KAISER ALUMINUM
& CHEMICAL CORPORATION

May 1, 1980

"Copied from Proctor's book"
HF

Thomas B. Bonney
Aluminum Company of America
1501 Alcoa Building
Pittsburgh, PA 15219

Dear Mr. Bonney:

Inclosed is the Toxicology of Principal Environmental Contaminants portion of the IPAI Health Committee's Sampling and Analytical Task Force publication.

Sincerely,

Thomas J. Walker (typ)
Thomas J. Walker

TJW/bjh

cc: H.M. Cole
E. Nordheim
A. Steineggar, M.D.
P. Martyn
N.H. Proctor

Absorbed Chlorine -
Hydrogen chloride -
H.F.

TX TINER
RMC0021152

OCCUPATIONAL HEALTH SURVEILLANCE CHARTS

SUMMARY OF MAJOR AIRBORNE CONTAMINANTS AND PHYSICAL

STRESSES IN REDUCTION OPERATIONS

MEDICAL AND INDUSTRIAL HYGIENE CONSIDERATIONS

Airborne Contaminant	Principle Use or Source of Emission	Health Effects	Medical Surveillance (1)	TLV
ALUMINA	Potrooms; materials handling areas; kilns; refractories	Lung overload; no fibrosis	Interval History	10 mg/m^3 (2)
ALUMINUM Dust	Product; spray-coated anodes; buss and anode rods.	Pneumoconiosis; fibrosis of lung	Chest x-ray Lung function	5 mg/m^3
AMMONIA	Spent pot relinings; cryolite recovery and pot repair; dross reclamation; cleaners; pretreatment of printed packaging	Irritation of respiratory tract and mucous membranes	Chest x-ray Lung function	25 ppm (50 ppm ceiling)
ASBESTOS	Insulation; coverings; lagging materials; marinite; molten metal control; brake linings	Fibrosis of lung; bronchogenic carcinoma; mesothelioma; cancer of stomach, colon and rectum	Chest x-ray Lung function Sputum cytology (OSHA requirement-Annual medical surveillance)	2 fibers per ml 5 microns, with a ceiling of 10 fibers/cc not to exceed 15 min per hr up to 5 hrs per 8 hr day
CARBON DIOXIDE	Potrooms; anode baking pits; combustion sources; kilns	Asphyxiation	None	5000 ppm
CARBON MONOXIDE	By-product of incomplete combustion in: potrooms, furnaces, internal combustion engines; inert gas furnaces; kilns; enclosed spaces, tanks & silos	Asphyxiation; disturbed consciousness	Interval Exam *Carboxyhemoglobin if intoxication suspected	50 ppm

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Airborne Contaminant	Principle Use or Source of Emission	Health Effects	Medical Surveillance (1)	TLV
COKE and CALCINED COAL	Carbon, paste, and coke plants; materials handling; potrooms	Lung overload; eye and nose irritation	Interval History	10 mg/m^3 (2)
COPPER FUME and DUST	Anode rods and flex; electrical connectors; metal alloying; brazing	Metal fume fever; irritation of respiratory tract; nasal septum perforation	Interval History	0.2 mg/m^3 (fume) 1.0 mg/m^3 (dust)
FLUORIDES Gaseous, Liquid and Particulate	Cryolite plant; Potrooms; Pot repair; Solid fluxes; metal treatment; welding flux; dross recovery	Acute - nose-bleed, vomiting, respiratory irritation. Chronic - increased bone density; aggravates bronchitis/asthma	Chest x-ray Lung function Urinalysis **Fluoride in urine **Pelvis x-ray (q. 6 years)	2.5 mg/m^3
NUISANCE DUST	Materials handling throughout plant	Lung overload; eye and nose irritation	Interval History	10 mg/m^3 (2)
OIL MIST Particulate	Coolant or lubricants for mills, saws, metal forming, cans, refractories	Lipoid pneumonitis; dermatitis	Interval History	5 mg/m^3
OZONE	By-product in electical discharge and welding	Respiratory irritation	Chest x-ray Lung function	0.1 mg/m^3

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OCCUPATIONAL HEALTH SURVEILLANCE CHARTS

SUMMARY OF MAJOR AIRBORNE CONTAMINANTS AND PHYSICAL

STRESSES IN REDUCTION OPERATIONS

MEDICAL AND INDUSTRIAL HYGIENE CONSIDERATIONS

Airborne Contaminant	Principle Use or Source of Emission	Health Effects	Medical Surveillance (1)	TLV
1 PARTICULATE POLYCYCLIC ORGANIC MATTER (FORMERLY CTPV)	Binder for electrodes, Soderberg potrooms, carbon and paste plants; pot relining; tar bonded/impregnated brick; magnesium anode sealer	Photochemical skin burns; possible cancer of lungs, leukemia, lymphomas	Chest x-ray Lung function CBC Skin inspection Urinalysis ****Spotum cytology	0.2 mg/m^3 (benzene soluble fraction)
3 PHOS PHINE	Gaseous by-product of dross and water	Severe irritation of lungs; delayed pulmonary edema; narcosis; nausea; vomiting	Interval History	0.3 ppm
2 SILICA	Clay refractories; diatomaceous earth (filter media); and blasting; finished carbon	Silicosis	Chest x-ray Lung function Tuberculin test	0.05 mg/m^3
2 SULPUR DIOXIDE	By-product of combustion of coke, coal, anodes, oil and gas; surface shield in magnesium production; waste water treatment	Respiratory irritation; synergistic with dust	Chest x-ray Lung function	5 ppm
1 WELDING FUME	By-product of welding; principally oxone, oxides of nitrogen and metal (s) welded	Respiratory irritation; metal fume fever	Chest x-ray Lung function	Variable 5 mg/m^3 as iron oxide or aluminum oxide

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(1) An interval medical history should be obtained with each procedure.

Frequency of medical examination depends upon severity of exposure and health status of the individual; annual re-examination is recommended if exposures exceed action level.***

All procedures to be performed only by a physician approved by the Medical Director, or by designated paramedical personnel under the direct supervision of such a physician. Results to be interpreted only by an approved physician.

* Selection of testing laboratory and analytical method to be approved by Medical Director, specimens to be collected only by an approved physician or under his direct supervision

** Medical examinations are to be repeated on an annual basis, except that the radiologic examinations of the pelvis shall be conducted every 6 years when the average of preshift urinary fluoride concentrations for the preceding 6 years exceed 4.0 mg/liter.

Urinary postshift fluoride analysis shall be made available at an interval not exceeding every 3 months to at least one-fourth of all workers subject to occupational exposure to fluoride in dust. Participating workers shall be rotated to provide all exposed workers the opportunity for urinalysis every year. Spot urine samples shall be collected at the conclusion of the workshift after 4 or more consecutive days of exposure. Urinary preshift analysis for fluoride shall be made available to all exposed workers at least annually. Preshift spot samples shall be collected at the start of the workshift at least 48 hours after a previous occupational exposure. Results shall be corrected for a specific gravity of 1.024.

If an individual's postshift urinary fluoride level exceeds 7.0 mg/liter, preshift spot urine samples shall be collected within 2 weeks at the start of a workshift at least 48 hours after a previous occupational exposure and a repeat postshift spot sample shall be collected at the conclusion of the workshift. This shall be done at the end of the work-week in which the preshift sample is collected. If the fluoride level of the second sample is above either the preshift limit of 4.0 mg/liter or the postshift limit of 7.0 mg/liter, steps shall be taken to evaluate dietary sources, personal hygiene, basic work practices, and environmental controls.

National Institute for Occupational Safety and Health, U.S. Department of Health, Education, and Welfare: Criteria for a recommended Standard . . . Occupational Exposure to Inorganic Fluorides, HEW Publication No. (NIOSH) 76-103, U.S. Government Printing Office, Washington, D.C., 1975.

*** Action level is defined as 0.5 times the 8-hour time-weighted TLV.

**** At the time of initial assignment to an area where there is exposure to Particulate Polycyclic Organic Matter (Coal Tar Pitch Volatiles) there shall be a sputum cytology examination. Thereafter, sputum cytology examinations shall be conducted annually for employees with 5 or more years of exposure to PPOM/CTPV or who are 45 years of age or older. Specimens are to be collected only by personnel trained for this purpose.

(2) Federal MESA, Section 55.5 (Dec. 19, 1970) and the American Conference of Governmental Industrial Hygienists recommends 10 mg/m^3; Federal OSH Standards, Section 1910.93 allows 15 mg/m^3 (August 13, 1971).

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ALUMINUM OXIDE

AL_2O_3

Synonyms: Alumina

Physical Form: White powder. The six common crystalline forms of aluminum oxide are as follows:[1]

1. Gibbsite (*alpha*-$Al_2O_3.3H_2O$), the prominent constituent of many bauxites and is also formed in the precipitation step of the Bayer process for producing alumina
2. Bayerite (*beta*-$Al_2O_3.3H_2O$), does not occur in nature
3. Boehmite (*alpha*-$Al_2O_3.3H_2O$), found in many bauxites
4. Diaspore (*beta*-$Al_2O_3.3H_2O$), found in some bauxites
5. Gamma alumina (*gamma*-Al_2O_3), a name applied to numerous anhydrous aluminas with ill-defined structures. One can readily obtain this form of alumina, which is the main constitutent of "activated" alumina, by dehydrating boehmite at about 400°C.
6. Corundum (*alpha*-Al_2O_3), found in nature and can also be produced synthetically by heating *gamma*-alumina for a few hours at 1200°C.[1]

~~(Also: Production of aluminum~~

Exposure: Inhalation

Toxicology: Alumina is a nuisance dust and does not injure the pulmonary system.

Nuisance dusts have little adverse effects on lungs and do not produce significant organic disease or toxic effect when exposures are kept under reasonable control.[2] The nuisance dusts have also been called (biologically) "inert" dusts, but the latter term is inappropriate to the extent that there is no dust which does not evoke some cellular response in the lung when inhaled in sufficient amounts. However, the lung-tissue reaction caused by inhalation of nuisance dusts has the following characteristics: the architecture of the air spaces remains intact; collagen (scar tissue) is not formed to a significant extent; and the tissue reaction is potentially reversible. Excessive concentrations of nuisance dusts in the workroom air may seriously reduce visibility, may cause unpleasant deposits in the eyes, ears, and nasal passages, or may cause mild injury to the skin or mucous membranes by chemical or mechanical action *per se*, or by the rigorous skin cleansing procedures necessary for their removal.[2]

In animals, experiments with alumina have shown that the type of reaction in lung tissue is dependent on the form of alumina and its particle size as well as on the species of animal used. For example, intratracheal administration into rats of *gamma*-alumina of 2 μ average size caused only a mild fibrous reaction (loose reticulin).[3] However, intratracheal administration of *gamma*-alumina of 0.02 to 0.04 μ size into rats produced reticulin nodules which later developed into areas of dense collagenous fibrosis.[4] The latter alumina, administered by the same route in mice and guinea pigs, caused development of a reticulin network with occasional collagen; in rabbits, however, only a slight reticulin network was observed.[5] Intratracheal administration in rats of another form of alumina, *corundum*, of particle size less than 1 μ, caused the development of compact nodules of reticulin.[5]

In rats, inhalation of massive levels of *gamma*-alumina with an average particle size of 0.005 to 0.04 μ for up to 285 days caused heavy desquamation of alveolar cells, secondary inflammation, but only slight evidence of fibrosis.[6] The dust concentration in the exposure chamber was described as being so high that visibility was reduced, and a few breaths of the atmosphere by the investigators caused bronchial irritation and persistent cough.[6]

Diagnosis: No reported signs and symptoms.

