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Texas Preventable Disease NEWS

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contents:

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WINTER WARNING: CARBON MONOXIDE POISONING

Carbon monoxide (CO) asphyxiation is the leading cause of toxic chemical-related death in the US today. Each year, an estimated 10,000 persons seek medical attention or lose at least one day of work or other normal activity because of CO intoxication. At least 1,500 people die each year from accidental exposure to high concentrations of the gas, and approximately 2,300 persons per year use CO to commit suicide.[1]

Carbon monoxide is a common and lethal gas produced by the incomplete combustion of liquid, solid, or gaseous fuels. Its concentration is increased by inadequate ventilation, insufficient fresh air intake, and defective air-fuel mixture. It is colorless, odorless, tasteless, and nonirritating. Therefore, if the ambient concentration is high, symptoms of acute, high-level carbon monoxide poisoning may appear without warning (Table 1). However, unless the amount in the air is very great, the prodromal symptoms usually give some warning of poisoning.[2]

Table 1. Continuum of signs and symptoms of CO poisoning in relation to the percentage of carboxyhemglobin (COHb) in the blood.[7]

Concentration (%)	Sign or Symptom
<10%	Either no symptoms or shortness of breath during vigorous exercise
10-20	Mild headache and shortness of breath during moderate exercise
20-30	Throbbing headache, irritability, emotional instability, impaired judgment, defective memory, rapid fatigue
30-40	Severe headache, weakness, nausea and vomiting, dizziness, dimness of vision, confusion
40-50	Hallucinations, severe ataxia, accelerated respirations
50-60	Syncope or coma with intermittent convulsions, tachycardia with a weak pulse, pallor or cyanosis
60-70	Increasing depth of coma with incontinence (fecal and urinary)
70-80	Profound coma with depressed or absent reflexes, weak thready pulse, shallow and irregular respirations and complete quiescence
>80	Rapid death from respiratory arrest

Artificial sources of carbon monoxide include poorly maintained or improperly vented gas-fired water heaters and furnaces, unvented or improperly vented space heaters and portable stoves, pyrolysis of some vinyl plastics and smoking. An unusual source of CO that has been identified is propane-fueled ice-surfacing machines in indoor skating rinks.[3] Burning charcoal also produces large quantities of carbon monoxide gas, enough to produce severe poisoning or death. For this reason, people should never use charcoal grills such as hibachis as a source of heat in sleeping areas.[4]

CO is absorbed through the lungs and combines with hemoglobin to form carboxyhemoglobin (COHb). The affinity of carbon monoxide for hemoglobin is approximately 200 times greater than that of oxygen, thus hemoglobin which is saturated with CO is unavailable to transport oxygen. Death may occur when blood concentrations of COHb reach 60% to 80% (Table 1).

Carboxyhemoglobin gives blood a cherry red color and its presence in capillary blood may give an abnormal red color to skin, nail beds, and mucous membranes. The victim's red coloration and lack of cyanosis may lead to delays in diagnosis and treatment if this pathognomonic sign of CO poisoning is not recognized.

Populations at special risk of carbon monoxide poisoning include: those with chronic obstructive or restrictive lung disease, anemia, chronic heart disease; obesity, or alcoholism. An enhanced metabolic rate increases the severity of symptoms; thus, children succumb more rapidly than adults when exposed to a given concentration of CO.[5,6] In pregnancy, the fetus may be extremely susceptible to the effects of carbon monoxide, suffering serious and/or permanent damage to the central nervous system. Infants born to women acutely exposed to high concentrations of CO while pregnant may display neurological sequelae, including gross brain damage. Persistent exposure of a fetus to

the low levels of carboxyhemoglobin that may be produced if a woman smokes during pregnancy may also decrease the infant's mental capacities.[5] Acute exposure of pediatric patients may also produce residual neurologic injury.[6]

Management of carbon monoxide poisoning. Oxygen is the specific antagonist to carbon monoxide. Following removal of the patient from the CO source, respiration must be supported, sometimes by artificial means. The rate of in vivo conversion of COHb to oxygenated hemoglobin (equilibration) may be accelerated significantly by increasing oxygen delivery to the tissues. This may be accomplished by administration of a mixture of 95% O_2 and 5% CO_2. Further acceleration of exchange of CO for O_2 may be accomplished by use of hyperbaric oxygen. For instance, when breathing ambient air at one atmosphere, equilibration of O_2 takes 320 minutes. Increasing the oxygen mix to 95% decreases the time to equilibration to about 80 minutes, while use of a hyperbaric oxygen chamber to deliver oxygen at three atmospheres will decrease equilibration to less than 25 minutes. Moribund patients have also been treated with exchange transfusions.[7]

Aspiration of saliva or vomitus during the acute episode may lead to bronchopneumonia during the convalescent period. Myocardial infarction, with or without coronary thrombosis, may occur within the first week following an acute exposure. Signs of nerve or brain damage may appear for up to three weeks following an acute poisoning episode. Permanent sequelae of CO poisoning may include neuropathies and motor and mental defects, some of which may mimic Parkinson's disease or multiple sclerosis.[7] Prolonged posthypoxic unconsciousness may severely damage vital organs. Extensive demyelination of white matter, bilateral necrosis of the globus pallidus, and necrotic lesions of Ammon's horn have been identified in human brain tissue after CO exposure.[5]

Prevention

Important techniques for prevention of carbon monoxide poisoning include the following:

1. Provide adequate ventilation when using wood stoves and fireplaces and insure proper installation, adjustment, and operation of all flame-burning appliances. Never use an oven or gas range to heat the house.

