CONTROL TECHNOLOGY FOR ETHYLENE OXIDE STERILIZATION IN HOSPITALS

Vincent D. Mortimer, Jr.
Sharon L. Kercher

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health (NIOSH)
Division of Physical Sciences and Engineering
Cincinnati, Ohio 45226

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PREFACE

Under the authority of the Occupational Safety and Health Act of 1970 (Public Law 91-596), the National Institute for Occupational Safety and Health (NIOSH) conducts research to prevent occupational health problems through the application of control technology in the workplace. The goal of this program is to assist in preventing hazardous exposures to workers and to document successful approaches and applications of control measures.

In 1982-83, the Engineering Control Technology Branch, Division of Physical Sciences and Engineering, NIOSH, conducted a feasibility study of the use of engineering controls in hospitals. As a result of research recommendations from that study and in response to the hospitals' need to control worker exposure to ethylene oxide, a study of the control of ethylene oxide (EtO) emissions from sterilizers in the hospital setting was conducted from 1984-86. The goals of this study were to evaluate and document effective engineering controls which selected hospitals have implemented, and to disseminate useful information and practicable recommendations on effective methods for controlling occupational EtO exposure.

This report examines control methods and systems for EtO sterilization in hospitals. Nine sterilizer control systems were evaluated in eight hospitals during week-long, industrial hygiene surveys. Individual in-depth survey reports (listed in Appendix C) which include more detailed information on specific characteristics of each control system are available from the National Technical Information Service, Port Royal Road, Springfield, Virginia 22161.

As a follow-up to this report, a hazard and operability (HAZOP) study was conducted on a model sterilizer installation. The HAZOP was performed primarily to evaluate the potential for catastrophic release of ethylene oxide due to failure of one or more sterilizer components, installation inadequacies, or worker actions. It has the advantage of looking at what could happen, whereas the field studies focus on conditions present at the time of the survey. The recommendations of the HAZOP complement those of the field study and are summarized as an appendix to this report.

ABSTRACT

This report examines control methods and systems for EtO sterilization in hospitals. Nine sterilizer control systems were evaluated in eight hospitals during week-long, in-depth surveys. Three emission sources typically account for most of the EtO routinely released into the work environment. First, most of the EtO gas mixture from the chamber is released to the indoor atmosphere at the air gap located at the connection of the drain to the outlet of the water sealed vacuum pump. Second, the opening of the sterilizer door at the completion of the cycle may result in a very short high exposure to the sterilizer operator followed by an increase in the workroom EtO concentration. Third, the load transfer procedure provides the closest contact with EtO for the sterilizer operator: pulling the load from the sterilizer, transporting the load to the aerator, and inserting the load into the aerator. EtO exposures from hospital sterilizers can be controlled to not exceed a ceiling limit of 5 ppm and to average less than 0.1 ppm for a full shift. All but one of the hospitals surveyed in this study had short-term exposures less than 2 ppm and full-shift exposures less than 0.1 ppm.

The extent of control needed by a hospital will depend on a number of factors such as the composition and size of the sterilized load, the location of the sterilizer and the time constraints on sterilization, the type of sterilizer and the types of controls selected, and the level to which EtO exposures are to be controlled. In-chamber aeration, which substantially eliminates any exposure, is the best control. When it is not possible to fully use in-chamber aeration, cycle modifications, local ventilation above the sterilizer door, and a ventilated enclosure around the sterilizer drain are the next most effective techniques for reducing exposures. General ventilation did not seem to be as important as other control techniques in controlling EtO exposures.

ABBREVIATIONS

AAMI	Association for the Advancement of Medical Instrumentation
ASHRAE	American Society of Heating, Refrigerating, and Air Conditioning Engineers
BI	Biological indicator
cfm	cubic feet per minute
cfm/ft^2	cubic feet per minute per square foot
CS	Central service
dc	Door-cracked period
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
do	Door-open period
EtO	Ethylene oxide
ft^3	Cubic feet
$ft^3/hour$	Cubic feet per hour
ft^3/min	Cubic feet per minute
ft/min	Feet per minute
HAZOP	Hazard and operability study
IPPB	Intermittent, positive pressure, breathing
IR	Infrared
LEV	Local exhaust ventilation
LOD	Limit of detection
LT	Load transfer
mL/min	Milliliters of air per minute
NFPA	National Fire Prevention Association
NIOSH	National Institute for Occupational Safety and Health
OSHA	Occupational Safety and Health Administration
PEL	Permissible exposure limit
ppm	Parts per million parts of air
ppm-min	Parts per million minutes
SCBA	Self-contained breathing apparatus
STEL	Short-term exposure limit

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INTRODUCTION

Ethylene oxide (EtO) is a currently indispensable sterilant for certain medical items in health-care facilities. However, in addition to being bactericidal, it is also potentially hazardous to workers. Acute exposures may cause irritation of the eyes, nose, and throat, burns of the skin, and allergic sensitization.[1] Animal toxicity studies have shown EtO to be a mutagen and a carcinogen,[2] which may have implications for chronic, low-level exposures. Some studies of exposed workers have indicated increased mutagenic activity in human cells, and an increase in the incidence of leukemia and adverse reproductive effects.[3]

Much information on EtO is available from many sources; but no comprehensive study had been done on EtO emissions from hospital sterilizers and the control of hospital worker exposures. Many recommendations have been based on anecdotal observations, rather than conclusions drawn from industrial hygiene sampling and engineering measurements made in a connected series of surveys. This study was designed to assess the relative importance of the various EtO emission sources associated with gas sterilizers, and to determine, to the extent possible, the effectiveness of certain control measures on limiting health-care worker exposure to EtO.

THE NEED FOR CONTROLS

Although less than 2 percent of all EtO produced in the United States is used as a sterilant (most of it is used in the chemical industry),[4,5] this small usage probably results in most of the employee exposures to EtO.[6] The Occupational Safety and Health Administration (OSHA) has reported that EtO is used as a sterilant in 7,700 sterilizers in 6,300 hospitals.[7] It has been estimated that approximately 75,000 U.S. health-care workers employed in sterilization operations are potentially exposed to EtO, and an additional 25,000 other employees working in adjacent areas may be incidentally exposed because inadequate control measures allow the spread of EtO.[4]

Hospitals and other health-care facilities routinely use EtO as an agent to sterilize medical devices and equipment. Its use is especially important in the sterilization of heat-sensitive items which cannot be sterilized by steam. There is no suitable substitute at the present time for EtO sterilization within hospitals, and, therefore, controlling exposures by other means is essential.