Differential Diagnosis: None is specific.

Special Tests: None is specific.

Treatment: Normal measures should be in place to prevent extraordinary overexposure.

Medical Control: Preplacement screening questionnaire with emphasis on detecting a history of chronic respiratory disease. Such persons may be at increased risk from dust exposure.

References

1. Godard, H. P., Jepson, W. B., Bothwell, M. R., and Kane, R. L.: The Corrosion of Light Metals, pp. 4–5. New York: John Wiley & Sons, 1967.
2. A.C.G.I.H.: Nuisance particulates. TLVs Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1977, pp. 5–6, 51. Cincinnati, 1977.
3. Stacy, B. D., *et al.*: Tissue changes in rats' lungs caused by hydroxides, oxides and phosphates of aluminum and iron. J. Pathol. Bact., *77*:417, 1959.
4. King, E. J., Harrison, C. V., Mohanty, G. P., and Nagelschmidt, G.: The effect of various forms of alumina on the lungs of rats. J. Pathol. Bact., 69:81, 1955.
5. Engelbrecht, F. M., *et al.*: Tissue reactions to injected aluminum and alumina in the lungs and livers of mice, rats, guinea-pigs and rabbits. J. Pathol. Bact., *77*: 407, 1959.
6. Klosterkotter, W.: Effects of ultramicroscopic gamma-aluminum oxide on rats and mice. A.M.A. Arch. Ind. Health, *21*:458, 1960.

ALUMINUM POWDER

Al

Synonyms: Pyro powder
Physical Form: Flake powder
Uses: Fine flake aluminum paint
Exposure: Inhalation

Toxicology: The inhalation of very fine aluminum powder in massive concentrations causes pneumoconiosis in some individuals.

In humans the symptoms of long-term overexposure have included dyspnea, cough, and weakness. Typically, there may be radiographic evidence of fibrosis and occasional pneumothorax. At autopsy, there is generalized interstitial fibrosis, predominantly in the upper lobes, with pleural thickening and adhesions. Particles of aluminum are found in the fibrotic tissue. A fatal case of pulmonary fibrosis from inhalation of a heavy concentration of fine aluminum dust was reported in a 22-year-old male worker; autopsy revealed a generalized nonnodular fibrosis and interstitial emphysema with right ventricular hypertrophy. There had been work exposure to varying concentrations of a wide range of particle sizes, but the quantity of dust in the atmosphere below 5 μ was of the order of 10 mg/m^3.[1]

Of 27 workmen with long exposures to aluminum powder, six were found to have evidence of pulmonary fibrosis. The fine dust was more dangerous than the coarse; of the 12 men exposed to fine aluminum powder, two died and two others were affected. Of 15 who worked exclusively with coarser powder, two had radiologic changes but no symptoms.[2]

Fine metallic aluminum powders inhaled by hamsters and guinea pigs caused no pulmonary fibrosis; in rats that inhaled the dust, small scars resulted from foci of lipid pneumonitis. Alveolar proteinosis developed in all three species; it resolved spontaneously, and the accumulated dust deposits cleared rapidly from the lungs after cessation of the exposure.[3] The failure of inhaled aluminum powder to cause pulmonary fibrosis in experimental animals parallels the clinical experience in the United States, where pulmonary fibrosis has not been observed in aluminum workers.[3]

It has been suggested that the explanation of pulmonary disease among powder workers in other countries may lie in the duration of exposure, the size of the particles, the density of the dust, and the presence or absence of stearin coatings on the particles; individual susceptibility also may be a factor.[3]

Diagnosis: Signs and symptoms include dyspnea, cough, lethargy, anorexia and tachypnea; radiographic abnormalities.

Differential Diagnosis: Differentiate from other diseases which are associated with diffuse infiltration of the lungs: Hamman-Rich syndrome, Löffler's syndrome, fungus infections, sarcoidosis, scleroderma, and other rare types of diffuse interstitial fibrosis.

Special Tests: Radiography, tuberculin skin test, and sputum culture.

Treatment: Removal from exposure may improve the dyspnea in the symptomatic worker, despite the fact that the chest roentgenogram does not improve.

Medical Control: Preplacement and annual physical examination with emphasis on the respiratory system; 14" × 17" chest roentgenogram.

References

1. Mitchell, J.: Pulmonary fibrosis in an aluminum worker. Br. J. Ind. Med., *16*:123, 1959.
2. Mitchell, J., Manning, G. B., Molyneux, M., and Lane, R. E.: Pulmonary fibrosis in workers exposed to finely powdered aluminum. Br. J. Ind. Med., *18*:10, 1961.
3. Gross, P., Harley, R. A., Jr., and de-Treville, R. T. P.: Pulmonary reaction to metallic aluminum powders. Arch. Environ. Health, *26*:227, 1973.

AMMONIA

NH_3

Synonyms: Ammonia gas

Physical Form: Colorless gas

Uses: Refrigeration; petroleum refining; manufacture of fertilizers, nitric acid, explosives, plastics and other chemicals

Exposure: Inhalation

Toxicology: Ammonia is a severe irritant of the eyes, respiratory tract, and skin.

Exposure to and inhalation of concentrations of 2500 to 6500 ppm cause severe corneal irritation, dyspnea, bronchospasm, chest pain, and pulmonary edema, which may be fatal; production of pink frothy sputum often occurs. Consequences can include bronchitis or pneumonia; some residual reduction in pulmonary function has been reported.[1]

In a human experimental study which exposed ten subjects to various vapor concentrations for five minutes, 134 ppm caused irritation of the eyes, nose, and throat in most subjects, and one person complained of chest irritation; at 72 ppm, several reported the same symptoms; at 50 ppm, two reported nasal dryness, and at 32 ppm only one reported nasal dryness.[2] In a survey of eight workers in a blueprint shop, ammonia concentrations of 4 to 29 ppm caused "barely noticeable" to "moderate" eye irritation; no respiratory irritation was reported.[3]

Tolerance to usually irritating concentrations of ammonia may be acquired by adaptation, a phenomenon frequently observed among workers who became inured to the effects of exposure; no data are available on concentrations that are irritating to workers who are regularly exposed to ammonia and who presumably have a higher irritation threshold.

Liquid anhydrous ammonia in contact with the eyes may cause serious eye injury or blindness; on the skin, it causes first- and second-degree burns, which are often severe and, if extensive, may be fatal. Vapor concentrations of 10,000 ppm are mildly irritating to the moist skin, while 30,000 ppm or greater cause a stinging sensation and may produce skin burns and vesiculation.[4]

The TLV is set at a level to prevent irritation to the eyes and respiratory tract.[5]

Diagnosis: Signs and symptoms include eye, nose, throat irritation; dyspnea, bronchospasm, chest pain, pulmonary edema, pink frothy sputum; skin and eye burns and vesiculation.

Differential Diagnosis: In mild exposure resulting in mucous membrane irritation, the symptoms may mimic a viral upper respiratory tract infection. The latter may be characterized by fever, myalgias, and lymphocytosis. As the tracheobronchial tree and pulmonary parenchyma become involved, the symptoms and signs must be differentiated from cardiogenic pulmonary edema, severe viral or bacterial pneumonia, and adult respiratory distress syndromes.

Special Tests: Diagnostic studies should include electrocardiogram, sputum gram stain and culture, differential white blood count, and arterial blood gas analysis.

Treatment: If the liquid is splashed in the eyes or on the skin, immediately flush with water. If severe exposure is suspected, immediate hospitalization and observation for 72 hours for delayed onset of severe pulmonary edema are advisable.

Refer to Therapeutic Maneuvers in Treatment of Respiratory Irritants, Chapter 6. Obtain chest X-ray and examine for infiltrates. Perform analysis of arterial blood gases. Maintain P_{O_2} above 60 mm Hg by instituting, in stepwise fashion, the following measures as needed:

1. Administration of 60 to 100 per cent oxygen by mask or cannula
2. Intubation and mechanical ventilation
3. Positive end expiratory pressure breathing

Fluid balance must be maintained; use of a diuretic may be required. Steroids may be administered on a short-term basis (two to four days) to decrease the inflammatory response of the lung.

Medical Control: Preplacement and annual physical examinations with emphasis on examination of the respiratory system, eyes, and skin; pulmonary function testing, such as FVC and FEV (1 sec.); 14" × 17" chest roentgenogram.

References

Principal

1. Department of Labor: Exposure to Ammonia, Proposed Standard. Federal Register, *40*:54684, 1975.
2. National Institute for Occupational Safety and Health, U.S. Department of Health, Education and Welfare: Criteria for a Recommended Standard. Occupational Exposure to Ammonia (NIOSH). pp. 74–136. Washington, D.C.: U.S. Government Printing Office, 1974.
3. Hygienic Guide Series: Anhydrous ammonia. Am. Ind. Hyg. Assoc. J., *32*:139, 1971.

Supplemental

4. Patty, F. A.: Alkaline materials. *In* Fassett, D. W., and Irish, D. D. (eds.): Toxicology, vol. 2. *In* Patty, F. A. (ed.): Industrial Hygiene and Toxicology, 2, pp. 859–862. New York: Interscience, 1963
5. A.C.G.I.H.: Ammonia. Documentation of the TLVs for Substances in Workroom Air, ed. 3, p. 289. Cincinnati, 1976.

ASBESTOS

Synonyms: Asbestos is a generic term applied to a number of hydrated mineral silicates, including chrysotile, amosite, crocidolite, tremolite, and anthophyllite

Physical Form: Fibers of various sizes, colors, and textures

Uses: Thermal and electrical insulation; fire smothering blankets and safety garments; filler for plastics; cement

Exposure: Inhalation

Toxicology: Asbestos causes asbestosis, cancer of the lungs and digestive tract, and mesothelioma.

