

Nitrogen Dioxide

Introduction

Nitrogen oxides include nitrous oxide, nitric oxide, nitrogen dioxide, nitrogen trioxide, nitrogen tetroxide, nitrogen pentoxide, nitric acid, and nitrous acid. A thorough discussion of the chemical and toxic properties of each oxide can be found in the NIOSH Criteria Document on the oxides of nitrogen⁷⁵. This chapter is concerned with the chemical agent nitrogen dioxide (NO₂) which exists in equilibrium with nitrogen tetroxide (N₂O₄) at body temperature.

Exposure to nitrogen dioxide is through inhalation. There is often only mild irritation of the upper respiratory tract, apparently because little of the inhaled gas enters into solution until it reaches the moist alveolar (air cell) spaces of the lungs. Therefore, at the time of exposure, there may be little pain or shortness of breath, and a seriously damaging dose can be delivered to the lungs while a worker is not immediately aware of the danger.

Depending upon the concentration and duration of exposure, toxic reactions to nitrogen dioxide can range from mere mucosal irritation to chemical pneumonitis, acute pulmonary edema (excess fluid in lung tissue), or death. There is a latent period of from 3 to 30 hours from the time of initial exposure to the onset of potentially fatal pulmonary (lung) symptoms.

Chronic exposure to low levels of nitrogen dioxide (5 to 20 ppm) may result in mild irritation to the eyes, nose, and throat, continued pulmonary irritation, coughing, and possible lung damage, especially to the alveolar tissue. Chronic bronchitis and a clinical condition in which the blood's ability to transport oxygen is reduced (methemoglobinemia) may result.

The following is a listing of common names for nitrogen dioxide followed by a listing of occupations with potential exposure to nitrogen dioxide:

Common Names

dinitrogen tetroxide
liquid dioxide
nitrito
nitro

nitrogen oxide
nitrogen peroxide
nitrogen tetroxide

Occupations with Potential Exposures to Nitrogen Dioxide

acid dippers
aniline makers
arsenic acid makers
artificial leather makers
braziers
bright dip workers
bronze cleaners
celluloid makers
copper cleaners
cotton bleachers
dental workers
diesel equipment operators
dye makers
electroplaters
electric arc welders
etchers

explosive makers
explosive users
farm workers
fertilizer makers
firemen
flour bleachers
food bleachers
gas shrinking operators
gas welders
glass blowers
heat treaters
jet fuel makers
jewelry makers
lacquer makers
lithographers

auto garage workers
auto painters
blueprinters
brass cleaners
medical technicians
metal cleaners
mine workers
nitrate workers
nitric acid workers
nitrite workers
nitrogen dioxide workers
nitrous acid workers
nurses
organic chemical synthesizers
oxalic acid makers
oxidized cellulose compound
makers
pharmaceutical makers
phosphoric acid makers
photoengravers
phthalic acid makers
physicians
picklers
pipe fitters
plasma torch operators
raw silk bleachers
rocket fuel makers
silo fillers
sulfuric acid makers
textile (rayon) bleachers
tunnel workers
welders

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

The following should be considered:

- smoking history,
- periodic chest X-rays,
- periodic pulmonary function tests such as 1 second forced expiratory volume (FEV₁) and forced vital capacity (FVC), and
- methemoglobin studies.

The differential diagnosis includes:

- Pneumonia,
- acute bronchitis, and
- X-ray findings may mimic tuberculosis.

A respiratory questionnaire (Appendix C) can be useful in evaluating the extent and importance of respiratory symptoms such as:

- Breathlessness,
- sputum production,
- chest pain,
- cough, and
- wheezing.

Nonoccupational Exposure

It should also be considered that exposure to nitrogen dioxide can be from a home hobby or activity such as:

- Automotive and related hobbies,
- welding, especially arc, and
- use of a gas heater or stove.

Air pollution may also be a factor, especially in cities with heavy vehicle use.

NOTE: Nitrogen dioxide is contained in both tobacco smoke and smog. Smokers in smoggy areas are likely to be at increased risk of developing chronic obstructive pulmonary disease.

Signs and Symptoms

Local

- irritation of eyes
- irritation of mucus membranes of upper respiratory tract
- yellowish or brownish staining of skin and teeth (may indicate nitric acid exposure)

Systemic

Nitrogen dioxide fumes can cause 2 types of upper respiratory tract injuries:

- Severe pulmonary irritation, progressing to pulmonary edema, which may occur within 24 hours of prolonged and/or concentrated exposure
- insidious bronchiolar damage, causing life-threatening respiratory tract obstruction in 1 to 4 weeks after only mild to moderate exposure to the toxic gas.

Methemoglobinemia may also occur.

Acute Exposure

- discomfort, uneasiness, or indisposition, often indicative of infection (malaise)
- bluish or grayish discoloration of the skin (cyanosis)
- cough
- expectoration of blood (hemoptysis)
- rapid breathing (tachypnea)
- labored or difficult breathing (dyspnea)
- chills
- fever
- headache
- nausea
- vomiting
- unconsciousness
- collapse and death from respiratory failure
- bronchial irritation, a 5- to 12-hour symptom-free period, followed by sudden onset of acute pulmonary edema may also occur

Nitrogen oxides formed from green silage may produce "silo-filler's disease" and or bronchiolitis fibrosa obliterans. It may develop within a few days or 6 weeks and is accompanied by:

- Fever,
- severe and progressive dyspnea, and
- cyanosis.

Silo filler's disease may also progress to chronic pulmonary obstruction.

Chronic Exposure

- pulmonary dysfunction
 - decreased vital and breathing capacities
 - decreased lung compliance
 - increased residual volume
- low arterial oxygen saturation
- dyspnea on exertion
- moist rales and wheezes
- sporadic cough with mucopurulent expectoration (consisting of mucus and pus)
- methemoglobinemia, usually mild and transient. Persons with genetic susceptibility may develop toxic levels of methemoglobin

Laboratory and Clinical Examinations

Additional data that will assist in arriving at a correct diagnosis are:

- Chest X-ray of acute exposure shows diffuse, reticular fine, nodular infiltration, or numerous scattered nodular densities (1 to 5 millimeters in diameter),
- decreased blood pH,
- decreased serum proteins, and
- increased urinary hydroxyproline and acid mucopolysaccharides.

