at concentrations greater than 0.5 ppm, but there was no apparent correlation below 0.5 ppm. Nervousness, headache, insomnia, and

shyness showed little relationship to chlorine exposure. Anxiety and dizziness showed moderate correlation with exposure level ( $p = 0.020$ ). Histories of neurologic illness and use of alcohol were unrelated to chlorine levels. There was no correlation of exposure with either tremors or abnormal reflexes. The gastrointestinal system and skin showed no dose-related effects. Leukocytosis ( $p < 0.05$ ) and low hematocrits ( $p < 0.017$ ) exhibited some relation to chlorine exposure. In summary, with the exceptions of anxiety, dizziness, leukocytosis, and lower hematocrits, dose-related effects were not found at exposures ranging from 0.006 to 1.42 ppm chlorine.

The use of prevalence studies, such as those reported in the foregoing reports, [51,52] to define the relationship between chronic exposure to chlorine and the development of symptoms or signs, suffers from certain conceptual as well as methodologic difficulties. The exposure pattern, even excluding acute episodes, may be variable over time. The results of air-monitoring performed during a study do not necessarily reflect chlorine exposure prior to the study. Acceptance of volunteer workers for medical examinations may produce a group of those exposed to chlorine potentially different from the pool of all exposed workers. The pulmonary function effects attributed to exposure to chlorine closely resemble those produced by smoking and by other respiratory irritants, some of which commonly occur in combination with chlorine; adjustment for all of those known exposures is difficult, and the possibility remains that unknown exposures exist. Selection of subjects to represent some defined group may pose problems. Complete follow-up information is difficult to

secure. Analysis of sufficient air samples to calculate TWA's for each exposed employee is rarely done. Some of the adverse effects of chlorine exposure are subtle and thus require fairly large studies to make them visible.

In spite of these difficulties, the authors [46-52] in the seven studies described did attempt to document chronic changes following chlorine exposure by means of prevalence studies. Three [46,47,50] concluded there were no observable effects by the methodology used. Chester et al [51] found that previous accidental exposure to chlorine in workers who smoked was associated with a decreased MMF. Patil et al [52] noted some apparent dose-response relationships, but none involving the respiratory tract. Leduc [48] uncovered two cases of pulmonary edema and two fatalities attributed to chlorine by industrial physicians. Capodaglio et al [49] found a lower DLCO in persons who reported having had previous accidental exposure at work. None of the seven epidemiologic studies made any measurements which would clarify whether or not small airway disease had occurred.

#### Animal Toxicity

In 1920, Underhill [53] described effects of chlorine on dogs. Animals not subjected to any previous testing were exposed to chlorine gas for 30 minutes at 50-2,000 ppm. They first showed general excitement, as indicated by restlessness, barking, urination, and defecation. Irritation was distinctly visible, as indicated by the blinking of eyes, sneezing, copious salivation, retching, and vomiting. Later, their respiration

became labored with frothing at the mouth. Although the dogs frequently drank large quantities of water, they refused food. With increased concentrations of chlorine, the respiratory distress increased until death occurred, usually within 24 hours, apparently from asphyxiation. Table III-4 shows that at a chlorine concentration of 800 ppm half the animals died within 3 days, while at 900 ppm exposure 87% died within this time.

Animals which died after 3 days were classified as "delayed" deaths. The animals so classified did not exhibit the signs of acute exposure, ie, labored and distressed breathing, for more than 1 or 2 days. They showed signs of loss of appetite, extreme depression, and weakness. In the majority of cases, deaths classed as "delayed" resulted from secondary factors, chiefly bronchopneumonia following the subsidence of acute pulmonary edema. The author considered "the minimum lethal toxicity of chlorine gas under the conditions of the experiment" to occur between 800 and 900 ppm chlorine.

Underhill [53] conducted further experiments on 40 of 43 dogs surviving the first gassing (Table III-4) with chlorine. He reported 53 original survivors, as shown in Table III-5; however, his tabular data presented only 43 survivors. The discrepancy was not explained.

Two interpretations of these results were suggested by the author: the first gassing either rendered the animals less susceptible to the effects of subsequent exposure or killed the weaker individuals. When the deaths from the first gassing were added to those from the second gassing, the final percentage of dying was practically identical with the original standard toxicity figures, a finding supporting the second hypothesis.

TABLE III-4

## MORTALITY IN DOGS EXPOSED TO HIGH CONCENTRATIONS OF CHLORINE

	Concentration of Chlorine, ppm													
	50- 250		400- 500		500- 600		600- 700		700- 800		800- 900		900- 2000	
	N*	%	N*	%	N*	%	N*	%	N*	%	N*	%	N*	%
Deaths														
1st day	0	0	0	0	0	0	4	19	3	17	12	52	10	71
2nd day	0	0	1	6	1	10	5	24	4	22	6	26	3	21
3rd day	0	0	0	0	1	10	0	0	2	11	2	9	0	0
Delayed deaths	1	11	4	24	2	20	5	24	2	11	1	4	0	0
Recoveries	8	89	12	70	6	60	7	33	7	39	2	9	1	7
Total number exposed	9		17		10		21		18		23		14	

\*N is the number of dogs calculated from percentages and total exposed.

From reference 53

In 1920, Winternitz et al [54] examined 326 dogs at post mortem which had been gassed with chlorine. The dogs were those which had died in the course of the study conducted by Underhill [53]. The salient features of pathologic changes in dogs dying within the first 24 hours after gassing (acute deaths) included severe injury to the mucous membranes of the upper

TABLE III-5  
MORTALITY IN DOGS REEXPOSED TO CHLORINE

Concentration of Chlorine at First Gassing (ppm)	No. of Dogs Surviving First Gassing	Concentration of Chlorine at Second Gassing (ppm)	No. of Dogs Exposed	No. of Acute Deaths	No. of Delayed Deaths
Less than 200	8	738-882	6	5	0
400-600	19	750-860	15	9	1
600-800	13	643-1065	12	2	1
800-900	10	809-851	7	2	0

From reference 53

respiratory tract with irregular dilation and contraction of the bronchi resulting in alternating patches of acute emphysema and atelectasis in the lungs. All tissues of the respiratory tract showed extreme congestion and edema. An acute inflammatory reaction began within a few hours of exposure, developing into pneumonia. Animals autopsied 2-5 days after gassing, (delayed deaths) showed an increase in the intensity of inflammation and development of lobular pneumonia which was frequently complicated by abscess formation and gangrene. Bronchiolar spasm was most pronounced in this group. Dogs in a group autopsied 5-15 days post mortem (late deaths) suffered pulmonary damage of a severity between that of the dogs in the delayed death group and that of the recovered dogs. Death of dogs in this group was usually due to pulmonary infection, pneumonia, and bronchitis, the most striking feature of which was the tendency of the inflammatory exudate to organize. This was cut short by the death of the animal.

Animals surviving for 15-193 days after gassing [54] showed marked emphysema, which was associated with an organizing exudate in the bronchioles, "bronchiolitis obliterans." The bronchioles sometimes contained a small amount of purulent exudate, but the larger mass of exudate was organized, composed of fibroblasts and blood vessels with some mononuclear cells. Not infrequently, the lung tissue surrounding such a bronchus showed an organizing pneumonia. Aside from the areas of organizing pneumonia which were found around the bronchi, the alveoli were, in many places, filled with a cellular exudate. This exudate was not composed of polynuclear leukocytes, or red cells, but almost entirely of large mononuclear cells with watery, vacuolated protoplasm similar to the desquamated cells frequently found in the more chronic forms of pneumonia in man.

Winternitz et al [54] thought that the patchy distribution of alveolar damage as seen in these animal studies represented effects of chlorine which reached the terminal sacs through airways that were not occluded by spasm.

Faure et al [32] studied 35 guinea pigs exposed to chlorine at 200 ppm for 15-30 minutes, repeating the pathologic observations of Winternitz et al. [54] The clinical reactions of the animals were not given.

Barbour [55] found that four dogs exposed to chlorine at concentrations of 24-30 ppm for 30 minutes showed clinical signs of "irritation," but that they returned to an apparently normal condition immediately after removal from the chamber. During the gassing, lacrimation and profuse salivation usually occurred as well as mild

retching and vomiting. The effects upon pulse and respiratory rates were variable. When exposed to chlorine at 180-200 ppm for 30 minutes, three dogs showed the irritant effects described above followed by general depression of muscle activity and dyspnea before the gassing was terminated. No evidence of bronchitis or edema was obtained and the animals became apparently normal again after a few hours. When chlorine at 800-900 ppm was used, it killed at least 85% of the dogs which were exposed for half an hour. It is not clear how many dogs were exposed at this concentration. These studies were also carried out to assess the possible effect of chlorine on body temperature. At 24-30 ppm, there were rises in body temperature which averaged 0.8 C and lasted 3-24 hours during gassing of four dogs. At 180-200 ppm, the three dogs had decreases in temperature which averaged 0.7 C during gassing. Return to normal began promptly; the average duration of hypothermia was 6 hours. At 800-900 ppm, the temperature drop in 20 dogs averaged 1.0 C/half hour. The temperature continued to fall at the same rate for another 1 1/2 hours, after which it declined more gradually until death ensued. After being gassed at 800-900 ppm, dogs were unable to regulate their body temperatures when exposed at moderately high external temperatures (35-40 C) or at low ones (ordinary room temperatures), a range over which ungassed animals maintained their normal temperature.

Gunn [56] found that exposing cats and rabbits to chlorine at concentrations of 1 part/5,000 parts (200 ppm) to 1 part/10,000 parts (100 ppm) produced a reflex constriction of the bronchi lasting about 1 minute. The rate of respiration increased concomitantly.

Bell and Elmes [57] used specific pathogen-free 14-week-old rats (SPF rats) to determine whether chlorine exposure at 40 ppm for 3 hours daily for a total of 43 hours (discrepancy not explained) had any immediate effect or altered the effect of exposure to chlorine at 117 ppm at age 30 weeks (3 hours daily until about half had died, 29 hours). They presented the details about the exposure in another paper. [58] At 40 ppm, exposed rats coughed, sneezed, and huddled together; after 3 hours, their noses were running and sometimes blood-stained. Exposure to chlorine at 40 ppm did not make death from chlorine at a subsequently higher concentration (177 ppm) more likely.

A second experiment [57] compared the effect of chlorine at high concentrations on SPF rats and those with spontaneously occurring lung diseases. Female SPF and diseased rats were exposed as separate groups to chlorine at 118 ppm for 3 hours followed by 14 hours at 70 ppm. Male SPF and diseased rats were exposed initially to chlorine at 34 ppm for 3 hours daily with incremental increases to 170 ppm; the total duration for male rat exposures was about 60 hours at a mean chlorine concentration of 90 ppm. It was concluded that the presence of preexisting lung disease increased the likelihood of death from exposure to chlorine at high concentrations ( $p < 0.01$ ). In diseased animals, the cellular response to heavy exposure was much more severe than that in SPF animals. Proliferation of goblet cells and aspiration of mucus were more intense and extensive in the diseased stock following exposure to chlorine. In animals dying during exposure, diseased rats had a significantly higher incidence of emphysema than did SPF stock. The most noticeable difference between

the two groups lay in the reactions of the alveolar part of the lungs to the aspiration of bronchial mucus and debris. In both experiments, the SPF animals showed no increase in number of polymorphonuclear cells, while in 11 of the 29 diseased animals dying during exposure there were extensive areas of acute inflammation with infiltration by polymorphonuclear leukocytes in relation to the aspirated mucus; in another 10, there were smaller patches of acute pneumonia.

