

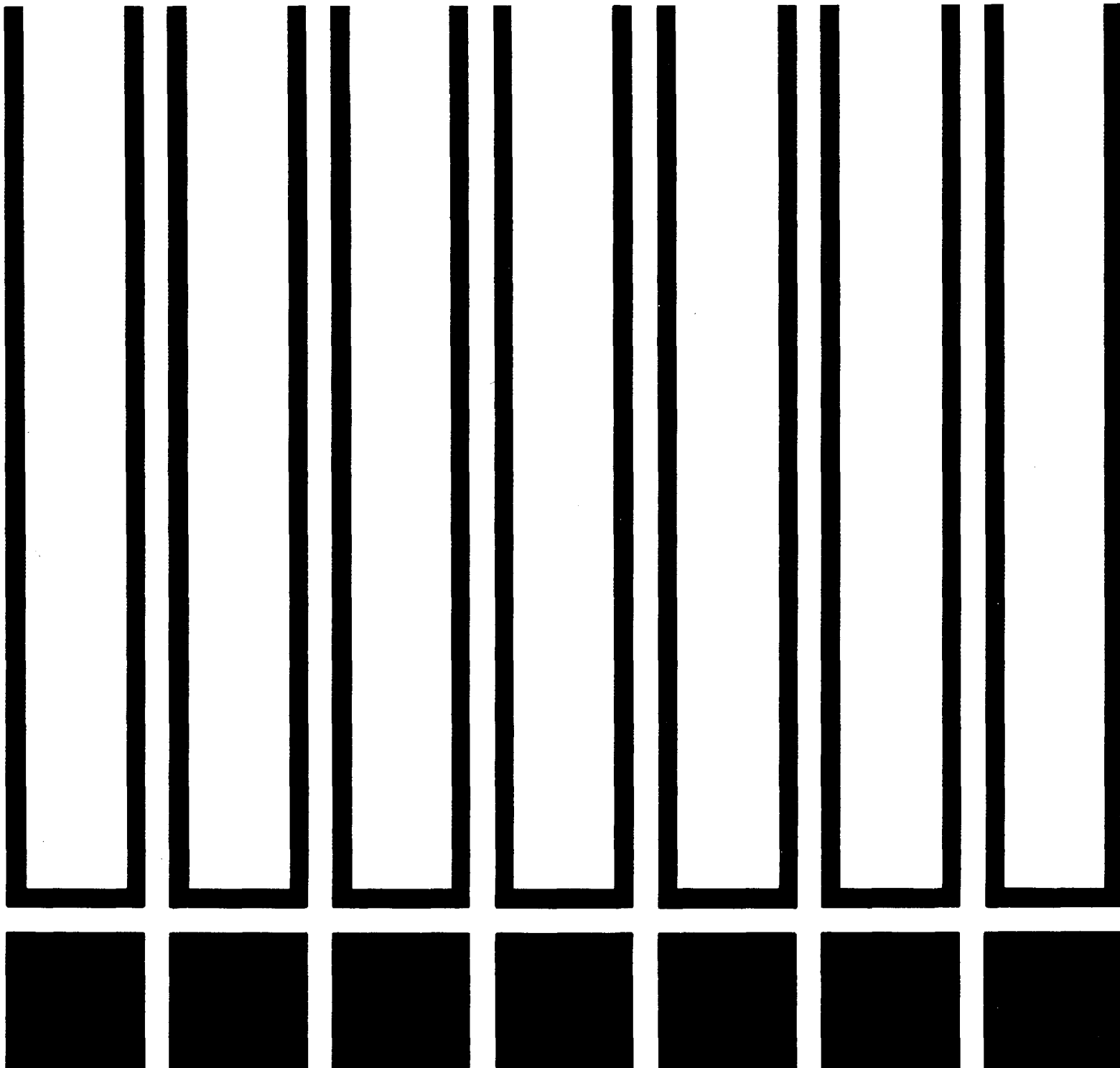
**NIOSH**

**criteria for a recommended standard . . . .**

**occupational exposure to**

**TUNGSTEN and**

**CEMENTED TUNGSTEN CARBIDE**



**U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE**

Public Health Service    Center for Disease Control

National Institute for Occupational Safety and Health

**criteria for a recommended standard....**

**OCCUPATIONAL EXPOSURE  
TO  
TUNGSTEN and  
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**U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE**  
Public Health Service  
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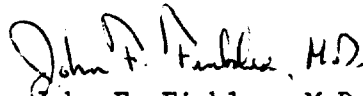
## PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. The National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices, to provide relevant data from which valid criteria for effective standards can be derived. Recommended standards for occupational exposure, which are the result of this work, are based on the health effects of exposure. The Secretary of Labor will weigh these recommendations along with other considerations such as feasibility and means of implementation in developing regulatory standards.

It is intended to present successive reports as research and epidemiologic studies are completed and as sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on tungsten and cemented tungsten carbide by members of the NIOSH staff and the valuable constructive comments by the Review Consultants on tungsten and cemented tungsten carbide, by the ad hoc committees of the American Academy of Industrial Hygiene and the American Medical Association, and by

Robert B. O'Connor, M.D., NIOSH consultant in occupational medicine. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and professional societies that reviewed this criteria document on tungsten and cemented tungsten carbide. A list of Review Consultants appears on page vi.



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The Division of Criteria Documentation and Standards Development, National Institute for Occupational Safety and Health (NIOSH), had primary responsibility for development of the criteria and recommended standard for tungsten and cemented tungsten carbide. Sonia Berg of this Division served as criteria manager. Stanford Research Institute (SRI) developed the basic information for consideration by NIOSH staff and consultants under contract CDC-99-74-31.

The Division review of this document was provided by Richard A. Rhoden, Ph.D. (Chairman), J. Henry Wills, Ph.D., and Howard L. McMartin, M.D., with Gregory Ness (Division of Surveillance, Hazard Evaluations, and Field Studies), and Charles C. Hassett, Ph.D. John J. McFeters and Mary E. Cassinelli (Division of Physical Sciences and Engineering) submitted written comments.

The views expressed and conclusions reached in this document, together with the recommendations for a standard, are those of NIOSH. These views and conclusions are not necessarily those of the consultants, other federal agencies or professional societies that reviewed the document, or of the contractor.

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CRITERIA DOCUMENT:  
RECOMMENDATIONS FOR AN OCCUPATIONAL  
EXPOSURE STANDARD FOR TUNGSTEN AND CEMENTED TUNGSTEN CARBIDE

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I. RECOMMENDATIONS FOR A STANDARD FOR TUNGSTEN  
AND CEMENTED TUNGSTEN CARBIDE

The National Institute for Occupational Safety and Health recommends that employee exposure to elemental tungsten and its compounds and to cemented tungsten carbide dusts in the workplace be controlled by adherence to the following sections. The standard is designed to protect the health and provide for the safety of employees for up to a 10-hour work shift, 40-hour workweek, over a working lifetime. Compliance with all sections of the standard should prevent adverse effects (except sensitization) of these materials on the health and provide for the safety of employees. The recommended environmental limits for the standard are measurable by techniques that are reproducible and available to industry and government agencies. Sufficient technology exists to permit compliance with the recommended standard. Although NIOSH considers the workplace environmental limits to be safe levels based on current information, employers should regard them as the upper boundaries of exposure and make every effort to maintain exposures as low as is technically feasible. The criteria and standard will be subject to review and revision as necessary.

Approximately 70% of US tungsten production is eventually used in the manufacture of cemented tungsten carbide, where tungsten carbide is mixed with cobalt and/or other metals, including nickel, and their oxides or carbides to form a material valued for its hardness and wear resistance. The term "insoluble tungsten," used throughout this document, refers to elemental tungsten and all insoluble tungsten compounds (Table XII-1). The

term "soluble tungsten" refers to those compounds of tungsten which are soluble in water and therefore readily translocated from the lungs, skin, or gastrointestinal tract to internal organs of experimental animals (Table XII-1). For the purpose of compliance with the recommended standards, insoluble tungsten compounds include all those for which water solubility is listed as "insoluble" or less than 0.01 g/100 cc. Soluble tungsten compounds are those listed as "very soluble," "soluble," "slightly soluble," equal to or greater than 0.01/100 cc or "decomposes." Those compounds for which no solubility information is listed should be considered soluble unless it can be demonstrated that they are insoluble in water. As a rough guide for control of airborne concentrations of tungsten compounds, the standard for insoluble tungsten should apply whenever processes within a building or independent structure involve only those stages which produce insoluble tungsten compounds from other insoluble tungsten compounds, eg, starting with ammonium-p-tungstate through tungsten carbide (Figure XII-1). Whenever soluble tungsten compounds are starting, intermediate, or final products, the standard for soluble compounds should apply throughout that building, unless it can be demonstrated through differential sampling and analysis that both limits are being met as appropriate.

The term "cemented tungsten carbide" or "hard metal" refers to a mixture of tungsten carbide (WC), cobalt, and sometimes other metals and metal oxides or carbides. The tungsten carbide content of hard metal is generally 80% or more, and the cobalt content is generally less than 10%, but it may be as high as 25%. When the cobalt content exceeds 2%, its contribution to the potential health hazard is judged to exceed that of

tungsten carbide and all other components, and the recommended standard for such mixtures is based on the current US federal standard for occupational exposure to cobalt, 0.1 mg/cu m. If a future NIOSH recommendation for an occupational exposure limit for cobalt differs from the US federal standard for cobalt, this new recommendation should be considered to replace the current recommendation for an occupational exposure limit for dusts of cemented tungsten carbide containing more than 2% cobalt. If nickel is used as a binder rather than cobalt and the nickel content of the mixture exceeds 0.3%, then the NIOSH recommended occupational exposure limit for nickel of 15µg/cu m shall apply. The recommended limits for airborne tungsten are different for "insoluble tungsten," "soluble tungsten," and "cemented tungsten carbide" because of their differing potential biologic effects and inherent toxicities. These criteria and the recommended standards shall apply to places of employment involved in the manufacture, use, storage, or handling of any of the defined materials.

The "action level" is defined as one-half the appropriate recommended time-weighted average (TWA) environmental limit. "Occupational exposure to cemented tungsten carbide, elemental tungsten, or its compounds" is defined as exposure at a concentration greater than the appropriate action level; ie, except for the case of cemented tungsten carbide products in which nickel is the binder, when the NIOSH definition of occupational exposure to nickel shall apply [1]. Exposures to airborne tungsten concentrations equal to or less than one half of the workplace environmental limits, as determined in accordance with Section 8, will not require adherence to the following sections, except for Sections 3(a), 4(b), 4(c), 5, 7, and 8. If exposure to other chemicals occurs, the employer shall also comply with the

provisions of applicable standards for these other chemicals.

