FIRST AIDS IN OZONE THERAPY
(Inhalatory exposition and accidental over dose).

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1. Title: First aids in ozone therapy (Inhalatory exposition and accidental over dose).

1.1. Brief background

There are a number of good experimental studies showing that exposure by inhalation to prolonged tropospheric ozone damages the respiratory system and extra-pulmonary organs. In 3addition, judiciously practiced ozonetherapy is becoming very useful either on its own or applied in combination with orthodox medicine in a broad range of pathologies (Bocci, 2006). In the regular practice of ozone therapy, patients and physician may be accidentally exposed to ozone by inhalation. In addition, in sensible patients, ordinary therapeutic dose range may originate an acute side effects.

1.2. Purpose

The purpose of this SOP is to describe the procedure to provide the first aids in case of inhalatory exposition or accidental over dose / side effects during the regular ozone therapy application. Aids intervention in case of direct intravenous (DIV) application are not considering due to DIV is a non recommended way of ozone administration according to the Madrid Declaration ISCO3/QAU/01/03.

1.3. Scope

This procedure specified the diagnosis and aids measurement in case of accidental inhalatory exposition to ozone or during the ozone therapy practice. In addition, it specified the devices, kit and emergency drugs required during the ozone therapy practice. Also, specified the recommended working environmental level of ozone and summarized the main important toxicological data of ozone.

1.4. Acronyms, abbreviations and definitions

CARB	California Air Resources Board
DIV	Direct Intravenous
EPA	Environmental Protection Agency, USA
FCV	Forced vital capacity
FEV1	Forced expiratory volume in one second
HSDB	Hazardous Substances Data Bank
IOA	International Ozone Association
LOAEL	lowest-observed-adverse-effect level
NIOSH	National Institute of Occupational Safety and Health, USA
NOAEL	No-observed-adverse-effect level
NRC	National Research Council, USA
PEFR	Peak expiratory flow rate
RPE	Respiratory protective equipment
SOP	Standard Operation Procedure
WEL	Workplace exposure limit

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2. Responsibility

Physician
Applications of aids measurement and monitoring
Recording all data on medical records
Recording a report of Side Effect (ISCO3/REC/00/03)
Reporting any late complications
Patient follow-up

Nurses
Accommodate the patients
Preparation of the clinical procedure
Supervision of patients, and vital signs control (temperature and pressure)
Detects and alerts the doctor to anomalies due to possible reactions
Notification of possible complications

3. Effects on health

Since ozone is a highly reactive substance, any adverse health effects will be found essentially at the sites of initial contact: the respiratory tract (nose, throat and airways), the lungs, and at higher concentrations, the eyes. The principal health effects are produced by irritation of and damage to the small airways of the lung. However, people have considerable variation in sensitivity.

Uncontrolled exposure to relatively high levels of ozone could lead to more severe health effects, including lung damage. At the levels of exposure likely to be normally found in the workplace the main concern is irritation of the (upper) airways, characterized by coughing and a feeling of tightness in the chest.

4. The workplace exposure limit (WEL)

The current WEL_2 for ozone is 0.2 ppm in air averaged over a 15-minute reference period. If exposure to ozone cannot be prevented, then the principles of good control practice need to be applied to ensure that the workplace exposure limit is not exceeded.

OSHA Permissible Exposure Limit: 8 h Time Weighted Average **0.1 ppm**
ANSI/ASTM: 8 h TWA **0.1 ppm**, Short Term Exposure Limit **0.3 ppm**
ACGIH: 8 h TWA **0.1 ppm**; STEL **0.3 ppm**
NIOSH: Exposure Limit Ceiling Value **0.1 ppm** light; **0.08 ppm** moderate; **0.05 ppm**, heavy; Light, moderate, heavy work TWA <= 2 h, **0.2 ppm**
Immediately Dangerous to Life or Health **5 ppm**

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4.1. Device security

Medical ozone generator: Manufacturers and suppliers of ozone devices, should provide recommendations on the installation and proper use of such equipment, and in normal use it is unlikely that you will need to do more than comply with the recommendations to ensure the WEL is not exceeded. The preferred option is to put the equipment in a dedicated room. Where this is not practicable, it may be necessary to install the equipment in a well-ventilated area. However, if it is not installed in accordance with the manufacturer's recommendations you should make a more detailed assessment of the potential risks. Device should fit the ISCO3/DEV/00/01 recommendations.