Epidemiology

When considering exposure to nitrogen dioxide, both concentration and exposure time must be evaluated. There is sufficient data to conclude that the primary irritant effects of nitrogen dioxide are dose-related.

It should be noted that the acute and usually delayed effects of higher concentrations of nitrogen dioxide are well established but the critical concentration needed to produce either acute or pulmonary edema or bronchiolitis fibrosa obliterans is not known. In addition, subacute and chronic responses to low levels of exposure to nitrogen dioxide are not well-established or defined in the human.

Muller⁷⁷ reported a study of 7 workers or guests who were exposed to nitrogen dioxide during the final blast which would connect 2 parts of a tunnel under construction. No environmental measurements are available. Two of them were hospitalized shortly after the explosion, and the third was hospitalized 12 days later after exposure during a second explosion. After the acute disease, the most common complaints were bronchitis, cough, sputum, and exertional dyspnea. Seven months after the accident, 4 of the exposed group had a 1 second forced expiratory volume (FEV₁) of less than 70%. Within 7 to 14 months after the accident, 5 recovered completely; 2 who suffered from a mild bronchitis previous to the accident had a worsening of symptoms afterwards and were unable to carry out normal duties. The following table from the report presents latency periods and bridging symptoms.

**LATENCY PERIODS AND BRIDGING SYMPTOMS
FROM 7 CASE STUDIES OF ACCIDENTAL
NITROGEN DIOXIDE EXPOSURE**

CASE	LATENT PERIOD	BRIDGING SYMPTOMS
1	4 weeks	Dull pressure in chest Fatigue, moderate dyspnea Irritating cough Temperature Severe dyspnea 24 days hospitalization
2	none	none (gradual worsening of respiration)

3	18 days	Fatigue, dyspnea Irritating cough Temperature Suffocation 18 days hospitalization
4	4 days	Temperature Bronchitis and cough Moderate dyspnea a few days sick at home
5	2-1/2 days	Sanguinolent sputum Feeling of suffocation Acute dyspnea 39 days hospitalization
6	none	none
7	14 hours	Pressure in chest Vomiting Dyspnea 1 week sick at home

(Muller, 1969)

NOTE: Case 2 became gradually short of breath but never was seriously sick. Case 6 had neither a latent period nor bridging symptoms, although he had respiratory symptoms for about 6 months.

The following report of a dose-response relationship in quotes has been taken from the National Institute for Occupational Safety and Health:⁶⁰

In a study of 70 workers, "aged 26 to 48, exposed for 6 to 8 hours daily for 4 to 6 years in a chemical plant to what was described as oxides of nitrogen, Kosider et al.⁷⁸ reported concentrations between 0.4 and 2.7 ppm as nitrogen dioxide. Sampling and analysis methods were not reported. A control group of 80 workers of similar ages who were not exposed to nitrogen oxides was selected, and workers smoking more than 10 cigarettes daily were excluded from both groups. Workers exposed to nitrogen dioxide complained of sporadic cough with mucopurulent expectoration and dyspnea on exertion. Fine bubbling rales and 'whistling' sounds were heard in some men, primarily over the lower lungs. There were no chest X-ray abnormalities noted."

Over a period of 18 months, Tse and Bockman⁷⁹ observed 4 firemen with acute toxic reactions due to accidental inhalation of nitrogen dioxide which originated from a leak in a chemical plant. Environmental measurements were not made but reports indicated the presence of dense, reddish brown fumes. Pulmonary function data for the firemen who experienced every phase of the illness including the eventual development of chronic pulmonary insufficiency follow:

PULMONARY FUNCTION DATA

	Predicted Normal	Date		
		11/2/66	5/9/67	2/28/68
Vital Capacity (ml)	3,943	3,000	2,750	2,650
RV/TLC^a (3%)	23	45	46	53
FEV₁^b (% of vital capacity)	79	51	61	70
FEV₃^c (% of vital capacity)	95	73	87	100
Maximum Mid-Expiratory Flow (liters/min)	130-395	45	60	65
Maximal Breathing Capacity (liters/min)	133	62	86	71

Lung Compliance (liters/cm/H ₂ O)	0.2	0.112
Arterial pH	7.35-7.45	7.40	7.44	7.48
Oxygen Saturation (%)	99.5	94.3	94.3	86.8
Pao₂^d (mm Hg) air	90-100	75	75	55
100% oxygen	550	560	550	550
Paco₂^e air	40	29	35	35
Diffusing Capacity (DLCO) ^f ml/min/mm Hg	23.2-39.2	30.7	32.3	24.0

(Tse and Bockman, 1970)

- a - Residual volume/total lung capacity
- b - Forced expiratory volume in 1 second
- c - Forced expiratory volume in 3 seconds
- d - Arterial oxygen pressure
- e - Arterial carbon dioxide pressure
- f - Diffusing capacity of the lung for carbon monoxide

All 4 firemen experienced respiratory discomfort of varying degrees with or without abnormal X-ray findings about 4 to 6 weeks after exposure. No correlation between individual response and smoking was made; however, it was noted that the 1 fireman most severely affected had stopped smoking 14 years prior to exposure. The others who smoked about ½ package of cigarettes per day at the time of the accident eventually became asymptomatic.