Arloing et al [59] exposed guinea pigs to chlorine at 5 mg/cu m (1.7 ppm) for 5 hours daily over 87 days. There were four groups of animals as follows (no numbers or tests of significance were given): (1) exposed first to tubercle bacilli and then to chlorine, (2) first to chlorine and then to tubercle bacilli, (3) given tubercle bacilli but no chlorine, and (4) given chlorine but no tubercle bacilli. The method of achieving this concentration of chlorine was cited [60] but not given in detail. The animals were described as tolerating it perfectly well. Of the animals exposed only to chlorine, all survived over 300 days. There was considerable overlap between the survival times of the first three groups of animals (Table XIII-4), but regardless of how the tubercle bacilli were administered (subcutaneously, into ganglia or conjunctivae, or into trachea), the average length of survival decreased when the animals breathed chlorine.

In a series of experiments evaluating the effects of mixtures of mercury vapor and chlorine gas, Viola and Cassano [61] compared the toxic effects of mercury vapor alone with the effects of a mixture of chlorine gas and mercury vapor. Eighty Wistar rats (average body weight of 250 g)

were divided into two equal groups and exposed separately 5 hours/day, for 5 days/week, during a period of 3 months. The first group was exposed to airborne mercury at a concentration of 4.5 mg/cu m and the second group was exposed to airborne mercury at the same concentration mixed with 1-3 ppm chlorine. After about 6 weeks of exposure to mercury vapor (first group), the rats revealed hyperexcitement, sometimes followed by ataxia and tremor. The rats exposed to airborne mercury vapor mixed with chlorine gas showed mild dyspnea, cough, and diarrhea in the second week. After 2 months of treatment, 10 of the 40 rats in the first group and 4 of 40 rats in the second group had died. In an earlier experiment, [61] the authors had demonstrated a fourfold reduction of the mercury vapor concentration in a closed chamber when chlorine gas was added. A fine precipitate, stated to be mercurous chloride--a reaction product of mercury and chlorine gas--was visible on the floor of the chamber and was thought to have accounted for the mercury vapor reduction.

The authors [61] concluded that the addition of chlorine gas to an atmosphere containing mercury vapor not only reduced mercury absorption, but resulted in a different distribution of the metal in the body, thought to be due to the formation of mercurous chloride. The latter conclusion was supported by autoradiographic studies in which radioactive mercury vapor showed a much different distribution pattern in rats when compared with orally administered radioactive mercurous-203 chloride.

The preceding animal studies were not especially helpful in elucidating the effects of exposure to chlorine at low concentrations. Two studies [53,55] provided data indicating a mortality rate in dogs of 85-87%

after exposure to chlorine at concentrations of 800-900 ppm; one study [53] suggested a chlorine LC50 for dogs of 800 ppm after 3 days following a 30-minute exposure to chlorine. The remaining studies, [32,54,56,57,59,61] with one exception, [59] either did not provide any data on chlorine concentrations, or the concentrations were 40 ppm and higher. Arloing et al [59] exposed guinea pigs to chlorine at 1.7 ppm, 5 hours/day for 87 days. Guinea pigs challenged with tubercle bacilli, with and without a chlorine challenge, were compared with guinea pigs exposed to chlorine alone. In all cases, the animals challenged with either the tubercle bacillus or chlorine alone survived longer than animals exposed to both; and, animals exposed first to chlorine and then to tubercle bacilli died sooner than those first exposed to tubercle bacilli and then to chlorine; this suggests that an increased susceptibility to infection may occur after an exposure to chlorine.

#### Correlation of Exposure and Effects

All the historical studies [9-12] and case reports [19-37] -- and the epidemiologic studies [46-52] with the exception of the publication by Patil [52] -- share a common deficiency when one attempts to quantify an exposure they described. Even when air sampling had been done, the values for chlorine concentration were available only during a limited period of the worker's total exposure, while any chlorine effects could also be assumed to represent the impact of unknown amounts of additional exposure in the past. Nonetheless, epidemiologic inferences, supplemented by limited studies of acute human exposures, allow some correlations to be

made between exposure levels and effects.

Rupp and Henschler [16] observed that human adults suffered discomfort when the chlorine concentration had risen from zero to 1.3 ppm during 50 minutes. At 1.3 ppm, one subject had severe shortness of breath, cough, and a violent headache. For other subjects, cough began when the chlorine concentration had risen to 0.5 ppm. Itching in the nose occurred in subjects exposed to 0.2 ppm for 4-20 minutes, and headache was experienced at 1.0-ppm exposure after approximately 35 minutes. [16] Beck [17] was unable to continue the experiment beyond 20 minutes when one subject in 10 exposed to chlorine at 1 ppm complained of headache, burning in the conjunctivae and skin, a distinct taste, coughing, and a sense of not being able to inhale deeply. Tickling and stinging in the nose were reported by six others, and four had scratchiness and dryness of the throat. When the concentration of chlorine was gradually increased from zero, one of four subjects considered 0.42 ppm as the limit of "what might be required" of experimental subjects. The details of this individual's symptoms were not given. The same subject had the sensation of choking at 0.36 ppm. Three of the four subjects experienced stinging in the throat at 0.3 ppm. Among 13 subjects exposed to chlorine at 0.2 ppm, 7 reported slight tickling in the nose and throat, 1 had a slight cough, and 3 reported sensations in the conjunctiva.

Matt [38] reported that exposure to chlorine at a concentration of 1.3 ppm resulted in an unpleasant burning in the eyes and nose of one subject after 7 minutes. Exposure at 2.5 ppm resulted in severe burning of the eyes, mouth, and throat after 7 minutes.

The much higher thresholds of discomfort (15.1 ppm) and olfaction (3.5 ppm) determined by Fieldner et al [39] are unsupported by adequate documentation. Ryazanov [15] determined 0.3-0.4 ppm to be the threshold concentration for detection of the odor of chlorine by man. Rupp and Henschler [16] found that 51 of 60 persons perceived the odor of chlorine at a concentration of 0.104 ppm or less; 7 of 14 persons could detect it at 0.038 ppm. Beck [17] found an odor was perceived by all 10 subjects exposed to chlorine at 0.09 ppm (7 could identify the odor) while at 0.044 ppm, 4 of 10 subjects detected an odor (2 could identify it as chlorine).

According to CB Kramer (written communication, June 1974), the Dow Chemical Company reported subjective responses made by their industrial hygienists when sampling workroom atmospheres for chlorine. During sampling periods of 10 minutes or more, odor was perceived by an unspecified number of industrial hygienists at concentrations which averaged 1.1-41.0 ppm. A respiratory response of "minimal," "easily noticed," or "strong" was experienced at concentrations which averaged 1.92-41.0 ppm during the collection of 41 samples. "Minimal" or "easily noticed" eye irritation was experienced at concentrations which averaged 7.7-41.0 ppm during the collection of five samples. It was pointed out that tests by the same individual made late in the shift after previous exposure were frequently less discerning than those made earlier the same day, implying some degree of fatigue or adaptation of olfaction.

Chronic exposures were evaluated by Ferris et al [47] who observed workers exposed an average of 20.4 years to chlorine concentrations ranging from 0 to 64 ppm without observing any adverse effects from chlorine.

Patil et al [52] found no pulmonary effects in 332 workers from 25 plants exposed for an unspecified number of work-years to chlorine at a mean TWA concentration of 0.15 ppm (range: 0.006-1.42 ppm). Capodaglio et al [49] published data on workers with an average exposure of 10 years to chlorine at concentrations which averaged 0.3 ppm who showed some decrease in diffusion capacity; these decreases were associated, however, with histories of accidental acute exposures of unknown magnitude.

Chemical changes have been observed in the genetic material of bacteria following treatment with chlorinating agents. [41-43] The significance of these changes in relation to human populations has not yet been determined.

No evidence has been found to indicate that chlorine is a carcinogen.

[45]

#### IV. ENVIRONMENTAL DATA

##### Sampling and Analytical Methods

Colorimetric methods of analysis involving air sampling have generally used the following reagents: arsenous anhydride, [62] neutral and acidic iodide, [63] methyl orange, [64] and o-tolidine. [65]

Boltz and Holland [66] reviewed the o-tolidine method, the methyl orange method, and the Konig reaction method involving cyanogen chloride-pyridine and condensation of a glutaconic aldehyde with an aromatic amine. For the o-tolidine method, a 9-liter air sample filtered through 10 ml of absorber solution allowed the detection of 0.05 ppm chlorine [67]; for methyl orange, a 3-liter air sample filtered through 10 ml of absorber solution allowed detection of 0.05 ppm chlorine. [64] Both methods were pH-dependent and neither was specific for chlorine only; other oxidizing agents will produce the same effects. [66] These authors [66] found that iron(III), manganese(III), manganese(IV), and nitrite interfered with the determination by o-tolidine, although Johnson and Overby [68] introduced a stabilized, neutral, o-tolidine reagent which effectively eliminated these interferences. Oxidizing agents such as bromine were reported to interfere with determination by methyl orange, [64] although iron(III) and substances containing available chlorine such as chloramine did not. [66] Laitinen and Boyer [69] developed a methyl orange method for visible-UV spectrophotometric determination of chlorine in the presence of bromine. Microliter aliquots of bromine solution and chlorine solution were simultaneously injected into a nitrogen stream which was dispersed into 25

ml of methyl orange solution. Individual halogen mole fractions were determined with an 8% relative error and a  $\pm 11\%$  relative standard deviation. The cyanogen chloride-pyridine method [66] was observed to be fairly specific, detecting chlorine, bromine, and substances containing available chlorine or bromine, but it suffered from decreased sensitivity (50% of that of the o-tolidine method [66]), the use of noxious reagents, and lengthy color development (2 hours at room temperature, 40 minutes at 60 C). [66]

The color reaction of the methyl orange-chlorine system was seen immediately (bleaching) with color stability lasting at least 24 hours. [64] The o-tolidine system [66] developed maximum color with chlorine almost immediately; the color intensity decreased thereafter at a moderate rate. [66]

Instrumental methods have been primarily gas chromatography, [70-77] UV spectrophotometry, [69,78] colorimetry, [79] amperometry, [80] mass spectrometry, [81] catalytic combustion, [82] and the use of direct reading detector tubes. [83] Bethea and Meador [71] listed 15 gas chromatographic methods for chlorine including one for an on-line analyzer. The gas chromatographic methods were particularly suited to trace analyses but required specialized equipment and expertise. Measurements of absorbance of UV radiation may be useful for singular halide components [69] but are difficult with mixtures. [78] Analogous potential complicating effects occur with mass spectrometric and catalytic combustion procedures.

Sampling of air by syringe followed by a colorimetric analysis using permeation tube standards has been developed [84] but reproducibility

depends on operator technique.