Ozone air decontaminator devices: At low levels, ozone oxidizes airborne organic matter and inhibits the growth of bacteria (although it does not kill them unless very high ozone in air levels are used). Commercially available low output ozone generators are being marketed to improve air quality in occupied spaces. Several problems may arise from improper use of these ozone generators in occupied spaces and may exceed the WEL, and knowing that sensitive people might be affected with very low ozone levels and prolonged exposure times, it's recommended to use air ozone generators in unoccupied spaces or select in-duct ozone systems which filter and then treat the ambient air with ozone. Residual ozone gas must be converted back to oxygen before the treated air is recirculated inside occupied spaces.

5. Prevention and control of exposure

5.1 Prevention of exposure

Prevention of exposure to ozone should be the preferred approach. The release of ozone into the workplace can be prevented or substantially reduced by using a device with an efficient destructor and applying every clinical protocol in a secured manner.

5.2 Control measures

Adequate control should be achieved, as far as reasonably practical, by the use of process or engineering controls. Where these measures are not possible, you should consider further controls, such as improved application protocols and the use of respiratory protective equipment (carbon mask). Whatever controls are chosen, there is a need to check that they are effective and remain effective.

ACCIDENTAL LEAKAGE MEASURES: Turn off ozone generator, and ventilate the area. Evacuate the area until ozone levels subside.

5.3 Engineering control

The workroom should be equipped with adequate general ventilation.

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5.4 Respiratory protective equipment (RPE)

There will be situations where other control measures are either not reasonably practicable or fail to achieve adequate control (for instance, during application of ozone in sauna, ozone bags, cupping, vaginal insufflation and dental application). In these circumstances the use of RPE (carbon mask is necessary) in addition to any other controls is a valid strategy.

The RPE selected should be adequate and suitable for the environment and the user and manufactured to an appropriate standard. Physicians should be properly trained in its use, fit tested for tight-fitting respirators and supervised. The equipment should be appropriately stored, regularly cleaned and checked to ensure that it remains effective.

6. Monitoring exposure

When physicians consider that there might be wide variations of exposure at certain times and in certain operations, then they may need to measure exposure to confirm that engineering control is adequate to maintain the exposure at or below the WEL. Any monitoring regime should be planned carefully, and the advice of an occupational hygienist could prove to be useful.

6.1. Detector tubes

Short-term detector tubes capable of measuring ozone are available from a number of manufacturers. They provide an inexpensive and simple method for estimating the concentration of ozone in workplace air over a short time period, and can therefore be useful for making screening measurements to identify peak exposures or potential leaks from machines or control equipment. However, it is generally not valid to use detector tube measurements to calculate time-weighted average exposures for comparison with the WEL. Also, ozone measurements made with detector tubes can be relatively imprecise and are susceptible to positive interference from other oxidizing agents, for example chlorine and nitrogen dioxide.

For personal monitoring the use of a direct-reading instrument is recommended when assessing the pattern and duration of exposure.

6.2 Direct-reading instruments

A range of direct-reading instruments for measuring ozone are available commercially. Many are fixed-site or transportable instruments that are only suitable for source characterization and making background screening measurements. However, there are some portable instruments available that are suitable for measuring personal exposure in addition to source characterization, screening measurements and checking the effectiveness of controls. Since direct-reading instruments are continuously reading, they can be used for making measurements of time-weighted average exposure over short-term (15 min) or long-term (8 h) reference periods for comparison with the WEL.

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7. First aid

7.1 Exposition by inhalation

Symptoms: Headache, cough, dry throat, heavy chest, shortness of breath.

If someone is overcome by ozone inhalation, the following precautions should be adopted:

(a) Remove the person to a warm uncontaminated atmosphere and loosen tight clothing at the neck and waist.

(b) Keep the person at rest.

(c) If the person has difficulty in breathing, oxygen can be administered by a competent individual using the appropriate equipment. A competent individual is an occupational health professional or workplace first aider who has received training in oxygen administration and whose competency is assessed on a regular basis.

(d) If the person is not breathing normally then start cardiopulmonary resuscitation. Individuals undertaking this action should be either first aiders who hold a current qualification in workplace first aid or are occupational health professionals considered competent in accordance with the current local basic and/or advanced resuscitation protocols.

(e) Seek additional medical help if needed.