The probable results of excessive single exposures to nitrogen dioxide as determined by the American Industrial Hygiene Association are as follows:⁸⁰

**PROBABLE RESULT OF SINGLE
EXPOSURE TO NITROGEN DIOXIDE**

Exposure Time (min)	Concentration in Air (ppm)	Expected Effect in Humans
5	400	Pulmonary edema and death
15	200	
30	150	
60	100	
5	200	Pulmonary edema with possible subacute or chronic lesions in the lungs
15	100	
30	75	
60	50	
5	100	Respiratory irritation, chest pain
15	50	
30	40	
60	25	

(Emergency Exposure Limits, *AIHA J*, 1964)

Lowry and Schuman⁸¹ reported a study of 4 workers who were exposed to nitrogen dioxide after entering a silo or silo chute. Exposure occurred within 48 hours of filling of the silo. Although actual environmental measurements were not available, observed concentrations of nitrogen dioxide obtained during research experiments in agricultural science have ranged from 200 to 4,000 ppm. Irritating fumes were noted by all 4 workers who also experienced respiratory symptoms of varying degrees of severity and developed bronchiolitis fibrosa obliterans. Two of the workers died, 1 on the 27th and the other on the 30th day. One of the surviving 2 was hospitalized for about 1 week, the other for 3 weeks. Both were able to resume normal duties but tiny nodular densities throughout both lung fields were still detectable by X-ray 2 months after exposure.

Evidence of Exposure

Sampling and Analysis

The NIOSH approved air sampling method uses a solid sorbent tube (packed column). Two previous methods used are:

1. A commercially available field kit and
2. impingement.

Direct-reading indicator tubes are still in use for spot sampling and analysis.

The NIOSH approved method for air sample analysis uses gas chromatography. Three methods previously used are:

1. Alpha-naphthylamine-nitrate spectrophotometry analysis for the field kit method,
2. colorimetric intensity measurement, and
3. phenol-disulfonic method.

The methods are not intended to be exclusive but other methods should be justified.

Allowable Exposure Limits

The Occupational Safety and Health Administration (OSHA) has adopted standards that limit exposure to nitrogen dioxide to 5 ppm (or 9 milligrams per cubic meter) of air by volume, based on an 8-hour time-weighted average exposure. (NOTE: A reduction in the standard to 1 ppm as a ceiling value has been proposed by NIOSH to prevent acute irritant effects in the lungs of workers exposed to nitrogen dioxide. In addition, the prevention of repeated acute episodes of irritancy should lessen the risk of developing chronic obstructive lung disease.)

The American Conference of Governmental Industrial Hygienists recommends an exposure limit to nitrogen dioxide of 5 ppm (or 9 milligrams per cubic meter) expressed as a Ceiling Limit which should never be exceeded.

Conclusion

There are no specific tests for diagnosing nitrogen dioxide poisoning. Diagnostic criteria for occupational nitrogen dioxide poisoning are based on meeting the following:

1. Confirmed history of occupational exposure to nitrogen dioxide,
2. clinical findings as outlined in this guide,
3. blood platelet may increase 10 to 100% above normal,
4. methemoglobin determination may be helpful,
5. carbon dioxide in the blood may be increased,
6. X-rays may show chemical pneumonitis or pulmonary edema, and
7. pulmonary function tests.

X-ray findings and lung function tests results are of diagnostic value but diagnosis cannot be based on these findings alone.

The clinical findings or effects of inhalation exposure to nitrogen dioxide may simulate other diseases such as pneumonia, acute bronchitis, or even cerebral hemorrhage. These entities can usually be excluded by an accurate medical history.

The history of acute nitrogen dioxide exposure is characteristic: initial symptoms subside upon termination of exposure, followed by a sudden onset of pulmonary edema (excess fluid in lung tissue) after a latent period of 3 to 30 hours.

Noise

Introduction

Occupational hearing loss is a slowly induced deafness produced by loud sound in the workplace, over a period of time varying from months to years. Hearing loss may also be immediate, such as that caused by a sudden, loud explosion.

Exposure to intense noise for an extended period of time causes hearing loss which is either temporary, permanent, or a combination. Hearing loss is referred to as temporary threshold shift (TTS) or permanent threshold shift (PTS).

Temporary hearing loss means that the person's ability to hear will return to normal when he is absent from the source of the noise for a period of time. In cases of permanent hearing loss, there is never a return of hearing to the previous threshold.

Disability from hearing loss results from the decreased ability to identify spoken words or sentences. Speech is composed of frequencies between the range of 250 and 3,000 Hertz (Hz). Hertz is a unit of measurement of the frequency, sometimes referred to as cycles per second (cps).

The hearing level for speech is a simple arithmetic average of the hearing levels at frequencies of 500, 1,000, and 2,000 Hz. (Sataloff, J.; and Michael, P. 1973. *Hearing Conversation* Springfield, Illinois: Charles C. Thomas Co.) Healthy young ears are able to hear sounds through the frequency range from 20 to 20,000 Hz.

Hearing loss from repeated exposure to excessively loud noise usually occurs in the 4,000 Hz. area. Since this is above the frequency range of the normal spoken voice, an individual may suffer a decrease in hearing and not be aware of it.

A person's ability to hear high frequencies decreases with age just as his ability to read fine print decreases with age. The hearing deficiency is called presbycusis. The effects of age on hearing and vision are not the same for all individuals. This adds to the problem of determining if a hearing loss is occupational in origin, or the result of the aging process. However,

presbycusis tends to start in the 8,000 Hz. frequencies, whereas hearing loss due to noise is usually in the 4,000 to 6,000 Hz. range. Recruitment is present in early cases of deafness due to excessive noise, but not in presbycusis. Recruitment is the inability to understand speech in the presence of surrounding noise. The louder the words are spoken, the more difficult it is to understand them. Noise induced hearing loss usually is bilateral (exists in both ears).

In cases of occupational hearing loss, any accompanying hearing loss due to presbycusis is usually accounted for by allowing a reduction of ½ decibel (dB) for each year of age over the age of 40. The decibel (dB) is a unit for measuring the loudness or intensity of sound. For example, the sound pressure level (loudness) of conversation is between 60 or 70 dBA, a compressor is in the range of 120 dBA, and a turbojet engine 160 dBA. Because noise is not one frequency but is composed of a mixture of many frequencies, the so-called A-weighted technique is used for measurement of intensity. It is an average of the intensity of the different frequencies and is expressed as dBA.