The recommended environmental limits are based on data which indicate that insoluble tungsten compounds and cemented tungsten carbide may cause transient or permanent lung damage and skin irritation, while soluble tungsten compounds have the potential to cause systemic effects involving the gastrointestinal tract and CNS. No carcinogenic, mutagenic, teratogenic, or reproductive effects in humans have been reported. Compliance with the appropriate recommended environmental limits should eliminate the hazards associated with tungsten compounds and cemented tungsten carbide, except for a few individuals who may become sensitized to cobalt or nickel and have adverse reactions upon exposure to extremely small amounts of cemented tungsten carbide.

#### Section 1 - Environmental (Workplace Air)

##### (a) Concentrations

(1) Occupational exposure to insoluble tungsten shall be controlled so that employees are not exposed to insoluble tungsten at a concentration greater than 5 milligrams, measured as tungsten, per cubic meter of air (mg/cu m), determined as a TWA concentration for up to a 10-hour work shift in a 40-hour workweek.

(2) Occupational exposure to soluble tungsten shall be controlled so that employees are not exposed to soluble tungsten at a concentration greater than 1 milligram, measured as tungsten, per cubic meter of air (mg/cu m), determined as a TWA concentration for up to a 10-hour work shift in a 40-hour workweek.

(3) Occupational exposure to dust of cemented tungsten carbide which contains more than 2% cobalt shall be controlled so that employees are not exposed at a concentration greater than 0.1 milligram, measured as cobalt, per cubic meter of air (mg/cu m), determined as a TWA concentration for up to a 10-hour work shift in a 40-hour workweek.

(4) Occupational exposure to dust of cemented tungsten carbide which contains more than 0.3% nickel shall be controlled so that employees are not exposed at a concentration greater than 15 $\mu$ g of nickel per cu m air determined as a TWA concentration, for up to a 10-hour workshift in a 40-hour workweek as specified in NIOSH's Criteria for a Recommended Standard for Occupational Exposure to Inorganic Nickel.

(b) Sampling and Analysis

Environmental samples shall be collected and analyzed for tungsten as described in Appendices I and II, or for cobalt or nickel, as appropriate. Any methods shown to be at least equivalent in accuracy, precision, and sensitivity to the methods specified may also be used.

Section 2- Medical

Medical surveillance and counselling regarding its importance shall be made available to employees subject to occupational exposure to soluble tungsten, insoluble tungsten, or cemented tungsten carbide.

(a) Preplacement or initial examinations shall include:

- (1) Medical and work histories.
- (2) Physical examinations.
- (3) Specific clinical tests including, but not limited to, a 14- x 17-inch postero-anterior chest roentgenogram and pulmonary function

tests including the forced vital capacity (FVC) and the forced expiratory volume in 1 second (FEV 1).

(4) A judgment of the employee's ability to use positive pressure respirators.

(b) Periodic examinations shall be made available at least annually. These examinations shall include, but not be limited to:

(1) Interim medical and work histories.

(2) A physical examination and special tests as outlined in paragraph (a)(3) of this section.

(c) During or after examinations, applicants or employees having medical conditions which would be directly or indirectly aggravated by exposure to tungsten compounds or cemented tungsten carbide shall be counseled on the increased risk of impairment of their health from working with these materials.

(d) Initial medical examinations shall be made available as soon as practicable after the promulgation of a standard based on these recommendations.

(e) Employers shall ensure that pertinent medical records are kept for all employees exposed to tungsten compounds or cemented tungsten carbide in the workplace for at least 30 years after termination of employment. These records shall be made available upon request to the designated medical representatives of the Secretary of Health, Education, and Welfare, of the Secretary of Labor, of the employer, and of the employee or former employee.

### Section 3 - Labeling and Posting

All labels and warning signs shall be printed both in English and in the predominant language of non-English-reading workers. Illiterate workers and workers reading languages other than those used on labels and posted signs shall receive verbal information regarding hazardous areas and shall be informed of the instructions printed on labels and signs.

#### (a) Labeling

Containers of soluble tungsten compounds or cemented tungsten carbide shall carry labels which bear the trademarks of the products, the chemical name(s) of the compound(s) contained therein, and information on the effects of the particular product(s) on human health. The trademarks and pertinent information shall be arranged as in the following example:

TRADEMARK  
(CHEMICAL NAME)

MAY BE HARMFUL IF INHALED OR SWALLOWED

MAY CAUSE SKIN AND RESPIRATORY TRACT IRRITATION

Avoid unnecessary contact with eyes, skin, and clothing.  
Report any suspected skin irritation to your supervisor.  
Avoid prolonged or repeated breathing of dust/vapor.  
Use only with adequate ventilation.  
Wash hands thoroughly after handling, before eating or smoking.

#### (b) Posting

In areas where soluble tungsten compounds or cemented tungsten carbide are used, a sign containing information on the effects of the specific compounds on human health shall be posted in readily visible locations if employee exposures are above the action level. This information shall be arranged as in the following example.



TRADEMARK  
(CHEMICAL NAME)

MAY BE HARMFUL IF INHALED OR SWALLOWED

MAY CAUSE SKIN AND RESPIRATORY TRACT IRRITATION

Avoid prolonged or repeated breathing of dust/vapor.  
Avoid unnecessary contact with skin, eyes, and clothing.  
Report any suspected skin irritation to your supervisor.  
Ventilation should be sufficient to carry away dust or vapor.

Section 4 - Personal Protective Clothing and Equipment

(a) Respiratory Protection

(1) Engineering controls shall be used when needed to keep concentrations of airborne tungsten products at or below the appropriate TWA exposure limits. The only conditions under which compliance with the permissible exposure limits may be achieved by the use of respirators are:

(A) During the time necessary to install or test the required engineering controls.

(B) For operations such as nonroutine maintenance and repair activities causing brief exposure at concentrations above the TWA exposure limits.

(C) During emergencies when concentrations of airborne tungsten products may exceed the TWA exposure limits.

(2) When a respirator is permitted by paragraph (a)(1) of this section, it shall be selected and used in accordance with the following requirements:

(A) The employer shall establish and enforce a respiratory protective program meeting the requirements of 29 CFR 1910.134.

(B) The employer shall provide respirators in accordance with Tables I-1, I-2, and I-3 and shall ensure that employees use the respirators provided. The respiratory protective devices provided in conformance with Tables I-1, I-2, and I-3 shall be those approved by NIOSH or the Mining Enforcement Administration. The standard is specified in 30 CFR 11.

TABLE I-1

RESPIRATOR SELECTION GUIDE FOR INSOLUBLE TUNGSTEN COMPOUNDS\*

Concentration Range (mg/cu m, as tungsten)	Respirator Type Approved under Provisions of 30 CFR 11
Less than or equal to 25**	(1) Single-use dust respirator (2) Dust respirator with quarter-mask facepiece
Less than or equal to 50**	(1) Half-mask dust respirator (2) Supplied-air respirator with half-mask facepiece
Greater than 50**	(1) Full facepiece dust respirator (2) Supplied-air respirator with full face- piece, hood, or helmet (3) Powered air-purifying respirator (posi- tive pressure) with high efficiency filter*** (4) For abrasive-blasting with tungsten car- bide, supplied-air respirator with hood or helmet operated in pressure-demand or other positive pressure mode or with continuous flow.

\*In areas where ammonium-p-tungstate is used, a canister or cartridge which will remove ammonia is needed in addition to a particulate filter.

\*\*When an employee informs the employer that eye irritation occurs while wearing a respirator, the employer shall provide an equivalent respirator with full facepiece, helmet, or hood and ensure that it is used.

\*\*\*A high-efficiency filter is defined as one having a penetration of less than 0.03% when tested against a 0.3- $\mu$ m DOP aerosol.

TABLE I-2

## RESPIRATOR SELECTION GUIDE FOR SOLUBLE TUNGSTEN COMPOUNDS\*

Concentration Range (mg/cu m, as tungsten)	Respirator Type Approved under Provisions of 30 CFR 11
Less than or equal to 10**	(1) Half-mask dust and mist respirator (2) Supplied-air respirator with half-mask facepiece
Less than or equal to 50**	(1) Full facepiece dust and mist respirator (2) Supplied-air respirator with full facepiece (3) Self-contained breathing apparatus with full facepiece
Greater than 50**	(1) Powered air-purifying respirator (posi- tive pressure) with high-efficiency filter*** (2) Supplied-air respirator with full face- piece, hood, or helmet, continuous-flow or other positive-pressure type (3) Self-contained breathing apparatus with full facepiece in pressure demand or other positive pressure mode

\*Tungsten hexachloride in contact with water decomposes to tungsten and chlorine. Therefore, the dust and mist respirator is not satisfactory if the chlorine gas concentration exceeds the permissible level. When this occurs, the minimum acceptable respirator is a full facepiece respirator with a combination dust, mist, and chlorine canister.

\*\*When an employee informs the employer that eye irritation occurs while wearing a respirator, the employer shall provide an equivalent respirator with full facepiece, helmet, or hood and ensure that it is used.

\*\*\*A high-efficiency filter is defined as one having a penetration of less than 0.03% when tested against a 0.3- $\mu$ m DOP aerosol.

TABLE I-3

RESPIRATOR SELECTION GUIDE FOR MIXTURES OF TUNGSTEN AND COBALT

Concentration Range (mg/cu m, as cobalt)	Respirator Type Approved under Provisions of 30 CFR 11
Less than or equal to 1.0*	(1) Any dust respirator, except single-use or quarter-mask respirator* (2) Supplied-air respirator operated with a quarter-mask or half-mask facepiece
Less than or equal to 5.0	(1) Air-purifying respirator with full face- piece and high-efficiency filter** (2) Supplied-air respirator with full facepiece (3) Self-contained breathing apparatus with full facepiece
Less than or equal to 100*	(1) Powered air-purifying respirator, posi- tive pressure, with high efficiency filter** (2) Type C supplied-air respirator contin- uous-flow type, with half-mask facepiece
Less than or equal to 200	(1) Type C supplied-air respirator, contin- uous-flow type, with full facepiece, hood, helmet, or suit (2) Type C supplied-air respirator operated in pressure demand mode with full facepiece
Greater than 200	(1) Self-contained breathing apparatus with full facepiece operated in pressure demand or other positive pressure mode (2) Combination Type C supplied-air respira- tor with full facepiece operated in pressure demand mode with an auxiliary self-contained air supply

\*When an employee informs the employer that eye irritation occurs while wearing a respirator, the employer shall provide an equivalent respirator with full facepiece, helmet, or hood and ensure that it is used.

\*\*A high-efficiency filter is defined as one having a penetration of less than 0.03% when tested against a 0.3- $\mu$ m DOP aerosol.