Asbestosis is a lung disorder characterized by a diffuse interstitial fibrosis, at times including pleural changes of fibrosis and calcification.[1] Chest x-rays reveal a granular change, chiefly in the lower lung fields; as the condition progresses, the heart outline becomes shaggy, and irregular patches of mottled shadowing may be seen.[2] Asbestos *bodies may be found in the sputum, and the* patient exhibits restrictive pulmonary function.[1] Accompanying clinical changes may include fine rales, finger clubbing, dyspnea, dry cough, and cyanosis.[1]

The onset of asbestosis probably depends upon the asbestos dust concentration, the fiber morphology, and the length of exposure.[2] Asbestosis is a progressive disease which may develop fully in seven to nine years and may cause death as early as 13 years after the first exposure.[3] Usually, the pneumoconiosis becomes evident 20 to 40 years after the first exposure to asbestos.[3] Once established, the asbestosis progresses even after the exposures have ceased.[3] In its severe forms, death results from the inability of the body to obtain requisite oxygen or from the failure of the heart to pump blood through the scarred lungs.[1]

Bronchogenic carcinoma and mesothelioma of the pleura and peritoneum are causally associated with asbestos exposures; excesses of cancer of the stomach, colon, and rectum have also been observed.[3] Among 632 asbestos workers observed from 1943 to 1967, there were 99 excess deaths (above that expected on the basis of the U.S. white male population) for three types of malignancies—bronchogenic (63), gastrointestinal (26), and all other sites combined (10).[4] Concerning mesothelioma, 80 per cent of the cases studied in South Africa and the United Kingdom were shown to have had an occupational or paraoccupational association with asbestos fibers.[4] In the U.S., 14 deaths were reported among 532 asbestos insulation workers studied from 1943 to 1968, compared with no deaths, expected in the same number of similar individuals in the general population.[4]

Neoplasm, such as mesothelioma, may occur without radiologic evidence of asbestosis at exposure levels lower than those required for prevention of radiologically evident asbestosis.[4] Mesothelioma can occur after a short intensive exposure; cases of mesothelioma in children under 19 years of age indicate the latent time period for development of mesothelioma may be shorter than first estimated.[4] Mesothelioma tumors have yet to be successfully cured by any types of treatment, including chemotherapy, radiation, or surgery; death usually results within a year of diagnosis.[1] Information is insufficient at this time to set an exposure standard (other than zero) which could assure prevention of mesothelioma in all workers, since the disease may occur following a very limited exposure 20 to 30 years earlier.[1]

Cigarette smoking is strongly implicated as a cocarcinogen among asbestos workers.[2] The incidence of bronchogenic carcinoma among nonsmoking asbestos workers is not significantly greater than that of nonasbestos workers, while asbestos workers who smoke have a much higher incidence.[2] Calculations suggest that cigarette smoking asbestos workers have approximately eight times the risk of developing lung cancer compared with other smokers.[1]

The TLV was established to protect against asbestosis and reduce to an acceptably low risk the development of neoplasms.[5]

Diagnosis: Signs and symptoms include localized pleural thickenings, which may become radiopaque through calcification; restrictive pulmonary function; rales, dyspnea, cyanosis, dry cough, and finger clubbing.

Differential Diagnosis: Differentiate from other diseases which are associated with diffuse infiltration of the lungs: Hamman-Rich syndrome, Löffler's syndrome, fungus infections, sarcoidosis, scleroderma, and other rare types of diffuse interstitial fibrosis.

Differential diagnosis of pleural mesothelioma should include inflammatory pleurisy, primary lung cancer, and the presence of metastatic disease from a primary tumor elsewhere in the body.[12]

Special Tests: Radiography, tuberculin skin test, and sputum culture should be performed.

Treatment: None is specific.

Medical Control: Preplacement and annual physical examination, with emphasis on the pulmonary, cardiovascular, and gastrointestinal systems; chest roentgenograms; lung function tests to include FVC and FEV (1 sec.); sputum cytology.

References

Principal

1. Department of Labor: Occupational exposure to asbestos. Federal Register, *40*:47652, 1975.
2. Sax, N. I.: Dangerous Properties of Industrial Materials, p. 440. New York: Reinhold Book Corporation, 1975.
3. Joint ACGIH–AIHA Aerosol Hazards Evaluation Committee: Background documentation on evaluation of occupational exposure to airborne asbestos. Am. Ind. Hyg. Assoc. J., *36*:91, 1975.
4. National Institute for Occupational Safety and Health, U.S. Department of Health, Education and Welfare: Criteria for a Recommended Standard. Occupational Exposure to Asbestos, (HSM) 72–10267. Washington, D.C.: U.S. Government Printing Office, 1972.
5. A.C.G.I.H.: Asbestos. Documentation of the TLVs for Substances in Workroom Air, ed. 3, pp. 17–19. Cincinnati, 1976.

Supplemental

6. Selikoff, I. J., Churg, J., and Hammond, E. C.: Asbestos exposure and neoplasia. JAMA, *188*:22, 1964.
7. Selikoff, I. J., Hammond, E. C., and Churg, J.: Asbestos exposure, smoking and neoplasia. JAMA, *204*:106, 1968.
8. Selikoff, I. J., Hammond, E. C., and Seidman, H.: Cancer risk of insulation workers in the United States. Biological Effects of Asbestos, p. 209. Lyon: WHO International Agency for Research on Cancer, 1973.
9. Wright, G. D.: Asbestos and health in 1969. Am. Rev. Respir. Dis., *100*:467, 1969.
10. Stumphius, J., and Meyers, P. B.: Asbestos bodies and mesothelioma. Ann. Occup. Hyg., *11*:283, 1968.
11. Champion, P.: Two cases of malignant mesothelioma after exposure to asbestos. Am. Rev. Respir. Dis., *103*:821, 1971.
12. Legha, S. S., and Muggia, F. M.: Pleural mesothelioma: clinical features and therapeutic implications. Ann. Intern. Med., *87*:613, 1977.

CARBON DIOXIDE

CO_2

Synonyms: Carbonic acid gas

Physical Form: Colorless gas (solid is "dry ice")

Uses and Sources: Byproduct of ammonia production, lime kiln operations, and fermentation

Exposure: Inhalation

Toxicology: Carbon dioxide is usually considered a simple asphyxiant; however, it is also a potent stimulus to respiration and both a depressant and excitant of the central nervous system.

Numerous human fatalities have occurred after workers entered fermentation vats, wells, and silos where the air had been replaced largely by carbon dioxide.[1, 2, 4] Exposure to concentrations near 10 per cent (100,000 ppm) for a few minutes can produce coma and subsequent asphyxiation.[1, 3] Inhalation of concentrations from 7 to 10 per cent may produce dyspnea, headache, dizziness, sweating, restlessness, paresthesias, and malaise; 5 per cent may produce shortness of breath and headache in some individuals.[7] After several hours of exposure to 2 per cent (20,000 ppm), subjects develop headache and dyspnea on mild exertion.[3] Circulatory effects in humans exposed to carbon dioxide include an increase in heart rate and cardiac output.[3] Exposure to extremely high concentrations, 25 to 30 per cent, cause coma and convulsions within one minute of exposure.[7] Carbon dioxide at room temperature will not injure the skin, but frostbite may result from contact with dry ice or from the gas at low temperatures.[4]

It is important to note that, since carbon dioxide is heavier than air, pockets of the gas may persist in areas such as pits for some time unless ventilation is provided.

The TLV provides a good margin of safety from asphyxiation and systemic effects.[8]

Diagnosis: Signs and symptoms include headache, dizziness, restlessness, parasthesias; dyspnea; sweating, malaise; increased heart rate, elevation of systolic and diastolic blood pressure, increase in pulse pressure; coma; signs of asphyxiation; convulsions at high concentrations; frostbite from handling dry ice.

Differential Diagnosis: Extreme exposure may cause a "confused" state; differentiate from other causes, such as hypoglycemia, hyperglycemia, cerebrovascular accident, transient ischemic episodes, head injury, postepileptic confusion, hysteria, heat stroke, drug abuse, toxic encephalopathy, meningitis, or encephalitis.

Special Tests: If signs or symptoms of CNS depression occur, obtain blood glucose and rectal temperature and perform a complete neurologic examination and further specific neurologic examinations as indicated.

Treatment: In cases of relatively mild exposure, symptoms such as sweating, headache, dizziness, and shortness of breath will be relieved by removal to a normal atmosphere. In coma or asphyxiation due to oxygen deficiency, resuscitation and administration of oxygen may be necessary.

Medical Control: Preplacement screening questionnaire with emphasis on detecting a history of cardiovascular impairment. Such persons may be at increased risk from exposure to high levels of carbon dioxide.

References

Principal

1. National Institute for Occupational Safety and Health, U.S. Department of Health, Education and Welfare: Criteria for a Recommended Standard Occupational Exposure to Carbon Dioxide, (NIOSH) 76-194, pp. 17-105, 114-126. Washington, D.C.: U.S. Government Printing Office, 1976.
2. Williams, H. L.: Carbon dioxide poisoning—report of eight cases, with two deaths. Br. Med. J., *2*:1012, 1958.
3. Cullen, D. J., and Eger, E. I.: Cardiovascular effects of carbon dioxide in man. Anesthesiology, *41*:345, 1974.
4. Hygienic Guide Series: Carbon dioxide. Am. Ind. Hyg. Assoc. J., *25*:519, 1964.
5. Schulte, J. H.: Sealed environments in relation to health and disease. Arch. Environ. Health, *8*:438, 1964.

Supplemental

6. International Labour Office: Encyclopaedia of Occupational Health and Safety, vol. 1, p. 251. New York: McGraw-Hill, 1972.
7. Wollman, H., and Smith, T. C.: Carbon dioxide. *In* Goodman, L. S., and Gilman, A. (eds.): The Pharmacological Basis of Therapeutics, ed. 5, pp. 893-899. New York: Macmillan, 1975.
8. A.C.G.I.H.: Carbon dioxide. Documentation of the Threshold Limit Values for Substances in Workroom Air, ed. 3, pp. 296-298. Cincinnati, 1976.

CARBON MONOXIDE

CO 1977 TLV 50 ppm

Synonyms: Carbonic oxide; exhaust gas; flue gas

Physical Form: Odorless, colorless, tasteless gas

Source: Byproduct of incomplete combustion

Exposure: Inhalation

Toxicology: Carbon monoxide (CO) causes tissue hypoxia by preventing the blood from carrying sufficient oxygen.

Carbon monoxide combines reversibly with the oxygen-carrying sites on the hemoglobin molecule with an affinity ranging from 210 to 240 times greater than that of oxygen; the carboxyhemoglobin thus formed is unavailable to carry oxygen.[1-4] In addition, carboxyhemoglobin interferes with the release of oxygen carried by unaltered hemoglobin.

Since CO causes damage by hypoxia, symptoms are referable to those tissues with the greatest oxygen consumption—the brain and myocardium.[1] Although most injuries seen in survivors of CO poisoning are referable to the central nervous system, it is likely that myocardial ischemia is responsible for many CO-induced deaths.[1]

With exposure to high concentrations, such as 4000 ppm and above, transient weakness and dizziness may be the only premonitory warnings before coma supervenes; the most common early aftermath of severe intoxication is cerebral edema.[4-6] Exposure to concentrations of 500 to 1000 ppm causes the development of headache, tachypnea, nausea, weakness, dizziness, mental confusion, and, in some instances, hallucinations; the person is commonly cyanotic.[3-5] Because carboxyhemoglobin has a bright red color, an occasional individual will exhibit the unusual combination of hypoxia together with a bright red color of the fingernails, mucous membranes, and skin; however, this "cherry red cyanosis" is usually seen only at autopsy.[4, 5]

Exposure to 50 ppm for 90 minutes may cause aggravation of angina pectoris; exposed anginal patients show a negative inotropic effect (weakened force of myocardial contraction); 50 ppm for 120 minutes may cause aggravation of intermittent claudication.[7] The clinical effects of CO exposure are aggravated by heavy labor, high ambient temperature, and altitudes above 2000 feet; pregnant women are more susceptible to the effects of CO.[8]

The reaction to a given blood level of carboxyhemoglobin is extremely variable. Some persons may be comatose with a carboxyhemoglobin level of 38 per cent, while others may maintain an apparently clear sensorium with levels as high as 55 per cent.[5] Levels of carboxyhemoglobin over 60 per cent are usually fatal; 40 per cent is associated with collapse and syncope; above 25 per cent there may be electrocardiographic evidence of a depression of the S-T segment; between 15 to 25 per cent there may be headache and nausea; levels below 15 per cent rarely produce symptoms.[5, 10] The blood of cigarette smokers usually contains 2 to 10 per cent, sometimes as high as 18 per cent, carboxyhemoglobin. Nonexposed persons have an average level of 1 per

Cont....

cent; heme metabolism is an endogenous source of CO.