Excessive noise can cause physiological problems other than hearing loss. It can have an effect on emotions, produce irritability, increase blood pressure and heart rate, and produce nausea. These effects on the worker in a noisy environment are not well defined as an occupational illness, but may have an affect on the quality and efficiency of the work performed.

Occupations With Potential Exposures to Noise

boiler rooms	paper manufacture
chemical products manufacture	paper products manufacture
construction	petroleum refining
corrugated paper manufacture	plastics manufacture
demolition	plastic products manufacture
earth moving equipment operators	power plant operators
electrical equipment manufacture	printing
engine rooms	primary metal processing
fabricated metal product manufacture	quarrying
farm equipment operators	rubber manufacture
food processing	rubber products manufacture
	shipbuilding
	steel making
	stone products industries (cement mills)

foundries
furniture manufacture
glass manufacture
lumbering
metal forming
metal machining
metal working
mining — open pit
mining — underground
ordinance manufacturing

stone workers
textile manufacture
transportation equipment
operators
trucking
tunneling
wood products manufacture

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

In the Medical History, the following should be considered:

1. Any previous history of diseases or injury involving the auditory nerve, capable of causing hearing loss, either as a direct result of disease or injury, should be evaluated to determine if present findings are associated with previous disease or injury.
2. In cases of possible occupationally induced hearing loss, it is important to evaluate the claimant's medical history pertaining specifically to diseases and conditions of the ear and auditory nerve. Included are the following considerations:
 - Previous ear trouble and disease,
 - extent of known hearing loss,
 - dizziness,
 - tinnitus (ringing in the ears),
 - treatment with drugs (ototoxic drugs),
 - head injury, and
 - estimate of subject's own hearing ability.

In the occupational history, consider also that exposure to noise may be from a hobby or from home activities. Included are the following:

- Woodworking,
- metal working,
- loud music in any form from any source,

- auto repair,
- operating noisy equipment (tractors, lawn mowers, etc.),
- traffic,
- pistol, rifle, or shotgun firing,
- auto racing, and
- operating motorcycles, snowmobiles, or boats.

Signs and Symptoms

Early signs of hearing loss are:

- Inability to understand spoken words in a noisy environment,
- need to look at the person speaking to understand words,
- familiar music may not sound the same, and
- changes occur in routine audiometric examination.

NOTE: The American Academy of Ophthalmology and Otolaryngology *Guide for Determining Hearing Impairment* is a useful guide for assessing handicaps due to hearing loss.

Laboratory and Clinical Evaluations

Other Tests

A thorough clinical examination of the ear should include the following:

- External ear examination for scars or malfunctions,
- otoscopic examinations of ear drum (typanic membrane) for any abnormalities,
- examination of nose, throat, and nasopharynx for any abnormalities,
- eye reflexes are noted (pupil and cornea),
- examination with tuning fork,
- pure tone audiometric examination,
- bone conduction studies,
- speech reception testing for threshold and discrimination,
- recruitment and tone decay studies, and
- other tests may be conducted.

If baseline and/or periodic audiometric examinations were conducted by the employer, these test results should be obtained for comparison with present audiometric test results.

In addition, the following should be considered:

The audiometric (pure tone) examination is one of the best clinical means of measuring hearing loss, although other examinations as referred to above should also be completed. The audiometric examination should be administered only by trained, competent personnel, and the test results interpreted by a competent otologist or audiologist.

The frequencies monitored by audiometry should cover the range of 250 Hz through 8,000 Hz. Factors which may alter audiometric test results include the following:

- Faulty or maladjusted equipment,
- inaccurate or misunderstood instructions from the test operator,
- wax in the ears,
- head cold or allergy, and
- exposure to intense noise 18 hours or less prior to the test.

Epidemiology

The sources of hearing loss and other auditory damage are well documented in the scientific literature, and many studies have shown the levels and durations of noise that are liable to cause such effects.

The following reports of dose-response relationships are taken from NIOSH⁸². NIOSH summarized audiometric surveys carried out between 1960-1970 in the United States and other countries. The following sections in quotes are all from NIOSH:

Coles and Knight⁸³ reported a study of workers in diesel-engine testing. "Maximum noise level 116 dB. Of six men who worked continuously in the intense noise of the two-stroke test-house (average period 3½ years) all had losses of 45-60 dB in one or both ears at 3.4 and 6 KHz, and none could be accounted for by an aging factor."

Yaffe and Jones⁸⁴ reported a study of Federal penitentiary workers (textiles, wood products, sheet metal, brush, shoe and clothing manufacture, and printing) where octave band noise levels ranged from 75-110 dB. "Those levels which exceeded octave band criteria produced significant hearing threshold shifts at 3, 4, and 6 KHz after 24 months exposure. The locations producing the largest shifts were cotton mill twist and weaving departments, woolen mill weaving departments, and furniture mills."

Schneider⁸⁵ reported a study of 294 jobs in chemical works involving 691 individuals. "Data divided into 4 noise exposure groups based on octave band criteria indicated that the group exceeding criteria more than 10% of the time experienced a permanent threshold shift of 1 dB per year at 2, 3, and 4 KHz. For the group near criteria exposure, most of the hearing loss occurred within the first five or so years."

Brohm and Zlamal⁸⁶ reported a study of noise in the cabs of heavy trucks ranging from 90-110 dB. Examinations were made of 51 truck drivers and in each case a loss of hearing was determined."

Mancini and Stancari⁸⁷ reported a study of 50 fettlers. "Men working in 9 foundries with noise levels of 92-100 dB. In men who had been working for more than 5-6 years in noisy conditions almost all frequencies were involved; those who had worked less than 2-3 months in noisy conditions showed a loss varying from 30 to 50 dB at 400 Hz." Chadwick⁸⁸ reported a study of 12 men exposed to noise from industrial gas-turbine engine noise. "Noise levels reached as high as 113 dB flat... the low-tone loss in just over two years was in the region of 10 dB and from 2,000-4,000 Hz was in the order of 20 dB... the average loss for the speech frequencies was... eight times more than that to be expected in a more conventional industry with a known noise hazard."