(C) Respirators specified for use in higher concentrations of tungsten compounds or cemented tungsten carbide may be used in atmospheres of lower concentrations.

(b) Eye Protection

Eye protection shall be provided in accordance with 29 CFR 1910.133 for operations, such as grinding, which produce and scatter particulates into the air.

(c) Skin Protection

While most workers do not experience skin irritation as a result of exposure to tungsten compounds or cemented tungsten carbide, there are some who develop sensitivity. Fingerless gloves may be used during grinding of hard metal to protect the hands from abrasion. Protective sleeves of dust-proof material may be worn to prevent impact of hard-metal dust on the skin of the arms. In the absence of such gloves and sleeves, creams protective against abrasion may be applied liberally to the hands and arms to minimize contact of the skin with hard-metal dust. When skin irritation occurs, these workers should be referred to a physician for appropriate protective and therapeutic measures. When abrasive dust of tungsten carbide is likely to contact major parts of an employee's body, the employee should wear closely woven coveralls provided by the employer. The coveralls should be laundered frequently to minimize mechanical irritation from dust in the cloth.

Section 5 - Informing Employees of Hazards from Tungsten

(a) Each employee with potential for exposure to tungsten compounds or cemented tungsten carbide shall be informed at the beginning

of employment or assignment to such work areas and periodically thereafter of the hazards, relevant symptoms, appropriate emergency procedures, and proper conditions and precautions for the safe handling and use of these materials. Special mention should be made of possible sensitization to cobalt or nickel. Employees engaged in maintenance and repair shall be included in these training programs.

(b) The employer shall institute a continuing education program, conducted by persons qualified by experience or training, to ensure that all employees have current knowledge of job hazards, proper maintenance and cleanup methods, and proper respirator usage. The instructional program shall include a description of the general nature of the medical surveillance procedures and of the advantages to the employee of participating in these programs. As a minimum, instruction shall include the information in Appendix III, which shall be kept on file, readily accessible to employees at all places of employment where exposure to tungsten compounds and/or cemented tungsten carbide may occur.

(c) Required information shall be recorded on the "Material Safety Data Sheet" shown in Appendix III or on a similar form approved by the Occupational Safety and Health Administration, US Department of Labor.

#### Section 6 - Work Practices

(a) Control of Airborne Tungsten Compounds and Cemented  
Tungsten Carbide

Engineering controls, such as process enclosure and/or local exhaust ventilation, shall be used to keep concentrations of airborne tungsten products within the recommended environmental limits. Ventilation systems

shall be designed to prevent the accumulation or recirculation of these materials in the workplace environment and to effectively remove them from the breathing zones of employees. Wet process sharpening of cemented tungsten carbide material may not provide total control of dust, and local exhaust ventilation may also be required. Exhaust ventilation systems discharging to the outside air shall conform to applicable local, state, and federal air pollution regulations and shall not constitute a hazard to employees. Ventilation systems shall be subject to regular preventive maintenance and cleaning to ensure their continuing effectiveness. Continuous airflow indicators, such as water or oil manometers (marked to indicate acceptable airflow), are recommended and should be checked monthly. If such indicators are not used, the efficiency of the ventilation system shall be verified by airflow measurements taken at intervals determined by the documented maintenance history of the system.

(b) Control of Spills and Leaks

Only personnel properly trained in the procedures and adequately protected against the attendant hazards shall be assigned to shut off sources of tungsten, clean up spills, and repair leaks. Dry sweeping should be avoided.

(c) Handling and General Work Practices

(1) Care should be taken in pouring or scooping powdered materials to avoid excessive dust generation. If an employee experiences upper or lower respiratory tract irritation, special attention should be paid to work practices.

(2) Safety precautions are needed in the manufacture of filaments to protect against electrical hazards during high voltage

smelting and against explosion hazards and lacerations from very finely drawn wire.

#### Section 7 - Sanitation

For insoluble tungsten compounds, appropriate provisions of 29 CFR 1910.141 shall apply. In addition, the following precautions apply to all areas where a potential for exposure to soluble tungsten or cemented tungsten carbide exists:

(a) Eating and food preparation or dispensing (including vending machines) shall be prohibited in work areas containing soluble tungsten or cemented tungsten carbide.

(b) Employees who handle soluble tungsten, cemented tungsten carbide, or equipment contaminated with these materials shall be instructed to wash their hands thoroughly with soap or mild detergent and water before eating or smoking and shall be cautioned not to touch their faces or any other skin surfaces with contaminated fingers.

(c) Waste material contaminated with soluble tungsten or cemented tungsten carbide shall be disposed of in a manner which prevents harm to employees. The disposal method must conform with applicable local, state, and federal regulations and must not constitute a hazard to the surrounding population or environment.

#### Section 8 - Environmental Monitoring and Recordkeeping

As soon as practicable after the promulgation of a standard based on these recommendations, the employer shall conduct an industrial hygiene



survey at each location where tungsten compounds or cemented tungsten carbide are released into the workplace air to determine whether exposure to airborne concentrations of tungsten products is above the respective action levels. The employer shall keep records of these surveys, including the basis for concluding that air levels are at or below the appropriate action levels. Surveys shall be repeated at least once every 3 years and within 30 days of any process change likely to result in an increase of airborne concentrations of tungsten or its products.

If it has been determined that the environmental concentration of tungsten or its products may exceed the appropriate action level or TWA environmental limit, then the following requirements shall apply:

(a) Personal Monitoring

(1) A program of personal monitoring shall be instituted to identify and measure, or permit calculation of, the exposure of each employee occupationally exposed to any tungsten compound or cemented tungsten carbide. Source and area monitoring may be used to supplement personal monitoring.

(2) Samples representative of the exposure in the breathing zone of the employee shall be collected in all personal monitoring. Procedures for the calibration of equipment, sampling, and analysis of tungsten samples are provided in Section 1(b).

(3) For each TWA concentration determination, a sufficient number of samples shall be taken to characterize the employee's exposure during each work shift. Variations in the employee's work schedules, locations, and duties and changes in production schedules shall be considered in deciding when samples are to be collected. The number of TWA

determinations for an operation or process shall be based on the variations in location and job functions of employees relative to that operation or process.

(4) If an employee is found to be exposed above the appropriate action level, the exposure of that employee shall be monitored at least once every 3 months until the measured concentration is found to be below the action level in two successive analyses of samples taken at least 1 week apart.

(5) If an employee is found to be exposed above the appropriate TWA concentration limit, the exposure of that employee shall be measured at least once every 30 days, and control measures shall be initiated; the employee and others who work in the area while controls are being installed shall be notified of the exposure and of the control measures being implemented. Monitoring shall continue until two consecutive determinations, at least 1 week apart, indicate that the employee's exposure no longer exceeds the recommended environmental concentration limit; quarterly or triennial monitoring as applicable may then be resumed.

(b) Recordkeeping

Employers or their successors shall retain records of environmental monitoring for each employee for at least 30 years after the individual's employment has ended. These records shall include the name and social security number of the employee being monitored; job function and location within the worksite at the time of sampling; dates of measurements; sampling and analytical methods used and evidence for their accuracy; number, duration, and results of samples taken and TWA concentrations based

on these samples; and the type of personal protective equipment in use, if any. Records for each employee, indicating the dates of employment with the company and changes in job assignment, shall be kept for the same 30-year period. These records shall be made available to designated representatives of the Secretary of Health, Education, and Welfare and of the Secretary of Labor. Employees or former employees shall have access to information on their own exposures. The employees or their representatives shall be given the opportunity to observe any measurement conducted in accordance with this section and to receive a full explanation of the significance of the measurements.

## II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon that were prepared to meet the need for preventing occupational disease or injury arising from exposure to tungsten, tungsten compounds, and cemented tungsten carbide. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to "...develop criteria dealing with toxic materials and harmful physical agents and substances which will describe...exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health and to provide for the safety of employees exposed to hazardous chemical and physical agents. The criteria and the recommended standard should enable management and labor to develop better engineering controls and more healthful work practices. Compliance with the recommended standard should not be used as the final goal.

These criteria for a recommended standard for tungsten, tungsten compounds, and cemented tungsten carbide are part of a continuing series of documents published by NIOSH. The recommended standard applies to the processing, manufacturing, use, and handling of these materials in the occupational setting. The standard was not designed for the population-at-large, and any application to situations other than occupational exposures

is not warranted. It is intended to (1) protect against the development of systemic toxic effects and local effects on the eyes and skin, (2) be measurable by techniques that are reproducible and available to industry and government agencies, and (3) be attainable with existing technology.

The major concern in occupational exposure to tungsten, tungsten compounds, or cemented tungsten carbide is the potential for transient or permanent pulmonary damage. Irritation of the skin and upper and lower respiratory tract has also been associated with inhalation of, or skin contact with, these materials and should be considered in any work practices program.

There is little information now available on the toxic effects of tungsten on animals and man which is applicable to the setting of a standard for the industrial environment. Retrospective and prospective epidemiologic studies are needed to assess the potential occupational hazards from tungsten and its compounds. Also, the abilities of various tungsten compounds to irritate the skin and eyes need to be investigated. Additional short- and long-term inhalation studies on animals are necessary to assess the toxic effects of tungsten, particularly on the liver, kidneys, lungs, and central nervous system (CNS). Such studies should aim also to distinguish the effects of exposure to tungsten and its compounds from those produced by mixtures containing cobalt or nickel. Chronic studies are also needed to investigate the carcinogenic, mutagenic, and teratogenic potentials of tungsten.

### III. BIOLOGIC EFFECTS OF EXPOSURE

#### Extent of Exposure

Tungsten (atomic number 74) is listed in the periodic table under period 6 of the transition elements, along with chromium and molybdenum. Tungsten closely resembles molybdenum in all of its physical and chemical properties. In the pure metallic state, tungsten is grey and has a luster somewhat like that of steel. The physical and chemical properties and formulae of tungsten and of some of its important organic and inorganic compounds are given in Table XII-1 [2-6]. The commercial tungstate minerals are wolframite (Fe,Mn)WO<sub>4</sub>), scheelite (CaWO<sub>4</sub>), ferberite (FeWO<sub>4</sub>), and hubnerite (MnWO<sub>4</sub>). Worldwide, wolframite is the most important ore containing tungsten, whereas scheelite is the principal domestic ore [7]. Most tungsten deposits are low-grade and are upgraded by concentration techniques. The tungsten concentrates produced contain at least 60% tungstic oxide.

The total US consumption of tungsten concentrate in 1975 was 14.0 million pounds, of which 5.6 million pounds were domestically produced, 6.6 million pounds were imported primarily from Canada, Thailand, Peru, the People's Republic of China, and Portugal, and the remainder was released from government stockpiles [8].