Exposure of nonsmokers to 50 ppm (the TLV) for six to eight hours results in carboxyhemoglobin levels of 8 to 10 per cent.[1-3] Several investigators have suggested that the results of behavioral tests, such as tests of time discrimination, visual vigilance, choice response tests, visual evoked responses, and visual discrimination threshold, may be altered at levels of carboxyhemoglobin below 5 per cent.[2]

Transient central nervous system symptoms or rapid death are not the only results of CO poisoning.[1] Late, fatal demyelinization is a rare but dreaded complication.[1] Further, it is inappropriate to assume that, because a patient with CO poisoning shows improvement, residual mental damage may not have occurred.[1] A report of patients studied three years after CO poisoning indicated that 13 per cent showed gross neuropsychiatric damage directly attributable to their CO intoxication, 33 per cent showed a "deterioration of personality" after poisoning, and 43 per cent reported memory impairment.[9]

The TLV was set at a level to prevent systemic intoxication.[8]

Diagnosis: Signs and symptoms include headache, tachypnea, nausea, weakness, dizziness, mental confusion, hallucinations; cyanosis; depression of the S-T segment of electrocardiogram; syncope.

Differential Diagnosis: Premonitory symptoms at lower levels include headache, dizziness, and nausea; these are easily confused with the onset of many common minor disorders.[5]

Special Tests: Carbon monoxide in blood and breath may be analyzed, but, unless appropriate equipment is available to perform the tests quickly, such analyses are of little use in patient care except as retrospective confirmation of a diagnosis.[1] If the patient is comatose, it is advisable to determine the state of the electrolytes. Very frequently there is profound acidosis despite adequate pulmonary ventilation.[5]

Treatment: The presence of symptoms and a history of exposure to CO justifies the initiation of therapy consisting of the aggressive administration of oxygen within the limits of oxygen toxicity. With 100 per cent oxygen and a tightly-fitting mask, the elimination half-time of CO is 80 minutes.[5] If a hyperbaric chamber is available, it is the preferred method of treatment in severe intoxication. Expose to 3 atmospheres absolute, but for no more than 90 minutes to avoid convulsions from oxygen intoxication; at this level, the elimination half-time of CO is 23 minutes. Treat acidosis with intravenous sodium bicarbonate. Cerebral edema may be treated with corticosteroids and diuretics.[1]

Medical Control: Preplacement and annual physical examination with emphasis on the central nervous system and cardiovascular system. A baseline determination of carboxyhemoglobin has been suggested by some.[3]

References

Principal

1. Winter, P. M., and Miller, J. N.: Carbon monoxide poisoning. JAMA, *236*:1502, 1976.
2. National Institute for Occupational Safety and Health, U.S. Department of Health, Education and Welfare: Criteria for a Recommended Standard. Occupational Exposure to Carbon Monoxide (HSM) 73-11000. Washington, D.C.: U. S. Government Printing Office, 1972.
3. Hygienic Guide Series: Carbon monoxide. Am. Ind. Hyg. Assoc. J., *26*:431, 1965.

Supplemental

4. Swinyard, E. A.: Noxious gases and vapors. *In* Goodman, L. S., and Gilman, A. (eds.): The Pharmacological Basis of Therapeutics. ed. 5, pp. 900-904, 910, 911. New York: Macmillan, 1975.
5. Kindwall, E. P.: Carbon monoxide. *In* Zenz, C.: Occupational Medicine—Principles and Practical Applications. pp. 605-612. Chicago: Year Book Medical Publishers, 1975.
6. Patty, F. A.: Inorganic compounds of O, N, and C. *In* Fassett, D. W., and Irish, D. D. (eds.): Toxicology. vol. 2. *In* Patty, F. A. (ed.): Industrial Hygiene and Toxicology. ed. 2, pp. 924-936. New York: Interscience, 1963.
7. Goldsmith, J. R., and Aronow, W. S.: Carbon monoxide and coronary heart disease: a review. Environ. Res., *10*:236, 1975.
8. A.C.G.I.H.: Carbon monoxide. Documentation of the TLVs for Substances in Workroom Air. ed. 3. pp. 41-43. Cincinnati, 1976.
9. Smith, J., and Brandon, S.: Morbidity from acute carbon monoxide poisoning at a three-year follow-up. Br. Med. J., *1*:318, 1973.
10. Haldane, J. B. S.: Carbon monoxide as a tissue poison. Biochem. J., *21*:1068, 1927.

COKE AND CALCINED COAL

Synonyms: none
Physical Form: Black particulate
Uses: Potrooms
Exposure: Inhalation

Coke and calcined coal are classed as nuisance dusts. Nuisance dusts have little adverse effects on lungs and do not produce significant organic disease or toxic effect when exposures are kept under reasonable control. The nuisance dusts have also been called (biologically) "inert" dusts, but the latter term is inappropriate to the extent that there is no dust which does not evoke some cellular response in the lung when inhaled in sufficient amount. However, the lung-tissue reaction caused by inhalation of nuisance dusts has the following characteristics: (1) The architecture of the air spaces remains intact. (2) Collagen (scar tissue) is not formed to a significant extent. (3) The tissue reaction is potentially reversible. Excessive concentrations of nuisance dusts in the workroom air may seriously reduce visibility, may cause unpleasant deposits in the eyes, ears and nasal passages or cause injury to the skin or mucous membranes by chemical or mechanical action per se or by the rigorous skin cleansing procedures necessary for their removal.

Diagnosis: No reported signs and symptons.
Differential Diagnosis: None is specific.
Special Tests: None is specific.
Treatment: Normal measures should be in place to prevent extraordinary overexposure.
Medical Control: Preplacement screening questionnaire with emphasis on detecting a history of chronic respiratory disease. Such persons may be at increased risk from dust exposure.

References:

1. American Conference of Governmental Industrial Hygienists: "Nuisance aerosols," Documentation of the Threshold Limit Values for Substances in Workroom Air (3d ed., 3d printing), Cincinnati 1976, p. 190.

COPPER DUSTS AND MISTS

Cu

Synonyms: None

Physical Form: Dust; mist

Sources: Manufacture of copper castings, sheets, rods, tubing, and wire; metal alloys

Exposure: Inhalation

Toxicology: Inhalation of dusts and mists of copper and copper salts results in irritation of the upper respiratory tract and, occasionally, ulceration and perforation of the nasal septum.[1]

Copper acetate dust has caused complaints of sneezing, cough, digestive disorders, and fever.[2]

Apparent metal fume fever occurred in three men who were exposed to an extremely fine copper dust at concentrations of 0.075 to 0.120 mg/m³. Typical metal fume fever, a 24- to 48-hour illness characterized by chills, fever, aching muscles, dryness in the mouth and throat, and headache, is usually thought of as the result of exposure to metal fume (metal oxide) rather than dust. In this case, the fineness of the particles was apparently the precipitating factor.

Metal workers exposed to complex copper salts in dust form complained of metallic taste with irritation of nasal and oral mucosa; atrophic changes in the mucous membranes were noted in subjects exposed for long periods of time.[4]

Copper salts splashed in the eye cause conjunctivitis, corneal ulceration, and turbidity, and may produce palpebral edema.[5] Copper particles embedded in the eye result in a pronounced foreign body reaction with characteristic discoloration of ocular tissue.[6] Allergic contact dermatitis due to copper exposure, although rare, has been reported.[7] Greenish discoloration of the skin and hair of some copper workers has been observed.[3]

Dusts from copper and its compounds usually have an objectionable taste—a warning that tends to limit exposures before serious toxic intake can occur.[3]

Four studies have found increased incidences of lung cancer among workers in copper smelters.[3] The studies suggest that the cancer was caused by exposure to arsenic trioxide in dust and fumes produced by the various pyrometallurgic processes. They did not suggest that copper itself played any etiologic role in the cancer deaths.

The TLV was set to prevent irritation.[8]

Diagnosis: Signs and symptoms include irritation of nasal mucous membranes and pharynx; nasal ulceration and perforation; eye irritation; metallic taste; dermatitis from prolonged contact.

Differential Diagnosis: Differentiate from other causes of conjunctivitis and mucous membrane irritation, such as viral infection of the upper respiratory tract and allergies.

Special Tests: In the absence of pregnancy or some intercurrent disease, elevated urinary copper levels should be regarded as an indicator of excess copper exposure.[1]

Treatment: Institute appropriate procedures, such as removal from exposure and flushing of eyes and skin with water. If dermatitis occurs, see section in Chapter 5 on Treatment of Contact Dermatitis.

Medical Control: Preplacement screening questionnaire with emphasis on detecting a history of chronic respiratory or skin disease. Such persons may be at increased risk from exposure.

References

Principal

1. Cohen, S. R.: A review of the health hazards from copper exposure. J. Occup. Med., *16*:621, 1974.
2. Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council: Copper. pp. 55–58. Washington, D.C.: National Academy of Sciences, 1977.
3. Gleason, R. P.: Exposure to copper dust. Am. Ind. Hyg. Assoc. J., *29*:461, 1968.
4. Askergren, A., and Mellgren, M.: Changes in the nasal mucosa after exposure to copper salt dust: a preliminary report. Scand. J. Work Environ. Health, *1*:45, 1975.

Supplemental

5. Stokinger, H. E.: The metals (excluding lead). *In* Fassett, D. W., and Irish, D. D. (eds.): Toxicology, vol. 2. *In* Patty, F. A. (ed.): Industrial Hygiene and Toxicology. ed. 2, pp. 1033–1037. New York: Interscience, 1963.
6. Grant, W.: Toxicology of the Eye. ed. 2. pp. 311–319. Springfield: Charles C. Thomas, 1974.
7. Saltzer, E. I., and Wilson, J. W.: Allergic contact dermatitis due to copper. Arch. Dermatol., *98*:375, 1968.
8. A.C.G.I.H.: Copper as Cu. Documentation of the TLVs for Substances in Workroom Air. ed. 3, pp. 305–306. Cincinnati, 1976.

COPPER FUME

CuO

Synonyms: None

Physical Form: Fume

Sources: Copper and brass manufacture; welding of copper containing metals

Exposure: Inhalation

Toxicology: Copper fume causes irritation of the upper respiratory tract and metal fume fever, an influenza-like illness.

In humans, effects of copper fume include irritation of the upper respiratory tract, metallic or sweet taste, and, in some instances, discoloration of the skin and hair.[1] Exposure of workers to concentrations of 1 to 3 mg/m³ for short periods resulted in altered taste response but no nausea; levels from 0.02 to 0.4 mg/m³ produced no complaints.[1] Transient irritation of the eyes has followed exposure to a fine dust of oxidation products of copper produced in an electric arc.[2]

Typical metal fume fever, a 24- to 48-hour illness characterized by chills, fever, aching muscles, dryness in the mouth and throat, and headache, has been reported in several workers exposed to copper fume.[4,5] With metal fume fever, there is usually leucocytosis, which may amount to 12,000 to 16,000/cmm; recovery is usually rapid, and there are no sequelae.[3] Most workers develop an immunity to these attacks, but it is quickly lost; attacks tend to be more severe on the first day of the workweek.[3]

The TLV was set at a level to prevent irritation.[1]

Diagnosis: Signs and symptoms include irritation of eyes and mucous membranes; metallic or sweet taste; discoloration of skin and hair; influenza-like symptoms.