Ozone poisoning should be treated symptomatically. A period of medical observation may be necessary because of the risk of delayed lung damage. I.V. application of 1 g of Vitamin C and administration of oxygen (3 L/min) may help in the recovery of the symptoms. In case of chronic exposition, oral N-acetyl-cysteine (600 mg) may help.

7.2 Side effects after parenteral application

Symptoms	First Aid
Heaviness or local tension: typically during or shortly after penetration, caused by mechanical action of oxygen-ozone in muscle tissue	Usually spontaneous regression, does not need treatment
Muscle hematoma	Ice packs local / sodium pentosan polysulfate 0.1% ointment
Myofascial contractures	Diazepam 5 mg oral way.
Burning pain: the injection of oxygen / ozone can cause a burning pain (intense) that can take up to an hour if the concentration and amount exceed the optimal standards.	Generally it has spontaneous regression, if the pathology became particularly intense: use (Ketorolac tromethamine 30 mg) intravenous or diluted in 100 mL of saline.
Overtone vagal crisis with sweating, paleness face	Supine patient in Trendelenburg*
Crisis overtone vagal bradycardia, hypotension	Saline 250 mL I.V., Oxygen
Vasovagal reaction, triggered by algogen stimulus	Intravenous atropine 0.3 mg (1 mg atropine in saline 1:10)
Overtone vagal crisis with collapse or cardiorespiratory arrest	Call reanimation
In case of extreme bradycardia or cardiorespiratory arrest	Call anesthesia and reanimation. In the meantime: epinephrine (dilution 1:10 in saline) assisted ventilation and cardiac massage.

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Note: * In the **Trendelenburg position**, the body is laid flat on the back (supine position) with the feet higher than the head by 15-30 degrees, in contrast to the reverse Trendelenburg position, where the body is tilted in the opposite direction. This is a standard position used in abdominal and gynecological surgery. It was named after the German surgeon Friedrich Trendelenburg.

7.3 Exposition by others ways

Route of Entry	**Symptoms**	**First Aid**
Skin Contact	Irritation	Rinse with water
Eye Contact	Irritation	Rinse with water, remove contacts

7.4 Patients Follow-up

In all case report the side effect using the form ISCO3/REC/00/03. Check the patients up-to complete recovery.

8. References

8.1 SOP References

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9. Documentation and attachments

9.1 Ozone acute toxicity to humans

Likely routes of exposure: inhalation, eyes, skin exposure.

Effects of Acute Exposure: Discomfort, including headache, coughing, dry throat, shortness of breath, heavy feeling in chest (including possible pulmonary edema/fluid in the lungs); higher levels of exposure intensify symptoms. Irritation of skin and/or eyes is also possible.

Effects of Chronic Exposure: Similar to acute exposure effects, with possible development of chronic breathing disorders, including asthma.

Inhalation LC_{50}: mice, 12.6 ppm for 3 h; hamsters, 35.5 ppm for 3 h.

Impairment of lung function and subsequent impairment of exercise performance were measured in exercising adult athletes (age 19-30) exposed to 0.2 ppm (0.4 mg/m³) ozone for 1 hour (Gong *et al.*, 1986).

Determination of Acute Reference Exposure Levels for Airborne Toxicants March 1999 21.6% was observed; a 5.6% decrease in FEV_1 was observed in athletes following a 1-hour exposure to 0.12 ppm (0.24 mg/m³) ozone with exercise. Significant reductions in peak minute ventilation, oxygen uptake, and tidal volume were observed in athletes exposed to 0.2 ppm ozone, but not in those exposed to 0.12 ppm.

Healthy young males (age 19-30) exposed to ozone at concentrations as low as 0.12 ppm (0.24 mg/m³) for 2.5 hours exhibited statistically significant changes in forced vital capacity (FVC), FEV_1, forced expiratory flow rates at 75% to 25% of lung volume (FEF_{25-75}), and increased coughing (McDonnell *et al.*, 1983). Statistically significant increases in specific airway resistance (SRaw) and reporting of shortness of breath and pain upon deep inspiration were observed in subjects exposed to ozone at concentrations of 0.24 ppm (0.47 mg/m³) or greater. A more recent study (McDonnell *et al.*, 1991) reported decrements in FVC, FEV_1, and significant increases in SRaw and respiratory symptoms in 38 healthy young men following a 6.6-hour exposure to 0.08 ppm (0.2 mg/m³) ozone involving 5 h of exercise.