2. Do not operate gasoline-powered engines (eg, automobiles, lawnmowers) in confined spaces such as garages or basements.

3. Never burn charcoal inside a home, cabin, recreational vehicle, or tent.

4. Conversion of fuel-burning equipment from one type of fuel to another should be accomplished only by a qualified technician.[8]

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RECOMMENDATIONS FOR MEASLES REIMMUNIZATION DURING SCHOOL-BASED OUTBREAKS

The ACIP is expected to issue new recommendations for measles prevention and control. Changes will probably include recommendations to broaden the criteria to determine which students should be reimmunized when measles occurs in a school. For now, the following recommendations are given:

A school-based outbreak of measles is: the occurrence of **one or more** confirmed cases of measles that have been in attendance at the school during the period of communicability. The period of communicability is five days prior to rash onset and four days after rash onset. Any cases identified shall be excluded from attendance until at least five days after their rash onset.

Upon recognition of possible measles in the school, the following activities should be conducted:

1. Notify the TDH Immunization Division: 1-800-252-9152

2. Confirm the first suspected case through IgM testing, available via the TDH Immunization Division. Serologies submitted for IgM testing should be drawn about 4 days after the rash onset.

3. Students with no record of measles vaccination should be required to show immediate proof of immunization or be excluded until two weeks after the last date of rash onset on the school campus.

4. Identify students who were: a) immunized prior to 1980 with measles vaccine, and b)students immunized prior to fifteen months of age regardless of the year. These students are considered susceptible.

5. Immunize the susceptible students and any of their siblings vaccinated prior to 1980 or before 15 months of age with either monovalent measles vaccine or combined MMR or MR. Advise teachers and other school personnel born after 1956 to review their immunization status and offer revaccination to those who meet the same requirement for susceptibility as the students.

6. Reimmunization should be offered on a voluntary basis, but strongly encouraged.

7. Interview and investigate each suspected/confirmed measles case to make certain contacts to the suspected/confirmed case are immunized or, if appropriate, given immune globulin. Limited supplies of immune globulin are available from the TDH Immunization Division for administration to exposed indigent individuals--especially immunocompromised persons and susceptible pregnant women.

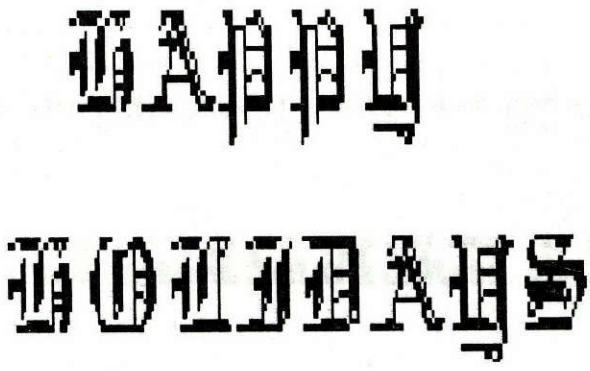
8. Notify personnel in unaffected schools whose students may be exposed to measles because of school-sponsored activities.

9. The decision to reimmunize students from an unaffected area who will be in contact with students from a school where measles is occurring must be made on a case by case basis. Points to consider include: the number of cases which have occurred, the time of last rash onset, what outbreak control measures have been taken, and whether the students will be in close proximity to contacts of known cases.

   Vaccine supplies are not unlimited. Decisions to reimmunize large groups of students with public vaccine in unaffected schools must be made in consultation with TDH. No student, however, should be discouraged from going to their private physician for measles reimmunization.

10. Cancellation or rescheduling of school events, or choosing another site should also be considered on a case by case basis and a decision should be made on the probability of exposure.

Note: There is no precise formula to use when estimating the probability of exposure to measles and the necessity to revaccinate visitors to an affected school or school event. Judgments can only be based on prior experience with similar situations and on current recommendations.



TEXAS MEASLES UPDATE

Texas continues to lead the nation in the number of reported measles cases for 1989. As of November 8, 1989, Texas has reported a cumulative total of 3,119 cases of measles. These case reports are from 97 counties throughout the state. Harris County has reported 49% (1,520 cases) of the measles morbidity. The following Texas counties have recently reported confirmed/suspected cases:

County	Latest Rash Onset	Affected Population	# Cases
Dallas	11/07/89	Pre-school, School-age, College	230
Nueces	11/07/89	All Age Groups	20
Refugio	11/06/89	Pre-school, School-age	4
Tarrant	11/04/89	Pre-school	1
Travis	11/07/89	Pre-school	1

Health care providers and school personnel should watch for signs/symptoms of measles among their patients and promptly report cases to the local health authority or the Texas Department of Health (TDH) Immunization Division toll-free in Texas at 1-800-252-9152. In addition, local health departments and public health regions are encouraged to contact the TDH Immunization Division in Austin to discuss outbreak control and case management.

Health care providers should make special arrangements for the isolation of patients with rash illnesses who present for care.

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