In 1975, the United States produced 12.634 million pounds of tungsten products and consumed 12.934 million pounds, including 72.5% in alloys for cutting and wear-resistant materials, 11.5% in mill products from metal powder, 9.2% in steel, 2.9% in super alloys, 2.4% in chemicals and

ceramics, 0.03% in cast iron, and 1.47% in miscellaneous products [8].

Fairhall [9] stated that the principal health hazards from tungsten and its compounds arise from inhalation of aerosols during mining and milling operations. The principal compounds of tungsten to which workers are exposed are ammonium-p-tungstate, oxides of tungsten ( $WO_3$ ,  $WO_5$ ,  $WO_2$ ), metallic tungsten, and tungsten carbide. In the production and use of tungsten carbide tools for machining, exposure to the cobalt used as a binder or cementing substance (Figure XII-1) [10,11] may be the most important hazard to the health of the employees. Since the cemented tungsten carbide industry uses such other metals as tantalum, titanium, niobium, nickel, chromium, and vanadium in the manufacturing process, the occupational exposures are generally to mixed dusts. Potential occupational exposures to sodium tungstate are found in the textile industry, where the compound is used as a mordant and fireproofing agent, and in the production of tungsten from some of its ores, where sodium tungstate is an intermediate product (Figure XII-1). Potential exposures to tungsten and its compounds are also found in the ceramics, lubricants, plastics, printing inks, paint, and photographic industries. Some of the occupations with potential exposures to tungsten and its compounds are listed in Table XII-2 [12].

NIOSH estimates that at least 30,000 employees in the United States are potentially exposed to tungsten and its compounds, based on actual observations in the National Occupational Hazards Survey.

### Historical Reports

In 1927, at Essen, Germany, it was discovered that, if tungsten carbide was mixed with cobalt, a very serviceable cemented tungsten carbide (ie, hard metal) was obtained [7].

About 10 years after the commercialization of cemented tungsten carbide, the first report appeared on the associated health problems of the exposed workers. In 1940, Jobs and Ballhausen [13] examined 27 German workers exposed to unspecified concentrations of hard-metal dusts for 2 years. Radiographs of the chests of eight men showed areas of fine nodulation in the lungs suggestive of early pneumoconiosis.

In 1945, Schwartz et al [14] investigated the incidence of dermatitis among employees in a tungsten carbide plant for the US Public Health Service. About 20 of the 1,200 workers examined had red, papular dermatitis on the face, neck, and extremities. Although the dust was abrasive, patch tests of six workers showed that it was sensitivity to cobalt that had caused the dermatitis in those six.

Fairhall and associates [15], in 1947, conducted an epidemiologic survey of 1,802 workers in the cemented tungsten carbide industry for the US Public Health Service. They found inflammation of the conjunctivae and of the mucous membranes of the respiratory tract. Radiographs of 36 workers' lungs revealed granular or conglomerate markings, but 23 (64%) of these men had previously worked in unspecified mining or metal fabricating industries. Some workers were found to have pruritus due to sensitivity of the skin to cobalt.

In 1953, Miller et al [16] were the first to report cases of "hard-metal disease" (interstitial pulmonary fibrosis) in hard-metal grinders.



Three men who had ground finished tools for 6.5-8.5 years complained of persistent coughing, with production of sputum and exertional dyspnea. Radiologic chest examinations showed that prominent hilar shadows and lung markings abated in one worker, but persisted for more than a year in the other two workers after they had been removed from the work atmosphere. However, the clinical signs and symptoms disappeared in all three men when they were no longer exposed to the workplace dust. The authors recognized that the workers were exposed to various combinations of powdered metals but concluded that cobalt was the most likely cause of the disease.

#### Effects on Humans

Occupational exposures to tungsten and its compounds have been reported to have primary effects on the respiratory system. In the cemented tungsten carbide (hard-metal) industry, exposure to a combination of cobalt and tungsten sometimes has resulted in hard-metal disease. The pulmonary involvement presented in a number of reports was characterized clinically by upper respiratory tract irritation, coughing, exertional dyspnea, and weight loss. As the condition of the workers worsened with prolonged exposure in this environment, extrinsic asthma, diffuse interstitial pneumonitis, fibrosis, pneumoconiosis, or combinations of these were evident.

Dorsit et al [17], in 1970, described three workers exposed to tungsten carbide and cobalt as having a "true, occupational, pulmonary disorder." These men, aged 30, 36, and 57 years, had been employed for 5, 11, and 16 years, respectively, in work involving the use of diamond and tungsten carbide drills, rectifiers, and sharpeners. Workplace dust

particles were under 3  $\mu\text{m}$  in diameter, and the concentration of dust in the air varied from 380,000 to 5,600,000 particles/liter of air (2.3-62.3 mg/cu m). The workers received examinations which included blood analyses, chest X-ray studies, and pulmonary function tests. All three workers had dry coughs, progressive dyspnea on exertion and subsequently at rest, roentgenographic signs of nodular reticulation of the lungs (diffuse interstitial fibrosis) of varying degrees, decreased lung capacities, oxyhemoglobin desaturation, hypercapnia, respiratory alkalosis, polycythemia, and weight loss.

Of 380 workers in the same factory, 29 men who had been employed for up to 17 years were examined for clinical signs and symptoms of respiratory disorders [17]. Of these, 15 had dry coughs, 5 showed throat irritation, 9 had worktime dyspnea, and 3 had allergic asthma (some had more than 1 symptom).

The authors [17] also reviewed published reports of exposure to cemented tungsten carbide dust and, from this study and their own work, concluded that affected workers were characterized as 30- to 50-year-old males exposed for at least 3 years. Clinical findings included dyspnea on exertion, a dry cough, cyanosis of lips and fingers, rales, polycythemia with a concurrent rise in hemoglobin, and weight loss. Observations among workers affected included progressive decreases in circulatory and cardiac function, concurrent pulmonary infections (possibly tuberculosis), and death within 4 years of initial onset of symptoms. The authors also pointed out that pulmonary function tests were more indicative of the severity of interstitial fibrosis than were radiographs. They concluded that a mixture of tungsten carbide and cobalt appeared to be the causative

agent and advised extremely close supervision of workers in the hard-metal industry.

In 1971, Coates and Watson [18] reported 12 cases of diffuse, interstitial lung disease among tungsten carbide workers. The group consisted of 11 men and 1 woman, with a mean age of 40.5 years (range 22-55 years), who were engaged in various processes in the manufacture and grinding of cemented tungsten carbide. The exposure levels of tungsten were not given, but the processes generated particles averaging less than 2.0  $\mu\text{m}$  in diameter and produced concentrations of airborne cobalt greater than 0.1 mg/cu m. In addition to tungsten and cobalt, the processes also used titanium carbide, tantalum carbide, chromium carbide, and nickel. The mean duration of exposure before the development of symptoms was 12.6 years, with a range of 1 month to 28 years. Eight of the 12 workers died. Seven of the 12 workers were examined by one of the authors; 6 of those examined were tested for vital capacity, FEV1, and maximum voluntary ventilation (MVV).

The authors [18] noted that the pattern of clinical signs and symptoms was strikingly similar in all the workers, varying mainly in intensity. The early stages of respiratory involvement were characterized by a cough with scanty sputum, followed by dyspnea on exertion. An average weight loss of 10 pounds was noted in seven patients. Ventilatory tests of six patients showed that the average vital capacity was 50% of the predicted values (range 37-70%); the FEV1 was between 85 and 100% of the predicted values, and the average MVV was 76% of the predicted values (range 45-92%). These findings were interpreted to indicate a severe, restrictive ventilatory pattern without airway obstruction. From the

findings of low carbon monoxide diffusing capacity and decreased oxygen saturation of the arterial blood, the authors concluded that the patients had a diffuse, interstitial, fibrotic response to the xenobiotic material that had entered their respiratory systems. In two individuals, pulmonary function changes preceded abnormal chest X-ray findings. Serial chest X-ray studies showed progressive, bilateral, nodular, and linear densities with gradual involvement of major portions of the lungs. Radiographic evidence of nonspecific, diffuse interstitial fibrosis was found.

Light-microscopic examinations of the aforementioned lung specimens showed interstitial cellular infiltration with slight to marked fibrotic tissue reaction, cystic air spaces lined by an epithelium showing a metaplastic change from a squamous to a cuboidal type, and desquamation into the alveoli of type II alveolar pneumocytes [18]. Large mononuclear and occasional multinucleated, giant cells were present. Electron microscopic findings were consistent with the changes observed by light microscopic examination. By X-ray diffraction analysis, the presence of tungsten carbide was detected in all of the lung samples analyzed. Mass spectrometric analysis revealed the presence of both tungsten carbide and cobalt. One lung biopsy sample for which quantitative data were reported contained 3.0  $\mu\text{g}$  of tungsten, 2.0  $\mu\text{g}$  of titanium, and 0.1  $\mu\text{g}$  of cobalt/g of wet lung. The authors found no correlation between the amount of cobalt in the lung specimens and the severity of the disease from which the decedent had suffered. The type of interstitial lung disease seen in these people reportedly resembled the hypersensitivity pneumonitis of "farmer's lung" and beryllium granulomatosis.

Coates and Watson [18] also briefly reported on five tungsten carbide workers with normal chest roentgenographs who were described as having occupational asthmatic bronchitis. Data such as age, sex, duration of exposure, and the nature of the work involved were not described. These workers had episodic attacks of wheezing cough which abated when they left work and recurred on their return. The authors referred to this form of lung disease as "sensitization" related to preexisting lung diseases, allergic background, or both. The authors stated that no evidence existed that this disease might progress to interstitial lung disease. They emphasized that determining workers' susceptibility to interstitial lung disease and diagnosing the disease in its early stages would help to protect workers' health. Although they presented no evidence that cobalt was responsible for the observed respiratory disorders, they believed it to be the most probable cause.

In 1974, Bech [19] described clinical and radiologic findings in 12 workers with diagnoses of hard-metal disease and in 1 worker with allergic asthma among 375 hard-metal tool grinders employed in a cemented tungsten carbide plant. These 12 men ranged in age from 33 to 69 years, with an average age of 54.3 years. Although the exposure concentrations and the compositions of the dusts to which the men were exposed were not given, the typical tools contained "80-90% tungsten, 8-18% titanium carbide, 5-25% cobalt," and occasionally small quantities of tantalum, vanadium, molybdenum, and chromium. The 12 workers with hard-metal disease had been engaged in tool grinding for an average of 25.8 years (range 15-51 years), whereas the worker who had allergic asthma had worked at tool grinding for only 2 years. Eight of the men worked only part time as tool grinders.