Currently, certain chlorine-specific tubes have been evaluated and certified by NIOSH in accordance with the provisions of 42 CFR 84 (1974). In order to be certified, detector tubes must exhibit (1) accuracy within  $\pm 35\%$  at half of the NIOSH test concentration (NTC) and within  $\pm 25\%$  at 1, 2, and 5 times the NTC (for chlorine, the NTC was 1 ppm); (2) channeling (beveled stained-unstained interface) of less than 20%; and (3) tube reader deviation (standard deviation estimate of three or more independent readers) of less than 10% of the average of the readers.

A method has been developed which uses an electrolytic cell through which chlorine-laden air passes. Chlorine oxidizes the bromide ions in the electrolyte; this causes an increase in current in the amperometric measuring circuit of the cell. Current is measured directly by the concentration of chlorine in the range of 0-50 ppm. [80] A procedure was developed [70] which used the combination of a gas chromatograph, an ion-selective electrode, and a combustion furnace. It detected as little as 36 ng chloride after conversion from chlorine. A combination pyrolysis-furnace/microcoulometric cell was found to be accurate to  $\pm 2.5\%$  with a detection limit of 3 ng but it was sensitive to chlorinated hydrocarbons. [79] Other electrometric methods, [85-87] continuous colorimetric methods, [88, 89] and sensitized test paper methods are mentioned in the literature. [90] In general, automatic and continuous monitoring methods are effective for a narrow range of specific industrial applications, eg, process or fixed position area monitoring, but they are not suited for typical work situations where breathing zone concentrations must be determined.

Although the o-tolidine method is the most sensitive procedure for determining trace amounts of chlorine, [66] the methyl orange method is not affected by iron III or compounds containing available chlorine such as chloramine, and yet has 70% of the sensitivity of o-tolidine. [66] In addition, o-tolidine has been mentioned as a suspected carcinogen. [91,92]

The method of choice for atmospheric sampling and analysis of elemental chlorine in working environments is the methyl orange procedure. [64] In this procedure, 10 ml of methyl orange sampling solution is placed in a fritted bubbler, and a volume of air is drawn through at a rate of 1-2 liters/minute for 15 minutes. Absorbance is then measured with a spectrophotometer. This procedure is designed to cover the range of 5-10 mg of free chlorine/10 ml of sampling solution. For a 30-liter air sample, this corresponds to approximately 0.05-1.0 ppm in air. The method has an accuracy of +5%. Reagent stability is good and preparation is not lengthy. Samples remain stable for 24 hours (see Appendix II). Equipment and apparatus needed are uncomplicated, and sampling and analysis are straightforward and easily interpreted.

Minimal performance criteria required for this recommended method and for any proposed alternative method should provide at least one-half the recommended environmental limit as a level of reliable detection. This is required for the purpose of identifying work areas subject to periodic air sampling.

#### Environmental Levels and Engineering Controls

Few studies have been published concerning workroom airborne

concentrations of chlorine and the extent of engineering controls required to reduce exposures. In 1964, this scarcity of information prompted the environmental health study of a chlorine plant described by Pendergrass.

[93] In the plant studied, chlorine was produced by the electrolysis of brine in 180 Hooker-type cells. The chlorine unit consisted of a cell house, a purification area, a compressor area, and a cell renewal building. The cell house and purification areas were of primary concern in this study. The building housing the cells was about 60 x 300 feet with a high ceiling and partial side walls. The purification area was about 25 feet from the cell house and was not enclosed. In normal operations, exposure could occur when chlorine was released to the workroom atmosphere during routine replacement of cells, replacement of connectors between cells and headers, discharge of caustic and brine from cells, and from leaks in old cells. Release of chlorine in the purification area was most likely to occur at the primary cooler. Larger amounts of chlorine were occasionally released to the work area when increased gas pressure blew a water seal in a header. Over a 5-month period, 2,785 air samples of 8 hours' duration each were collected and analyzed. The 8-hour average levels at various locations ranged from less than 0.1 ppm to greater than 6.0 ppm. Of the 2,785 samples, 91.2% were less than 1.1 ppm chlorine and 98.9% were less than 1.0 ppm chlorine. The concentrations of chlorine in those samples which exceeded 1.0 ppm were not given. Controls were not mentioned, but the system described by Pendergrass apparently was essentially an enclosed system.

Ferris et al [47] used detector tubes to determine chlorine concentrations in the workplace air of a pulp and paper mill that manufactured its own chlorine. Thirty-three samples indicated the existence of workplace airborne concentrations of chlorine that ranged from "trace" to 64 ppm. (Mean concentrations ranged from "trace" to 7.4 ppm.) No sampling locations were given and no samples were related to any specific job category. Existence of controls was not mentioned.

Concern with the possible long-term effects of exposure to chlorine at low airborne concentrations resulted in an epidemiologic study by Patil et al [52] of diaphragm cell workers in 25 chlorine plants. Each plant was surveyed every 2 months throughout the study-year. TWA exposures to chlorine ranged from 0.006 to 1.42 ppm with a mean of 0.15 ppm. Of the personnel studied, 98.2% were exposed to chlorine at TWA levels below 1.0 ppm. Controls were not mentioned, nor were sampling locations designated.

Feiner and Marlow, [94] reporting on industrial hygiene in pulp mills, stated that the need for control of chlorine by ventilation in pulp mill-bleaching plants was minimal when chlorine was accurately metered in proportion to the volume of stock to be bleached. However, they recommended covers for bleach chests, hoods for rinse washers, and exhaust ventilation of the enclosures as precautionary measures. The authors did not provide air sampling data to support their statement.

Elkins [95] reported one sample of "hazardous concentration" out of four samples taken in textile- and paper-bleaching processes. "Hazardous concentration" was assumed to indicate that the threshold limit value of 1 ppm was exceeded. No further data were given.

Joyner and Durel [25] reported on a spill of about 6,000 gallons of liquid chlorine. Three hours after the spill, the contaminated area was approximately 200 yards in length along a highway. Chlorine at concentrations of 10 ppm was found in the fringes of this area. About 7 hours after the spill, chlorine at a concentration of 400 ppm was found in more heavily contaminated areas 75 yards from the spill. Two and one-half hours later, after treatment of the spill had begun, the airborne chlorine levels dropped to 8 ppm. Joyner and Durel stated [25] that minor first-degree burns of the facial skin resulted from exposure to the gaseous chlorine. In a verbal communication of July 1974, Joyner stated that there was no opportunity for persons to contact the liquid; therefore, he was certain that the skin irritation was caused by gaseous chlorine.

Capodaglio et al [49] investigated the respiratory function of workers engaged in chlorine production by means of the electrolysis of brine in mercury cells. They noted that no special precautions were taken to control chlorine in the plant air, although ventilation was present to minimize mercury exposure. Presumably this would have also prevented exposure to chlorine. The authors [49] stated that natural and forced ventilation "assured 40 hourly exchanges" in a 40,000-cu m shed. Under these conditions, 18 samples taken for an unspecified period of time showed the average airborne chlorine concentration to be 0.298 ppm. Sixteen spot samples showed an average chlorine concentration of 0.122 ppm.

Smith et al [96] reported that most chlorine cell rooms had airborne chlorine levels well below 1 ppm, usually in the 0.1 to 0.3-ppm range. No supporting data were given.

The TI-2 Chemical Industry Committee of the Air Pollution Control Association [97] mentioned that chlorine-manufacturing and processing equipment was normally operated with a slightly negative gauge pressure, thus preventing leaks of chlorine into cell room atmospheres. Pressure fluctuations occurring in the system from power outages or compressor failures could have caused chlorine leakage until cells were shutdown.

Connell and Fetch [98] described vacuum-operated systems for water chlorination. These systems removed much of the hazard which could result from leaks in pressurized chlorine piping.

Many references recommend ventilation of chlorine storage and handling rooms. [98-105] Several of these [99,102,104] recommend a ventilation rate of one air change every 1-4 minutes. Ventilation for bleach mixing rooms and rooms for storage of chlorine containers in pulp, paper, and paperboard mills is required by 29 CFR 1910.261. It has been recommended that chlorine be used only in fully or partially enclosed systems. [99]

## V. DEVELOPMENT OF STANDARD

### Basis for Previous Standards

In order to obtain data on industrial contaminants which might affect Massachusetts' workers, Elkins [106] prepared in 1939 a list of existing threshold concentrations or maximum allowable concentrations (MAC's), added some tentative proposals for Massachusetts, and sent the list to 19 American and 8 foreign experts. Suggestions and criticisms were received from all but two of the American and four of the foreign experts. The results were tabulated and considered in detail by the Massachusetts Dust and Fume Code Committee. One ppm was proposed for chlorine as a maximum allowable concentration. There was no written explanation provided to determine if this was intended as a TWA or as a ceiling value.

In 1945, Cook [107] compiled a list of standards and recommendations for MAC's of industrial atmospheric contaminants. The author noted that 1 ppm was the MAC value for exposure to chlorine in the workplace air in California, Connecticut, Massachusetts, New York, Oregon, and Utah. According to Cook, 2 ppm was the standard promulgated by the American National Standards Association (now the American National Standards Institute Inc). The American National Standards Institute Inc order department, however, has no record of a standard prior to 1945 (written communication, March 1976).

Cook [107] reported that early work had indicated 1 ppm should be the maximum allowable concentration for chlorine, and that this recommendation had been generally followed in industry. However, Cook [107] proposed 5

ppm rather than 1 ppm based on data referred to in the US Bureau of Mines technical paper 248. [39] This paper described research conducted by the Chemical Warfare Service, American University Experiment Station, and purportedly showed that 15.1 ppm chlorine was necessary to cause throat irritation and 30.2 ppm was necessary to cause coughing, while the chlorine concentration least detectable by odor was 3.5 ppm. This value was likely a TWA concentration since Cook stated that in every case the concentrations given were considered allowable for prolonged exposures, usually assuming a 40-hour week.

In 1947, the American Conference of Governmental Industrial Hygienists (ACGIH) [108] adopted an MAC for chlorine of 2 ppm. It was not stated whether this MAC was intended as a ceiling concentration or as a TWA concentration. The April 1948 meeting of this same organization [109] adopted 1 ppm as a threshold limit value (TLV). This TLV for chlorine was clearly specified as a TWA concentration.

In their documentation of TLV's [110] published in 1962, the ACGIH cited reviews by Heyroth [111] and Henderson and Haggard [112] to explain its selection of 1.0 ppm as the TLV for chlorine. Heyroth [111] cited data from an unpublished dissertation that men could work without interruption in air containing 1-2 ppm chlorine. A translation of this dissertation by Matt [38] has been reviewed in Chapter III under Effects on Humans. Heyroth listed 1 ppm as a "maximum permissible" limit in 13 states and 5 ppm in Ohio and Washington. Heyroth [111] also referred to the Principles of Exhaust Hood Design, [113] in which DallaValle suggested that the limit be less than 0.35 ppm. The basis for this limit was not identified.

Henderson and Haggard [112] recommended a maximum concentration of 0.35-1.0 ppm for prolonged exposure. The only reference cited by either Heyroth [111] or Henderson and Haggard [112] which gave any support to the TLV of 1 ppm was Matt, [38] as quoted by Heyroth. [111]

Henderson and Haggard [112] and a more recent edition of Heyroth [114] were used as a basis for the 1966 documentation [115] of the 1-ppm chlorine TLV. It was recommended as a ceiling value "to minimize chronic changes in the lungs, accelerated aging, and erosion of the teeth," but no data were given to document the occurrence of these chronic changes.