ETHYLENE OXIDE - BACKGROUND AND STERILIZATION CHARACTERISTICS

Chemically, EtO (C_2H_4O) is a polar molecule with a molecular weight of 44. At atmospheric pressure EtO has a boiling point of 5.3°F (10.7°C); and, thus, it is a liquid in cartridges and cylinders, but a gas in the sterilizer and in the workroom atmosphere. At standard temperature and pressure, the vapor density of EtO is 1.5. Ethylene oxide is flammable in air at concentrations above 3 percent or 30,000 parts EtO per million parts of air (ppm), and has a relatively high odor threshold of about 700 ppm.[1] EtO is soluble in water, organic solvents, and some organic solids; and it readily diffuses and penetrates most materials.

EtO was first used as a fumigant and pesticide early in this century. In 1929 its bactericidal properties were recognized by H. Schrader and E. Bossert.[9] A "Method of Sterilization" using EtO was patented by P. M. Gross and L. F. Dixon in 1937. C. L. Griffith and L. A. Hall patented a sterilization process using EtO in 1940 and 1943.[8] Existing steam autoclaves were used to first draw a vacuum on the chamber, then pure EtO was injected to sterilize the items. Beginning in 1949, C. R. Phillips and S. Kaye published a series of articles defining the four parameters necessary for EtO sterilization.[9] With the introduction of commercially available automatic equipment, EtO sterilization gained general acceptance in hospitals and industry. As medical technology advanced, the number of items which could not be sterilized with steam (plastics, rubber, drills, and implants) also grew, thereby increasing the need for EtO sterilization.

Four parameters affect the ability of EtO to sterilize products: temperature, concentration, humidity, and time. Most routine hospital sterilization is done at 120 to 140°F, however, sterilization of particularly heat-sensitive items can be performed at 100 to 105°F. Research has determined that 450 mg/liter is the minimum EtO concentration necessary for sterility. To be sure there is enough EtO, most sterilizers use EtO concentrations of 600 to 1,100 mg/liter. The proper amount of humidity is necessary for the sterilization to be effective. The amount of time required for sterilization depends on the temperature, taking less than 2 hours at approximately 130°F and over 5 hours at approximately 100°F.[9]

Because EtO is absorbed into the materials, an appropriate aeration time is required to allow the residual EtO to be released. Standard use items require 12 hours at 120°F. Implants and specialty items require longer aeration periods, and the manufacturer's instructions are followed.

Gas sterilizers have changed much in the past few years. A variety of emissions controls have been developed as more has been learned about the hazards of EtO.

ETHYLENE OXIDE TOXICITY

Ethylene oxide is a toxic chemical with acute and chronic health effects. EtO is a carcinogen in animals and a suspected carcinogen in humans.[3,6,10] It is an established mutagen in animal test systems and is associated with chromosomal aberrations and sister chromatid exchanges in humans.[2,11,12] Adverse reproductive effects, such as fetotoxicity and dominant-lethal mutation, have been demonstrated in several animal species,[6,13,14] and EtO exposure has been linked to spontaneous abortions and gynecological disorders in humans.[15,16] Additional evidence is now available which suggests that EtO exhibits a dose-rate effect.[17] This section summarizes many of the major toxicological investigations of EtO.

Acute Health Effects

Inhalation exposure to very high concentrations of EtO (600 ppm for 8 hours or 6,000 ppm for 12 minutes) is likely to result in severe injury or death. Individual response to particular exposure levels will vary. Immediate effects include watering eyes, salivation, nasal discharge, and shortness of breath. Exposure to more than a few hundred ppm for more than a few hours may include such delayed effects as: nausea, vomiting, diarrhea, convulsions, headache, drowsiness, dizziness, bronchitis, cardiac abnormalities, and possible death from secondary lung infection or systemic poisoning. Exposure to higher concentrations of more than a few thousand ppm for more than a few hours may cause death from fluid collecting in the lungs.[1] Exact exposure values for the onset of specific health effects in humans are not known because experiments have not been conducted on people and the values must be estimated from animal data.

Dermal exposure to concentrated liquid EtO may result in sensitization, edema, and frostbite. Dilute aqueous solutions of EtO may produce chemical burns and blisters. A small quantity of moisture may increase the irritant effects of

EtO, but copious rinsing of exposed skin is recommended if EtO comes in contact with the skin.[1]

Carcinogenicity

The potential carcinogenicity of EtO was not evident until the mid- to late-1970's. Since then, several important studies have established EtO as a carcinogen in animals.

A 2-year chronic inhalation bioassay study was conducted at the Bushy Run Research Center with male and female Fischer 344 rats exposed to EtO at concentrations of 10, 33, or 100 ppm for 6 hours per day, 5 days per week. The study reported a dose-related increase in the incidence of mononuclear cell leukemia for the exposed female rats. Exposed males experienced a dose-related increase in peritoneal mesotheliomas. A dose-related increase in cerebral gliomas was reported in both exposed males and females.[13]

NIOSH also conducted a 2-year inhalation study (Lynch et al.[18]) with male Fischer 344 rats and Cynomolgus monkeys, with exposures to EtO at concentrations of 50 or 100 ppm for 7 hours per day, 5 days per week. There was also a control group which received no experimental exposure to EtO. The NIOSH study confirmed the findings of the Bushy Run study, reporting dose-related increases in the incidence of mononuclear cell leukemia, peritoneal mesothelioma, and cerebral glioma in the exposed rats.

Intragastric administration of EtO to female Sprague-Dawley rats was performed by Dunkelberg during a 3-year study.[19] A dose-dependent increase in the incidence of squamous cell carcinomas of the forestomach was reported.