Filin⁸⁹ reported a study of drivers of self-propelled jumbos in underground ore mining. "Noise levels of 127 dB at frequencies between 1,000 Hz and 8,000 Hz. Hearing loss in 91 of 135 miners examined; after 10 years' work, 28 dB loss at 4,000 Hz."

Weston⁹⁰ reported a study of agricultural tractor drivers. "53 drivers of tractors of different horsepower; audiograms showed greater impairment in inland drivers where the tractors are of

higher power and exposure is for longer periods than on coast-plain farms. Noise levels ranged from 92 dB to 106 dB, occasionally as high as 114 dB."

Cohen⁹¹ reported a study where "hearing levels for heavy earth-moving equipment operators, paper bag workers, and airport ramp workers were compared with those of non-noise exposed groups. Noise encountered ranged from 80-120 dB (A-weighted sound level). The hearing loss levels of the heavy earth equipment operators were found to be significantly higher than the non-noise exposed groups. The paper bag workers had higher hearing loss levels but not as high as the earth equipment operators. The airport ramp personnel, however, had the lowest hearing loss levels, probably due to the intermittency of their exposures.

Burns⁹² reported a study of 759 employees in 32 various industrial factories with noise levels ranging from 78 to 109 dBA. "A relationship between noise level, exposure duration and hearing level was defined with two parameters: audiometric frequency and percentage of persons expected to exceed a specific hearing level. A-weighted sound level was found to be adequate for estimating hearing level for the industrial noises measured."

Stone⁹³ reported a study of "3,116 employees of 9 steam electric generating plants and 2 hydroelectric plants were tested. Noise levels from assorted equipment ranged from 91 to 127 dBA, the more intense values associated with coal hoppers, turbine generators and pumps, and forced draft fans. Prevalence of hearing impairment (defined by hearing levels averaging more than 15 dB (reASA 1951) at test frequencies of 0.5, 1, and 2 KHz) varied from 4.7 percent for the younger workers having less than two years of service to 31.9 percent for the oldest workers with 26 years or more experience. Boilermakers, heavy equipment operators, and conveyor car operators as classes had high incidences of hearing impairment."

Evidence of Exposure

Measurement Methods

The current basis for evaluating continuous industrial noise exposures is the A-weighted sound level measurement. The A-weighted network is one of the several standardized frequency

weighting networks on most sound measuring equipment. The A-scale is thought to rate noise in a similar manner as the human ear. Measurements are A-weighted, slow response for the evaluation of continuous noise. If only octave band analyses are available, equivalent A-weighted levels can be calculated for comparison to current standards.

There is a wide variety of instrumentation available for the evaluation of noise from very simple equipment to extremely sophisticated equipment used by acoustical engineers and consultants. The Occupational Safety and Health Administration (OSHA) proposes that noise level measurements for steady-state or continuous noise be made "with a sound level meter confirming as a minimum to the requirements of the ANSI Z1.4-1971, Type 2, and set to an A-weighted slow response or with an audio-dosimeter of equivalent accuracy and precision." Measurements should be taken as close as possible to the hearing zone of the worker whose noise exposure is being evaluated.

For the measurement of impact noise (such as that from a drop hammer), an impact noise meter with peak hold capability should be used. This type meter should conform to the requirements of ANSI Z1.4-1971, Type 1.

Sound level measuring instrumentation should be calibrated with an acoustical calibrator the day of the study, preferably before and after the noise measurements.

Allowable Exposure Limits

The OSHA allowable limits for continuous noise are as follows:

DURATION PER DAY HOURS	SOUND LEVEL dBA SLOW RESPONSE
8	90
6	92
4	95
3	97
2	100
1½	102
1	105
½	110
¼ or less	115

OSHA indicates that "when the daily noise exposure is composed of two or more periods of noise exposure of different levels, their combined effect should be considered, rather than the individual effect on each.

"If the sum of the following fractions $C_1/T_1 + C_2/T_2 + \dots C_n/T_n$ exceeds unity, then the mixed exposure should be considered to exceed the limit value. C_n indicates that the total time of exposure at a specified noise level, and T_n indicates the total time of exposure permitted at that level."

The OSHA allowable limit for impact noise should not exceed 140 dB peak sound pressure level. NOISE ABOVE these limits may cause damage, and the exact level of safety has not yet been determined.

Conclusion

A careful otologic examination and hearing evaluation as outlined above are necessary for an accurate diagnosis. Criteria for diagnosing occupational hearing loss due to exposure to noise include the following:

1. Time and nature of onset of the loss,
2. pattern of hearing loss for different frequencies,
3. confirmed history of occupational exposure of many months or years to noise level in excess of accepted standards, and
4. clinical findings of otologic examination and medical history.

Functional hearing impairment exists when there is no organic cause for the apparent deafness, and the inability to hear results chiefly from psychological or emotional factors.

Acoustic trauma is hearing loss resulting from a loud noise, such as an accidental explosion. If the causative noise occurs on the job, the hearing loss would be occupational.

Crystalline Silica

Introduction

The crystalline form of silica, silicon dioxide, is widely distributed in nature and constitutes a major portion of most rocks and their products such as soils and sands. Silica occurs in three principal crystalline forms: Quartz, tridymite, and cristobalite. During many industrial operations such as drilling blast holes and grinding stone objects, a dust of silica particles can be formed. Inhalation of these very sharp, insoluble particles into the lungs can produce the disease silicosis, which is a form of pneumoconiosis characterized by the formation of small, discrete fibrous nodules in the lungs.