Differential Diagnosis: Differentiate from other causes of conjunctivitis and mucous membrane irritation, such as viral infection of the upper respiratory tract and allergies.

Metal fume fever is characterized by a history of recent exposure to metal fume and the transient nature of the influenza-like illness, often occurring upon first exposure of the workweek, with development of tolerance during the workweek.

Special Tests: Differential white cell count.

Treatment: Institute appropriate procedures, such as removal from exposure and flushing of eyes and skin with water. Treat the influenza-type illness symptomatically; recovery is usually complete within 48 hours of onset.

Medical Control: Preplacement screening questionnaire with emphasis on detecting a history of chronic respiratory disease. Such persons may be at increased risk from exposure.

References

1. A.C.G.I.H.: Copper as Cu. Documentation of the TLVs for Substances in Workroom Air. ed. 3, pp. 305–306. Cincinnati, 1976.
2. Grant, W. M.: Toxicology of the Eye. ed. 2, pp. 312–320. Springfield: Charles C Thomas, 1974.
3. McCord, C. P.: Metal fume fever as an immunological disease. Ind. Med. Surg., *29*:101, 1960.
4. Committee on Medical and Biologic Effects of Environmental Pollutants: Copper. pp. 55–58. Washington, D.C.: National Academy of Sciences, 1977.
5. Cohen, S. R.: A review of the health hazards from copper exposure. J. Occup. Med., *16*:621, 1974.

FLUORIDE

Synonyms: None

Physical Form: Dust

Sources: Grinding, drying, and calcining of F-containing minerals and acidulation of these minerals; metallurgical processes, such as aluminum reduction and steel-making, involving fluoride fluxes or melts; kiln firing of brick and ceramic materials; melting of raw material in glassmaking

Exposure: Inhalation; ingestion

Toxicology: Fluoride causes irritation of the eyes and respiratory tract; absorption of excessive amounts of fluoride over a long period of time results in increased radiographic density of bone.[1]

Workers exposed to an airborne fluoride concentration of 5 mg/m³ complained of eye and respiratory tract irritation and nausea.[2] The lethal oral dose of sodium fluoride for humans is approximately 5 g.; effects from ingestion are diffuse abdominal pain, diarrhea, and vomiting; excessive salivation, thirst, and perspiration; painful spasms of the limbs; and sometimes albuminuria.[3, 4]

Most absorbed fluoride is excreted rapidly in the urine. A portion is stored in bone, but a nearly equal amount is mobilized from bone and excreted.[1, 5] Some storage of fluoride occurs from the ingestion of as little as 3 mg/day.[6] Repeated exposure to excessive concentrations of fluoride over a period of years results in increased radiographic density of bone and eventually may cause crippling fluorosis (osteosclerosis due to deposition of fluoride), now an exceedingly rare phenomenon.[1] The gross changes in the skeleton are quite distinctive and characteristic; as the amount of fluoride in the bone increases; exostoses may develop, especially on the long bones; the sacrotuberous and sacrosciatic ligaments begin to calcify, vertebrae occasionally fuse together, and typical stiffness of the spinal column develops.[1]

The absorption of 20 to 80 mg of fluoride daily may be expected to lead to crippling fluorosis in ten to 20 years; this condition has not been reported in the United States from industrial exposure.[7] Evidence from several sources indicates that urinary fluoride concentrations not exceeding 5 mg/liter in preshift samples taken after two days off work are not associated with detectable osteosclerosis and that such changes are unlikely at urinary levels of 5 to 8 mg/liter.[7] Preshift urinary fluoride concentration is considered to be a measure of the worker's body (skeletal) burden of fluoride, while the postshift sample is taken to be representative of exposure conditions during that workshift.[1]

Repeated or prolonged exposure of the skin to fluoride-bearing dusts and fumes may cause dermatitis.[8]

Mottled appearance and altered form of teeth are produced only when excessive amounts of fluoride are ingested during the period of formation and calcification of teeth; this period occurs during the first eight years of life in humans. After calcification has been completed, fluoride does not have an adverse effect on the teeth.[4]

The TLV was set at a level to prevent irritation.[2]

See separate monograph on hydrogen fluoride (p. 290).

Diagnosis: Signs and symptoms include irritation of eyes and respiratory tract. Stiffness of the spine results from severe overexposure for many years.

Differential Diagnosis: Differentiate from other causes of conjunctivitis and mucous membrane irritation, such as viral infection of the upper respiratory tract and allergies.

The calcification of the sacrotuberous and sacrosciatic ligaments is indisputable evidence of flourosis, as it is not seen in other diseases.[1]

Special Tests: Urinary postshift fluoride analysis should be made available at an interval not exceeding every three months to at least one fourth of all workers subject to occupational exposure to fluoride.[9] Participating workers should be rotated to provide all exposed workers the opportunity for urinalysis every year. Spot urine samples are collected at the conclusion of the workshift after four or more consecutive days of exposure.

If an individual's postshift urinary fluoride level exceeds 7.0 mg/liter, preshift spot urine samples should be collected within two weeks at the start of the workshift at least 4 hours after a previous occupational exposure and a repeat postshift spot sample is collected at the conclusion of the workshift. If the fluoride level of the second sample is above either the preshift limit of 4.0 mg/liter or the postshift limit of 7.0 mg/liter, steps should be taken to evaluate dietary sources, personal hygiene, basic work practices, and environmental controls.

Treatment: Institute appropriate procedures; such as removal from exposure, washing of skin areas, and irrigation of eyes with water.

Medical Control: Preplacement and annual physical examination with emphasis on examination of the eyes, respiratory tract, skeletal system, kidneys, and skin; FVC and FEV (1 sec.); 14" × 17" chest roentgenogram. Preplacement pelvic x-ray with gonadal shielding. Women should have a pelvic x-ray only during menses, owing to danger to the fetus in undetected pregnancy. Additional pelvic x-ray is performed when the average of preshift urinary fluoride concentrations for the preceding six years exceeds 4.0 mg fluoride/liter of urine.[9]

References

1. Hodge, H. C., and Smith, F. A.: Occupational fluoride exposure. J. Occup. Med. *19*:12. 1977.
2. A.C.G.I.H.: Fluoride as F. Documentation of the TLVs for Substances in Workroom Air. ed. 3, pp. 116–117. Cincinnati 1976.
3. World Health Organization: Fluorides and Human Health. pp. 225–271. Geneva 1970.
4. Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council: Fluorides. pp. 163–221. Washington, D.C., 1971.
5. Dinman, B. D., *et al.*: Prevention of bone fluorosis in aluminum smelter workers. J. Occup. Med., *18*:7. 1976.
6. Largent, E. J.: Rates of elimination of fluoride stored in the tissues of man. A.M.A. Arch. Ind. Hyg. Occup. Med. *6*:37; 1952.
7. Biological Monitoring Guides: Fluorides. Am. Ind. Hyg. Assoc. J., *32*:274. 1971.
8. Hygienic Guide Series: Fluoride-bearing dusts and fumes (inorganic). Am. Ind. Hyg. Assoc. J., *26*:426, 1965.
9. National Institute for Occupational Safety and Health, U.S. Department of Health, Education and Welfare: Criteria for a Recommended Standard. Occupational Exposure to Inorganic Fluorides. (NIOSH 76–103, pp. 19–100. Washington, D.C. U.S. Government Printing Office. 1975.

HYDROGEN FLUORIDE

HF

Synonyms: None

Physical Form: Gas, liquefying at 19.5°C.; aqueous solution is hydrofluoric acid

Uses: Catalyst for production of high-octane gasoline HC; aqueous solution for frosting, etching, and polishing glass, and for removing sand from metal castings

Exposure: Inhalation

Toxicology: Hydrogen fluoride (HF) as a gas is a severe respiratory irritant; in solution it causes severe and painful burns of the skin.

Inhalation of HF produces transient choking and coughing. After an asymptomatic period of one to two days, fever, cough, dyspnea, cyanosis, and pulmonary edema may develop. Death from pulmonary edema occurred within two hours in three of six workers splashed with a 70 per cent solution, despite prompt showering with water. The HF concentration in the breathing zone was estimated to be above 10,000 ppm.[1] A chemist exposed to HF splashes on the face and upper extremities developed pulmonary edema three hours after exposure and died ten hours later.[2]

In human subjects, exposure to 120 ppm for one minute caused conjunctival and respiratory irritation with stinging of skin.[3]

Humans exposed to 30 ppm for several minutes experienced mild irritation of the eyes, nose, and respiratory tract.[3]

In humans, exposure to 2.6 to 4.8 ppm for periods up to 50 days caused slight irritation of nose, eyes, and skin, but there were no signs or symptoms of pulmonary irritation.[4]

Repeated exposure to excessive concentrations of fluoride over a period of years results in increased radiographic density of bone and eventually may cause crippling fluorosis (osteosclerosis due to deposition of fluoride in bone).[3] The early signs of increased bone density from fluoride deposition are most apparent in the lumbar spine and pelvis; those signs can be detected by x-ray.

Biologic monitoring of urinary fluoride concentration provides an indication of total fluoride intake. Data indicate that a postshift urinary flouride level of less than 8 mg/liter, averaged over an extended period of time, will not lead to osteosclerosis, although a minimal or questionable increase in bone density might develop after many years of occupational exposure.[3]

HF solutions (hydrofluoric acid) in contact with skin result in marked tissue destruction; the fluoride ion readily penetrates skin and deep tissue, causing necrosis of soft tissues and decalcification of bone; the destruction produced is excruciatingly painful.[5-7] The process of tissue destruction and neutralization of the hydrofluoric acid is prolonged for days, unlike other acids, which are rapidly neutralized.[5-7] Because of the insidious manner of penetration, a relatively mild or minor exposure can cause a serious burn. When skin comes in contact with solutions of less than 20 per cent, the burn manifests itself by pain and erythema, with a latent period of up to 24 hours; with 20 to 50 per cent solutions, the burn becomes apparent one to eight hours following exposure; solutions above 50 per cent cause immediate pain, and tissue destruction is rapidly apparent.[5]

Severe eye injuries from splashes may occur. In one case of eye burns from a fine spray of hydrofluoric acid in the face, considerable loss of corneal epithelium occurred, despite immediate and copious flushing with water and irrigation for three hours with a 0.5 per cent solution of benzethonium chloride; within 19 days there was recovery of normal vision.[8]

The TLV was set at a level to minimize irritation of eyes and nose and to prevent fluorosis.[9]

Diagnosis: Signs and symptoms include severe eye, nose and throat irritation, delayed fever, cyanosis, and pulmonary edema; severe and painful skin and eye burns from splashes of solutions; prolonged or repeated exposure to low concentrations of the gas may cause nasal congestion and bronchitis.