Between 1965 and 1968, [116-119] the 1-ppm TLV was considered a ceiling value by the ACGIH. A revised second edition of the Documentation citing Heyroth [114] listed a threshold limit of 1 ppm as adopted by the ACGIH and deleted its discussion of concentrations proposed by different states and its reference to DallaValle. [113]

The 1971 documentation of threshold limit values [120] acknowledged that relatively few studies provided data useful in developing a TLV and proceeded to give a general review of proposed limits without specifically supporting its TLV as a TWA concentration of 1 ppm. Thus it stated that Heyroth [114] and Flury and Zernik [121] had proposed 1 ppm, Henderson and Haggard [112] had suggested 0.35-1 ppm, Cook [107] had suggested 5 ppm, and Rupp and Henschler [16] had proposed 0.5 ppm. This documentation [120] discussed the results of studies by McCord, [36] Ferris et al, [47] and Kowitz et al [28] in which adverse effects were found in humans after exposure to chlorine. However, the exposure levels in these studies [36,47] varied from negligible to 15 ppm and did not give support to the

TLV of 1 ppm. The Kowitz et al report [28] concerned a chlorine accident and did not quantify exposures.

In 1971, the Pennsylvania Department of Environmental Resources [122] adopted a 1-ppm TLV which was a TWA concentration and it also adopted a short-term limit of 3 ppm for 5 minutes. [123] Heyroth [114] and Imperial Chemical Industries, Great Britain, (no specific reference listed) were cited as a basis for the documentation of these short-term limits. Heyroth [114] reported that chlorine at 3-6 ppm caused a reaction, but that men could work without interruption at 1-2 ppm. The Imperial Chemical Industries recommendation [123] stated that exposure to chlorine at 4 ppm for more than a short time might lead to symptoms of illness.

A number of occupational airborne chlorine limits have been set by foreign countries and international groups. East Germany, Hungary, Poland, [124] and Bulgaria [125] recommended a permissible concentration of 1 mg/cu m (about 0.3 ppm) while West Germany [124] recommended 2 mg/cu m (0.5 ppm) and Czechoslovakia, Great Britain, Yugoslavia, [124] Finland, [125] and Japan [126] recommended 3 mg/cu m (about 1 ppm). The Czechoslovak limit of 3.0 mg/cu m was a suggested mean value and was asserted to be "considerably lower than the concentration which molests unaccustomed persons." A peak concentration (MAC) of 6 mg/cu m (about 2 ppm) was also established which was stated to be low enough to prevent lung edema after short exposure. Documentation was not given for the suggested MAC.

In the Soviet Union, [127] a mandatory maximum permissible concentration of 0.001 mg/liter (about 0.3 ppm) in the workroom air was established by the Main State Health Inspector of the USSR, January 10,

1959, Regulation No. 279-59. Maximum permissible concentrations were considered ceiling values, and they could only be exceeded with the permission of the State Sanitary Inspection of the USSR if the workers were in an industrial area for an "unspecified short period."

Rupp and Henschler [16] investigated the relevant literature and conducted studies on the effects of chlorine at low concentrations on man. Exposures to chlorine at concentrations of 0.5 ppm and higher were reported to be disturbing. The authors proposed an MAK value of 0.5 ppm to the Committee for Testing of Industrial Substances Injurious to Health of the German Research Association. It is not clear whether this MAK value was intended to be a TWA or a ceiling concentration. This value was accepted by the committee in November 1961. Prior to 1961, the MAK value in Germany had been 1 ppm. [16]

In 1971, the Japanese Subcommittee on Permissible Concentrations of Hazardous Substances [126] recommended the continued acceptance of the 1961 TWA concentration of 1 ppm chlorine. The recommendation was based on reports and data from human and animal experiments as well as from experience in the industry. No documentation was provided.

In 1963, the Second International Symposium on Permissible Limits for the Air of Workplaces [128] adopted an MAC of 1 ppm which was considered as a ceiling value. No basis or documentation was given to support the MAC.

ANSI [129] adopted an 8-hour TWA concentration of 1.0 ppm in 1974. At the same time, a maximum peak acceptable concentration of 3.0 ppm for 5 minutes and an acceptable ceiling of 2.0 ppm were established. In both cases, these concentrations were acceptable only insofar as the 8-hour TWA

was not exceeded. The primary references cited in support of the acceptable concentrations were: Patil et al, [52], Kowitz et al, [28] Weill et al, [26] Kaufman and Burkons, [35] and Heyroth. [114]

The present federal standard (29 CFR 1910.1000) for chlorine is an 8-hour TWA of 1 ppm and is based on the ACGIH TLV for 1968. [119]

#### Basis for the Recommended Environmental Limit

Exposure to high concentrations of chlorine can be fatal. The LC50 for dogs is approximately 800 ppm for 30 minutes. [53,55] Humans have died after accidental exposures to high chlorine concentrations. [19,23,24,29] These were accidental exposures and the chlorine concentrations were not reported.

Accidental massive exposures to chlorine have on occasion been associated with ECG changes. Four studies [22,27,31,34] mentioned ECG's of patients exposed to chlorine at high concentrations. Chasis et al (22) obtained serial tracings on 12 patients and found either no abnormality or evidence of preexisting heart disease. Uragoda's [31] patient had one PVC (premature ventricular contraction) every three normal beats when first examined with fewer PVC's on a subsequent examination, 27 days later. In 48 ECG's taken in cases of chlorine exposure, Leube and Kreiter [34] found several instances of significant sinus tachycardia, isolated ventricular extrasystoles, and signs of repolarization disturbance of the left ventricle. Gervais et al [27] found one instance of transient right-heart block. A lack of data made it impossible to estimate a possible dose-response relationship.

Chronic lung disease was reported in persons who had accidental exposure to chlorine at high concentrations. [24,28] Hoveid [24] relied on statements by exposed individuals about their health. These statements were made an unspecified time after exposure, without other confirmation. He assigned 20% of the persons to the category of those having "difficulties believed to be a reasonable consequence of the accident." Kowitz et al [28] performed a series of pulmonary function tests on 11 persons after they were discharged following hospitalization for exposure to chlorine and found that, even after 3 years, their lung volumes were still low. The study did not provide a quantitative estimate of the exposure, although the acute respiratory distress had been severe in 7 of the 11, and acute symptoms were documented in the remaining 4. [28]

Rupp and Henschler, [16] in exposing subjects to concentrations of chlorine increasing from zero to 1.3 ppm over 50 minutes, demonstrated that itching in the nose began at 0.06 ppm after approximately 4 minutes for one subject, cough began at 0.5 ppm within 25 minutes, and headache began at about 1 ppm; beyond 1 ppm, all test subjects felt the stay was uncomfortable. Between 1.0 and 1.3 ppm, one subject had severe shortness of breath, cough, and a violent headache, the latter probably an individual variation in sensitivity to chlorine. When 20 subjects were exposed to concentrations of airborne chlorine averaging 0.027 ppm, tickling in the nose occurred; at concentrations averaging 0.058 ppm, tickling involved the throat as well, and at concentrations averaging 0.452 ppm conjunctival burning was present, which progressed to a feeling of pain in a few

subjects after 15 minutes. There were no data given for effects at concentrations of chlorine between 0.5 and 1.0 ppm.

In a similar study, Beck [17] found that 4 of 10 subjects, after exposures of up to 30 minutes, experienced some tickling and stinging in the nose at 0.09 ppm, and one had a weak cough. At 0.2 ppm, 7 of 13 had tickling and stinging in the nose and throat and 3 had slight conjunctival burning. At 1 ppm, 7 of 10 had symptoms of upper respiratory irritation. In one subject, the exposure had to be terminated in 20 minutes because it was unbearable. With gradually increasing concentrations of chlorine, three of four subjects exposed felt a stinging in the throat at 0.3 ppm, and at 1.4 ppm, one subject felt neck pain and conjunctival irritation.

Matt [38] experienced an unpleasant burning in the eyes and nose when he exposed one subject to chlorine at a concentration of 1.3 ppm. He concluded, however, that uninterrupted work was possible at this level.

In contrast, subjective responses of industrial hygienists from the Dow Chemical Company [CB Kramer, written communication, June 1974] suggested that chlorine at a higher concentration was required to produce a respiratory response or eye irritation. During air sampling periods of 10 minutes or more, average chlorine concentrations of 1.92-41.0 ppm produced a "minimal", "easily noticed," or "strong" respiratory response. Eye irritation was considered "minimal" at an average concentration of 7.7 ppm (one air sample) and "easily noticed" at concentrations of 8.7-41.0 ppm (4 samples). The above values were qualified, however, by the observation that a previous exposure of the same individual on the same day resulted in a less discerning response subsequently.

Several epidemiologic studies [46-52] have attempted to relate previous industrial exposure to the frequency of pulmonary abnormalities and symptoms found. The study by Ferris et al [47] indicated that no specific adverse effects resulted from repeated exposures to chlorine at concentrations ranging from 0 to 64 ppm over a period averaging 20.4 years. Insufficient data were provided, however, to determine TWA exposures. The most extensive prevalence study, which was conducted by Patil et al [52] and which was the only one reporting time-weighted averages, reported TWA concentrations of chlorine were 0.44 ppm or less for all but 21 of 332 workers. For these 21, the TWA concentrations ranged from 0.52 to 1.42 ppm; 15 were 0.52-1.00 ppm and 6 were 1.00-1.42 ppm, and their durations of exposure ranged from 2 to 14 years. No dose-response relationship (extent of exposure to chlorine vs pulmonary symptoms or signs) could be established for any of the 332 workers. There are no prospective epidemiologic studies relating the degree of exposure to chlorine in industry with the incidence of either mild chronic symptoms or chronic disability.

It is concluded that the existing federal standard should be lowered. Exposures to chlorine at concentrations of 1.3 ppm for 7 minutes, [38] 0.2-1.0 ppm for 30 minutes or less, [17] and 0.5 ppm or more for 1 hour or less [16] have resulted in the development of symptoms of both ocular and respiratory irritation. Exposure to chlorine at concentrations of approximately 0.5 ppm resulted in conjunctival pain in several subjects after 15 minutes. On the other hand, it was reported [52] that 311 workers exposed to chlorine TWA concentrations of 0.44 ppm or less for an average

of 11 years did not show any significant dose-related pulmonary or ocular effects when compared with a control group. Considering this evidence as well as the fact that further research is needed (see Appendix III) to clarify the relationship between chlorine dose and effect, a ceiling concentration of 0.5 ppm chlorine, measured over a sampling period of 15 minutes, is recommended as an environmental limit.

It is recognized that many workers handle small amounts of chlorine or work in situations where, regardless of the amounts used, there is only negligible contact with the substance. Under these conditions, it should not be necessary to comply with many of the provisions of this recommended standard, which has been prepared primarily to protect worker health under more hazardous circumstances. Concern for worker health requires that protective measures be instituted below the enforceable limit to ensure that exposures stay below that limit. For these reasons, "exposure to chlorine" has been defined as exposure at or above one-half of the environmental limit, thereby delineating those work situations which do not require the expenditure of health resources for environmental and medical monitoring and associated recordkeeping. One-half of the environmental limit has been chosen on the basis of professional judgment rather than on quantitative data that delineate nonhazardous areas from areas in which a hazard may exist. However, because of nonrespiratory hazards such as those leading to skin burns or irritation or eye contact, it is recommended that appropriate work practices, training, and other protective measures be required regardless of concentrations of chlorine in air.