Hogstedt et al. have studied Swedish workers exposed to EtO.[20] In 1979, he reported 3 cases of leukemia in 230 workers in a factory sterilizing hospital equipment; the expected leukemia rate was 0.2 cases based on Swedish national statistics. Exposures were estimated to have been less than 30 ppm TWA.

Hogstedt et al. reported, also in 1979, significant increases in the mortality of workers in an EtO production plant.[21] The workers had at least 1 year of exposure to EtO and had worked in the plant for at least 10 years. Compared with Swedish national rates, the workers experienced 9 cases of stomach cancer where 3.4 were expected, 2 cases of leukemia with an expected rate of 0.14, and 12 cases of circulatory system disease with 6.3 cases expected. Similar increased mortality was observed for production workers with at least 10 years exposure to EtO and 20 years since first exposure.

In 1986, Hogstedt reported on a follow-up of the first two studies.[22] In the 733 workers, 8 cases of leukemia had occurred with 0.8 expected, and 6 cases of stomach cancer were reported with 0.65 cases expected, again based on Swedish national statistics. Workers were estimated to have been exposed to low EtO concentrations.

Morgan et al. reported a study of EtO exposed workers in a chemical production plant with no indication of increased leukemia mortality.[23] The study population was small, and based on national statistics, the expected leukemia

rate was 0.14. The authors indicated their study would have detected only a greater than 10-fold increase in the risk of leukemia. The study reported a significant increase in pancreatic cancer and Hodgkin's disease.

Based on the animal and human studies, both NIOSH and OSHA concluded that EtO is a carcinogen in animals and increases the risk of cancer deaths in humans.[6,10]

Mutagenicity and Cytogenicity

Ethylene oxide is an alkylating agent and causes mutation of cells and/or chromosomes. EtO is a mutagen in all microbial and plant test systems and in submammalian test systems tested so far, including barley, rice, wheat, viruses, Tradescantia, Salmonella typhimurium, Escherichia coli, Neurospora crasa, and Drosophila melanogaster.[2] EtO has also been shown to be mutagenic in mice, rabbits, and monkeys.

Generoso et al. reported the results of two studies in which male mice were injected intraperitoneally with EtO.[24] The first group was injected with 150 mg/kg EtO (maximum tolerated dose) then caged with female mice for 22 days. The second group was injected with either 30 or 60 mg/kg EtO for 5 days per week for 5 weeks, then caged with 3 female mice for 1 week. Dominant-lethal effects were observed in both groups, and a dose-related increase in the occurrence of heritable translocations was reported for the male offspring of exposed mice.

The NIOSH 2-year inhalation study with Cynomolgus monkeys exposed to 50 or 100 ppm for 7 hours per day, 5 days per week showed an increased frequency of chromosomal aberrations in peripheral lymphocytes and an increase in sister chromatid exchanges.[18] Yager and Benz reported a dose-related increase in sister chromatid exchanges in peripheral lymphocytes for New Zealand white rabbits exposed to EtO in an inhalation study in which rabbits were either not exposed (control group) or exposed to 10, 50, or 250 ppm for 6 hours per day, 5 days per week for 12 weeks.[26]

Studies of humans exposed to EtO concur with the results of animal studies. Abrahams reported a study of workers exposed to EtO in the manufacture and distribution of health-care products.[11] Exposure data indicated that workers were exposed to less than 50 ppm TWA (the OSHA PEL at the time), but that occasionally the 75 ppm short-term limit recommended by NIOSH had been exceeded. Results of the study showed an increase in chromosomal aberrations in peripheral lymphocytes and an increase in sister chromatid exchanges in the exposed workers.

In an extensive study of its workers, Johnson and Johnson found chromosomal aberrations and sister chromatid exchanges in workers exposed to 1 to 10 ppm EtO TWA.[6] One group of workers characterized by high exposures, 5 to 200 ppm, evidenced a persistent high rate of sister chromatid exchanges even after cessation of exposure.

Yager et al. studied a small group (14 workers) of hospital sterilizer operators.[12] Short-term exposures averaged 82 ppm over 3.5 minutes. The

workers exhibited an exposure related increase in the frequency of sister chromatid exchanges which was correlated with both EtO exposure and smoking.

Reproductive Effects

The reproductive effects of EtO have been studied in animals and to a lesser extent in humans. Ethylene oxide has been shown to decrease fertility and to cause malformed fetuses when administered at specific times during gestation in rats and mice. NIOSH studies demonstrated that EtO exposure adversely affected the sperm counts of monkeys.[25] Studies of humans have not been definitive, but increased spontaneous abortions and gynecological disorders have been indicated.

Snelling et al. exposed male and female Fischer 344 rats to 10, 33, 100 ppm EtO in air for 6 hours per day, 5 days per week for 12 weeks, and for 6 hours per day, 7 days per week during a 2-week mating period.[13] Females were then exposed per the latter regimen for days 0-19 of gestation. No effects were observed in the dams or litters of the groups exposed at 10 or 33 ppm. In the group exposed to 100 ppm, decreases in pups per liter and the number of implantation sites were statistically significant. In the same exposure group, the percentage of pregnant females and the percentage of males proven fertile were also lower than for unexposed controls.

LaBorde and Kimmel reported results of intravenous administration of EtO to CD-1 mice.[14] Pregnant mice received doses of 75 or 150 mg/kg once during gestation, either days 4-6, days 6-8, days 8-10, or days 10-12. All exposure groups experienced a reduction in mean fetal weight. Exposure groups for days 6-8 and days 10-12 experienced an increase in the number of malformed fetuses per litter. Defects in the thoracic and cervical skeleton were the most common.

Hardin et al. conducted a study of EtO teratogenicity in Sprague-Dawley rats and New Zealand white rabbits.[27] Both rats and rabbits were exposed to a concentration of 150 ppm EtO for 7 hours per day. Four groups experienced different exposure regimens ranging from a control group with no exposure to a group exposed to EtO for 3 weeks prior to breeding and on each day of gestation. Rats evidenced embryo and fetal toxicity as well as an increase in the incidence of reduced skeletal ossification. The study did not detect evidence of embryo or fetal toxicity or developmental defects in exposed rabbits.