A statistically significant 3% decrease in FEV_1 was observed in male children (age 8-11) following a 2.5 h exposure to 0.12 ppm (0.24 mg/m³) ozone with intermittent exercise (McDonnell *et al.*, 1985). No significant increase in cough was noted as a result of ozone exposure.

A review by Lippmann (1993) reported that the ozone-associated lower airway response in the normal population engaged in outdoor recreational activity is greatly underestimated by

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1 to 2 h controlled chamber exposure studies, which indicate very little or no functional decrement at 0.120 ppm (249 μg/m³) ozone. One study cited by Lippmann (1993) reported significant ozone-associated decrements in FVC, FEV_1, peak expiratory flow rate (PEFR), FEF_{25-75}, and FEV_1/FVC in healthy adults following outdoor exercise in ambient ozone concentrations of 0.021-0.124 ppm (41-243 mg/m³) for an average of 29 min (Spektor *et al.*, 1988). In subjects with low ventilation rates (<60 L/min), the effects observed were about two times greater than those reported in chamber studies using comparable ventilation rates. Recent studies have confirmed that asthmatics react more severely than normal subjects to ozone (Scannell *et al.*, 1996) and that there is a wide variability in spirometric responsiveness (as measured by changes in FVC, FEV_1, and FEF_{25-75}) among individuals to ozone (Weinmann *et al.*, 1995).

Predisposing Conditions for Ozone Toxicity

Medical:	Persons with preexisting respiratory conditions, such as asthma or chronic obstructive lung disease, may be more sensitive to the adverse effects of ozone exposure (CARB, 1987a). Persons doing vigorous exercise or manual labor outdoors are likely to have increased ventilation rates and to be exposed to a higher dose of ozone and thus may be at increased risk for ozone toxicity.
Chemical:	Co-exposure to some aeroallergens and respiratory irritants, such as sulfur dioxide, may exacerbate the adverse respiratory effects of ozone in asthmatics (CARB, 1987a).

9.1.1 Acute toxicity to laboratory animals

The 3-hour LC_{50} values for rats, mice, guinea pigs, and rabbits are reported as 21.8 ppm, 21 ppm, 51.7 ppm, and 36 ppm (42.7, 41, 101, and 71 mg/m³) ozone, respectively (Mittler *et al.*, 1956).

A 21% increase in mortality over controls was observed in mice challenged with aerosolized streptococci concurrent with a 3-h exposure to 0.1 ppm (0.2 mg/m³) ozone (Miller *et al.*, 1978). Mice challenged with streptococci immediately following the 3-h ozone exposure, however, did not exhibit a significant increase in mortality. Due to the abundance of human exposure studies, additional animal studies were not summarized here.

9.1.2 Reproductive or developmental toxicity

No reports of human reproductive or developmental toxicity due to ozone were located in the literature (Shepard, 1994). Increased resorption rates were observed following exposure of pregnant rats to 1.97 ppm (3.86 mg/m³) ozone 8 h per day on days 6-9, 9-12, or 6-15 of gestation (Kavlock *et al.*, 1979). A later study from the same laboratory reported that

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pregnant rats exposed to 1.0 ppm or 1.5 ppm (2 mg/m³ or 2.9 mg/m³) ozone on days 17-20 of gestation had offspring which exhibited retardation of reflex development and slowing in open field behavior (Kavlock *et al*., 1980).

Veninga (1967) reported blepharophimosis (inability to open the eye to the normal extent) and jaw anomalies in mouse fetuses following maternal exposure to 0.2 ppm (0.4 mg/m³) ozone 7 h per day, 5 days per week. Because the original reference was not available for review, key experimental details (including the days of gestation during which exposure occurred) are not known.

Comparisons of pregnant, lactating, and virgin female rats exposed to 1 ppm (2 mg/m³) ozone for 6 h demonstrated enhanced sensitivity to ozone-induced pulmonary inflammation in pregnant and lactating rats (Gunnison *et al*., 1992). Pulmonary lavage fluid indicators of inflammation measured include total protein, LDH, total leukocytes, total PMN, and β-glucuronidase activity.