## VI. WORK PRACTICES AND ENGINEERING CONTROLS

Safety precautions for the manufacture, storage, transportation, handling, and use of chlorine are well-defined and are the subject of a considerable body of literature. [40,99, 101-105,130-136] These references are concerned primarily with prevention and control of hazards arising from emergency situations involving escape of relatively large amounts of chlorine from leaks or accidentally ruptured containers or pipelines. Reports of work practices written specifically for the prevention of low-level exposures are limited. The type of ventilation needed for the control of chlorine in storage areas and in the workplace depends upon vapor density, dead air spaces, temperature, convection currents, and wind direction. [99] Use of fully or partially enclosed processes is recommended. [99,137] If full enclosure of the process cannot be used for control, good engineering practices, such as those recommended in Industrial Ventilation--A Manual of Recommended Practice, [138] should be followed in order to control continuous low-level exposures and to minimize excursions.

### Emergency Assistance

If in-plant emergency teams cannot cope with chlorine emergencies, the nearest supplier, manufacturer, or designated source of information must be called for assistance. [102,134,139] Phone numbers of persons for such assistance must be prominently posted in areas where emergencies are likely to occur. Information to be furnished to the supplier, manufacturer

or other information source when assistance is requested should include:

User company name, address, telephone number, and persons to contact for further information.  
Travel directions to emergency site.  
Type and size of container or other equipment involved.  
Nature, location, and extent of emergency.  
Corrective measures being applied. [102]

Standardized kits for the control of leaks have been designed and manufactured. The Chlorine Institute maintains current listings of the locations of these kits. [139,140] The Chlorine Institute should be contacted for the nearest location if chlorine emergency kits are not readily available locally.

#### Training and Drills

The value of drills and training in handling emergencies and in using equipment for personal protection and control of escaping chlorine was emphasized in the literature. [99,101,102,105, 134,141,142] Danielson [143] reported on a chlorine spill caused by a rail car bumping into a tank car discharging chlorine. A total of 55 tons of chlorine could have been released into the atmosphere; however, only a few tons escaped because of quick action by employees and supervisory personnel. Danielson [143] credited the quick action to rigorous and thorough training and drills.

#### Leaks

Studies by the Bureau of Mines [144] indicate that pinhole leaks in chlorine containers are rapidly enlarged by corrosion if moisture is

present. Furthermore, the control of chlorine leaks or spills by the use of water is not effective because of the limited solubility of chlorine in water. [144] Even the coldest water will supply sufficient heat to cause an increase in the evaporation rate of chlorine. [134] Therefore, water must never be used on leaking containers of chlorine, or to control spills. It is illegal to ship a leaking container of chlorine. [136]

Daily checks must be made for leaks in pressurized chlorine systems and containers. [134] Leaks may be detected by using the vapor from strong ammonia water. A white cloud will be formed near leaks. [99, 105,130,134] If leaking chlorine cannot be removed through regular process equipment, it may be absorbed in alkaline solutions. [99,130,134,136] These solutions can be prepared as described in Table XIII-3. [130] The quantities listed in the table are chemical equivalents and it is desirable to provide excess over these amounts in order to facilitate absorption.

Emergency leak kits designed for standard chlorine containers are available at various locations throughout the country. These kits operate on the principle of capping off leaking valves or, in the case of cylinders and ton containers, of sealing off a rupture in the side wall. [130] A record of kit locations is maintained by the Chlorine Institute. [139,140] If possible, users of chlorine should have their own appropriate emergency leak kits readily available for use at the process location. It should be noted, however, that the use of leak kits requires some training prior to use in an emergency situation.

Chlorine containers must be used on a first-in, first-out (FIFO) basis, [99] because valve packings may harden during prolonged storage and

cause leaks when containers are finally used.

Because of the potential danger of excessive hydrostatic pressure in chlorine containers, such containers are filled only partially with liquid chlorine, leaving sufficient gas-filled space to act as an expansion chamber. [40,100] Accordingly, gaseous chlorine is discharged from a cylinder if the cylinder is in the upright position, and liquid chlorine is discharged if the cylinder is inverted. Gaseous chlorine is discharged from the upper valve and liquid chlorine from the lower valve in a ton container. To minimize a leak in a container, the container should be oriented so that gaseous chlorine is discharged instead of liquid. The volume of gaseous chlorine formed by vaporization of liquid chlorine is about 450 times its original volume as a liquid. [99,102,134,137]

#### Protective Clothing and Equipment

Whenever liquid or gaseous chlorine is handled or used, it may come in contact with the skin and eyes, or be inhaled. For this reason, personal protective clothing and equipment are necessary. While not specific for chlorine, safety glasses or goggles, hard hats, and safety shoes should be worn or be available as dictated by the special hazards of the area or by plant practice. [130] Personnel working in areas where chlorine is handled or used should be provided with suitable escape-type respirators. Supplied-air and self-contained breathing apparatus should be used when the concentration of chlorine is not known, as in an emergency. [130]

Canister-type gas masks have limitations. In chlorine concentrations

of 2% (20,000 ppm), a canister will protect the user for about 10 minutes. [145, 30 CFR 11] Canisters should be discarded and replaced whenever they are used, or when the shelf life, as indicated by the manufacturer, expires. Canister masks do not protect in atmospheres deficient in oxygen and should not be used except for escape in chlorine concentrations exceeding 1%. [99,102,134,136,146] Self-contained breathing apparatus or supplied-air full-face respirators should be worn when atmospheres contain more than 1% chlorine or where oxygen deficiency may exist. Workers required to use respiratory protection must be thoroughly trained and drilled in its use. [99,105,136,146] When the concentration of chlorine is not known, as in an emergency, canister masks must not be used.

#### Fire and Explosions

Chlorine is classified as nonflammable and nonexplosive. However, it will support combustion of certain materials, [99,102,134] reacting explosively in some cases. At elevated temperatures, it reacts vigorously with most metals. [102] Carbon steel, for example, ignites in an atmosphere of chlorine at 483 F (250 C). [102] Fusible plugs are placed in chlorine containers to prevent rupturing of containers from excessive hydrostatic pressure caused by high temperatures. The fusible metal is designed to melt between 158 F and 165 F (70-74 C). [102] For these reasons, it is important to avoid application of heat to chlorine containers.

Explosions have occurred during the chlorination of synthetic rubber, [147,148] during the manufacture of chlorine, [149,150] and in chlorine absorption systems. [151] The last two incidents [150,151] were caused by

a mixture of hydrogen and chlorine which was in excess of the explosive limits. Determination of the explosive limits of chlorine-hydrogen mixtures indicates variations of from 3% hydrogen in pure chlorine to 8% hydrogen in a pressurized gas mixture containing 19% chlorine. [152] It is important that precautionary measures be taken to prevent chlorine from coming into contact with materials with which it may react.

#### Hydrostatic Rupture of Containers and Systems

Liquid chlorine has a very high coefficient of thermal expansion. [102,146] A 50 F (28 C) rise in temperature causes a volume increase of about 6%. [145] If liquid chlorine is trapped in a pipeline between two valves, increasing temperature will cause very high pressures, leading to possible hydrostatic rupture of the line. Accordingly, precautions must be taken to avoid this. It is important that liquid chlorine lines be at the same or higher temperature as the chlorine being fed into the line to prevent condensation, and that the lines be equipped with adequate expansion chambers, pressure relief valves, or rupture discs discharging into a receiver or a safe area. [102,153-155] Some expansion chambers are heated to ensure that chlorine does not condense therein and destroy the effectiveness of the vapor cushion. [154] Should it become necessary to evacuate a chlorine line equipped with expansion chambers, it is important that the vacuum not be broken with liquid or gaseous chlorine, a procedure which would render the expansion chambers ineffective. Dry air or nitrogen must be used for breaking such vacuums. [153]

### Warning Properties

The readily identifiable odor of chlorine and the attendant disagreeable reactions it produces appear to be one means by which workers are warned of impending excessive exposure. [40,99,101-103,105,137,141,156] However, determinations of the threshold of odor have given varying results. For example, Ryazanov [15] found the threshold of odor of chlorine to be 0.3-0.45 ppm, while Fieldner et al [39] and Leonardos et al [14] reported it to be 3.5 ppm and 0.314 ppm, respectively. The variation of these results probably reflects differences in methods of determination, and possibly differences in the development of odor adaptation. [16-18,99,101] While a noticeable odor of chlorine may indicate a potentially hazardous exposure, it should not be relied on as a quantitative indication.

### Specific Gravity of Chlorine Gas

Gaseous chlorine is about 2.5 times as heavy as air. [102] Therefore, in the absence of air currents, [99] leaking chlorine tends to accumulate in low spots. Storage areas should be constructed with this property in mind, eliminating low spots unless they are specifically engineered for the purpose of chlorine collection. Personnel evacuation plans should consider the slope of the terrain and the prevailing wind direction when describing evacuation routes and sites. [99,134] Ventilation systems should remove contaminated air at the lower levels of rooms and replacement air should enter at the higher levels. [99,102]

### Unmanned Chlorinators

Chlorinators used for water treatment are often unattended. [90] Allen and Angvik [90] described an alarm system based on the use of sensitized paper at remote unmanned chlorination stations which darkened upon contact with escaping chlorine gas. When the chlorine concentration reached 3 ppm, a visual warning signal appeared in a central manned station. Remedial personnel with proper emergency leak kits and respiratory protection equipment were then dispatched to the site of the leak. All chlorination stations were equipped with ventilation to clear the building of excessive chlorine. Consideration should be given to the installation of such alarm systems for unmanned areas where chlorine is stored or used.

### Materials of Construction

Materials which will come into direct contact with liquid or gaseous chlorine in storage, conveying, process, or other systems must be carefully selected to avoid excessive corrosion or more serious consequences. Dry chlorine may be handled in a wide variety of materials but moist chlorine is extremely corrosive. Chlorine will vigorously react with many metals at elevated temperatures. [98,99,102,104,131-134,137,155] Ventilation has been used to prevent airborne chlorine from corroding equipment. [157] Ventilation systems for transporting chlorine should be constructed of corrosion-resistant materials.

### Unusual Sources

Excessive exposure to chlorine may occur when solutions of hypochlorites are mixed with materials such as toilet bowl cleaners [4,158] or vinegar. [5] Maintenance and custodial personnel should be warned of this possibility and instructed not to mix hypochlorites with any other material. Chlorine exposure may also occur when chlorinated hydrocarbons are decomposed thermally [6] or by ultraviolet radiation from electric arcs. [7,8]

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VIII. APPENDIX I  
METHOD FOR SAMPLING CHLORINE IN AIR

General Requirements

In order to evaluate conformance with the environmental limit, chlorine concentrations in air shall be determined within the worker's breathing zone. Sampling procedures shall conform with the following criteria:

(a) Samples collected shall be representative of the individual worker's exposure.

(b) Sampling data sheets shall include:

(1) The date and time of sample collection.

(2) Sampling duration.

(3) Volumetric flowrate of sampling.