The silicosis nodule is composed of circular bundles of collagen (a fibrous insoluble protein) resulting in fibrous nodules measuring 1 to 10 millimeters in diameter. The nodules are found in lymphatics around blood vessels, beneath the pleura (the membrane covering the thoracic cage and lungs), and in groups of lymph nodes within the chest cavity. The upper lobes and hilar lymph nodes are more severely affected than the lung bases. The nodules may fuse to become "progressive massive fibrosis."

Silicosis may be of an acute or chronic nature. Acute silicosis refers to a rapidly developing lung disease which may occur in workers exposed to high levels of respirable free silica over a relatively short period ranging from a few weeks to 4 or 5 years. Eight to 18 months may lapse from the time of first exposure to the onset of symptoms which include progressive dyspnea (labored or difficult breathing), fever, cough, and weight loss. After development of symptoms, survival time is likely to be short. This disease has been most often reported in manufacturers and packers of abrasive soap powders, in sand-blasters working in enclosed tanks, and in high-power drillers of tunnel rock.

Chronic pulmonary silicosis, the type commonly encountered in industry, is similar to acute silicosis but usually develops after many years of exposure to silica dust and may take many more years to progress. This disease occurs most frequently in the mining industry but is also seen in other industries such as

potteries, foundries, stone cutting and finishing, tile and clay producing, and glass manufacturing.

Both acute and chronic silicosis have a definite tendency to progress whether or not the worker remains exposed to dust. Tuberculosis is a common complication of silicosis. Silicosis is also associated with pulmonary hypertension and cor pulmonale (hypertrophy or failure of the right ventricle).

Lung function tests and chest X-rays classed according to the ILO U/C system (international classification of radiographs of the pneumoconioses) are useful in diagnosing and following the progression of silicosis.

Exposure to crystalline silica can result in the occupational dermatosis, silica granuloma (a granular tumor or growth usually of lymphoid and epitheloid cells).

The common names of some minerals that contain varying amounts of crystalline silica follow:

Common Names

agate	jasper
amethyst	muscovite
beach sand	pegmatite
chalcedony	quartz
chert	quartzite
chrysoprase	rock crystal
citrine quartz	rose quartz
cristobalite	sand
diatomaceous earth	sandstone
feldspar	sardonyx
flint	silica flour
free silica	silican hydride
ganister	tridymite
granite	tripoli
gritstone	

The following is a list of trade names of products that either consist of or contain silica:

Trade Names

A 175	Cab-o-Sil H-5
Acticel	Cab-o-Sil L-5
Aerogel 200	Cab-o-Sil MS-7
Aerogel	Cab-o-Sil M-5
Aerosil	Cabosil N 5
Aerosil 175	Cabosil ST-1
Aerosil 200	Carplex
Aerosil 300	Carplex 30
Aerosil 380	Carplex 80
Aerosil A 175	Celite
Aerosil A 300	Celite Superfloss
Aerosil BS-50	Coloidal Silica
Aerosil E 300	Colloidal Silicon Dioxide
Aerosil K 7	Corasil II
Aerosil M-300	Crystallite A 1
Aerosil TT 600	Diatomaceous Silica
Aerosil-Degussa	Dicalite
Amorphous Silica Dust	Dri-die
Aquafil	Extrusil
C.I. 77811	Fossil Flour
C.I. Pigment White 27	Gasil
Cab-o-Sil	HK 125
HK 400	Silica (SiO ₂)
HI-Sil-C	Siliceous Earth
Iatrobeads 6RS8060	Silicic Anhydride
KS 160	Silicon Dioxide
KS 300	Silicon Oxide (SiO ₂)
KS 404	Silikil
Ludox	Silikolloid
Ludox HS 40	Siloxid
Manosil VN 3	Sipur 1500
Milowhite	Snowtex
Min-U-Sil	Snowtex 30
Minusil 5	Snowtex N
Minusil 30	Snowtex O
Nalcast P1W	Snowtex OL
Nalco 1050	Snowtex C
Nalfloc	Super-Cel
Nalfloc N 1050	Superfloss
Neosil	Suprasil
Neosyl	Syton 2X
Nipsil VN 3	Syton WL
OK 412	TK 900

Porasil
Positive Sol 130M
Positive Sol 232
Pregel
Protek-Sorb
Quso G 30
Quso 51
RD 8
Santocel CS
Santocel 62
Santocel Z
Si-O-Lite
Siflox
Silanox
Silanox 101

Tokusil Gu-N
Tokusil TPLM
U 333
Ultrasil VN 3
Ultrasil VH 3
Ultrasil VN 2
Verticurine
Vitasil 220
Vulcasil S
Wessalon S
White Carbon
Zeofree 80
Zipax
Zorbax SIL

Occupations with Potential Exposures to Crystalline Silica

abrasive blasters	cement mixers
abrasives makers	ceramic workers
agriculture	chemical glass makers
auto garage workers	chippers
bisque-kiln workers	coal miners
brick layers	construction workers
brickmakers	cosmetics makers
buffers	cutlery makers
buhrstone workers	diatomaceous earth calciners
carborundum makers	electronic equipment makers
casting cleaners, foundry	enamellers
cement makers	farming
fertilizer makers	quartz workers
fettlers	refractory makers
flint workers	road constructors
foundry workers	rock crushers
furnace liners	rock cutters
fused quartz workers	rock drillers
glass makers	rock grinders
glaze mixers, pottery	rock screeners
granite cutters	rubber compound mixers
granite workers	sand cutters
grinding wheel makers	sand pulverizers
grindstone workers	sandblasters
hard rock miners	sandpaper makers
insecticide makers	sandstone grinders
insulators	sawyers

jewelers
jute workers
kiln liners
lithographers
masons
metal buffers
metal burnishers
metal polishers
miners
mortar makers
motormen
oil purifiers
oilstone workers
optical equipment makers
paint mixers
polishing soap makers
porcelain workers
pottery workers
pouncers, felt hat
pulpstone workers
quarry workers

scouring soap workers
silica brick workers
silicon alloy makers
silver polishers
slate workers
smelters
sodium silicate makers
spacecraft workers
stone bedrubbers
stone cutters
stone planers
street sweepers
subway construction workers
tile makers
tooth paste makers
tube mill liners
tumbling barrel workers
tunnel construction workers
whetstone workers
wood filler workers

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

The following should be considered:

- Other pneumoconioses,
- sarcoidosis (a chronic granulomatous disease),
- tuberculosis,
- fibrosing alveolitis (hardening of lung tissue),
- carcinomatous lymphangitis (spread of cancer via lymph channels), and
- pulmonary hemosiderosis (iron deposits in lung tissue).