Differential Diagnosis: In mild exposure resulting in mucous membrane irritation, the symptoms may mimic a viral upper respiratory tract infection. The latter may be characterized by fever, myaligias, and lymphocytosis.

As the tracheobronchial tree and pulmonary parenchyma become involved, the symptoms and signs must be differentiated from cardiogenic pulmonary edema, severe viral or bacterial pneumonia, and adult respiratory distress syndrome.

Special Tests: Diagnostic studies should include electrocardiogram, sputum gram stain and culture, differential white blood cell count, and arterial blood gas analysis. The determination of fluoride concentration in the urine may be used to gauge the degree of absorption of hydrogen fluoride following suspected overexposure.

Treatment: The high risk of either immediate or delayed onset of pulmonary edema following inhalation of the gas requires that oxygen be administered under pressure immediately after a severe exposure and continued as long as necessary; close observation should be continued for 24 to 48 hours. See section on Therapeutic Maneuvers in Treatment of Respiratory Irritants, Chapter 6. Obtain chest x-ray and examine for infiltrates. Perform analysis of arterial blood gases. The arterial P_{O_2} must be maintained above 60 mm Hg. Along with oxygen administration, this may require intubation, mechanical ventilation, and positive end expiratory pressure breathing. Fluid balance must be maintained; use a diuretic if necessary. Steroids may be administered on a short-term basis (two to four days) to decrease the inflammatory response of the lungs.

Persons who have had contact with hydrofluoric acid should be subjected immediately to a drenching shower of water. Contaminated clothing should be removed as rapidly as possible, even while the person is under the shower. It is essential that the exposed area be washed with copious quantities of water for a sufficient period of time to remove all hydrofluoric acid from the skin or eyes. The affected area is immediately dressed at the time of injury with a soft, bulky dressing liberally soaked with iced zephiran or hyamine chloride (0.2 per cent in distilled water or in mildly denatured 70 per cent ethanol).[5-7] If the offending agent contained less than 20 per cent hydrofluoric acid, treatment need not go beyond iced zephiran or hyamine chloride soaks for one to four hours. Meticulous follow-up is recommended.

If the concentration of the acid was greater than 20 per cent, if the burns appear to be

deep, or if there is exquisite pain, the painful areas should be cautiously injected with 10 per cent calcium gluconate. The calcium gluconate injections should be in small quantities in order not to distend the tissues, and they should be injected through a 30-gauge needle.[5] Block anesthesia is generally used if necessary; if not, by utilizing the patient's pain as a monitor, the smallest effective amount of calcium gluconate may be determined. Furthermore, the patient can accurately localize the areas requiring treatment. The calcium gluconate is infiltrated directly into the affected dermis and subcutaneous tissue through use of a technique similar to the infiltration of a local anesthetic agent. Approximately 0.5 ml of calcium gluconate/cm^2 of burned surface area is a rough guide to the usual effective dose. The infiltration is carried 0.5 cm away from the margin of the obviously injured tissue into the surrounding, apparently uninjured, area. After the calcium gluconate injection, the burnt area of patients with severe burns may be carefully debrided. The physician should not hesitate to remove the fingernail if there is any question of serious subungual exposure. If the debridement is performed, the patient should probably be hospitalized. The hand should be dressed in a soft, bulky dressing, elevated, and observed carefully for the next 48 hours. If the pain recurs, additional calcium gluconate injections should be given.

Medical Control: Preplacement and annual physical examinations, with emphasis on examination of the respiratory tract and skin; FVC and FEV (1 sec.); 14" × 17" chest roentgenogram. Preplacement pelvic x-ray only during menses, owing to danger to the fetus in undetected pregnancy. An additional pelvic x-ray, when the average of preshift urinary fluoride concentrations for the preceding six years exceeds 4.0 mg fluoride/liter of urine.[3]

References

Principal

1. Mayer, L, and Geulich, J.: Hydrogen fluoride (HF) inhalation and burns. Arch. Environ. Health, 7:445, 1963.
2. Kleinfeld, M.:Acute pulmonary edema of chemical origin. Arch. Environ. Health, *10*:942, 1965.
3. National Institute for Occupational Safety and Health, U.S. Department of Health, Education and Welfare: Criteria for a Recommended Standard. Occupational Exposure to Hydrogen Fluoride, (NIOSH) 76-143., pp. 106-115. Washington, D.C.: U.S. Government Printing Office, 1976.
4. Largent, E.J.: Fluorosis—The Health Aspects of Fluorine Compounds. pp. 34-39, 43-48. Columbus:Ohio State University Press, 1961.
5. Dibbell, D.G. *et. al.*: Hydrofluoric acid burns of the hand. J. Bone Joint Surg., 52*A*:931, 1970.

Supplemental

6. Reinhardt, C.F., Hume, W.G., Linch, A.L., and Wetherhold, J.M.: Hydrofluoric acid burn treatment. Am. Ind. Hyg. Assoc. J., 27:166, 1966.
7. Wetherhold, J.M., and Shepherd, F.P.: Treatment of hydrofluoric acid burns. J. Occup. Med., 7:193, 1965.
8. Grant, W.M.: Toxicology of the Eye. ed. 2, pp. 557-559. Springfield:Charles C Thomas, 1974.
9. A.C.G.I.H.: Hydrogen fluoride as HF. Documentation of the TLVs for Substances in Workroom Air. ed. 3, p. 131. Cincinnati, 1976.

NUISANCE DUSTS

Synonyms: Nuisance aerosols
Physical Form: Dust
Source: Ubiquitous
Exposure: Inhalation

Toxicology: Nuisance dusts have little adverse effects on lungs and do not produce significant organic disease or toxic effect when exposures are kept under reasonable control.[1]

The nuisance dusts have also been called (biologically) "inert" dusts, but the latter term is inappropriate to the extent that there is no dust which does not evoke some cellular response in the lung when inhaled in sufficient amounts.[1] However, the lung-tissue reaction caused by inhalation of nuisance dusts has the following characteristics: the architecture of the air spaces remains intact; collagen (scar tissue) is not formed to a significant extent; and the tissue reaction is potentially reversible.

Excessive concentrations of nuisance dusts in the workroom air may seriously reduce visibility, may cause unpleasant deposits in the eyes, ears, and nasal passages, or cause injury to the skin or mucous membranes by chemical or mechanical action *per se* or by the rigorous skin cleansing procedures necessary for their removal.

The TLV was set at a level to prevent irritation.[1]

Diagnosis: Signs and symptoms include irritation of eyes and upper respiratory tract; dermatitis.

Differential Diagnosis: Differentiate from other causes of conjunctivitis and mucous membrane irritation, such as viral infection of the upper respiratory tract and allergies.

Special Tests: None is specific.

Treatment: Institute appropriate procedures, such as removal from exposure, irrigation of eyes with water, and washing of skin with soap and water. If dermatitis occurs, see section in Chapter 5 on Treatment of Contact Dermatitis.

Medical Control: None is specific.

References

1. A.C.G.I.H.: Nuisance aerosols. Documentation of the TLVs for Substances in Workroom Air, ed. 3, p. 190. Cincinnati, 1976.

OIL MIST (mineral)

Synonyms: Petrolatum *liquid*; *mineral oil*; paraffin oil

Physical **Form:** Colorless, oily, odorless, and tasteless liquid

Uses: Lubricating oil; solvent for inks in printing industry

Exposure: Inhalation

Toxicology: Mineral oil mist is of low toxicity.

A single case of lipid pneumonia suspected as caused by repeated exposure to very high concentrations of oil mist was reported in 1950; this was in a cash register serviceman whose heavy exposure occurred over 17 years of employment.[1]

A review of exposures to mineral oil mist averaging below 15 mg/m³ (but higher in some jobs) in several industries disclosed a striking lack of reported cases of illness related to these exposures.[2] A study of oil mist exposures in machine shops, at mean concentrations of 3.7 mg/m³ and maximum of 110 mg/m³, showed no increase in respiratory symptoms or decrement in respiratory performance attributable to oil mist inhalation among men employed for many years.[3] Similar results were found in a five-year study of 460 pressmen exposed to a respirable concentration of less than 5 mg/m³.[4, 5]

There is no evidence to suggest any relation between inhalation of oil mist and lung cancer. However, there are some reported cases of skin cancer from contact with certain oils.[2]

The TLV was set at a level which contains a safety factor against minor lung changes.[6]

Diagnosis: No reported signs and symptoms.

Differential Diagnosis: Although so-called lipid pneumonia may be confused with bacterial or viral pneumonia, the likelihood of contracting lipid pneumonia from occupational exposure to mineral oil mist appears small.

Special Tests: None is specific.

Treatment: None is specific.

Medical Control: Preplacement screening questionnaire with emphasis on detecting a history of chronic respiratory disease. Such persons may be at increased risk from severe exposure.

References

1. Proudfit, J. P., Van Ordstrand, H. S., and Miller, C. W.: Chronic lipid pneumonia following occupational exposure. A.M.A. Arch. Ind. Hyg. Occup. Med. *1*:105, 1950.
2. Hendricks, N. V. *et al.*: A review of exposures to oil mist. Arch. Environ. Health, *4*:139, 1962.
3. Ely, T. S., Pedley, S. F., Hearne, F. T., and Stille, W. T.: A study of mortality, symptoms, and respiratory function in humans occupationally exposed to oil mist. J. Occup. Med., *12*:253, 1970.
4. Lippmann, M., and Goldstein, D. H.: Oil-mist studies, environmental evaluation and control. Arch. Environ. Health, *21*: 591, 1970.
5. Goldstein, D. H., Benoit, J. N., and Tyroler, H. A.: An epidemiologic study of an oil mist exposure. Arch. Environ. Health, *21*:600, 1970.
6. A.C.G.I.H.: Oil mist (mineral). Documentation of the TLVs for Substances in workroom Air, ed. 3, pp. 191–192. Cincinnati, 1976.

OZONE

O_3

Synonyms: Triatomic oxygen

Physical Form: Blue gas

Sources: Inert-gas-shielded arc welding; around ozoning devices used for air and water purification; around high-voltage electric equipment

Exposure: Inhalation

Toxicology: Ozone is an irritant of the mucous membranes and the lungs.

Effects of ozone exposure range from irritation of the throat to severe pulmonary edema and hemorrhage.[1–4] Exposure to 0.05 ppm to 0.1 ppm for 13 to 30 minutes causes irritation and dryness of the throat; above 0.1 ppm effects are changes in visual acuity (decrease in the scotopic and mesopic ranges), increase in peripheral vision, choking, cough, substernal pain, and dyspnea.[2, 3] More severe exposure also causes headache, dizziness, and a burning sensation in the eyes.[1] Signs are usually minimal or absent except in cases of severe poisoning; in such cases, signs of pulmonary edema may appear and, a few hours following exposure, signs of bronchopneumonia may be present; the respiratory ailment usually resolves within one to two weeks.[1] When exposure is to levels such as 0.6 to 0.8 ppm for two hours, lung function is impaired for the duration of exposure and for up to 24 hours afterward.[1]

Systemically, ozone has been reported to mimic the effects of ionizing radiation, which raises the possibility of ozone causing damage to chromosomal structures.[1–3]

Animals develop a tolerance to the acute effects of ozone. Exposure of laboratory animals to 0.3 ppm for one hour will permit the animals to withstand multilethal doses for months afterwards.[5] It is not known with certainty that this tolerance phenomenon occurs in humans.[2]

The TLV was set at a level to prevent radiomimetic effects.[6]

Diagnosis: Signs and symptoms include irritation and dryness of throat, choking, cough; substernal pain, dyspnea, pulmonary edema; headache, dizziness; burning sensation in the eyes.