9.1.3 Reference for exposure levels

Derivation of Acute Reference Exposure Level and Other Severity Levels (for a 1h exposure)

Reference Exposure Level (protective against mild adverse effects): 0.09 ppm (180 mg/m³)

Level Protective Against Severe Adverse Effects: No recommendation is made due to the limitations of the database.

U.S.EPA (1975) has identified a significant harm level of 0.6 ppm (1.2 mg/m³). U.S.EPA states that "at this exposure-time combination [0.6 ppm (1.2 mg/m³) ozone for a 1-h exposure], it is judged that acutely incapacitating symptoms will be experienced by significant portions of the population, especially those engaged in light to moderate exercise, and that the health status of particularly vulnerable cardiopulmonary subjects may be seriously compromised. "The key study, on which this level is based, is a study of 10 subjects who reported substernal soreness (6/10), cough (8/10), and marked shortness of breath during a 2-h exposure to 0.75 ppm (1.5 mg/m³) ozone involving alternating 15-min periods of exercise and rest (Bates *et al*., 1972). The authors concluded that an ozone concentration of 0.75 ppm (1.5 mg/m³) produced serious adverse effects under conditions of mild exercise. The choice of the significant harm level is unacceptable as a level protective against severe health effects for exposure of the general public due to the lack of the presentation of a formal protocol for its derivation by U.S.EPA (1975).

Level Protective Against Life-threatening Effects

No recommendation is made due to the limitations of the database.

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The NIOSH-IDLH for ozone (NIOSH, 1995) is 10 mg/m³ (5 ppm) based on acute inhalation toxicity data in humans (Deichmann and Gerarde, 1969; Kleinfeld *et al*., 1957). According to NIOSH, "Pulmonary edema developed in welders who had a severe acute exposure to an estimated 9 ppm ozone plus other air pollutants (Kleinfeld *et al*., 1957). It has been reported that on the basis of animal data, exposure at 50 ppm for 60 min will probably be fatal to humans (King, 1963)." The derivation of this value is not clearly explained.

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9.2 Ozone levels and their effects

Data from IOA

ppm (µgN/mL)		**Effects**
0.001 ($2.49\ 10^{-6}$)		Lowest value detectable by hypersensitive humans. Too low to measure accurately with elaborate electronic equipment.
0.003 ($6.42\ 10^{-6}$)		Threshold of odor perception in laboratory environment, 50 per cent confidence level.
0.003 - 0.010 ($6.42\ 10^{-6}$ -$2.14\ 10^{-5}$)		The threshold of odor perception by the average person in clean air. Readily detectable by most normal persons. These concentrations can be measured with fair accuracy. Ozone levels measured in typical residences and offices equipped with a properly operating electronic air cleaner when outdoor ozone level is low. Infiltrating outdoor ozone could cause higher indoor concentrations.
0.020 ($4.28\ 10^{-5}$)		Threshold of odor perception in laboratory environment, 90 per cent confidence level.
0.001 -0.125 ($2.49\ 10^{-6}$ -$2.67\ 10^{-4}$)		Typical ozone concentrations found in the natural atmosphere. These levels of concentration vary with altitude, atmospheric conditions and locale.
0.020 - 0.040 ($4.28\ 10^{-5}$ -$8.56\ 10^{-5}$)		Representative average total oxidant concentrations in some major cities in 1964. Approximately 95 % or greater of these oxidants are generally accepted to be ozone.
0.040 ($8.56\ 10^{-5}$)		CSA limit for devices for household use. Measured as sustained concentration in test room.
0.050 ($1.07\ 10^{-4}$)		Maximum allowable ozone concentration recommended by ASHRAE in an air conditioned and ventilated space.
0.050 ($1.07\ 10^{-4}$)		Maximum allowable ozone concentration produced by electronic air cleaners and similar residential devices according to the proposed amendment of the Federal Food, Drug and Cosmetic Act. (Note: Keep this figure in mind when selecting an ozone type air purifier).
0.100 ($2.14\ 10^{-4}$)		The maximum allowable ozone concentration in industrial working areas: permissible human exposure - 8 h per day, 6 days a week.
0.100 ($2.14\ 10^{-4}$)		Continuous maximum ozone concentration allowable (per U.S. Navy in confined quarters such as atomic submarines).
0.100 ($2.14\ 10^{-4}$)		Maximum allowable limit for industrial, public, or occupied spaces in England, Japan, France, the Netherlands and Germany.
0.15 -0.51 ($3.21\ 10^{-4}$ -$1.09\ 10^{-3}$)		Typical peak concentrations in American cities.
0.200 ($4.28\ 10^{-4}$)		Prolonged exposure of humans under occupational and experimental conditions produced no apparent ill effects. The threshold level at which nasal and throat irritation will result appears to be about 0.300 ppm.
0.300 ($6.42\ 10^{-4}$)		The ozone level at which some sensitive species of plant life began to show signs of ozone effects.
0.500 ($1.07\ 10^{-3}$)		The ozone level at which Los Angeles, California, declares its Smog Alert No. 1. Can cause nausea in some individuals. Extended exposure could cause lung edema (an abnormal accumulation of serous fluid in connective tissue or serous cavity). Enhances the susceptibility to respiratory infections.
1.0 - 2.00 ($2.14\ 10^{-3}$ -$4.28\ 10^{-3}$)		Los Angeles, California, declares its Smog Alert No. 2 at 1.00 ppm ozone concentration and Smog Alert No. 3 at 1.500 ppm. When this range of ozone concentration was inhaled by human volunteers for 2 h, it caused symptoms which could be tolerated without incapacitation with the symptoms subsiding after a few