(4) A description of the sampling location.

(5) Ambient temperature and pressure.

(6) Other pertinent information (eg, worker's name, shift, work process).

Breathing Zone Sampling

(a) Breathing zone samples shall be taken as near as practicable to the worker's face without interfering with his freedom of movement. Care should be taken that the bubbler is maintained in a vertical position during sampling.

(b) A portable, battery-operated, personal sampling pump capable of being calibrated to 5% at the required flow in conjunction with a midjet fritted bubbler (coarse porosity) holding 10 ml of sampling solution shall be used to collect the sample.

(c) The sampling rate shall be accurately maintained at 1-2 liters/minute for a period of 15 minutes.

(d) A "blank" bubbler should be handled in the same manner as the bubblers containing the samples (ie, fill, seal, and transport) except that no air is sampled through this bubbler.

#### Calibration of Sampling Trains

Since the accuracy of an analysis can be no better than the accuracy of the volume of air which is measured, the accurate calibration of a sampling pump is essential to the correct interpretation of the volume indicator. The frequency of calibration is dependent on the use, care, and handling to which the pump is subjected. In addition, pumps should be recalibrated if they have been misused, or if they have just been repaired or received from a manufacturer. If the pump receives hard usage, more frequent calibration may be necessary. Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples.

The accuracy of calibration is dependent upon the type of instrument used as a reference. The choice of calibration instrument will depend largely upon where the calibration is to be performed. For laboratory testing, a 1-liter buret (soapbubble flowmeter) or wet-test meter is

recommended, although other standard calibrating instruments, such as a spirometer, Marriott's bottle, or dry gas meter, can be used.

Instructions for calibration with the soapbubble flowmeter follow. However, if an alternative calibration device is selected, equivalent procedures should be used. The calibration setup for personal sampling pumps with a midget bubbler is shown in Figure XIII-1.

(a) Check the voltage of the pump battery with a voltmeter both with the pump off and while it is operating to assure adequate voltage for calibration. If necessary, charge the battery to manufacturer's specifications.

(b) Fill the bubbler with 10 ml of the sampling solution.

(c) Assemble the sampling train as shown in Figure XIII-1.

(d) Turn the pump on and moisten the inside of the soapbubble meter by immersing the buret in the soap solution and drawing bubbles up the inside of the buret until they are able to travel the entire length of the buret without bursting.

(e) Adjust the pump rotameter to provide a flowrate of 1.5 liters/minute.

(f) Check the water manometer to ensure that the pressure drop across the sampling train does not exceed 13 inches of water (approximately 1 inch of mercury).

(g) Start a soapbubble up the buret and, with a stopwatch, measure the time it takes for the bubble to travel a minimum of 1.0 liter.

(h) Repeat the procedure in (g) above at least three times, average the results, and calculate the flowrate by dividing the volume

between the preselected marks by the time required for the soapbubble to travel the distance.

(i) Data required for the calibration include the volume measured, elapsed time, pressure drop, air temperature, atmospheric pressure, serial number of the pump, date, and name of the person performing the calibration.

(j) Corrections to the flowrate may be necessary if the pressure or temperature when samples are collected differs significantly from that when calibration was performed. Flow rates may be calculated by using the following formula:

$$q \text{ (actual)} = q \frac{\text{(indicated)} \times P \text{ (calibrated)} \times T \text{ (actual)}}{P \text{ (actual)} \times T \text{ (calibrated)}}$$

where:        q = volumetric flowrate  
              P = pressure  
              T = temperature (Kelvin or Rankine)

(k) Use graph paper to record the air flow corrected to 25 C and 760 mmHg as the ordinate and the rotameter readings as the abscissa.

IX. APPENDIX II  
METHOD FOR ANALYSIS OF AIR SAMPLES

Principle of Method [64]

Near a pH of 3.0, the color of a methyl orange solution nearly ceases to vary with increasing acidity. [64] At this pH, the dye may be quantitatively bleached by free chlorine and the extent of bleaching determined colorimetrically. The optimum concentration range is 0.05-1.0 ppm chlorine in ambient air (145-2900  $\mu\text{g}/\text{cu m}$  at 25 C and 760 mmHg).

Apparatus

- (a) Spectrophotometer suitable for measurement at 505 nm, preferably accommodating 5-cm cells.
- (b) Midget fritted bubblers (coarse porosity) of 25-ml capacity.
- (c) Sampling pump capable of a flowrate of 1-2 liters/min.

Reagents

Reagents must be ACS analytical reagent grade. Distilled water should conform to ASTM Standard for Reference Reagent Water, a blank standard.

- (a) Methyl orange stock solution, 0.05%. Dissolve 0.500 g reagent grade methyl orange (sodium 4'-dimethylaminoazobenzene-4-sulfonate) in distilled water and dilute to 1 liter. This solution is stable indefinitely if freshly boiled and cooled distilled water is used.

(b) Methyl orange reagent, 0.005%. Dilute 100 ml of stock solution to 1.0 liter with distilled water. Prepare fresh for use.

(c) Sampling solution. Dilute 6 ml of 0.005% methyl orange reagent to 100 ml with distilled water. Add 3 drops (0.15-0.20 ml) of 5.0 N hydrochloric acid. One drop of butanol may be added to induce foaming and increase collection efficiency, although care must be taken to prevent the solution from foaming over during use. A practice test run is desirable.

(d) Acidified water. To 100 ml distilled water add 3 drops of 5 N hydrochloric acid.

(e) Potassium dichromate solution, 0.01000 N. Dissolve 0.4904 g anhydrous potassium dichromate, primary standard grade, in distilled water and dilute to 1.000 liter using a volumetric flask.

(f) Starch indicator solution. Prepare a thin paste of 1 g of soluble starch in a few milliliters of distilled water. Bring 200 ml of distilled water to a boil, remove from heat, and stir in the starch paste. Prepare fresh before each use.

(g) Potassium iodide, reagent grade.

(h) Sodium thiosulfate solution, 0.1 N. Dissolve 25 g of sodium thiosulfate pentahydrate in freshly boiled and cooled distilled water and dilute to 1.0 liter. Add 5 ml chloroform as preservative and allow to age for 2 weeks. If turbidity develops, discard the solution.

(i) Sodium thiosulfate solution, 0.01 N. Dilute 100 ml of the aged, 0.1 N sodium thiosulfate solution to 1.000 liter with freshly boiled and cooled distilled water using a volumetric flask. Add 5 ml chloroform

as preservative and store in a glass-stoppered bottle. Standardize before use with 0.01 N potassium dichromate as follows: to 80 ml distilled water add with constant stirring 1 ml concentrated sulfuric acid, 20 ml 0.01 N potassium dichromate, and approximately 0.1 g of potassium iodide. Allow to stand in the dark for 6 minutes. Titrate with 0.01 N thiosulfate solution. Upon approaching the end point (brown color changing to yellowish green) add 1 ml starch indicator solution and continue titrating to the end point (blue to light green). Repeat the standardization procedure two more times. Calculate the average normality of sodium thiosulfate from the three titrations.

$$\text{Normality of sodium thiosulfate} = \frac{2.000}{\text{mls of sodium thiosulfate used}}$$

dure two more times. Calculate the average normality of sodium thiosulfate from the three titrations.

(j) Chlorine solution, approximately 100 ppm (100  $\mu\text{g}/\text{ml}$ ). Prepare by serial dilution of household bleach (approximately 50,000 ppm) or by dilution of strong chlorine water made by bubbling chlorine gas through cold distilled water. The diluted solution should contain approximately 100 ppm of free (available) chlorine. Prepare 1 liter.

### Standards

(a) Prepare a series of six 10-ml volumetric flasks containing 0.6 ml 0.005% methyl orange reagent, 8.0 ml distilled water, and 20  $\mu\text{l}$  of 5.0 N HCl. Using Eppendorf pipets, carefully pipet 0, 5, 10, 25, 50, and 100  $\mu\text{l}$  chlorine solution (approximately 100 ppm) into the respective flasks, holding the pipet tip beneath the surface. Gently mix and dilute to volume with distilled water. The manner of addition of chlorine standards to

working solutions is important. The methyl orange solution is reported [69] to be less bleached by a rapid addition of halogen without stirring than by slow addition with vigorous mixing.

(b) Immediately standardize the 100-ppm chlorine solution as follows: to a flask containing 1 g potassium iodide and 5 ml glacial acetic acid add 50 ml chlorine solution, swirling to mix. Titrate with 0.01 N sodium thiosulfate until the iodine color becomes a faint yellow. Add 1 ml of starch indicator solution and continue the titration to the end point (blue to colorless). Standardize two additional 50-ml portions of the 100-ppm chlorine solution. Average these three values to obtain the exact titer of the approximately 100-ppm chlorine solution, then calculate the amounts of free chlorine added to each of the six volumetric flasks. One milliliter of 0.01 N sodium thiosulfate equals 354.6  $\mu$ g of free chlorine. Compute the amounts of free chlorine added to each flask.

(c) Transfer the standards prepared as in (a) above to absorption cells and measure absorbance. Construct a standard curve by plotting absorbance versus micrograms of chlorine.

#### Sample treatment

(a) Place 10 ml of sampling solution in the fritted bubbler and draw a measured volume of air through the bubbler at a rate of 1-2 liters/min for 15 minutes. Transfer the solution to a 10-ml volumetric flask and dilute to volume, if necessary, with acidified water. Measure absorbance at 505 nm in 5-cm cells using distilled water as a reference.

(b) The volume of sampling solution, the concentration of methyl

orange in the sampling solution, the amount of air sampled, the size of the absorbing vessel, and the length of the photometer cell can be varied to suit the needs of the situation as long as proper attention is paid to the corresponding changes necessary in the standardization procedure.

### Calculations

$$\text{ppm chlorine in air} = \frac{0.001 \times \mu\text{g chlorine found} \times 344.37}{\text{liters of air sampled}}$$

(at 25 C and 760 mmHg).

For different temperatures and atmospheric pressures proper correction for air volume must be made as follows:

$$\text{ppm chlorine in air} = \frac{0.001 \times \mu\text{g chlorine found} \times \frac{ABC}{D}}{\text{liters of air sampled}}$$

where: A = 22.4  $\mu\text{l}$  chlorine/ $\mu\text{mol}$  chlorine at STP

$$B = \frac{\text{sampling temperature (K)}}{273 \text{ K}}$$

$$C = \frac{760 \text{ mmHg}}{\text{atmospheric pressure (mmHg)}}$$

$$D = 71 \mu\text{g chlorine}/\mu\text{mol chlorine}$$

### Interferences

Free bromine, which gives the same reaction, interferes in a positive direction. Manganese(III) and manganese(IV) in concentrations of 0.1 ppm or above also interfere positively. In the gaseous state, interference from sulfur dioxide is minimal but, in solution, negative interference from sulfur dioxide is significant. Nitrites impart an off-color orange to the

methyl orange reagent. Nitrogen dioxide interferes positively, reacting as 20% chlorine. Sulfur dioxide interferes negatively, decreasing the chlorine by an amount equal to one-third the sulfur dioxide concentration.

[64]

#### Sensitivity and Range

The procedure given is designed to cover the range of 5-100  $\mu\text{g}$  of free chlorine/100 ml sampling solution. For a 30-liter air sample, this corresponds to approximately 0.05-1.0 ppm in air, the optimum range.