In the NIOSH inhalation study, Lynch et al. reported that cynomolgus monkeys, exposed to 50 or 100 ppm EtO for 7 hours per day, 5 days per week, for 2 years experienced decreased sperm counts and decreased sperm motility.[25] However, there was no increase in the percentage of morphologically abnormal sperm nor were any adverse effects noticed in the control group.

Other studies have evaluated rabbits, rats, and mice with varying exposure regimens. OSHA concluded that EtO in doses which produce maternal toxicity is fetotoxic in rabbits, mice and rats, and teratogenic in mice when exposure occurs during gestation.[6] At lower doses, EtO is fetotoxic in rats when both sexes have been exposed prior to and during gestation, and when females

are exposed during gestation. EtO induces dominant-lethal effects in several species, and effects sperm counts and sperm motility in monkeys.

Studies of human reproduction are very difficult and complex. Hemminki et al. conducted a retrospective study of hospital sterilizing personnel in Finland. Women exposed to EtO during sterilization operations from 1951 to 1981 were evaluated for occurrence of spontaneous abortions and compared to other members of the sterilizing staff and other hospital personnel who were not knowingly exposed to EtO in the course of their work.[15]

No exposure data were available prior to 1976. Exposures for the 25 years prior to 1976 were estimated to be the same as the conditions in that year. Most workers seemed to have been exposed only once or twice per day with short-term peak exposures of 20 ppm or more. Hemminki found the rate of spontaneous abortion for the exposed sterilizing staff to be significantly higher than the unexposed hospital personnel. Hemminki's methodology has been criticized; however, NIOSH concluded the results are suggestive of adverse effects on human reproduction and cannot be discounted.[10]

A study conducted by Yakubova et al. in the Soviet Union reported on gynecological disorders of women working in EtO production plants.[16] The exposed workers experienced an increased incidence of gynecological disorders primarily diseases of the cervix and uterus. Exposed workers also had an increased frequency of spontaneous abortion and toxemia.

Dose-Rate Effect

When Generoso et al. exposed male mice for 4 days at 600 ppm per day for 3 hours, 3 times as many offspring died as for those exposed at 300 ppm for 6 hours; and at 1,200 ppm for 1.5 hours, 6 times as many died.[17] Although the exposure levels were at much higher concentrations than typically encountered in the workplace, it is clear the study shows a dose-rate effect for EtO. Such an effect raises additional concerns about short-term exposures above recommended limits, even if the full-shift TWA exposure limits are met.

Conclusions

Based on the available EtO toxicity information, NIOSH and OSHA have concluded: (1) EtO is a carcinogen in animals and represents a significant cancer risk for exposed humans, (2) EtO is a mutagen in animals and affects human DNA, and (3) EtO adversely affects animal reproduction and evidence suggests that human reproduction may also be adversely affected.[6,10]

EXPOSURE LIMITS

In 1971, OSHA set the permissible exposure limit (PEL) at 50 ppm for an 8-hour time-weighted average exposure.[6] The PEL was based on the 1968 American Conference of Governmental Industrial Hygienists, ACGIH, threshold limit value, TLV.[28] The TLV had been established on the basis of a limited animal inhalation study showing no adverse effects from exposures less than 50 ppm. No indications of the carcinogenicity of EtO were available.

NIOSH conducted a special occupational hazard review in 1977 and recommended an exposure ceiling limit of 75 ppm (15 minutes) in addition to the 50 ppm PEL.[4] NIOSH reported on several studies of EtO's potential as a mutagen and the chemical bonding of EtO with DNA. At that time, no data were available on EtO's carcinogenicity.

In 1979, ACGIH issued a notice of intended change (adopted in 1981) to lower the TLV for EtO from 50 ppm to 10 ppm, 8-hour TWA.[29] This action was based on the growing number of in vitro studies reporting mutagenic responses to EtO exposure and to Hogstedt's studies in Sweden.

NIOSH issued Current Intelligence Bulletin #35 in 1981 which reviewed the growing body of literature on the mutagenic, carcinogenic, and reproductive effects of EtO exposure.[2] NIOSH recommended that the OSHA standard be reevaluated. Also in 1981, ACGIH proposed a reduction in the TLV from 10 ppm to 5 ppm and listed EtO as a suspected carcinogen based on the results of the Bushy Run study.[30]

In 1982, ACGIH proposed to further reduce the TLV to 1 ppm (adopted in 1984).[31] Also in that year, OSHA announced its intent to reevaluate the standard and began the formal promulgation process.[7]

OSHA developed a risk assessment and conducted hearings on EtO in 1983. Based on an estimated 60,000 hospital workers exposed to EtO over a 45-year employment period, OSHA estimated that 3,800 to 6,500 excess deaths could be expected at the 50 ppm PEL. For the proposed 1 ppm PEL, OSHA estimated 72 to 138 excess deaths could be expected.[6] NIOSH testified that it considered the risk imposed by exposure to EtO even at the 0.1 ppm level to be too great, and that exposures "should be reduced through engineering controls to the lowest feasible level." NIOSH recommended to OSHA that a ceiling limit of 5 ppm should be established and not be achieved for more than 10 minutes in any working day, and that the PEL should be set less than 0.1 ppm.[10]

In June 1984, OSHA issued the new standard PEL of 1 ppm, 8-hour TWA.[6] However, the inclusion of a short-term exposure limit (STEL) became a matter of contention. In early 1985, OSHA announced its decision not to establish a STEL.[32] Later that year, Generoso et al. reported a study of male mice exposed to very high concentrations of EtO during short time periods.[17] The results showed an increase in dominant-lethal response with an increase in dose rate. J. Donald Millar, NIOSH Director, is quoted as saying the study's results "strengthen our previous conviction" that a short-term limit is needed.[33] In July 1986, the U.S. Court of Appeals for the District of Columbia Circuit ordered OSHA to either adopt a STEL or explain why it's interpretation of the evidence on exposure patterns resulted in terming a STEL "irrelevant" in controlling TWA exposures.[34] In April 1988, OSHA amended its existing standard for occupational exposure to ethylene oxide (29 CFR 1910.1047) by adopting an excursion limit for EtO of 5 ppm averaged over a sampling period of 15 minutes.[35]

Potential for Overexposure

Small amounts of EtO can cause significant exposures. For example, 1 gram of EtO can initially create a concentration of over 20 ppm in a room approximately 10 by 10 feet with an 8-foot ceiling. Even after being diluted with supposedly adequate general room ventilation for 8 hours, this single gram could cause an average exposure of approximately 1 ppm. Most of the single-dose cartridges now being used in table-top sterilizers contain either 100 or 134 grams of EtO. An 8.8-ft^3 sterilizer which uses a mixture of 12 percent (by weight) ethylene oxide in dichlorodifluoromethane contains approximately 150 grams of EtO during sterilization. A typical large supply cylinder of the 12:88 gas mixture contains over 7,000 grams of EtO when full.