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		days. The symptoms were headache, pain in the chest, and dryness of the respiratory tract.
1.40 - 5.60 ($2.99\ 10^{-3}$ -$1.19\ 10^{-2}$)		The pinto bean exposed to 1.4 to 5.0 ppm ozone concentrations for 70 min showed some signs of severe injury to mature leaves.
5.00 -25.00 ($1.07\ 10^{-2}$ -$5.35\ 10^{-2}$)		Experimentation showed that a 3 hour exposure at 12 ppm was lethal for Guinea pigs. Welders who were exposed to 9 ppm concentration plus other air pollutants developed pulmonary edema. Chest X-rays were normal in 2 to 3 weeks, but 9 months later they still complained of fatigue and exertional dyspnea (labored respiration).
25.00 ($5.35\ 10^{-2}$)		Ozone concentrations that are immediately hazardous to human life are unknown but on the basis of animal experimentation, and exposure at 50 ppm concentration for 60 min would probably be fatal.

Note:
ppm = Parts per million volume air concentration
Unit conversion when P = 1.0 atm & T = 273.15 K :

1.0 ppm =	
1000	ppb (ppbv)
100	pphm (pphmv)
0.0001	Vol. %
$2.1414 \cdot 10^{-6}$	g/L
0.0021414	µg/mL
2141.4	$\mu g/m^3$
2.1414	mg/m^3
0.0021414	g/m^3
0.0021414	g/Nm^3
0.00016570923139381	Wt % (Air)
0.00014999992500004	Wt % (O_2)
$1.0 \cdot 10^{-6}$	mole fraction

Ozone Concentration in Air by Volume
1 g O_3 / m^3 = 467 ppm O_3
1 ppm O_3 = 2.14 mg O_3/m^3

Ozone Concentration in Air by Weight
100 g O_3/m^3 = 7.8% O_3 (Approximate)
1% O_3 = 12.8 g O_3/m^3 (Approximate)
1% O_3 = 7 284 ppm Ozone

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9.3 Content for first aid kits

List of suggested minimum content for first aid kits:

- a manual giving general guidance on first aid
- individually wrapped moist wipes or saline solution
- 20 individually wrapped sterile adhesive dressings (assorted sizes), appropriate to the type of work (dressings may be of a detectable type for food handlers)
- two sterile eye pads
- two individually wrapped triangular bandages (sterile)
- six safety pins
- two stretch bandages
- two Ice packs
- six medium sized, individually wrapped unmedicated wound dressings – approximately 12 cm x 12 cm
- two large sterile individually wrapped unmedicated wound dressings – approximately 18 cm x 18 cm
- two pairs of disposable gloves
- one resuscitation mask.
- Thermal cover

List of suggested minimun Drugs

Diazepam 5 mg, tablets
Ketorolac tromethamine 30 mg i.v.
Atropine 1 mg, i.v.
Epinephine 1 mg, i.v.
Vitamin C 1 g, i.v.
Saline 100 mL i.v.
Saline 250 mL i.v.
Sodium pentosan polysulfate 0.1% ointment

List of suggested Devices

Automatic external defibrillator.

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10. Change History

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11. Document Records

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