This quarterly Newsletter from the Poison Control Centre aims to provide you with the current information on the national and international issues related to poisoning, chemical safety and environmental health. The brief reviews on the management of toxic exposures will surely update your academic armamentarium on the subject. The news letter will function as a window to appraise you with the activities going on in the Centre on the one hand and receive feed back, information-needs and advice on the other. We are sure that it will be the most awaited publication on your desk.

Contents:

- **Scientific Articles / Reviews**
  - Poisons, poisoning and poison control centre.
  - Management of smoke inhalation in fire victims.
- **Current Concerns**
  - Potential risk for lead exposure in dental offices.
  - Contribution of environmental factors to the risk of male infertility.
- **Regulatory Issues**
  - FDA announces advisory on methyl mercury poisoning.
- **Regular Features**
- **Poison Control Centre news**.
- **Conferences / Training courses**.

**International Concerns**

However during the last few decades, with industrial, economic and agricultural revolution all over the world, there has been increased availability, and use of chemicals. It is estimated that more than 8 million chemicals are available today, of which 70,000 are in common use. Further, one to two thousand new chemical formulations and products are being added each year, some of these chemicals are highly toxic. There is growing worldwide incidence of poisoning and toxic exposures, estimated to cause half a million deaths per year. In addition, there have been increased frequency of major chemical accidents involving mass casualties and evacuations all over the world. Although the risk of toxic exposures and poisonings is universal, yet the magnitude and circumstances of exposure, morbidity and mortality vary from country to country depending upon the degree of industrialization and agricultural growth, awareness about safe use of chemicals, and availability of optimum facilities and expertise to manage poisoning. Intense international concern about the dangers of chemicals for human health and environment led to the establishment of International Programme on Chemical
Safety (IPCS) that provides internationally evaluated scientific information base to establish chemical safety programme in member countries. Poison control programme being an important element of the Chemical Safety Programme, provides framework for both prevention and management of poisoning and also for preparedness and response to chemical accidents.

The United Nations Conference on Environment and Development (UNCED) held in Rio de Janeiro in 1992, in its Agenda 21, Chapter 19, calls upon every country to promote the establishment and strengthening of poison control centres. The Intergovernmental Forum on Chemical Safety (IFCS) in 1996 emphasized that all governments should establish poison centres with related clinical and analytical facilities and promote harmonized system of poisoning data collection. The IPCS provides policy and technical guidelines for setting up the Poison Centres to all countries.

A software package (INTOX) along with the compact disc (INCHEM) developed by IPCS serve as excellent tools for running a poison centre. In addition, IPCS provides technical support and manpower development through training courses and workshops pertaining to poisons, poisoning and poison control centres.

**National Commitment**

The Sultanate of Oman with rapid expansion of industrial and agricultural activities, is confronting the risks of chemicals in addition to the existing risks from environment, pharmaceuticals and traditional remedies. To sustain development process and at the same time to protect human health and environment, the country, as a member of IPCS, has recently initiated National Chemical Safety Programme.

Poison Control Centre is an essential component of National Chemical Safety Programme, that ensures prompt and adequate diagnosis and treatment of poisoning, conducts toxicology-vigilance and prevention activities and participates in the preparedness planning and medical response to chemical accidents. The key initial step is the establishment of Poisons Information Centre which later evolves into the Poison Control Centre when clinical and analytical facilities are developed.

**Poisons Information Centre**

It is a specialized unit that provides information and advice concerning the diagnosis, prognosis, treatment and prevention of poisoning as well as about the toxicity of chemicals and the risks they pose to human health. The information service should be available 24 hours a day, 7 days a week throughout the year in principle, to whole community.

**Expected Benefits of a Poison Control Centre**

- Reduction of poisoning related morbidity, mortality and health care cost, and strengthening of chemical safety programme in the country.
- **Direct saving in health care cost by:**
  - early diagnosis and management.
  - avoiding unnecessary medical care and transport of non-toxic exposures.
  - directing severe poisoning cases to tertiary care hospitals without delay.
  - making specific antidotes and other therapeutic agents available immediately through coordination.
- **Indirect benefits by:**
  - sensitizing the communities to prevention of poisoning.
  - promoting control and regulation of chemicals, including labeling and packaging of products.

- providing education and training to the health care professionals.
- collection of epidemiological data on poisoning.
- obtaining information from abroad/WHO to respond to emergencies and other needs in a cost-effective manner.