All workers were evaluated by physical examination and chest radiography. Lung tissue obtained by biopsy from one worker, by biopsy and by autopsy from two workers, and by autopsy only from four workers were evaluated microscopically. Pulmonary function tests also were conducted on 8 of the 12 workers with hard-metal disease.

Of the 12 employees with hard-metal disease, 8 died of secondary consequences of their pulmonary disease [19]; four of the eight died of cor pulmonale and cardiac failure, whereas the other four died of emphysema. Except for differences in the degree and duration of the symptoms, the general course of the disease was similar in all 12 cases. Coughing (dry or with sputum production), exertional dyspnea, and occasional wheezing characterized the disease. Initially, the chest radiographic studies showed slight increases in lung markings and very sparse linear opacities in the mid- and lower zones. In later stages of the disease, there were marked radiologic changes with varying degrees of linear, micronodular, and nodular opacities in the midzones of the lung, prominent hilar shadows, and occasional honeycombing. Pulmonary function tests revealed restrictive ventilatory defects, with reduction in residual lung volume and total lung capacity (no values given). A low FEV1 and FEV and a marked reduction (50% of normal) in carbon monoxide uptake were noted occasionally. Examination of lung samples obtained by biopsy or autopsy revealed emphysema and varying degrees of interstitial fibrosis in seven workers. In one patient with hard-metal disease of recent onset, withdrawal from exposure and corticosteroid therapy produced clinical and radiographic evidence of improvement.

The worker with allergic asthma developed wheezing, tightness and discomfort in the chest, and exertional dyspnea which was later manifested in asthmatic attacks [19]. These signs and symptoms were least severe at the beginning of a workday and during the weekend. However, there were signs of bronchospasm upon auscultation of the chest. The asthmatic signs and symptoms receded 2-3 months after the worker's discharge from tool-sharpening work.

The author [19] concluded that, since hard-metal disease is progressive and potentially lethal, its early recognition is of the utmost importance. He also acknowledged that there was no correlation between the development of diffuse interstitial fibrosis and the onset of symptoms or the duration of exposure. The study does not contribute to the evaluation of the etiologic role of tungsten in the development of hard-metal disease.

In 1975, Baudouin et al [20] reported 5 cases of diffuse interstitial pulmonary fibrosis among 100 men of unspecified age who worked in a hard-metal factory where dusts of tungsten, titanium, tantalum, and niobium metals, of their carbides and oxides, and of cobalt metal were liberated during various operations. The concentrations, particle sizes, and specific compositions of these dusts were not indicated. The authors noted that most occupational exposures occurred in operations involving the mixing of powders and the grinding and machining of sintered pieces. The workers were screened for observable and radiographic anomalies of the chest. Of the 100 exposed workers, 15 (aged 22-65) had signs of respiratory irritation after 1 month-9 years of exposure. Five of these 15 workers developed pulmonary fibrosis within 6 months to 4 years after the onset of the irritative signs.

The investigators [20] found no predisposing factor in the personal or family backgrounds of the five subjects with pulmonary fibrosis. The irritation diminished or disappeared during vacations and weekends and reappeared during work. When the disease progressed to fibrosis, the authors found rapid weight loss, fine rales, and exertional dyspnea which was not reversible during vacations.

No significant changes were noted in the chest radiographs of the workers with irritative symptoms [20]. However, reticulonodular images that became gradually accentuated and more generally distributed were characteristic of the fibrotic stage. Spirography showed low volumes of residual air in the lungs, indicative of a constant, restrictive disorder. Lung diffusion studies showed resistance to transepithelial gas exchange, leading the authors to suggest that such studies could be used to diagnose fibrosis before the appearance of radiologic signs. Blood-gas analysis demonstrated decreased exchange of respiratory gases between the alveoli and the pulmonary capillaries. Baudouin and his associates [20] reported that immunologic examination revealed a cellular rather than a humoral immune deficiency.

In one worker, the progression of clinical, radiographic, blood-gas, and pulmonary function changes was similar to those described above, and both light and electron microscopic examinations of lung tissue removed for biopsy revealed that diffuse, interstitial fibrosis had evolved [20]. Electron microscopic examinations also revealed crystalline particles, possibly of tungsten carbide, in the macrophages. Examination of the ashed lung by X-ray fluorescence revealed the presence of a large amount of tungsten, of an amount of titanium greater than is usual, and of small



amounts of tantalum and niobium. The carbides of tungsten and titanium were demonstrated in the ashed lung by X-ray diffraction. The authors concluded that inhalation of dusts containing a mixture predominantly of tungsten metal or tungsten carbide caused diffuse interstitial fibrosis. Tungstosis was suggested as the name for this form of fibrosis, which was preceded by irritative symptoms and appeared 4-11 years after initial exposure. The authors suggested the registration of tungstosis as the 65th occupational disease in France. Although no component metal of the dusts was established as the causal agent, the authors [20] stated that tungstosis follows the inhalation of powders composed principally of tungsten metal or tungsten carbide.

Scherrer [21] described pulmonary interstitial fibrosis in three male workers exposed to tungsten, cobalt, titanium, tantalum, and niobium dusts at unspecified concentrations. The men, aged 34, 38, and 58 years, had been exposed for 3, 1, and 8 years, respectively. In each case, clinical symptoms, vital capacity, and chest radiographic findings were reported. A sample of lung was removed from the 38-year-old worker for biopsy, examined under a light microscope for histologic changes, and was analyzed spectroscopically and histochemically for the presence of tungsten and cobalt.

Although the course of the disease was somewhat different in each worker, the chief signs and symptoms were similar [21]. The illnesses of the three workers began with dyspnea, coughing, fever, and general malaise. The symptoms of the two younger workers were somewhat relieved when they left the dusty work environment. The condition of the 58-year-old man, whose illness had developed more slowly, continued to deteriorate even

after he left work. For the 38-year-old worker, a diagnosis of hard-metal pneumoconiosis (pulmonary interstitial fibrosis) was made on the basis of chest radiographs showing miliary reticulation, wet rales heard over most lung areas, decreased pulmonary functions, and hypoxemia and on the results of examination of specimens of lung removed for biopsy. In this worker, the alveolar septae were thickened by fibrotic processes, and the air spaces were decreased by excess mucus and hyperplasia of the alveolar epithelium. Macrophages containing numerous foreign materials were present. Both spectroscopic and histochemical analyses of biopsy specimens of lung indicated the presence of 200 times the normal amount of tungsten, but no cobalt was found. The 34-year-old worker was able to return to work in an area free of the dusty exposures; the other two were severely restricted in their ability to work.

Scherrer [21] recommended that vital capacities be determined as early as possible in suspected exposure cases, since these measurements seemed to permit the earliest detection of abnormalities in hard-metal pneumoconiosis or fibrotic interstitial lung disease. The disease was not attributed to any one component of the hard-metal alloy. However, the author noted that individual susceptibility to hard-metal pneumoconiosis was a significant factor.

In 1972, Rochemaure et al [22] described the development of fibrosis in a 46-year-old woman who had worked with hard-metal mixtures and thus had been exposed to tungsten carbide and cobalt at unspecified concentrations. She was a nonsmoker with no history of bronchopulmonary problems. Three years after beginning such work, she developed a persistent, dry cough that lasted for 12 months and became productive during the following 6 months.

At the end of this period, exertional dyspnea and a weight loss of 8 kg were noted. About 3 months later, the woman was hospitalized after X-ray studies of her chest revealed bilateral, reticulonodular opacities.

Except for crepitant rales, physical examination of the woman did not reveal any abnormalities [22]. Pulmonary function tests showed a decrease of more than 50% in vital capacity and decreased carbon monoxide diffusion (50% of the theoretical value). Blood-gas determinations showed a tendency toward respiratory alkalosis. Differential blood cell counts and liver function test results were normal. Chest radiographs revealed reticulonodular opacities on both sides of the chest, but mainly on the right side and in the basal areas of the lungs. Expectations contained mucus, many bronchial cells, and macrophages, and bronchial aspirates contained, in addition, large amounts of blood and some inflammatory, cellular elements.

Ocular microscopic examination of a specimen of lung taken for biopsy 2 months after initial hospitalization revealed fibrosis with deposits of collagen, lymphocytes, and plasmocytes around the bronchi and vessels, which caused thickening of the alveolar septa [22]. Hyperplastic squamous or cuboidal epithelial cells lined the damaged alveoli, which contained macrophages and leukocytes and showed some evidence of edema. Emission spectrographic analysis of the lung specimen revealed no detectable levels of tungsten, titanium, or cobalt. The investigators attributed these negative findings to the small sample size.

The patient's clinical, radiologic, and pulmonary function tests showed significant improvement when she left the work environment [22]. Coughing, dyspnea, and reticulonodular opacities recurred when the patient

resumed work 3 months later. Because of the severity of her signs and symptoms, the patient again stopped work after 3 weeks. At this time, pulmonary function tests showed that her vital capacity had decreased to 60% of the predicted value and that there was a 66% decrease in carbon monoxide diffusing capacity. The authors stated that the recurrence of her signs and symptoms when she returned to work indicated that tungsten carbide dust played an important role in her illness, but they pointed out that cobalt was the most toxic agent to which she had been exposed. The authors suggested that individual susceptibility differences might have caused only some workers to experience coughing and dyspnea and still fewer to develop pulmonary fibrosis.

In 1967, Bruckner [23] diagnosed extrinsic asthma in a 45-year-old grinder of tungsten carbide-tipped tools. The worker had smoked for 17 years before being employed in the tungsten carbide industry but had not smoked during the present employment. During the 4 years before the diagnosis, the subject had done rough grinding of tungsten carbide for about 16 months. He used silicon carbide and aluminum oxide wheels and had later performed fine, dry grinding using diamond wheels. When the worker changed to the latter type of grinding, he experienced shortness of breath, chest tightness, audible wheezing, and productive cough during work. His symptoms decreased or disappeared during weekends and vacations. The composition and concentration of dusts in the workplace air were not described. Because of progressive deterioration in his health, the worker was hospitalized twice for severe shortness of breath with audible wheezing and a cough productive of yellow sputum.