#### Precision and Accuracy

Chlorine concentrations have been measured by this procedure with an average error of less than  $\pm 5\%$  of the amount present. [64]

#### Storage

The color of the sampled solutions is stable for 24 hours if protected from direct sunlight, although certain agents [iron(III)] may induce kinetic responses resulting in a slow color change.

X. APPENDIX III  
RECOMMENDED RESEARCH

There is clear need for information in the following areas in order to set a limit for chlorine which is more reliably based on demonstrated dose-response relationships:

(a) Documentation of human response in the range between perception of odor and marked discomfort at concentrations below 5.0 ppm, with assessment of possible increasing tolerance effects with increasing duration of exposure.

(b) Epidemiologic studies correlating long-term effects with chlorine exposures in excess of 0.3 ppm.

(c) Studies correlating the combined effects of cigarette smoking and chlorine exposure.

(d) Animal studies of myocardial response to graded exposures to chlorine.

(e) Animal studies of the effect of low doses of chlorine on the animals' capacity to resist infection.

(f) Animal studies of the effects of mixtures of chlorine with other chemicals.

XI. APPENDIX IV  
MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material shall be provided in the appropriate block of the Material Safety Data Sheet (MSDS).

The product designation is inserted in the block in the upper left corner of the first page to facilitate filing and retrieval. Print in upper case letters as large as possible. It should be printed to read upright with the sheet turned sideways. The product designation is that name or code designation which appears on the label, or by which the product is sold or known by employees. The relative numerical hazard ratings and key statements are those determined by the rules in Chapter V, Part B, of the NIOSH publication, An Identification System for Occupationally Hazardous Materials. The company identification may be printed in the upper right corner if desired.

(a) Section I. Product Identification

The manufacturer's name, address, and regular and emergency telephone numbers (including area code) are inserted in the appropriate blocks of Section I. The company listed should be a source of detailed backup information on the hazards of the material(s) covered by the MSDS. The listing of suppliers or wholesale distributors is discouraged. The trade name should be the product designation or common name associated with the material. The synonyms are those commonly used for the product, especially formal chemical nomenclature. Every known chemical designation or

competitor's trade name need not be listed.

(b) Section II. Hazardous Ingredients

The "materials" listed in Section II shall be those substances which are part of the hazardous product covered by the MSDS and individually meet any of the criteria defining a hazardous material. Thus, one component of a multicomponent product might be listed because of its toxicity, another component because of its flammability, while a third component could be included both for its toxicity and its reactivity. Note that a MSDS for a single component product must have the name of the material repeated in this section to avoid giving the impression that there are no hazardous ingredients.

Chemical substances should be listed according to their complete name derived from a recognized system of nomenclature. Where possible, avoid using common names and general class names such as "aromatic amine," "safety solvent," or "aliphatic hydrocarbon" when the specific name is known.

The "%" may be the approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range or maximum amount, ie, "10-40% vol" or "10% max wt" to avoid disclosure of trade secrets.

Toxic hazard data shall be stated in terms of concentration, mode of exposure or test, and animal used, ie, "100 ppm LC50-rat," "25 mg/kg LD50-skin-rabbit," "75 ppm LC man," or "permissible exposure from 29 CFR 1910.1000," or, if not available, from other sources of publications such as the American Conference of Governmental Industrial Hygienists or the

American National Standards Institute Inc. Flammable or reactive data could be flash point, shock sensitivity, or other brief data indicating nature of the hazard.

(c) Section III. Physical Data

The data in Section III should be for the total mixture and should include the boiling point and melting point in degrees Fahrenheit (Celsius in parentheses); vapor pressure, in conventional millimeters of mercury (mmHg); vapor density of gas or vapor (air = 1); solubility in water, in parts/hundred parts of water by weight; specific gravity (water = 1); percent volatiles (indicated if by weight or volume) at 70 Fahrenheit (21.1 Celsius); evaporation rate for liquids or sublimable solids, relative to butyl acetate; and appearance and odor. These data are useful for the control of toxic substances. Boiling point, vapor density, percent volatiles, vapor pressure, and evaporation are useful for designing proper ventilation equipment. This information is also useful for design and deployment of adequate fire and spill containment equipment. The appearance and odor may facilitate identification of substances stored in improperly marked containers, or when spilled.

(d) Section IV. Fire and Explosion Data

Section IV should contain complete fire and explosion data for the product, including flash point and autoignition temperature in degrees Fahrenheit (Celsius in parentheses); flammable limits, in percent by volume in air; suitable extinguishing media or materials; special firefighting procedures; and unusual fire and explosion hazard information. If the product presents no fire hazard, insert "NO FIRE HAZARD" on the line

labeled "Extinguishing Media."

(e) Section V. Health Hazard Information

The "Health Hazard Data" should be a combined estimate of the hazard of the total product. This can be expressed as a TWA concentration, as a permissible exposure, or by some other indication of an acceptable standard. Other data are acceptable, such as lowest LD50, if multiple components are involved.

Under "Routes of Exposure," comments in each category should reflect the potential hazard from absorption by the route in question. Comments should indicate the severity of the effect and the basis for the statement, if possible. The basis might be animal studies, analogy with similar products, or human experiences. Comments such as "yes" or "possible" are not helpful. Typical comments might be:

Skin Contact--single short contact, development of burns; prolonged or repeated contact, pain and tissue destruction.

Eye Contact--burning and tearing

"Emergency and First Aid Procedures" should be written in lay language and should primarily represent first aid treatment that could be provided by paramedical personnel or individuals trained in first aid.

Information in the "Notes to Physician" section should include any special medical information which would be of assistance to an attending physician including required or recommended preplacement and periodic medical examinations, diagnostic procedures, and medical management of overexposed workers.

(f) Section VI. Reactivity Data

The comments in Section VI relate to safe storage and handling of hazardous, unstable substances. It is particularly important to highlight instability or incompatibility to common substances or circumstances such as water, direct sunlight, steel or copper piping, acids, alkalies, etc. "Hazardous Decomposition Products" shall include those products released under fire conditions. It must also include dangerous products produced by aging, such as peroxides in the case of some ethers. Where applicable, shelf life should also be indicated.

(g) Section VII. Spill or Leak Procedures

Detailed procedures for cleanup and disposal should be listed with emphasis on precautions to be taken to protect workers assigned to cleanup detail. Specific neutralizing chemicals or procedures should be described in detail. Disposal methods should be explicit including proper labeling of containers holding residues and ultimate disposal methods such as "sanitary landfill," or "incineration." Warnings such as "comply with local, state, and federal antipollution ordinances" are proper but not sufficient. Pertinent specific local requirements shall be identified.

(h) Section VIII. Special Protection Information

Section VIII requires specific information. Statements such as "Yes," "No," or "If necessary" are not informative. Ventilation requirements should be specific as to type and preferred methods. Respirators shall be specified as to type and NIOSH or US Bureau of Mines approval class, ie, "Supplied air," "Organic vapor canister," "Suitable for dusts not more toxic than lead," etc. Protective equipment must be

specified as to type and materials of construction.

(i) Section IX. Special Precautions

"Precautionary Statements" shall consist of the label statements selected for use on the container or placard. Additional information on any aspect of safety or health not covered in other sections should be inserted in Section IX. The lower block can contain references to published guides or in-house procedures for handling and storage. Department of Transportation markings and classifications and other freight, handling, or storage requirements and environmental controls can be noted.

(j) Signature and Filing

Finally, the name and address of the responsible person who completed the MSDS and the date of completion are entered. This will facilitate correction of errors and identify a source of additional information.

The MSDS shall be filed in a location readily accessible to workers potentially exposed to the hazardous material. The MSDS can be used as a training aid and basis for discussion during safety meetings and training of new employees. It should assist management by directing attention to the need for specific control engineering, work practices, and protective measures to ensure safe handling and use of the material. It will aid the safety and health staff in planning a safe and healthful work environment and in suggesting appropriate emergency procedures and sources of help in the event of harmful exposure of employees.

--


## MATERIAL SAFETY DATA SHEET

I PRODUCT IDENTIFICATION		
MANUFACTURER'S NAME	REGULAR TELEPHONE NO. EMERGENCY TELEPHONE NO.	
ADDRESS		
<b>TRADE NAME</b>		
<b>SYNONYMS</b>		
II HAZARDOUS INGREDIENTS		
MATERIAL OR COMPONENT	%	HAZARD DATA
III PHYSICAL DATA		
BOILING POINT (760 MM HG)		MELTING POINT
SPECIFIC GRAVITY (H <sub>2</sub> O=1)		VAPOR PRESSURE
VAPOR DENSITY (AIR=1)		SOLUBILITY IN H <sub>2</sub> O % BY WT
% VOLATILES BY VOL		EVAPORATION RATE (BUTYL ACETATE=1)
APPEARANCE AND ODOR		

<b>IV FIRE AND EXPLOSION DATA</b>				
FLASH POINT (TEST METHOD)		AUTOIGNITION TEMPERATURE		
FLAMMABLE LIMITS IN AIR, % BY VOL.		LOWER		UPPER
EXTINGUISHING MEDIA				
SPECIAL FIRE FIGHTING PROCEDURES				
UNUSUAL FIRE AND EXPLOSION HAZARD				
<b>V HEALTH HAZARD INFORMATION</b>				
HEALTH HAZARD DATA				
ROUTES OF EXPOSURE				
INHALATION				
SKIN CONTACT				
SKIN ABSORPTION				
EYE CONTACT				
INGESTION				
EFFECTS OF OVEREXPOSURE				
ACUTE OVEREXPOSURE				
CHRONIC OVEREXPOSURE				
EMERGENCY AND FIRST AID PROCEDURES				
EYES				
SKIN				
INHALATION				
INGESTION				
NOTES TO PHYSICIAN				

<b>VI REACTIVITY DATA</b>	
CONDITIONS CONTRIBUTING TO INSTABILITY	
INCOMPATIBILITY	
HAZARDOUS DECOMPOSITION PRODUCTS	
CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION	
<b>VII SPILL OR LEAK PROCEDURES</b>	
STEPS TO BE TAKEN IF MATERIAL IS RELEASED OR SPILLED	
NEUTRALIZING CHEMICALS	
WASTE DISPOSAL METHOD	
<b>VIII SPECIAL PROTECTION INFORMATION</b>	
VENTILATION REQUIREMENTS	
SPECIFIC PERSONAL PROTECTIVE EQUIPMENT	
RESPIRATORY (SPECIFY IN DETAIL)	
EYE	
GLOVES	
OTHER CLOTHING AND EQUIPMENT	

**IX SPECIAL PRECAUTIONS**

PRECAUTIONARY  
STATEMENTS

OTHER HANDLING AND  
STORAGE REQUIREMENTS

PREPARED BY \_\_\_\_\_

ADDRESS \_\_\_\_\_

DATE \_\_\_\_\_

## XII. APPENDIX V

### PULMONARY FUNCTION ABBREVIATIONS USED BY AUTHORS

DLCO	diffusing capacity of lung for carbon monoxide
FEV	forced expiratory volume
FEV 0.5	forced expiratory volume at 0.5 second
FEV 1	forced expiratory volume at 1 second
FEV 3	forced expiratory volume at 3 seconds
FRC	functional residual capacity
FVC	forced vital capacity
Glaw	lower airway conductance in liter/sec/cm H <sub>2</sub> O/liter
MMF	maximum midexpiratory flow rate
MVV	maximum voluntary ventilation
PaCO <sub>2</sub>	partial pressure of carbon dioxide in arterial blood
PaO <sub>2</sub>	partial pressure of oxygen in arterial blood
PO <sub>2</sub>	partial pressure of oxygen
PEFR	peak expiratory flow rate
Raw	airway resistance
RV	residual volume
TLC	total lung capacity
VC	vital capacity