Differential Diagnosis: Ozone intoxication can mimic the common cold, influenza, sinusitis, bronchial asthma, bronchopneumonia, pulmonary embolism, and myocardial infarction.[1]

Special Tests: Diagnostic studies should include electrocardiogram, sputum gram stain and culture, differential white blood cell count and arterial blood gas analysis.

Treatment: If exposure to dangerous levels is suspected, immediate hospitalization and observation for 72 hours for delayed onset of severe pulmonary edema is advisable.

Refer to Therapeutic Maneuvers in Treatment of Respiratory Irritants, Chapter 6. Obtain chest x-ray and examine for infiltrates. Perform analysis of arterial blood gases. Maintain P_{O_2} above 60 mm Hg by instituting, in stepwise fashion, the following measures as needed:

1. *Administration of 60 to 100* per cent oxygen by mask or cannula
2. Intubation and mechanical ventilation
3. Positive end expiratory pressure breathing

Fluid balance must be maintained; use of a diuretic may be required. Steroids may be administered on a short-term basis (two to four days) to decrease the inflammatory response of the lung.

Medical Control: Preplacement and annual physical examination with emphasis on the respiratory tract; 14" × 17" chest roentgenogram; FVC and FEV (1 sec.).

References

Principal

1. Nasr, A. N. M.: Ozone poisoning in man: clinical manifestations and differential diagnosis—a review. Clin. Toxicol. *4*: 461, 1971.
2. Tabershaw, I. R., Ottoboni, F., and Cooper, W. C.: Oxidants: air quality criteria based on health effects. J. Occup. Med., *10*:464, 1968.
3. Jaffe, L. S.: Photochemical air pollutants and their effects on men and animals. II. Adverse effects. Arch. Environ. Health, *16*:241, 1968.
4. Committee on Medical and Biologic Effects of Environmental Pollutants: Ozone and other photochemical oxidants. pp. 402–415. Washington D.C.: National Academy of Sciences, 1977.

Supplemental

5. Stokinger, H. E., and Scheel, L. D.: Ozone toxicity: immunochemical and tolerance-producing aspects. Arch. Environ. Health, *4*:327, 1962.
6. A.C.G.I.H.: Ozone. Documentation of the TLVs for Substances in Workroom Air, ed. 3, pp. 194–195. Cincinnati, 1976.

PHOSPHINE

PH_3

Synonyms: Hydrogen phosphide; phosphoretted hydrogen; phosphorus trihydride

Physical Form: Colorless gas

Uses: Insecticide used for fumigation; preparation of phosphonium halides

Exposure: Inhalation

Toxicology: Phosphine is a severe pulmonary irritant.

Workers exposed intermittently to concentrations up to 35 ppm but averaging below 10 ppm complained of nausea, vomiting, diarrhea, chest tightness, cough, headache, and dizziness; no evidence of cumulative effects was noted.[1] Single severe exposures cause similar signs and symptoms, as well as excessive thirst, muscle pain, chills, sensation of pressure in the chest, dyspnea, syncope, and stupor.[2] In a few cases of exposure, dizziness and staggering gait have also occurred.[1] From 1900 to 1958 there were 59 reported cases of phosphine poisoning, with 26 deaths; the effect most frequently reported was marked pulmonary edema.[2]

Phosphine has a fishy or garlic-like odor detectable at 2 ppm; the odor threshold does not provide sufficient warning of dangerous concentrations.[3]

The TLV was set to prevent adverse effects.[4]

Diagnosis: Signs and symptoms include nausea, vomiting, abdominal pain, diarrhea; sensation of pressure in the chest, dyspnea; muscle pains, chills; stupor or syncope.

Differential Diagnosis: In mild exposure resulting in mucous membrane irritation, the symptoms may mimic a viral upper respiratory tract infection. The latter may be characterized by fever, myalgias, and lymphocytosis.

As the tracheobronchial tree and pulmonary parenchyma become involved, the symptoms and signs must be differentiated from cardiogenic pulmonary edema, severe viral or bacterial penumonia, and adult respiratory distress syndrome.

Special Tests: Diagnostic studies should include electrocardiogram, sputum gram stain and culture, differential white blood cell count, and arterial blood gas analysis.

Treatment: If phosphine has been inhaled, immediate hospitalization and observation for 72 hours for delayed onset of severe pulmonary edema is advisable.

Refer to Therapeutic Maneuvers in Treatment of Respiratory Irritants, Chapter 6. Obtain chest x-ray, examine for infiltrates, and perform anaylsis of arterial blood gases. Maintain P_{O_2} above 60 mm Hg by instituting, in stepwise fashion, the following measures as needed:

1. Administration of 60 to 100 per cent oxygen by mask or cannula
2. *Intubation and mechanical ventiliation*
3. Positive end expiratory pressure breathing

Fluid balance must be maintained; use of a diuretic may be required. Steroids may be administered on a short-term basis (two to four days) to decrease the inflammatory response of the lung. Treat central nervous system effects symptomatically.

Medical Control: Preplacement and annual physical examination with emphasis on the respiratory system, 14" × 17" chest roentgenogram; FVC and FEV (1 sec.).

References

Principal

1. Jones, A. T., Jones, R. C., and Longley, E. O.: Environmental and clinical aspects of bulk wheat fumigation with aluminum phosphide. Am. Ind. Hyg. Assoc. J., *25*:376, 1964.
2. Harger, P. N., and Spolyar, L. W.: Toxicity of phosphine, with a possible fatality from this poison. A.M.A. Arch. Ind. Health, *18*:497, 1958.

Supplemental

3. Hygienic Guide Series: Phosphine, Am. Ind. Hyg. Assoc. J., *25*:314–316, 1964.
4. A.C.G.I.H.: Phosphine. Documentation of the TLVs for Substances in Workroom Air, ed. 3, pp. 209–210. Cincinnati, 1976.

SILICA (amorphous, including diatomaceous earth [D.E.])

SiO_2

Synonyms: Diatomaceous earth; diatomite; diatomaceous silica; infusorial earth; ripoli

Physical Form: Solid; soft, chalky powder

Uses: Production of filters, polishes, absorbents, insulators

Exposure: Inhalation

Toxicology: Amorphous silica, including natural diatomaceous earth, is usually considered to be of low toxicity; however, pure amorphous silica is rarely found and diatomaceous earth usually contains some amount of crystalline silica; processing of amorphous silica by high temperature calcining alters the silica from the benign amorphous to the pathogenic crystalline form which causes fibrosis.[1]

In a study of diatomaceous earth workers, those employed in the quarry for more than five years and exposed only to natural diatomaceous earth had no significant roentgenologic changes; of others employed for more than five years in the milling process and exposed to calcined material, 17 per cent had simple pneumoconiosis and 23 per cent had the confluent form, probably the result of fibrogenic action of the crystalline silica formed by calcination of the naturally occurring mineral.[1, 2]

In humans, calcined diatomaceous earth pneumoconiosis is characterized roentgenographically by fine linear and/or minute nodular shadows, either or both of which may be accompanied by conglomerate fibrosis; in the simple phase of the disease, the upper lobes are affected more than the lower lobes and the condition progresses by an increase in the apparent number of the nodules, which rarely attain the density or size of nodules often seen in quartz silicosis.[3] In the early confluent stage of the disease, the linear and nodular changes in the upper lung fields become more circumscribed and homogeneous; histologically, there is an absence of the focal, discrete, hyaline nodules or the whorled pattern of collagenous fibers of typical silicosis.[2, 3]

In two different industrial processes, exposure to freshly vaporized amorphous silica among electric arc furnace workers resulted in pulmonary fibrosis in some, although the fume was contaminated by other substances in both situations.[5, 6]

Repated exposure of guinea pigs to natural diatomaceous earth for periods up to 50 weeks to average concentrations ranging from 60 to 124 mg/m³ caused thickening of the alveolar septa by infiltration of macrophages, accumulation of large numbers of multinuclear cells containing dust particles, and lymphadenopathy, but no proliferation of connective tissue.[4]

The recommendation is made that concentrations be kept below the TLV, based on good industrial hygiene practice.[7]

Diagnosis: Signs and symptoms include roentgenologic signs of pneumoconiosis from exposure to calcined material or material contaminated with crystalline silica.

Differential Diagnosis: Differentiate from the following other diseases wich are associated with diffuse infiltration of the lungs: Hamman-Rich syndrome, Löffler's syndrome, fungus infections, sarcoidosis, scleroderma, and other rare types of diffuse interstitial fibrosis.

Special Tests: Radiography, tuberculin skin test, and sputum culture.

Treatment: Removal from exposure diminishes the risk of occupational pulmonary disease.

Medical Control: Preplacement and annual physical examination with emphasis on the respiratory system; 14" × 17" chest roentgenogram; FVC and FEV (1 sec.).

References

1. Dutra, F. R.: Diatomaceous earth pneumoconiosis. Arch. Environ. Health, *11*: 613, 1965.
2. Oechsli, W. R., Jacobson, G., and Brodeur, A. E.: Diatomite pneumoconiosis: roentgen characteristics and classification. Am. J. Roentgenol. Radium Ther. Nucl. Med., *85*:63, 1961.
3. Smart, R. H., and Anderson, W. M.: Pneumoconiosis due to diatomaceous earth—clinical and x-ray aspects. Ind. Med. Surg., *21*:509, 1952.
4. Tebbens, B. D., and Beard, R. R.: Experiments on diatomaceous earth pneumoconiosis. I. Natural diatomaceous earth in guinea pigs. A.M.A. Arch. Ind. Health, *16*:55, 1957.
5. Jephcott, C. M.: Chemical aspects of Shaver's disease. *In* Pneumoconiosis—Beryllium, Bauxite Fumes. pp. 489–497. New York: Hoeber Medical Division (Harper & Row, 1950.
6. Vitums, V. C. *et al.*: Pulmonary fibrosis from amorphous silica dust, a product of silica vapor. Arch. Environ. Health, *32*:62, 1977.
7. A.C.G.I.H.: Silica (amorphous). Documentation of the TLVs for Substances in Workroom Air, ed. 3, pp. 396–397. Cincinnati, 1976.

COAL TAR PITCH VOLATILES

Synonyms: CTPV; particulate polycyclic aromatic hydrocarbons

Physical Form: CTPV refers to the volatile matter emitted when coal tar or coal tar pitch is heated. CTPV contain thousands of organic substances, including benzo(a)pyrene and other polynuclear aromatic hydrocarbons (PNAs).

Sources: Pitch melting operations; carbon electrodes using coal tar pitch as a binder

Exposure: Inhalation; skin contact

Toxicology: CTPV have caused cutaneous photosensitization and irritation of the eyes.