**Poison Control Centre is established in the Directorate of Environmental Health & Malaria Eradication. You can call at 562898 or 566510 between 7:30 - 14:00 for any poisons information.**

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**ROLE AND FUNCTIONS OF POISON CONTROL CENTRE**

**MANAGEMENT OF SMOKE INHALATION IN FIRE VICTIMS**

Smoke inhalation is an important cause of airway injury, acute mortality and prolonged morbidity in fire victims. Smoke contains a mixture of toxic inhalants depending upon the temperature, burnt material and pyrolysis products (Table 1).

The irritant gases cause direct cytotoxic injury to the airway and/or alveolar mucosa, while asphyxiants interrupt the oxygen utilization at cellular level causing systemic effects. Carbon dioxide and particulate matter in smoke act as simple asphyxiants by displacing the available oxygen. Thermal injury of oropharynx usually coexists with the toxic upper airway injury.

**Pathophysiology and Clinical Features**

The smoke-induced pathophysiological changes ultimately lead to hypoxia. There could be an immediate compromise in the oxygenation at cellular level (CO, HCN) or it may follow an upper airway obstruction progressing to acute respiratory failure. Highly water-soluble gases rapidly produce irritant effect on the mucous membranes causing conjunctivitis, rhinitis, skin erythema, sore throat, cough, wheezing and hoarseness. High level exposure may produce severe caustic mucosal injury that together with thermal injury stimulates the release of mediators of inflammation and oxygen free radicals, which in turn cause supraglottic oedema, reflex laryngospasm and ultimately upper airway obstruction. Symptoms such as cough, sore throat, hoarseness and stridor.
Serious intoxication in persons with coronary artery disease. Myocardial infarction may get precipitated with reversible CO to Hb and other heme molecules is picture, because of inter-individual strictly correlate with patient's clinical cardiac arrhythmia, coma and death may ensure. The levels of COHb at room temperature is 1.5 hours while with reduced to 1-2 minutes. Hydrogen cyanide (HCN) is another highly toxic constituent of smoke. It is an intracellular toxin that inhibits the final step of oxidative phosphorylation by binding with cytochrome a-1. The cyano-cytochrome complex is relatively stable; however, it can be dissociated in presence of thiosulfate by the mitochondrial enzyme, sulfurtransferase. Since, cyanide produces cellular hypoxia, the signs and symptoms are quite similar to CO poisoning (headache, nausea, dyspnoea confusion, syncope, seizures, coma, and rapid cardiovascular collapse). The bitter almond odour typical of cyanide exposure may not be detected and red venous blood also may not be specific to HCN exposure.

Management

History, initial rapid survey and evaluation of vital signs help to determine the severity and potential for clinical deterioration. Some of the risk factors that affect the prognosis include open or close space exposure, duration of exposure, associated burn injury, alcohol intoxication and past medical history of lung or coronary artery disease. Signs and symptoms of respiratory distress and tissue hypoxia chiefly direct the course of therapy. Asymptomatic patients, with no risk factors, are kept under observation for 1-2 hours, while those with risk factors are observed for 1-2 hours after direct visualization of vocal cords. In all symptomatic patients the goal of treatment is to treat hypoxia. Cases with mild symptoms of sore throat and hoarseness are given 1% oxygen and are observed for 1-2 hours. Patients with severe intoxication may present with only upper airway injury or both upper and lower airway damage along with systemic toxicity. Fiberoptic endoscopy to assess the airway injury and removal of debris followed by endotracheal intubation and continuous delivery of 1-2% free flow oxygen is recommended in all such cases. In a comatose patient, dextrose and naloxone are given intravenously and blood samples are analyzed, carboxyhemoglobin, methemoglobin and ethanol levels and for other routine tests. However, ABG may be falsely normal in CO poisoning because it is calculated from arterial PO2, which is unaffected by COHb. Pulse oximetry for measuring oxygen saturation is also falsely normal in CO poisoning. Therefore, in such patients CO oximetry is done to measure COHb. Other investigations include ECG, X-ray and CT of chest, and Xenone ventilation scanning, if available, to detect parenchymal injury. In case of bronchospasm, ß adrenergic agonist aerosol is given. Role of corticosteroids is not established.