Physical examinations disclosed evidence of respiratory distress, hyperresonance of the chest to percussion, a depressed diaphragm, and use of the accessory muscles of respiration [23]. Skin tests revealed a positive intracutaneous reaction to house and mattress dust. The dust in the patient's work atmosphere was not among the substances used in skin tests. The chest radiographs were reported to be within normal limits. Films of the paranasal sinuses showed marked to moderate clouding with some signs of scarring from an old disease. Except for eosinophilia, all the values of the blood and urine analyses were within the normal ranges. When the patient returned to work following his second hospitalization, severe asthmatic episodes recurred within 1-3 minutes after he began work. The patient stopped working until a respirator designed to remove particles with a diameter of 0.6  $\mu\text{m}$  or more was made available to him. However, he continued to experience asthmatic symptoms when wearing the respirator. Bruckner concluded that either cobalt dust or a combination of tungsten carbide and cobalt dust was responsible for the patient's symptoms. He suggested that the patient's reaction was mediated through a hypersensitivity mechanism because he exhibited the symptoms within 1-3 minutes after beginning work. Although Bruckner found eosinophilia in the patient, he did not point it out as evidence of hypersensitivity.

Schwartz and colleagues [14], in 1945, reported the results obtained in studies of 1,200 persons who worked with tungsten, tantalum, and titanium carbides, carbon, and cobalt in a cemented tungsten carbide manufacturing plant. No specifics regarding age and sex were given. Work assignments for the group were also not mentioned. Approximately 20 of the workers were found to have an erythematous, papular type of dermatitis

which was limited primarily to the sides of the neck, flexor portions of the forearm, and backs of the hands. In a few cases, the dermatitis was generalized and two sweepers each had contact dermatitis on the heel and ankle of one foot. This was the foot which was kept advanced during sweeping and was therefore exposed to the most dust. All of the affected workers had been employed for at least a month before the dermatitis appeared.

Patch tests were performed on six of the workers with dermatitis and on two control subjects not exposed to the aforementioned dusts. The reactions were read at 24 and 48 hours. The tests revealed that no subject with dermatitis reacted to the oxides of tungsten, tantalum, or titanium, or to carbon [14]. However, all six had positive reactions to metallic cobalt powder, to the unfused tungsten carbide, to mixtures or powders of cobalt, and to other materials used to make the hard metal. The control subjects showed no reaction to any test material. The authors concluded that, since the distribution of the skin eruptions was most marked in the areas of friction, the abrasiveness of the dust facilitated the development of sensitization. They concluded that sensitivity to cobalt caused the dermatitis and that this sensitivity was accentuated by the abrasiveness of the dust.

In 1963, Skog [24] described the incidence of skin disorders among workers in the Swedish hard-metal industry. Of 360 workers (sex and age not specified), 34 who had various skin disorders were examined by a dermatologist. They worked with tungsten carbide, titanium carbide, tantalum-niobium carbide, and cobalt. The durations of their exposures were not mentioned. The industry employed good general and local exhaust

ventilation and cleaning procedures, and the workers had used soap and cold cream with an organic solvent for the past 10 years to facilitate cleaning the hands and forearms. All workers with skin problems were given further dermatologic examinations, and 26 of them were given patch tests with various substances, including tungstic oxide, titanium oxide, cobalt chloride, and finished hard-metal powder.

The examinations resulted in the following diagnoses: contact eczema, 16; pruritus without skin lesions, 8; folliculitis, 6; and neurodermatitis, 5 [24]. The eczema and itching were localized on the areas of the body with high dust contact, namely on the face and extremities. Of the observed skin disorders, only eczema and itching were attributed to the occupational exposures.

Patch tests on 14 workers with contact eczema revealed that 3 of them were sensitive to cobalt chloride and 2 of them were sensitive to finished, hard-metal powder as well. Seven of the eight workers with pruritus and the five with neurodermatitis who were patch-tested showed no reaction. Although an allergic reaction to cobalt was noted in a few workers, the author attributed both eczema and itching to the combined irritant effect of hard-metal dust and intensive cleaning of the skin.

#### Carcinogenicity Reports

The following two studies are the only ones dealing with the incidence of cancer in cemented tungsten carbide workers. No human studies are available that indicate the carcinogenic potential of either soluble or other insoluble tungsten compounds.

Bech et al [25] reported bronchial carcinoma in a worker suffering

from hard-metal disease. The 63-year-old man began working as a core maker when he was 13 years old and held this job in various iron foundries for 32 years. He subsequently worked in the hard-metal industry for 17 years. His smoking history was not reported. During the latter employment period, he worked in all of the operations of the hard-metal industry, including powder mixing. No information was provided on the level of his exposure and the nature of the metal components to which he was exposed. After 13 years of working in the hard-metal tool industry, the worker reported to the hospital with a 3-year history of persistent dry cough, shortness of breath, and pain and tightness in the chest. The incipient fibrosis was detected by radiographic examination. The fibrosis increased during the next 5 years, and a mass in the lower lobe of the right lung was diagnosed as a neoplasm. The workman died 3 years after this diagnosis. At autopsy, most of the right lung was found to be invaded by anaplastic adenocarcinoma. There was a general appearance of nonspecific fibrosis which the authors considered to be unrelated to the carcinoma. Some neoplastic invasion of the capsule of the right kidney was also observed. The worker had been exposed to a number of industrial dusts of unknown compositions and concentrations. Therefore, the tumor cannot be attributed definitely to exposure to tungsten.

In 1963, Collet and associates [26] described a case of pulmonary fibroadenomatosis in a 57-year-old man who worked in the manufacture of sharp-edged tools. The study presented the results of physical examination, bronchoscopic examination, pulmonary function tests, and chest radiographs. During the 14 years before the manifestation of the disease, the man had been exposed to unspecified amounts of dusts of tungsten



carbide powder, cobalt, tantalum, titanium, and cobalt oxalate, and to trichloroethylene vapor. After the man had worked in such an environment for 8 years, chest roentgenographs showed reticulation of the bases of both lungs and of the central portion of the left lung. After 2 more years, fever and breathing difficulties were reported; a year later, exertional dyspnea had become progressively worse. These symptoms persisted during the next three years, and the patient also developed an increased heart rate and pulmonary rates. In addition, his vital capacity was only 65% of the theoretical value, while the carbon monoxide diffusion capacity measured 19% of the theoretical value. At the end of this period, he died of respiratory failure and cardiovascular collapse. Seven months before the man's death, a sample of lung was taken for microscopic study and for spectrographic analysis for tungsten, tantalum, titanium, cobalt, and nickel.

Microscopic study of the lung sample taken 7 months before the worker's death led to a diagnosis of pulmonary fibroadenomatosis with alteration and obliteration of capillaries resulting in an endarteritis [26]. Proliferations of smooth muscle, elastic fibers, mucous-producing cells, and macrophages were also evident. The epithelial cells of the alveoli and septa were altered by stratification metaplasia, fibrous tissue, and collagen deposits. The small alveolar cells were completely absent. Electron microscopy confirmed the previously described findings and revealed that the most common changes were mitochondrial and nuclear. The lung tissue was stated to contain significant amounts (no values given) of tungsten, nickel, and titanium. The other elements, presumably tantalum and cobalt, were reported to be present only in traces. On the basis of

the microscopic findings, the authors concluded that the lesion was not malignant, although it had some questionable features. They believed that the fibroadenomatosis observed in their patient was the result of occupational exposures to dusts arising in the tungsten carbide industry. While this is a possibility, the unexplained spectrographic findings of significant amounts of nickel in the lung and the worker's exposure to trichloroethylene vapor make the basis for this conclusion equivocal.

#### Epidemiologic Studies

Studies of workers exposed to dusts of tungsten and its products in the cemented tungsten carbide industry show that the effects are chiefly respiratory. Most of the reports do not distinguish the effects of tungsten products from those due to cobalt. However, one study [27] found that the effects of exposure to mixtures containing cobalt are more severe than the effects of exposure to tungsten compounds alone.

In 1967, Mezentseva [28] reported a study of the effects on workers of exposure to airborne dusts of tungsten trioxide, tungsten dioxide, metallic tungsten, and tungsten carbide. The author determined the air levels of dusts present in a workshop where malleable tungsten was prepared and at the following stages of hard-metal production: grinding, loading and unloading, reduction of tungsten, carbonization, and sifting. Dust particle sizes, determined by what was described as the shadow method, were reported for unspecified processes in the production of hard metal; apparently, particle sizes were not determined in the malleable-tungsten workshop. Total airborne dust levels for hard-metal production were 8.3-83 mg/cu m. The diameters of 72-82% of the particles were less than 4  $\mu$ m. In

the manufacture of malleable tungsten, the airborne dust concentrations were 1.3-60 mg/cu m in various stages of processing.

The author [28] described the results of physical and radiographic examinations of 54 workers engaged in processes associated with the formation of the dust of tungsten or of its compounds, but it was not clear from the report whether these workers were employed in the hard-metal industry or in the manufacture of malleable tungsten. The physical and radiographic examinations were not described in detail. The workers examined were 29-45 years old, but other details such as smoking history and sex, which might have had an impact on the results, were not presented. No control group was mentioned. The author did not mention the average number of years these workers were employed in operations that might generate significant air levels of tungsten dust. Of the 54 workers examined, 5 showed early radiologic signs of diffuse pulmonary fibrosis; one of these had been employed for 19 years, another for 24 years, and the rest for no more than 2-3 years. Where such wide variation in exposure durations exists, it is possible that other preexisting conditions which were not described may have contributed to the cause of the signs and symptoms.

Kaplun and Mezentseva [27], in 1959, reported the effects on worker health of exposure to dusts generated in the production of hard metal. The dust concentrations in various operations of tungsten carbide manufacture and in various stages in the production of tungsten carbide-cobalt hard metal were determined gravimetrically. The concentrations of cobalt dust generated in the production of hard metal from the tungsten carbide-cobalt mixture were also monitored. Records of periodic medical examinations of

36 tungsten carbide workers who came into contact only with tungsten and its compounds were analyzed separately from those of 247 workers exposed to mixed dusts of cobalt and tungsten carbide. No control group was mentioned. Details of medical examinations and the age, sex, duration of exposure, and medical histories of the workers were not reported.

In the various processing operations in the production of tungsten carbide, the dust concentrations were 8.6-107 mg/cu m [27]. The dust concentrations in the production of tungsten carbide-cobalt hard metal were 3-186 mg/cu m, and the cobalt concentrations were 0.27-1.75 mg/cu m. The authors noted that 4 of the 36 workers who came into contact only with tungsten compounds showed radiographic evidence of early diffuse interstitial fibrosis. No other changes were reported. Analysis of medical examinations of 247 workers exposed to mixed dusts of tungsten and cobalt showed that 117 workers had some damage to the upper respiratory tract. Of these, 33 had indications of incipient diffuse interstitial fibrosis, and chronic bronchitis was diagnosed in 35. It was noted that a significant fraction (no values given) of the workers had hypotension, unspecified changes in the blood, or an impaired sense of smell. This group of 117 workers also complained of loss of appetite, nausea, and coughing. The authors concluded that the harmful health effects in the manufacture of hard metal were produced by mixed dusts of tungsten, titanium, and cobalt. They also suggested that evaluation of the biological action of such mixed dusts should be based on the content of the most toxic component, cobalt.