A respiratory questionnaire, such as that in Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms.

Nonoccupational Exposure

Potential nonoccupational sources of silica dust include:

- Ceramics, pottery and related hobbies,
- work with plasters, mortars, or cements having a high silica content, and
- rock working hobbies (carving, cutting, chiseling).

Signs and Symptoms—Simple Silicosis

Simple silicosis may be nonspecific early in the course of the illness and have little effect on ventilatory capacity. Generally, the only finding is nodulation of the lungs as seen on a chest X-ray. Approximately 20 to 30% of the persons with simple silicosis go on to develop complicated silicosis despite removal from a silica environment.

Signs and Symptoms—Complicated Silicosis

- Cough and sputum (productive cough),
- labored or difficult breathing (dyspnea),
- wheezes (rhonchi),
- crackling sound (crepitations), on examination of the lungs,
- chest pain,
- bluish or grayish discoloration of the skin (cyanosis),
- decreased pulmonary function, and
- chest X-ray shows nodulation of the lungs.

Progression of involvement is related to continued exposure, increasing age, smoking, and pulmonary infections. Severe pulmonary fibrosis may occur in 20 to 30% of workers who develop silicosis. In advanced cases, the following may occur:

- Chronic bronchitis,
- obstructive pulmonary disease (emphysema),
- cardiac failure may occur, and
- death may occur from respiratory failure.

Silicosis may also co-exist with tuberculosis. It should also be noted that silicosis favors the growth of the tubercle bacilli. However, silicosis may suppress the usual features of epithelioid cell proliferation, giant cell formation, and lymphocytic reaction.

Laboratory and Clinical Examinations

Additional data that will assist in arriving at a correct diagnosis are:

Pulmonary Function

- reduced forced vital capacity (FVC)
- decreased 1 second forced expiratory volume (FEV₁)
- reduced diffusing capacity
- reduced maximal breathing capacity

NOTE: These test results indicate impairment of lung function; there are no lung function tests which specifically assay for silica.

Pulmonary impairment including oxygen desaturation on exercise progresses rapidly in complicated silicosis. Associated chronic bronchitis may be a key factor in the decreased pulmonary function.

Chest X-Ray

The following classifications are used in charting the possible progression of silicosis:

- Simple silicosis: multiple opacities of various sizes and densities (from less than 1.5 to 10 millimeters) may be diffused over the entire lung field; the opacities may be calcified. Hilar nodes may develop “egg shell” calcification.
- Complicated silicosis: conglomerate masses are greater than 1 centimeter in diameter and are usually found in upper and middle zone. Large sausage-shaped masses which may be surrounded by emphysematous bullae may appear in advanced cases.
- Caplan’s syndrome: occurs when larger nodules appear against the background of simple silicosis; rheumatoid disease may be associated.

Radiographs should be classified by the ILO U/C scheme.

NOTE: Because silicosis shares the X-ray appearance of at least 20 other chest diseases, X-ray findings alone cannot be the basis of diagnosis. However, most workers' compensation insurance acts use X-ray criteria for compensation purposes.

Acute, high exposure can result in death without any X-ray evidence of silicosis. It should also be noted that nodular densities can be induced by silica, iron, tin, and barium without associated fibrosis.

Epidemiology

The relationship between crystalline silica and respiratory impairment, including silicosis, has been demonstrated in various epidemiologic studies. The available information indicates that 1 or more of the following factors may have important etiologic significance in the development of lung disease: The particle size of the crystalline silica dust, the concentration of the free crystalline silica, possible synergistic action of other ions present, differences in individual susceptibility, and the presence of a concomitant infection (especially tuberculosis). This should be taken into consideration when interpreting the following studies:

Musk et al.⁹⁴ reported a 4-year study of 688 granite shed workers who were exposed to mean silica dust concentrations less than the threshold limit value of 100 micrograms per cubic meter for respirable free silica. Excessive average yearly decrements in pulmonary function were observed: 75 to 84 milliliters per year for forced vital capacity (FVC) and 53 to 67 milliliters per year for 1 second forced expiratory volume (FEV₁). Observed decrements were independent of exposure group (i.e., cutter, sculptor, polisher, sandblast area worker, etc.) and could not be accounted for by cigarette smoking. In 528 additional granite shed workers, decrements in ventilatory capacity were measured for 1, 2, or 3 years and were of the same order of magnitude.

Prospective studies of lung function in working populations and in the general population have shown that FEV₁ and FVC in healthy men decrease at a rate of less than 40 milliliters per year after the age of 25 years with a greater rate of decline for cigarette smokers than for those who have never smoked. Subjects with chronic obstructive pulmonary disease exhibit a rate of decline of FEV₁ and FVC of approximately 80 milliliters per year.⁹⁴

In a study of 727 mine workers from a representative group of metal mines, Dreessen et al.⁹⁵ reported an incidence of silicosis in 25% of the workers exposed for more than 6 years to silica dust concentrations of 10 to 23 million particles per cubic foot (mppcf) having a free silica content of 20 to 40%. No cases of silicosis were observed in workers whose exposures did not exceed an average of 18 mppcf and whose employment exposure did not exceed 10 years. The severity of pulmonary fibrosis among cases of silicosis increased greatly with increasing length of employment.