It does not take a supply-line rupture or some other catastrophic event to cause high concentrations of EtO. One overexposure situation (observed by NIOSH, but not part of this study) caused by inadequate ventilation was sufficient to elicit symptoms of acute exposure.[36] Because EtO cannot be detected by its odor until concentrations exceed approximately 700 ppm, it is possible for workers to be overexposed without knowing it. It seems imperative that adequated controls be instituted and that exposures be periodically monitored to make sure the controls are working.

BASIS FOR CONTROL - PROCESSES AND EMISSION SOURCES

HOSPITAL STERILIZATION

Department Description

Ethylene oxide sterilization of medical equipment and surgical items may occur in one or more medical departments such as surgery or respiratory therapy. The most common practice is to centralize all sterilization in one area. Typically, hospitals refer to this department as central service; supply, processing, and distribution; or sterile reprocessing. In this report, the term central service (CS) will be used.

The CS department usually had four functional areas: decontamination, preparation and packaging, sterilization, and storage. Physical layout depended on the size of the hospital. In the hospitals studied, decontamination was always performed in a separate room as an infection control measure. The other three activities were sometimes performed in separate rooms, sometimes all were in the same room, and some hospitals had a combination of separate and common rooms.

A variety of sterilizers and locations was encountered, including small table-top units, large freestanding sterilizers enclosed in a cabinet, and large sterilizers recessed in a mechanical access room. A few were located in small rooms isolated from the rest of the department. And, although most of the sterilizers surveyed in this study were in a CS department, one hospital had a sterilizer in the surgery department and two sterilizers in the respiratory therapy department.

Nine of the sterilizers discussed in this report used a gas mixture of 12 percent EtO by weight in dichlorodifluoromethane delivered to the sterilizer from cylinders. The other two sterilizers used small, single-use cartridges of pure EtO. None of the sterilizers which use the glass ampoules of liquid EtO were surveyed in this study, because our walk-through surveys suggested that they represent a relatively small segment of installed sterilizers.

The number of workers in the sterilization departments of the surveyed hospitals ranged from four to ten, depending on the size of the hospital. Usually, one worker on each shift was responsible for operating both the steam and EtO sterilizers, one or two workers prepared and packaged the clean items, and one or two workers were assigned to decontaminate incoming soiled items. The sterilizer operator also assisted in the preparation and packaging of items when not operating the sterilizers. Duties were assigned on a rotating basis; and, depending on the number available, some workers might perform more than one, perhaps even all, of the mentioned tasks. Most of the workers were assigned to the day shift. Most of the hospitals ran a load during the latter

part of the day shift, so that the purge phase and transfer to the aerator would occur during the evening shift when fewer workers were present, yet not too late so that the load would be ready to be removed from the aerator when the day-shift workers arrived the next morning.

Process Description

Although practices varied from one hospital to another, the typical process started with the items arriving in the decontamination area. Here the items were cleaned, washed, and dried. The instruments were inspected and usually reassembled before being packaged.

For the heat-sensitive equipment and other items destined for gas sterilization, the items were packaged in special paperbacked polyethylene bags which were heat-sealed or wrapped in linen cloths and sealed with an EtO exposure indicator tape. The packaged items were then arranged on cart-racks or in baskets by the sterilizer operator and recorded on a sterilization log sheet. A biological indicator (BI) was placed in the load to provide quality assurance that sterilization had been achieved.

The sterilizer operator prepared the sterilizer, set the operating temperature, and checked the cycle pressure chart. Next, the load was inserted into the sterilizer chamber. This was either done by rolling a wheeled rack off a special cart, setting baskets on shelves fitted inside the chamber, or, in a few instances, placing individual items on the shelves. For the sterilizers which used the single-use cartridges, EtO, the operator placed the EtO gas cartridge inside the receptacle in the chamber then closed the door.

With the door securely closed, the cycle began. Cycle parameters varied depending on the items to be sterilized, the particular sterilizer, and any controls which had been added. The basic sterilization cycle was common to all of the sterilizers studied: initial chamber evacuation and humidification, charging the chamber with EtO, dwell period during which the actual sterilization took place, and chamber evacuation. Figures 1 to 3 illustrate the pressure and time relationships of the sterilizer cycles of interest. Most loads were sterilized at 130°F, and the dwell period was about 2-1/2 hours. Some items were particularly heat sensitive and were sterilized at 100°F with a dwell period of about 5 hours.

For all sterilizers, a buzzer indicated the completion of the basic cycle. At this point, one of three procedures were followed: (1) For the sterilizer with the in-chamber aeration feature, the operator simply allowed the cycle to continue uninterrupted to the aeration mode. (2) Some operators unloaded the sterilizer immediately and transferred the load to the aerator. (3) Other operators opened the sterilizer door a few inches and left the area for approximately 15 minutes before returning to unload the sterilizer. The latter practice is recommended by the manufacturers of the sterilizers studied and is known as the "door-cracked" period.