Exposure to coal tar and coal tar pitch has caused phototoxic skin reactions.[1,2] Characteristically there is a short induction period of a few hours, followed by the appearance of an exaggerated sunburn on areas exposed to the sun or ultraviolet light—usually the face and hands. Erythema and swelling subside after removal from exposure; hypermelanosis is commonly observed. Intimate contact with coal tar oils and pitch without adequate personal hygiene causes acne, folliculitis, or both.

Although allergic dermatitis is readily induced by PNAs in guinea pigs, it is only rarely reported in humans from occupational contact with PNAs; these have resulted largely from the therapeutic use of coal tar preparations.[3] Skin cancer has occurred in rats and mice from repeated application of coal tar or certain of its components such as benzo(a)pyrene. Skin carcinomas have been observed in occupations in which there is contact with pitch, tar, tar products, and products of fractionation and distillation of oil.[1]

In a study of 34 workers engaged in a roofing operation, 23 (68 per cent) complained of skin reactions.[2] Seventeen workers had eye disorders: eight described slight burning, five had burning and slight conjunctival erythema, and four had burning, conjunctival erythema, lacrimation, and palpebral edema. The severe eye symptoms usually began as burning and lacrimation three to four hours after the beginning of work and led to conjunctivitis on exposure to sunlight.

In a mortality study of 5939 pitch workers (roofers), the ratio of lung cancer deaths in pitch workers to the number of such deaths expected on the basis of U.S. mortality data was as follows: 0.92 for workers exposed less than 20 years; 1.5 for those exposed 30 to 39 years, and 2.47 for those exposed 40 years or longer.[4] Smoking histories were not determined.

The TLV was set at a limit to minimize exposure to the carcinogens in CTPV.[5]

Diagnosis: Signs and symptoms include photosensitization dermatitis and irritation of eyes.

Differential Diagnosis: The transient nature of the phototoxic skin reaction together with a history of recent occupational exposure to CTPV should aid determination of etiology.

Differentiate from other causes of conjunctivitis and mucous membrane irritation such as viral infection of the upper respiratory tract and allergies.

Special Tests: None is specific.

tive procedures such as thorough washing of skin daily. If dermatitis occurs, refer to Chapter 5, Treatment of Contact Dermatitis.

Medical Control: The NIOSH criteria document suggests preplacement and annual physical examinations with emphasis on the skin, mucous membranes of the oral cavity, respiratory tract, liver, and kidneys; 14" × 17" chest roentgenograms (PA and lateral); FVC and FEV (1.0 sec); sputum cytology.[5]

References

1. Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council: Particulate Polycyclic Organic Matter, pp. 185–193. Washington, D.C.: National Academy of Sciences, 1972.
2. National Institute for Occupational Safety and Health, U.S. Department of Health, Education, and Welfare: Criteria for a Recommended Standard. Occupational Exposure to Coal Tar Products, (NIOSH) 78-107, pp. 1–119. Washington, D.C.: U.S. Government Printing Office, 1978.
3. Scala, R.: Toxicology of PPOM. J. Occup. Med., *17*:784, 1975.
4. Hammond, E. C., Selikoff, I. J., Lanther, P. L., and Seidman, H.: Inhalation of benzpyrene and cancer in man. Ann. NY Acad. Sci., *271*:102, 1976.
5. A.C.G.I.H.: Coal tar pitch volatiles (benzene soluble fraction). Documentation of the TLVs for Substances in Workroom Air, ed. 3, pp. 57–58. Cincinnati, 1976.

SULFUR DIOXIDE

SO_2

Synonyms: Sulfurous anhydride; sulfurous oxide

Physical Form: Colorless gas

Uses: Manufacture of sulfuric acid; as a bleach; casting of nonferrous metal; food processing, manufacture of sodium sulfite

Exposure: Inhalation

Toxicology: SO_2 is a severe irritant of the eyes, mucous membranes, and skin.

The irritant effects of SO_2 are caused by the rapidity with which it forms sulfurous acid on contact with moist membranes.[1-3] Approximately 90 per cent of all SO_2 inhaled is absorbed in the upper respiratory passages, where most effects occur; however, it may produce respiratory paralysis and may also cause pulmonary edema.[2] Exposure to concentrations of 10 to 50 ppm for five to 15 minutes causes irritation of the eyes, nose, and throat, rhinorrhea, choking, cough, and, in some instances, reflex bronchoconstriction with increased pulmonary resistance.[2]

It is estimated that 10 to 20 per cent of the healthy young adult population is hypersusceptible to the effects of sulfur dioxide, while the phenomenon of adaptation to irritating concentrations is a recognized occurrence in experienced workers.[2] Workers repeatedly exposed to 10 ppm experienced upper respiratory irritation and some nosebleeds, but the symptoms did not occur at 5 ppm; in another study, initial cough and irritation did occur at 5 ppm and 13 ppm, but subsided after five minutes of exposure.[2, 3]

In a human experimental study with the subjects inhaling through the mouth, brief exposure to 13 ppm caused a 73 per cent increase in pulmonary flow resistance; 5 ppm resulted in a 40 per cent increase; 1 ppm produced no effects.[4]

Exposure of the eyes to liquid SO_2 from pressurized containers causes corneal burns and opacification resulting in a loss of vision.[2] Liquefied SO_2 on the skin produces skin burns from the freezing effect of rapid evaporation.[2]

The TLV was set at a level to prevent respiratory irritation.[5]

Diagnosis: Signs and symptoms include irritation of the eyes, nose, throat, rhinorrhea; choking, cough; reflex bronchoconstriction; eye and skin burns.

Differential Diagnosis: In mild exposure resulting in mucous membrane irritation, the symptoms may mimic a viral upper respiratory tract infection. The latter may be characterized by fever, myalgias, and lymphocytosis.

As the tracheobronchial tree and pulmonary parenchyma become involved, the symptoms and signs must be differentiated from cardiogenic pulmonary edema, severe viral or bacterial pneumonia, and adult respiratory distress syndrome.

Special Tests: Diagnostic studies should include electrocardiogram, sputum gram stain and culture, differential white blood cell count, and arterial blood gas analysis.

Treatment: If severe exposure is suspected, hospitalization and observation for 72 hours for delayed onset of severe pulmonary edema are advisable.

Refer to Therapeutic Maneuvers in Treatment of Respiratory Irritants, Chapter 6. Obtain chest x-ray and examine for infiltrates. Perform analysis of arterial blood gases. Maintain P_{O_2} above 60 mm Hg by instituting, in stepwise fashion, the following measures as needed:

1. Administration of 60 to 100 per cent oxygen by mask or cannula
2. Intubation and mechanical ventilation
3. Positive end expiratory pressure breathing

Fluid balance must be maintained; use of diuretic may be required. Steroids may be administered on a short-term basis (two to four days) to decrease the inflammatory response of the lung.

If eyes are irritated, flush with water; flush skin with water if splashed with liquefied SO_2.

If dermatitis occurs, see section in Chapter 5 on Treatment of Contact Dermatitis.

Medical Control: Preplacement and annual physical examination with emphasis on the eyes, respiratory tract, and skin; 14" × 17" chest roentgenogram; FVC and FEV (1 sec.).

References

Principal

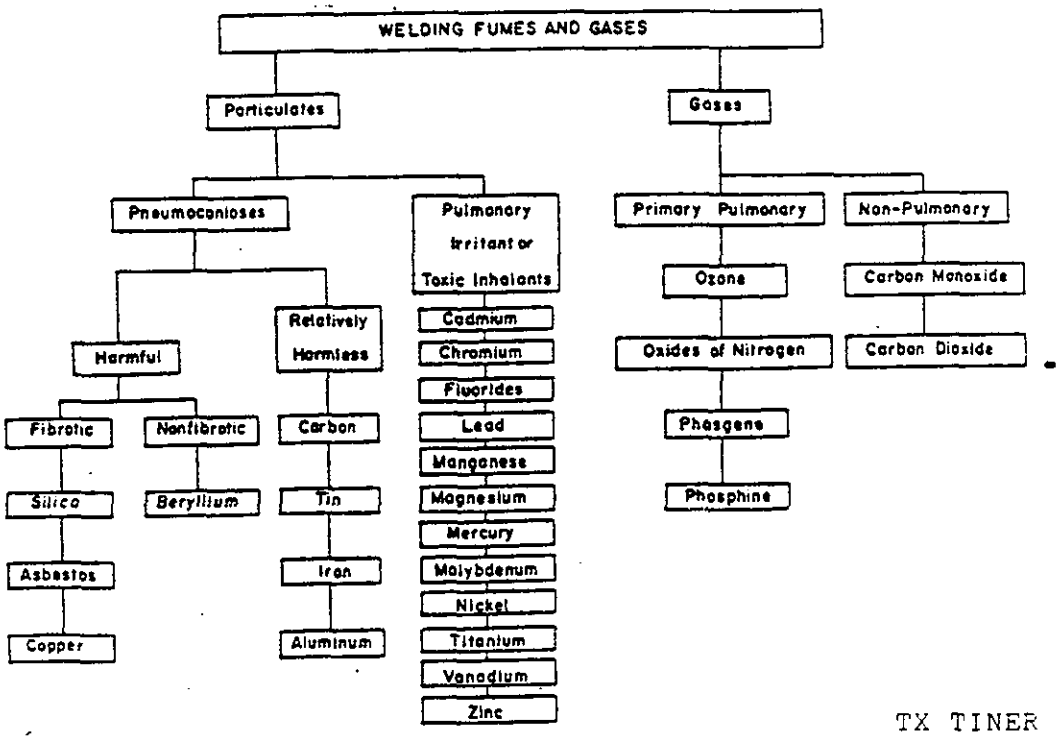
1. National Institute for Occupational Safety and Health, U.S. Department of Health, Education and Welfare: Criteria for a Recommended Standard. Occupational Exposure to Sulfur Dioxide. pp. 16–54. Washington, D.C.: U.S. Government Printing Office, 1974.
2. Department of Labor: Occupational exposure to sulfur dioxide. Federal Register, *40*:54520, 1975.

Supplemental

3. Hygienic Guide Series: Sulfur dioxide. Am. Ind. Hyg. Assoc. Quart., *16*:332, 1955.
4. Whittenberger, J. L., and Frank, R. N.: Human exposures to sulfur dioxide. Arch. Environ. Health, *7*:244, 1963.
5. A.C.G.I.H.: Sulfur dioxide. Documentation of the TLVs for Substances in Workroom Air, ed. 3, pp. 238–239. Cincinnati, 1974.

METAL WELDING FUME

In welding and related operations (cutting, brazing), various kinds of particulate and gaseous contaminants are produced. Their nature depends on the welding method, the composition of the metal to be welded, welding electrodes, fluxes and coatings. The following figure is a schematic representation of some of the possible effluents present in welding fumes, and it shows how they can be characterized into various toxicologic subgroups. The particulate fraction can be resolved into two general groups--those which produce pneumoconioses and those which can be classified as pulmonary irritants or toxic inhalants. Some of the materials are difficult to classify and additional exposure data are needed in order to place them in a specific classification. Efforts were made to include all of the common fume and gas constituents to which welders or welding operators might be exposed in the course of their daily activities; several less commonly encountered constituents were included for completeness of coverage.



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References:

1. American Welding Society Technical Activities Committee's Task Group on Welding Fume Research: The Welding Environment, American Welding Society, 2501 N.W. 7th Street, Miami, Florida 33125, 1973, pp. 3-4