Flinn et al.⁹⁶ reported a study of 2,516 workers who manufactured pottery products in 9 potteries. Workers were exposed to dust containing from 1 to 39% quartz and having an average particle diameter of 1.2 micrometers. The following table summarizing data from the report is taken from the NIOSH Criteria Document on crystalline silica.⁹⁷

**RELATION OF DUST CONCENTRATION AND
LENGTH OF EMPLOYMENT IN THE
POTTERY INDUSTRY TO SILICOSIS***

Dust Concentration (million particles per cubic foot)	Years in Pottery Industry				
	0-9	10-19	20-29	30-39	Over 40
0-3.9:					
Cases of silicosis	-	1	1	-	-
Workers exposed	481	223	65	21	8
Percentage	0	0.4	1.5	0	0
4-7.9:					
Cases of silicosis	1	6	26	27	29
Workers exposed	321	198	110	53	34
Percentage	0.3	3	24	51	85
8-15.9:					
Cases of silicosis	-	8	5	10	10
Workers exposed	176	119	25	17	14
Percentage	0	7	20	59	71

Over 16:

Cases of silicosis	13	33	10	5	4
Workers exposed	363	174	21	7	5
Percentage	4	19	48	71	80

(Flinn et al., 1939 and NIOSH, 1974)

*Includes 1st, 2nd, and 3rd stage cases.

Flinn et al. suggested that new cases of silicosis would not develop if the dust concentration in potteries could be brought below 4 mppcf.

Rajhans and Budlovsky⁹⁸ reported a study of 1,166 production workers in 10 brick and tile plants in Ontario in which no cases of silicosis were found. Workers had been exposed for 1 to 30 years to mean workplace dust concentrations ranging from 12 to 1,026 mppcf. Average respirable dust concentrations ranged from 1.05 to 4.26 milligrams per cubic meter and had a free silica content of approximately 13%. Rajhan and Budlovsky suggested that progression of the silicotic process was inhibited by the 14% alumina content in the clays and tiles used to manufacture brick and tile. In an earlier study in 3 British brick plants, Keatinge and Potter⁹⁹ reported similar findings and concluded that excessive occupational hazards were not associated with brick making.

Theriault et al.^{100,101,102} reported a comprehensive study of approximately 800 workers from 13 occupational groups in 49 granite sheds. Granite dust and quartz were reported to cause significant decreases in FVC, FEV₁, and total lung volume but not in residual volume. Of 784 workers, 233 had X-rays classed as abnormal which showed opacities compatible with pneumoconiosis.¹⁰³ Workers with abnormal X-rays had been exposed to an average of 2.3 times more dust than workers having normal X-rays. Theriault et al. concluded that pulmonary function measurements are more sensitive indicators of the effect of granite dust than chest roentgenograms.

In a 1-year study of 869 workers in 5 diatomite plants, Cooper and Cralley¹⁰⁴ suggested that nearly all presumptive abnormal chest roentgenograms found in 156 workers were associated

with exposure to calcined diatomite containing 15 to 61% cristobalite. The extent and severity of pneumoconiosis also appeared to correlate with length of exposure. For all plant operations, airborne dust concentrations ranged from 1 to 66 mppcf, and the median particle size was 1.1 micrometers.

Evidence of Exposure

Sampling and Analysis

The NIOSH approved air sampling method uses mechanical filtration. Three methods previously used are:

1. Impingement,
2. cascade impactor, and
3. electrostatic precipitator

The NIOSH approved methods for samples analysis are:

1. Gravimetric plus X-ray diffraction,
2. gravimetric plus colorimetric analysis, and
3. gravimetric plus infrared spectrophotometry.

Three methods previously used are:

1. Electron microscopic,
2. exo-electron emission, and
3. differential thermoanalysis.

The above methods are not intended to be exclusive, but other methods should be justified.

Allowable Exposure Limits

Standards adapted by the Occupational Safety and Health Administration (OSHA) have recommended that limits for dusts containing greater than one percent of silicon dioxide (SiO_2) are to be calculated from the following formula:

- quartz (respirable mass fraction)
(microscopic counting)

$$\frac{250^*}{\% \text{ SiO}_2 + 5} \quad \text{million particles per cubic foot}$$

- quartz (respirable mass fraction)
(gravimetric analysis)

$$\frac{10^{**}}{\% \text{ SiO}_2 + 2} \quad \text{milligrams per cubic meter}$$

- quartz (total dust)
(gravimetric analysis)

$$\frac{30}{\% \text{ SiO}_2 + 2} \quad \text{milligrams per cubic meter}$$

- cristobalite
1/2 the value calculated from the mass or count formula for quartz
- tridymite
1/2 the value calculated from the mass or count formula for quartz

*The percent of crystalline silica in the formula is the amount determined from airborne samples except in those instances in which other methods have been shown to be applicable.

**Both concentration and percent quartz for the application of this limit are to be determined from the fraction passing a size selector with the following characteristics.

AERODYNAMIC DIAMETER

(um) (unit density sphere)	Percent Passing Selector
2.0	90
2.5	75
3.5	50
5.0	25
10.0	0

(NOTE: NIOSH has recommended a reduction in the standard to 50 micrograms per cubic meter as respirable free silica based on an 8-hour time-weighted average exposure.)

Conclusion

Diagnostic criteria for occupational silicosis are based on meeting the following:

- 1. Confirmed history of occupational exposure to free silica of:
 - a. a particle size capable of producing the disease (pathologic)**
 - b. sufficient intensity of exposure**
 - c. sufficient duration of exposures****
- 2. X-ray findings as outlined above (in accordance with ILO U/C International Classification of Radiographs of Pneumoconioses 1971)**
- 3. clinical findings compatible with silicosis as outlined above**
- 4. lung function test results that are indicative of respiratory dysfunction associated with the formation of fibrous tissue within the tissue spaces (interstitial fibrosis)**

Lung biopsy and lung sections collected after death remain the only unequivocal methods of making a definitive diagnosis.