Once the door was fully opened, the load was removed from the sterilizer. Typically, the wheeled rack containing the sterilized goods is pulled from the

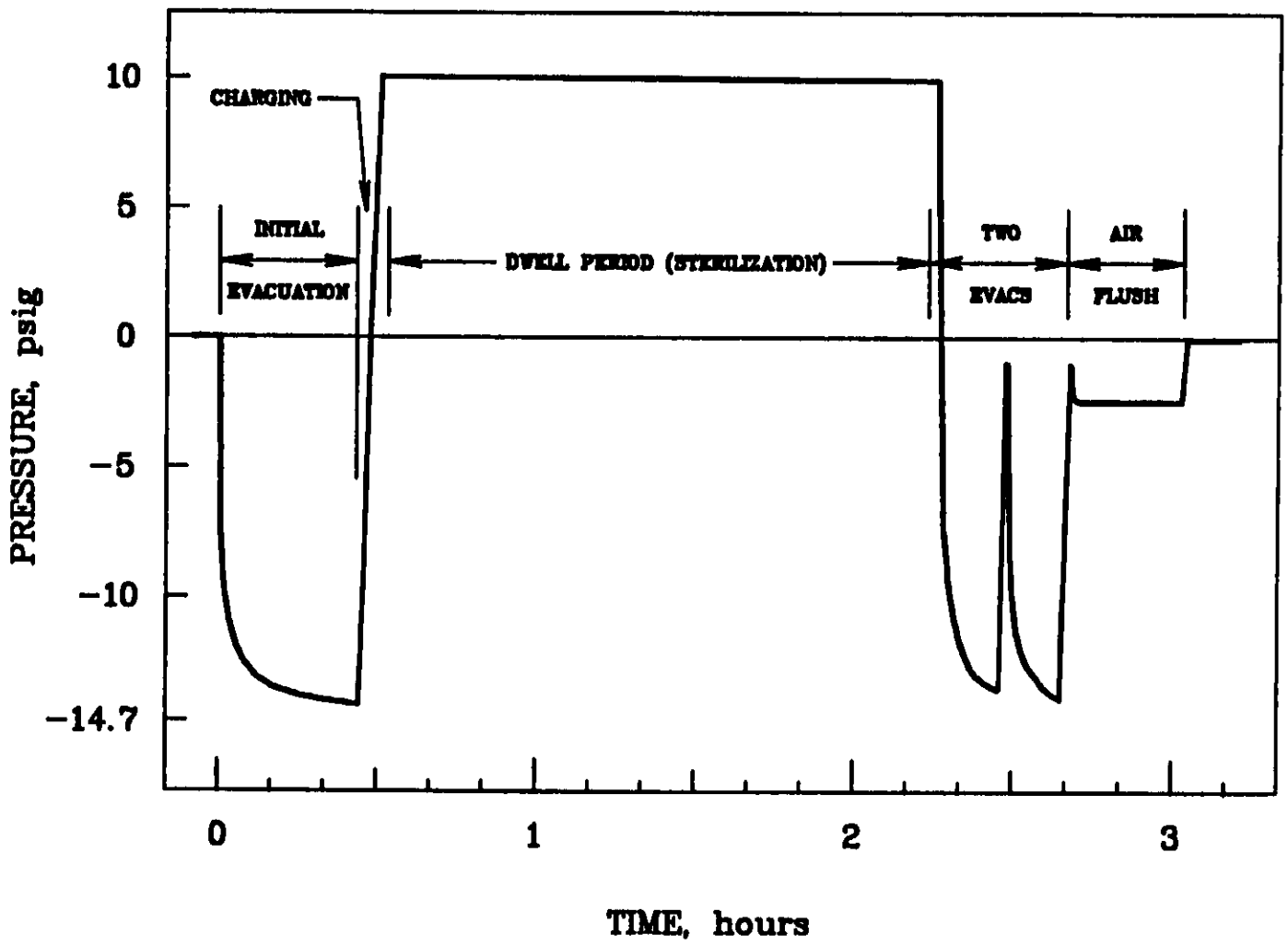

Figure 1. Typical cycle for a 12:88 sterilizer drawing two deep vacuum purges (approximately 0.9 atm) at the end of sterilization followed by one or more closed-door air flush periods.