Patients with signs and symptoms of pulmonary oedema are given positive end expiratory pressure (PEEP) assisted ventilation to maintain oxygenation. There is no role of furosemide in these patients unless evidence of hypervolemia due to over treatment for burns or there is associated myocardial failure. Antibiotics are given to treat pneumonia and sepsis. Seizures and cardiac arrhythmias are treated as per the standard protocols. In CO poisoning with specific CNS signs and symptoms, or when COHb levels are >20%, or if the patient is pregnant, hyperbaric oxygen is considered, but not at the expense of resuscitation and burn care.

In cyanide poisoning, though oxygen is relatively ineffective, still it is continued as it promotes removal of CN from cytochrome oxidase and also will help in treating associated CO poisoning. The Lilly cyanide kit containing amyl nitrite, sodium nitrite and sodium thiosulfate is used judiciously in these patients. The smoke inhalation might lead to significant methemoglobinemia due to presence of oxides of nitrogen, the use of amyl nitrite or sodium nitrite may further decompensate the oxygenation in presence of elevated COHb. It is therefore, recommended to use only sodium thiosulfate of the kit in the recommended doses or instead use another antidote as hydroxocobalamin or Dicobalt EDTA. The coexisting methemoglobinemia should be left untreated as it provides some protection against HCN poisoning. Only those patients, who show specific signs of methemoglobinemia (deep cyanosis despite oxygenation) and have metHb levels >5%, may be considered for treatment with methylene blue. Over administration of methylene blue can cause hemolysis and even would release free cyanide to worsen toxicity. Thus, this antidote should be used selectively with caution in fire victims. A secondary survey of patients is carried out for concomitant injuries (skin, eyes, bones), burns and underlying medical problems, and treated accordingly. The patients with severe CO exposure should be followed up for 6 months.
A white powder, widely used in the boxes employed to store the dental intraoral radiograph films, contains \( \geq 99\% \) of lead as lead oxide. This could be a potential source of lead exposure in dental clinics. The lead may be transferred to patients’ mouth during a radiograph procedure both from the dental hygienist’s fingers as well as from the films. A mock dental radiograph procedure exercise conducted by the Wisconsin Division of Public Health, revealed that the wipes from the tips of dental hygienists’ fingers, contained \( \geq 99\% \) of lead that could have been transferred to the patients mouth. The study highlighted a new potential source of exposure to lead, especially in children undergoing repeated dental radiographs for dental carries. Further, it advises the health care providers, who may discover high blood lead levels of unexpected origin in a child, to explore the possibility of a dental radiograph done within the past 30 days (approximate half-life of lead in blood). The report also stresses that the lead lined radiograph storage boxes may be unnecessary in view of the advanced radiograph technology with reduced scatter radiation, available currently. Abstracted from Public Health Dispatch, MMWR: Morbidity and Mortality Weekly Report, Vol. 50, Oct 2001

**FDA ANNOUNCES ADVISORY ON METHYL MERCURY IN FISH**

Seafood can be an important part of a balanced diet for pregnant women as it is a good source of high quality protein and other nutrients and is also low in fat. However, some fish may contain high levels of methyl mercury that can harm the developing foetal brain. Mercury occurs naturally in the environment and can also be released in the air or water through industrial pollution. The bacteria in the water transform the mercury into methyl mercury, which gets absorbed in fish as they feed on aquatic organisms. Nearly all fish contain trace amounts of methyl mercury that are not harmful to humans. However, bigger fish that feed on other smaller fish, accumulate large quantities of methyl mercury and pose risk of damage to the developing nervous system, when consumed by pregnant women, lactating mothers and young children. The Food and Drug Administration (FDA) announces its advisory to the pregnant women and women of childbearing age, who may become pregnant, not to eat large fish as swordfish, king mackerel, tilefish and shark. FDA advises these women to select a variety of other kinds of fish including shellfish, canned fish, smaller ocean or farm-raised fish and that these women can safely eat \( \geq 7 \) ounces per week of cooked fish thereby prevent the risk of methyl mercury poisoning in the unborn child.

*Source: FDA Consumer Advisory, March 2001*

### Brain Teasers

- How did Cleopatra commit suicide?
- When was the first case of lead poisoning described?
- Which chemical warfares were used in the World War I?
- What caused Minamata disease?
- Where did Methyl isocyanate gas disaster occur?

Find the answers in next issue.

### Forthcoming Conferences / Training Courses / Workshops during 2002 in Muscat

- International Training Course on Prevention & Management of Poisoning.
- National Workshop & Training Course on Occupational Health.
- National Workshop on Analytical Toxicology & Antidotes.

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