Basing their recommendations on the percentage of cobalt present in the mixed dusts at the plants studied, the authors [27] advised that the

mixed dust concentrations in the air should not exceed 2 mg/cu m. Although this is the first report of an attempt to distinguish the health effects of tungsten and its compounds from those produced by a mixture of tungsten carbide and cobalt, the derivation of the limit of 2 mg/cu m for mixed dusts is not clear. In the absence of details on the duration of exposure and preexisting conditions for each group, no meaningful distinction can be made between the results from the exposures to the two types of dust. The higher incidence of pulmonary disorders in the group exposed to mixed dusts of tungsten carbide and cobalt may be related to total dust concentrations that were somewhat higher than those found for the group with pure tungsten exposures.

Lichtenstein et al [29], in 1975, reported the results of a study of the effects of airborne tungsten carbide and cobalt on the health of workers in an operation that involved grinding tool bits and inserts made of two commercial grades of cemented tungsten carbide. One grade contained 72% tungsten carbide, 8% titanium carbide, 11.5% tantalum carbide, and 8.5% cobalt; and the other contained 94% tungsten carbide and 6% cobalt. About 70% of the tool-grinding activity involved the latter grade. Approximately 75% of the tool work involved regrinding old carbide tips, and 25% involved grinding new carbide tips. The air was sampled with filters in the workers' breathing zones, and the filter contents were analyzed for tungsten and cobalt by atomic absorption spectrometry. The concentrations of tungsten ranged between 0.2 and 12.8 mg/cu m, while those of cobalt were 0.04-0.93 mg/cu m. Mean concentrations were 5.16 mg/cu m for tungsten and 0.28 mg/cu m for cobalt. Of the 25 samples taken, 40% exceeded 5 mg/cu m for tungsten and 60% exceeded 0.1 mg/cu m for cobalt.

To evaluate the employees' health status, the authors [29] examined 22 tungsten carbide grinders, 31-60 years old, including 6 nonsmokers, 10 smokers, and 6 ex-smokers who had ground tungsten carbide tools for 1-30 years, for a mean duration of 11 years.

Radiographic examinations revealed no evidence of pulmonary fibrosis in any of the workers. For all tool grinders, the mean FVC and FEV<sub>1</sub>, expressed as percentages of the predicted values (based on normal values from persons of the same sex, age, and physical stature), were 88.7 and 95.1, respectively, and the mean FEV<sub>1</sub>/FVC was 86.8%. The small number of workers tested, especially when subdivided into smokers and nonsmokers, did not permit a statistical evaluation of the pulmonary function test data. However, the authors suggested that the reduced FVC, the near-normal FEV<sub>1</sub>, and the elevated FEV<sub>1</sub>/FVC ratio may be indicative of an early stage of restrictive ventilatory impairment.

The mean red blood cell counts of all 22 grinders were within the normal range [29]. Exertional dyspnea was recorded for 1 smoker and 2 ex-smokers, while 3 of 6 nonsmokers and 5 of 10 smokers complained of productive coughs. The authors concluded that, in this study, cobalt exposure provided a good index of the total dust exposure, since concentrations of the tungsten dust above its TLV were recorded only when the concentration of cobalt dust exceeded its TLV. They did not observe increases in red blood cell counts or indices of pulmonary fibrosis. The authors noted that exposures to cobalt at levels above the TLV might occur in the cemented tungsten carbide tool-grinding industry during wet process grinding in the absence of local exhaust ventilation. Although the TLV's of both tungsten and cobalt were exceeded in the cemented tungsten carbide

tool grinding operations, the authors concluded that cobalt rather than tungsten exposures caused the observed changes. However, the data presented do not clearly distinguish the effects of the two metals.

In a medical surveillance program, developed as a consequence of the above study, Bartl and Lichtenstein [30] found one case of pulmonary fibrosis in the same population of cemented tungsten carbide tool grinders. The 34-year-old tool grinder had worked in a variety of jobs for 15 years. During this period, he had ground tools or drills for a total of 7 years, 42 months of which had involved work with tungsten carbide alloys. Approximately 9 years elapsed between the time he began working with tungsten carbide alloys and the examination at which the diagnosis of pulmonary fibrosis was made.

At this examination, his chest radiographs showed diffuse, bilateral, poorly delineated, parenchymatous densities distributed evenly in the upper and lower lung lobes [30]. His FVC and FEV 1 were 77 and 89% of normal, respectively. However, the carbon monoxide diffusion capacity was normal, and the worker did not develop shortness of breath during exercise. A sample of lung removed for biopsy was described as diffusely nodular with a slightly increased palpable density [30].

Microscopic examination of the same lung sample showed multi-focal pulmonary scarring associated with patchy interstitial fibrosis and nonspecific reactive changes in a hilar lymph node. The authors concluded that the observed interstitial fibrosis in this employee was consistent with the magnitude of his potential exposure to hard-metal dust containing cobalt.

Bech et al [25], in 1961, examined the medical histories of 255 hard-metal shapers, grinders, and powder workers who had been employed for 1 month-20 years. Chest radiographs of the 113 shapers, 120 grinders, and 22 powder workers were also examined. Their age, sex, and smoking history were not specified, and no control group was mentioned. Forced expiratory volume and airway resistance tests were performed on 19 volunteers (7 powder workers and 12 shapers) at the beginning and end of a working day. In addition, concentrations of airborne dust were measured (by gravimetric analysis) and the dust was analyzed for particle size and composition.

The breathing zone samples contained 195-1,230 particles/ml, the particles being less than 5  $\mu\text{m}$  in diameter. The dust contained 90% tungsten and 6% cobalt, the remaining 4% consisting of titanium, silica, aluminum, magnesium, and iron. An unspecified number of these workers complained of wheezing and tightness of the chest during the workday. Of the volunteers who underwent pulmonary function tests, two shapers who complained of wheezing and tightness exhibited considerably decreased (13.4-17.6%) ventilatory capacity accompanied by appreciably increased (24.3-31.8%) airway resistance. Among the 255 workers whose medical reports were examined, 1 case of hard-metal disease was diagnosed by chest radiograph; early signs of pulmonary fibrosis were noted in an unspecified number of radiographs.

Barborik [31] reported studies in 1966 on the health of 193 employees (104 men and 89 women) working in the production of hard metal. The workers, with average ages of 43 and 40 years (range 19-66 years) for men and women, respectively, were exposed to dust levels of 13-100 mg/cu m



while handling powders consisting of 70-90% tungsten carbide, 8-18% titanium carbide, and 5-25% cobalt. The composition of the airborne dust was not given. Although the particle sizes were not measured, the author concluded from data in published reports that most dust particles generated in this process were respirable. The average length of work experience was 6 years (range 1-13 years). The workers were examined for signs and symptoms of disease and for changes in chest radiographic findings.

Ninety workers (36 men and 54 women) complained of coughing, and half of this number had coughs that varied from irritant to barking [31]. Sixty-seven workers, including approximately equal numbers of men and women, complained of dyspnea. Other upper respiratory difficulties, such as burning or dryness of throat and anosmia, were less frequently reported. Moist or markedly crepitant rales were heard on auscultation in an unspecified number of workers. Of the 116 workers who were examined by spirometry, 25 showed moderate to severe disturbances in pulmonary ventilation. Roentgenologic abnormalities were reported in 31 of the workers. In 13 of the 31, the abnormalities were described as incipient, atypical, pulmonary reticulations, while in the remaining 18, they were said to be more clearcut and indicative of pronounced pulmonary fibrosis.

Barborik [31] concluded that metallic cobalt powder played a substantial role in the etiology of the disease and that its effect may have been potentiated by tungsten carbide. The author probably based his conclusion on the autopsy findings from one of the five case studies presented in the report. This worker had 12.5 and 78.6  $\mu\text{g}$  of cobalt/10 g of dry tissue in the lungs and hilar lymph nodes, respectively.

Vengerskaya and Salikhodzhaev [32] studied the effects of aerosols of tungsten, cobalt, and their compounds on workers in a hard-metal plant. The study included measurement of atmospheric concentrations of tungsten and cobalt at various operations by unspecified methods.

Of the 178 hard-metal workers (52 men and 126 women), 81% were about 30 years old [32]. About 84% of the workers had been engaged in hard-metal operations for 3 years. The concentrations of tungsten in the work atmosphere during various operations varied from 0.75 to 6.1 mg/cu m, while those of cobalt were 0.6-3.2 mg/cu m. No control group was described.

Among the 178 workers, 88 persons complained of dyspnea, coughing, pounding of the heart, headache, dizziness, nausea, loss of appetite, and impaired sense of smell [32]. The range of the mean tungsten concentrations was 0.8-1.1 mg% in the blood of 45 workers and 0.6-1.1 mg/liter in the urine of 40 workers. Tungsten was not detectable in the blood of 11 workers and in the urine of 7. Although it is not clear from the information given in the report, the workers may have been exposed to both soluble and insoluble tungsten compounds. The concentration of chlorides in the blood, measured in 30 workers, was slightly above normal in 20 and below normal in 10, while the chloride content of the urine was somewhat depressed in 32 of 39. Blood sugar levels were slightly higher than normal (no values given) in 8 of 37 workers examined, and the glucose tolerance curve in 8 of 14 workers was elevated. In 9 of 34 persons, hippuric acid elimination after loading with sodium benzoate was above 80% of the theoretical maximum conversion; it was 71-80% in 6, 61-70% in 8, and below 60% in 11 persons.

The authors [32] concluded that tungsten can be found in the blood and urine of workers exposed to aerosols of tungsten and cobalt, that the workers' blood sugars were somewhat elevated, and that there was some impairment of the detoxication of benzoic acid by the liver. They did not describe the methods used to determine tungsten concentrations either in the air or in body fluids. Therefore, the significance of the instances in which tungsten was undetectable cannot be assessed. The authors also did not elaborate on the absence of a relationship between the concentrations of tungsten in the body and the results of liver function tests or of blood changes. They did not measure cobalt concentrations in tissue and body fluid when they were aware that the exposure levels of cobalt exceeded the maximum permissible concentration (MPC) of 0.5 mg/cu m. Furthermore, they neither tried to define the causal factors involved in the study nor explained what the measured changes implied.

In 1962, Heuer [33] reported the respiratory effects in 208 workers engaged in hard-metal production, where dust levels of 277-4,064 mg/cu m were generated. Thirty-nine of the 208 employees worked in the mixing room, where the airborne dust was composed of 67.8% tungsten, 21.2% cobalt, 2.25% iron, 1.75% titanium, and 7% volatiles (not described). In the granulating room, 99 workers were exposed to dust containing 76.1% tungsten, 7.6% cobalt, 0.3% iron, and 16% volatiles. The remaining 90 workers were engaged in processing the finished hard metal. The airborne dust in this area was not analyzed for its components. The average size of 99.2-99.9% of the particles generated in various operations during hard-metal production was below 5 $\mu$ g.