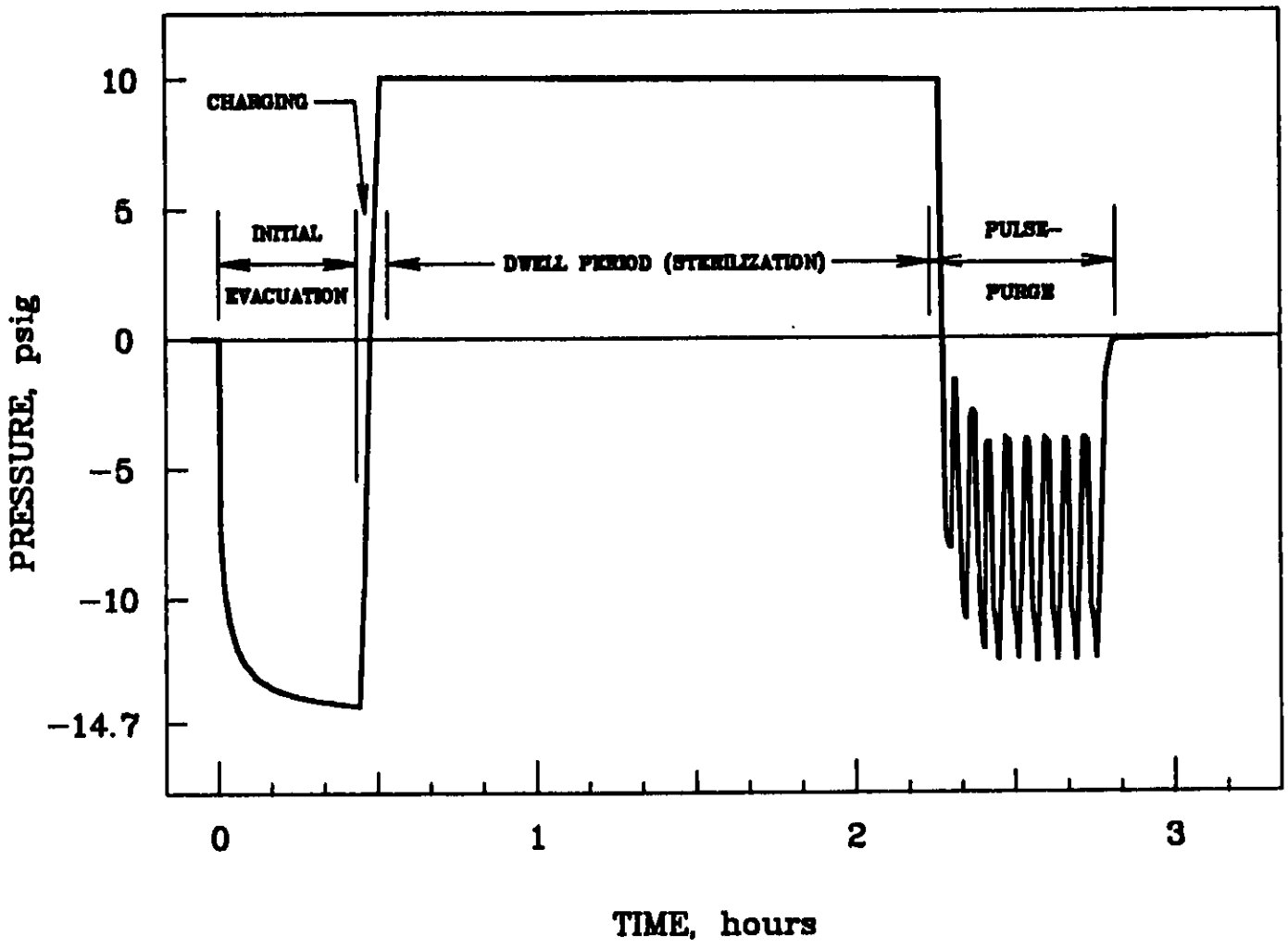

Figure 2. Typical cycle for a 12:88 sterilizer drawing "pulsating" purges (approximately 0.5 atm at a rate of 1 per minute) for a period of 30 minutes. For clarity of illustration, only nine pulses are shown in the 30-minute pulse purge period.

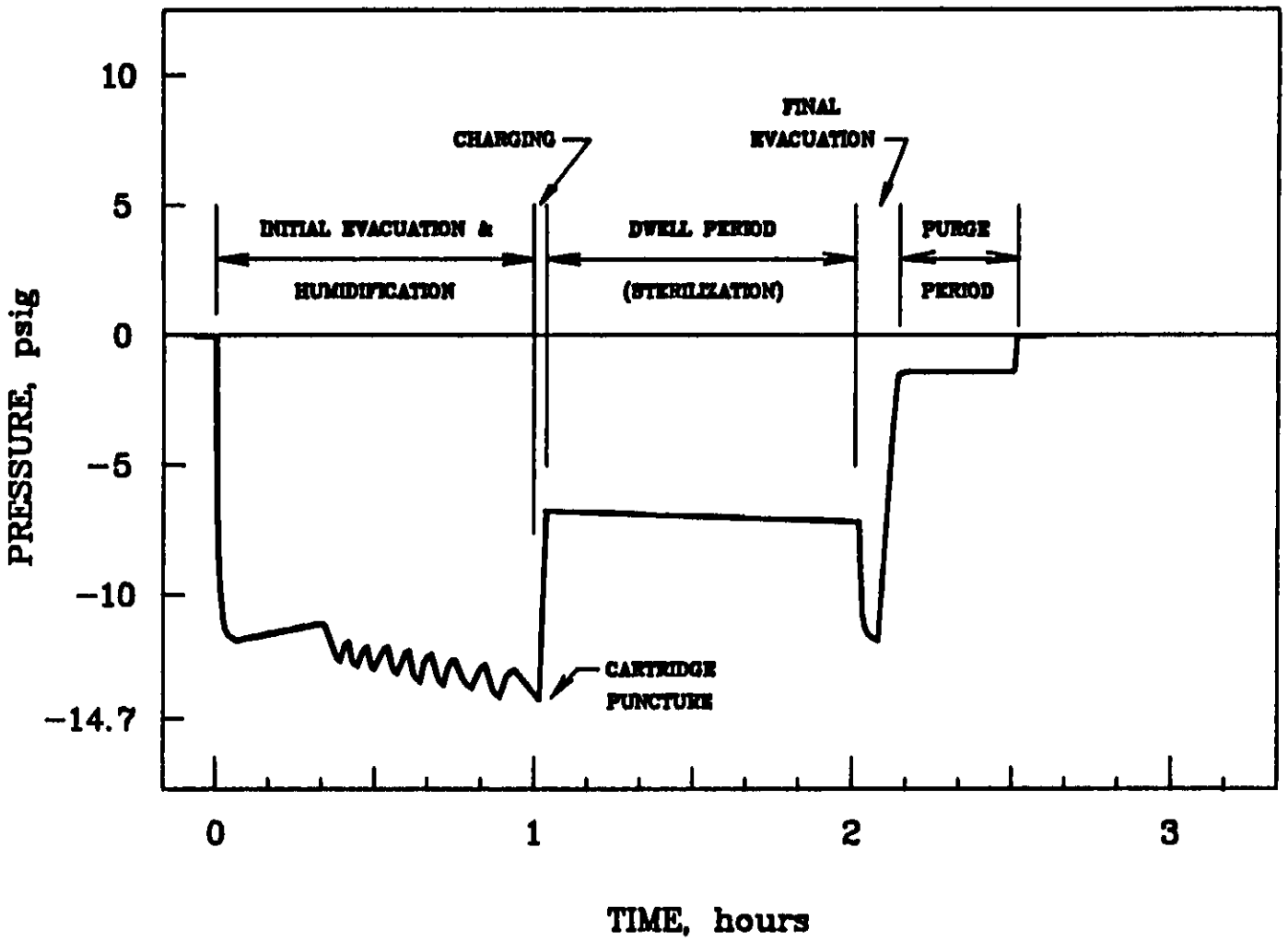

Figure 3. Typical cycle for a 100 percent EtO sterilizer showing a single vacuum draw (approximately 0.3 atm) followed by a closed-door fresh air purge period.

sterilizer onto a cart. The cart is rolled to the aerator, where the rack is pushed off of the cart into the aerator chamber. For departments using baskets to transfer items, the baskets were pulled off the shelves of the sterilizer chamber and carried to the aerator to be set on shelves. One hospital unloaded a cart-rack from the sterilizer, then manually placed baskets and some individual items on shelves in the aerator. Other than closing the aerator door, the final step was to record the date and time that aeration was started so it could be determined when the items could be removed from the aerator.