## XIII. SELECTED TABLES AND FIGURE

TABLE XIII-1

## CHEMICAL AND PHYSICAL PROPERTIES OF CHLORINE

---

Molecular weight	70.906 g/mole
Vapor pressure, 21 C	6.0 kg/sq cm gage (85.3 psig)
Specific volume, 21 C, 1 atm	0.34 liters/g
Boiling point, 1 atm	-34 C
Freezing point (bp), 1 atm	-101 C
Specific gravity of gas at 0 C, 1 atm (air = 1)	2.49
Specific gravity of liquid at 20 C	1.41
Density of gas at 0 C, 1 atm	3.214 g/liter
Density of liquid at 0 C, 3.65 atm	1468 g/liter
Critical temperature	144 C
Critical pressure	78.64 kg/sq cm absolute (76.1 atm)
Latent heat of vaporization at bp	68.8 calories/g
Solubility in water at 20 C, 1 atm	7.30 g/liter
Color of gas	Yellowish green
Color of liquid	Clear amber
Flammability	Nonflammable
Reactivity	Highly reactive
Odor	Disagreeable, strong, suffocating, pungent, irritating, characteristic

---

Adapted from reference 105

TABLE XIII-2

## OCCUPATIONS WITH POTENTIAL EXPOSURE TO CHLORINE

---

Aerosol propellant makers	Iron detinners
Alkali salt makers	Iron dezinkers
Aluminum purifiers	Laundry workers
Benzene hexachloride makers	Methyl chloride makers
Bleachers	Paper bleachers
Bleaching powder makers	Petroleum refinery workers
Bromine makers	Phosgene makers
Broommakers	Photographic workers
Carpetmakers	Pulp bleachers
Chemical synthesizers	Rayon makers
Calcium chloride makers	Refrigerant makers
Chlorinated solvent makers	Rubber makers
Chlorinated hydrocarbon insecticide makers	Sewage treaters
Chlorine workers	Silver extractors
Colormakers	Sodium hydroxide makers
Disinfectant makers	Submarine workers
Dyemakers	Sugar refiners
Ethylene glycol makers	Sulfur chloride makers
Ethylene oxide makers	Swimming pool maintenance workers
Flour bleachers	Tetraethyl lead makers
Fluorocarbon makers	Textile bleachers
Gasoline additive workers	Tin recovery workers
Gold extractors	Vinyl chloride makers
Inkmakers	Vinylidene chloride makers
Iodine makers	Water treaters
	Zinc chloride makers

---

Adapted from reference 3

TABLE XIII-3

## RECOMMENDED ALKALINE SOLUTIONS FOR ABSORBING CHLORINE

Chlorine Container Capacity lb (net)	<u>Caustic Soda</u>		<u>Soda Ash</u>		<u>Hydrated Lime*</u>	
	100% lb	Water gal	lb	Water gal	lb	Water gal
100	125	40	300	100	125	125
150	188	60	450	150	188	188
2000	2500	800	6000	2000	2500	2500

\*Hydrated lime solution must be continuously and vigorously agitated during chlorine absorption.

From reference 130

TABLE XIII-4

SURVIVAL OF GUINEA PIGS INOCULATED WITH MYCOBACTERIUM TUBERCULOSIS,  
WITH AND WITHOUT EXPOSURE TO CHLORINE

Average Days (and Range) Survived				
Inoculation Route	TB Exposure Only	TB Exposure Before Chlorine	TB Exposure After Chlorine	Chlorine Exposure Only
Subcutaneous	200 (142-275)	145 (110-175)	123 (49-177)	
Conjunctival	250 (220-280)	205 (160-250)	190 (172-208)	
Intra-tracheal	99 (84-114)	70 (48-107)	-----	
Ganglionic	215 (193-232)	203 (110-264)	139 (127-169)	
None	-----	-----	-----	>300

From reference 59

TABLE XIII-5  
 CHLORINE EXPOSURE-EFFECT DATA  
 --HUMAN STUDIES

Reference	Exposure Concentration (ppm)	Number Exposed	Route of Administration	Effects
16	0.027 (mean) 0.014-0.054 (range)	20	Inhalation	Tickling of nose
16	0.058 (mean) 0.04-0.097 (range)	20	"	Tickling in throat
16	0.06-0.2	3	Inhalation of concentrations increasing from 0.0 to 1.3 ppm over 50 minutes	Itching in the nose
17	0.09	10	Inhalation	Tickling and stinging in the nose (4), cough (1), dryness in throat (1)
17	0.2	13	"	Slight tickling in the nose and throat (7), cough (1), sensations in the ocular conjunctiva (3)
17	0.3	4	Inhalation of concentrations increasing from 0.0 to 1.8 ppm	Stinging in the throat (3)
17	0.36	4	"	Sensation of choking (1)

TABLE XIII-5 (Continued)  
 CHLORINE EXPOSURE-EFFECT DATA  
 --HUMAN STUDIES

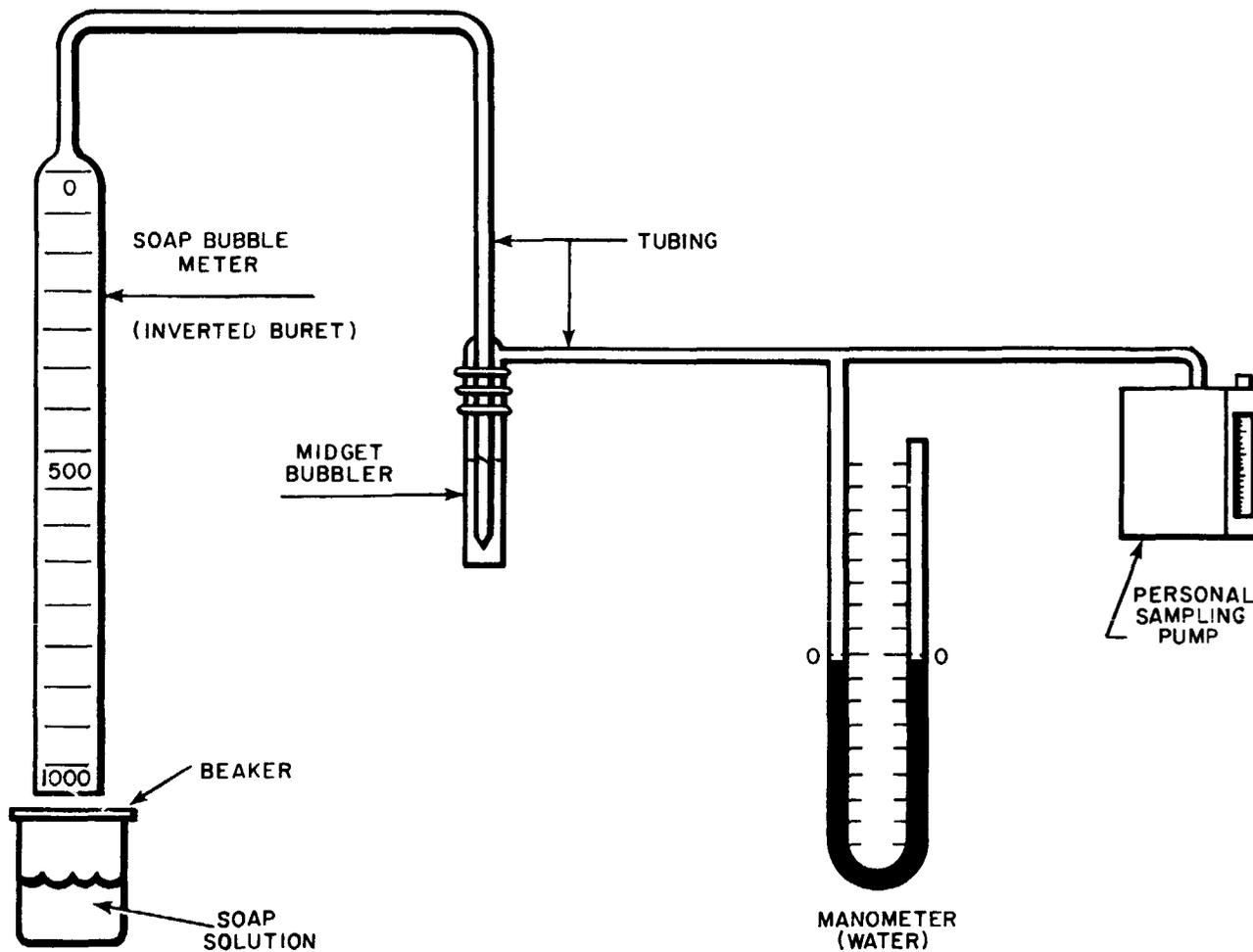
Reference	Exposure Concentration (ppm)	Number Exposed	Route of Administration	Effects
16	0.452 (mean) 0.35-0.72 (range)	19	Inhalation	Burning of conjunctiva, pain after 15 minutes.
16	0.5	3	Inhalation of concentrations increasing from 0.0 to 1.3 ppm over 50 minutes	Cough
16	1.0	3	Inhalation of concentrations increasing from 0.0 to 1.0 ppm over 35 minutes	Headache
17	1.0	10	Inhalation	Tickling and stinging in the nose (6), scratchiness and dryness in the throat (4), dull sensation in the teeth and a slight metallic taste (1), headache and pressure, burning of ocular conjunctiva and outer skin, coughing, constriction of breathing (1)
16	1.0-1.3	1	Inhalation of concentrations increasing from 0.0 to 1.0 ppm over 35 minutes	Severe shortness of breath and cough with violent headache

TABLE XIII-5 (Continued)  
 CHLORINE EXPOSURE-EFFECT DATA  
 --HUMAN STUDIES

Reference	Exposure Concentration (ppm)	Number Exposed	Route of Administration	Effects
38	1.3	1	Inhalation for 7 minutes	Burning sensation in eyes and nose
17	1.4	4	Inhalation of concentrations increasing from 0.0 to 1.8 ppm	Neck pain, substernal pain, conjunctival irritation (1), headache (1)
38	2.5	1	Inhalation for 5-16 minutes	Severe burning in eyes, itching in mouth and throat, nasal congestion, heavy coughing, breathing pains
38	3.5-4.0	1	Inhalation	Immediate burning of eyes, nasal congestion

Figure XIII-1

CALIBRATION SETUP FOR PERSONAL SAMPLING PUMP WITH MIDGET BUBBLER



DEPARTMENT OF  
HEALTH, EDUCATION AND WELFARE  
PUBLIC HEALTH SERVICE  
CENTER FOR DISEASE CONTROL  
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH  
ROBERT A. TAFT LABORATORIES  
4676 COLUMBIA PARKWAY, CINCINNATI, OHIO 45226



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