Two events were particularly variable, both from hospital to hospital and from operator to operator: the time at which the sterilizer door was closed after the load was removed, and the time at which the BI was removed from the sterile load. In some cases, the sterilizer door was closed before transferring the load to the aerator, and in other cases it was closed after the aerator door was closed. Similarly, usually the BI was pulled before the load was inserted into the aerator, but at other times, it was removed after aeration.

Generally the items were aerated approximately 12 hours at 120°F. When the aeration cycle was complete, the load was removed from the aerator. Sterile items were either stored on shelves in CS or returned to the using department.

ETHYLENE OXIDE EMISSION SOURCES

Ethylene oxide emission sources can be categorized into three groups: those with potential for creating immediately dangerous EtO exposures of a 1,000 ppm or more as the result of an accident or incident, those which might occasionally emit enough EtO to create exposures greater than 5 ppm, and those which may cause exposures of a few ppm or less.

Potential Release of Large Quantities of EtO

Three sources comprise the first group of infrequent but potentially hazardous emissions. First, the EtO supply container for the sterilizer, whether it is a large cylinder or a single-use cartridge, may release from one hundred to several thousand grams of EtO. The cylinders of the 12:88 mixture which a majority of hospitals use contain liquid under pressure and are connected through a valve to a supply line, another valve system, and the sterilizer chamber. Leaks or failure of any of the connections or the cylinders themselves could cause the contents of the entire cylinder to be discharged into the workroom atmosphere. It is known that EtO cylinders have leaked before being connected to the supply line and while in service, and that the entire contents of supply cylinders have been accidentally discharged due to human error. And even if the exposures in front of the sterilizer are controlled, the maintenance worker who changes the cylinder or supply line filters could be acutely exposed.

From the standpoint of occupational exposures, the cartridges of pure EtO are inherently safer due to the lack of external connections to the sterilizer and the much smaller quantity of EtO that they contain; the cartridge is punctured automatically after it is sealed inside the sterilizer and the cycle has

begun. However, if the cartridge were damaged accidentally or punctured outside the sterilizer, the quantity of EtO which it contains could create a dangerous concentration of EtO in the immediate area and department. The well in which the single-dose cartridge is located during sterilization was located outside the cabinet on early models of the single-dose cartridge sterilizers. With this configuration, workers could be sprayed with liquid EtO and/or exposed to the EtO vapor when the cartridge was punctured if it was not seated properly. The sterilizer manufacturer has recalled all sterilizers of this type and no longer supports their use.

The second potential source in this first group for releasing large quantities of EtO is the sterilizer itself, specifically the sterilizers using the 12:88 mixture. These sterilizers are pressurized to approximately 10 psig during the sterilization dwell period. Overpressurization of the chamber could result if the gas supply valve malfunctioned and was open when it was supposed to be closed. To counteract this, the chamber is fitted with an overpressure relief valve which could cause local concentrations of several hundred ppm in the indoor atmosphere if not properly vented, depending on the size of the sterilizer and the nature of the malfunction. The sterilizers which use the single-dose cartridge operate at a pressure below atmospheric throughout the entire cycle, and there is no relief valve.

Third, the sterilizer door gasket may develop leaks. This is especially true for the pressurized 12:88 sterilizers. Again, depending on the size of the sterilizer, the nature of the leak, and the effectiveness of the ventilation, local concentrations as high as several hundred ppm could develop.

The sterilizers which are supplied by single-dose cartridges are inherently less likely to leak EtO than the cylinder-supplied variety. Since they operate at negative pressure, if there were to be a door gasket leak, air would leak into the sterilizer chamber rather than EtO leaking out. However, the evacuation line carrying EtO downstream of the venturi is under pressure, and it is possible that it could be the source of an EtO leak.

Routine Sources Which May Cause High Concentrations

A second group consisting of three emission sources may account for most of the EtO released on an occasional basis. First, the sterilizer evacuation system for the 12:88 sterilizers depends on the evacuation of the EtO gas mixture through a water-sealed vacuum pump. At the discharge side of the pump, water and EtO are released to a sewer drain. Plumbing codes require an air gap between the discharge point of the vacuum pump and the sewer drain. Most of the EtO gas mixture from the chamber is released to the indoor atmosphere at this air gap. Depending on the control of the drain air gap, this may be the single most significant routine emission source.

Second, the opening of the sterilizer door at the completion of the cycle may provide the sterilizer operator with a very short high exposure followed by an increase in the workroom EtO concentration. In spite of the evacuation cycle, some EtO remains in the sterilizer and on the load. When the door is opened, this hot air containing EtO rises from the chamber. Without adequate local

exhaust ventilation, this EtO diffuses throughout the room, creating higher ambient concentrations for all workers in the department.

Third, the load transfer procedure provides the closest contact with EtO for the sterilizer operator: pulling the load from the sterilizer, transporting the load to the aerator, and inserting the load into the aerator. Residual EtO in the chamber and on the load may be released into the operator's breathing zone. Contact with the load may be prolonged by handling of baskets, individual items, and removal of the BI from the load.

Sources of Low Concentrations

A third group consisting of two emission sources may be responsible for EtO concentrations and exposures of a few ppm or less. The first source is opening the aerator door to retrieve items or to rearrange items on shelves to accommodate another load. This situation tends to occur mostly in departments with one aerator. In this case, production demands are such that the aerator must be used for loads with overlapping aerations times.

The second source involves cleaning the interior of the sterilizer chamber. This practice usually involves wiping the interior surface of the chamber with water. Often the worker must insert the head and upper body into the chamber in order to reach back surfaces. The sterilizer may retain EtO even after the load is removed, especially when the door is completely closed after each use. If cleaning is done soon after a load transfer, an elevated exposure could result.