During routine medical evaluations, 15 mixing room workers had complaints of cough, 14 of dyspnea, and 3 of asthma. While the physical examinations showed 5 cases of bronchitis and 14 cases of emphysema in these workers, chest radiographs revealed 3 cases of bronchitis, 10 cases of emphysema, and 23 cases of fibrosis that varied in severity. Forty of the 99 workers from the granulating room complained of coughs, and 34 reported dyspnea. Clinical signs and symptoms indicated 10 cases of bronchitis and 7 cases of emphysema among these 99 workers, while chest radiographs revealed the incidence of bronchitis in 9, emphysema in 6, and incipient or mild fibrosis in 18. Thirty-three of the 70 workers who handled the finished hard metal complained of coughs, and 16 reported dyspnea. Among these 70 workers, 10 cases of bronchitis and 2 cases of emphysema were diagnosed from clinical signs and symptoms, while chest roentgenograms showed that 7 had bronchitis, 13 had emphysema, and 14 had incipient or mild fibrosis.

On the basis of reported complaints, workers in the mixing room were the only ones to develop asthma, and they had the highest incidence of pulmonary fibrosis. The pulmonary fibroses found in these workers were more advanced or severe than those of the employees who worked in the granulating room or with the finished products. The author attributed the asthma and pulmonary fibrosis to exposure to high concentrations of dust containing the pure components of the hard metal. Analyses of the lung specimens obtained by autopsy from a mixing room worker who had severe fibrosis showed that dust particles accounted for 0.96% of the dry weight of the lung.

Chemical analyses of the dust from the lung revealed 14.4% tungsten, 9.8% calcium oxide, 9.2% phosphorus pentoxide, 5.6% silicon dioxide, 2.9% cobalt, 2.8% ferric oxide, 2.05% titanium, and 1.1% aluminum oxide, while 50.3% of the sample was lost during annealing.

The author noted that mixing room employees with mild to severe pulmonary fibrosis had worked for 4-8 years in hard-metal production. However, other employees who worked for 12 years in the same room had no adverse signs or symptoms. According to Heuer, this difference indicated that individual susceptibility was an important factor in the development of occupationally related pulmonary problems in hard-metal workers.

#### Animal Toxicity

Short- and long-term animal experiments revealed that the effects of inhalation or intratracheal introduction of insoluble tungsten compounds were limited to the respiratory system, and the effects of ingestion of soluble tungsten compounds such as sodium tungstate, were not clearly identifiable in any organs of the body. Few reports were found on the dermatologic effects of tungsten and its products in experimental animals.

Mezentseva [28] studied the effects of dusts of tungsten and its compounds on 55 white rats. Either metallic tungsten, tungsten trioxide, or tungsten carbide was injected intratracheally in single doses of 50 mg of material suspended in 0.5 ml of physiologic saline. Tungsten carbide was also administered by inhalation for 1 hour/day for 5 months to another group of animals. The tungsten carbide concentration in the exposure chambers was 600 mg/cu m, with up to 77% of the particles measuring less than 5  $\mu$ m in diameter. The age, weight, sex, strain, and number of rats

subjected to each experimental condition were not reported. Six rats served as controls. No details of their management were given. In the intratracheal studies, rats were killed 4, 6, or 8 months after administration of the suspensions and their lungs were examined microscopically. Animals exposed by inhalation were monitored for general health and body weight. At the end of the exposure period, all rats were killed and examined macroscopically for changes in unspecified internal organs.

At 4 months, the lungs of rats given metallic tungsten showed infiltration by macrophages, chiefly around the pulmonary blood vessels, and thickening of the walls between the alveoli [28]. Around the bronchi, large numbers of round cells were seen surrounding the dust particles at 6 months. Collagen fibers had overgrown these foci by 8 months; the endothelium was swollen and the walls of small vessels were thickened. Metallic tungsten did not cause macroscopic changes in the internal organs of rats at either 4, 6, or 8 months. No details of the general health and weight gain of the rats were given.

Four months after the administration of tungsten trioxide, the lungs showed considerable thickening of the walls between the alveoli and infiltration of macrophages around the vessels of the bronchi. The endothelium was swollen, with thickening of the walls of small blood vessels [28]. Eight months after the intratracheal injection, cellular proliferation persisted. Lesions surrounding the dust particles were characterized by fine, collagenous fibers and sclerosis of the vascular walls and peribronchial areas. In rats given tungsten trioxide, no macroscopic changes in the internal organs were observed either 4 or 8

months after the injection.

In rats administered tungsten carbide intratracheally, thickening of the walls between the alveoli and accumulation of macrophages around the bronchi and blood vessels were noted 4 months after treatment [28]. Lungs examined at 6 months showed cellular proliferation around the blood vessels, with overlying, collagenous fibers. At this examination, swelling of the endothelium and thickening of the walls of the small blood vessels were evident, and some hyperplasia of the tracheal lymph nodes containing free dust particles was observed. Microscopic findings in animals killed after 8 months were similar to those in animals killed at 6 months. The report did not discuss the effects of intratracheal administration of tungsten carbide on macroscopic changes of the other internal organs, on general health, or on weight.

Rats exposed to tungsten carbide dust by inhalation at 600 mg/cu m remained healthy and gained weight well during the experiment [28]. Those killed at the end of 5 months' exposure at this concentration showed no macroscopic changes in the internal organs. According to the author, the findings from microscopic examination of the lungs were essentially the same as those observed 6 months after intratracheal administration of tungsten carbide.

Mezentseva [28] concluded that metallic tungsten, tungsten trioxide, and tungsten carbide dusts did not cause severe changes. These changes were described as being more marked after the intratracheal administration of tungsten trioxide than after that of either metallic tungsten or tungsten carbide. From the experimental findings and medical data, the author concluded that these tungsten substances can cause a mild, diffuse

interstitial lung fibrosis. A permissible maximum level of exposure of 6 mg/cu m for tungsten dust was suggested.

Miller et al [16] studied the effects of intratracheally-introduced pure tungsten carbide on rat lungs. Tracheotomies were performed on 15 white rats under ether anesthesia, and 1 ml of a 10% suspension of tungsten carbide in isotonic saline was administered intratracheally. The age, sex, weight, and strain of the animals were not mentioned, the control group was not described, and the size of tungsten carbide particles in the administered suspension was not reported. An unspecified number of rats was killed at 2-week intervals for 18 weeks. The animals were examined macroscopically for exudate on the surface of the lungs, for fluid in the chest cavity, for the distribution of dust in the lungs, which were examined also for microscopic changes, and for alterations in reticulin and collagen contents.

Gross examination of the lungs 2 weeks after the tungsten carbide injections revealed that particles were present in all lobes, particularly the lower ones [16]. The lungs reportedly felt somewhat firmer than those from control animals killed at this time. The treated animals had neither exudates on the surfaces of the lungs nor fluid in the chest cavities. Microscopic examination at this time showed that, although particles were visible within the septa, most of the tungsten carbide remained free in the alveoli. Tungsten carbide was observed consistently within the alveoli throughout the experiment.

By 4 weeks, there had been some mobilization of the septal cells and engulfment of the tungsten carbide particles; these particles were found also in the perihilar lymph nodes and around the peribronchial and



perivascular connective tissue fibers [16]. Results of examinations 6-14 weeks postexposure were not reported. After 16- or 18-week examinations, findings were essentially unchanged from those at 4 weeks. Although more dust was found within the alveolar walls and lymphoid tissue of the lungs, dust was still present in the air sacs. Lung reticulin and collagen did not increase; there was no evidence of inflammation caused by bacteria.

The authors described the observed effects as responses to an inert dust and noted that tungsten carbide did not provoke a necrotizing or fibrosing response in rat lung parenchyma [16]. Commenting on these findings of Miller et al, Schepers [34] speculated that industrial cases of lung fibrosis and pneumoconiosis in tungsten carbide-tool and hard-metal industries might be caused by the coexisting exposures to cobalt.

Delahant [35] studied the effects of selected rare metals and other chemicals on guinea pig lungs in 1943 and presented the results in 1955. These experiments were undertaken to determine the particulate metallic component in cemented tungsten carbide that might provoke lung lesions. Dusts of tungsten metal or of tungsten carbide and carbon in a ratio of 94:6 were injected intratracheally into groups of six guinea pigs. The average weight of the guinea pigs was approximately 600 g. The age, sex, and strain of the animals were not given, and no control group was described. A 10% suspension of each dust in sterile isotonic saline was prepared. Although attempts were made to obtain dusts of 3  $\mu$ m in diameter or less, when this was not possible dusts were used as received. Thus, the particle size of the dusts injected in most cases was unknown. A total of 150 mg of each dust was injected intratracheally in three equal doses at weekly intervals. Animals were killed after 1, 4, 8, and 12 months, and

lung specimens were prepared for microscopic examination. None of the guinea pigs injected with either tungsten metal or tungsten carbide and carbon died during the experimental period.

Gross examinations of the lungs of animals given tungsten metal or tungsten carbide and carbon were performed [35]. Large, circumscribed pigmented lesions were found. Beneath the visceral pleura, widely distributed, small, discrete foci of pigmentation were occasionally observed. There was no appreciable temporal change in the character of the lesions. The author concluded from these findings that dusts of tungsten metal or of tungsten carbide and carbon produced relatively benign effects on lung tissue.

Schepers [36] also studied the effects of tungsten metal dust on the lungs of the guinea pigs exposed by Delahant [35]. One month after injection, microscopic examinations of the lungs showed proliferation of interstitial cells; moderate thickening of alveolar walls was most marked around massed tungsten particles. A considerable amount of macrophage infiltration into the alveoli was noted. Around the capillary blood vessels of the lung, numerous focal cellular lesions were observed. The mucosae of the bronchi and bronchioles were slightly inflamed, with some bronchioles partially or wholly closed. The investigator noted no effects on the lymphoid tissue.

Guinea pig lungs examined 1 year after the intratracheal introduction of tungsten metal dust were reported [36] to have residual lesions. These included persistent, focal, interstitial, cellular infiltration in relation to the retained particles. There were various degrees of peribronchial, peribronchiolar, and perivascular fibrocellular reactions, with

bronchiolitis obliterans and bronchial inflammation. Slight atrophic vesicular emphysema was also present. The author concluded that tungsten was probably a relatively benign substance. Commenting on the occupational significance of his findings, he remarked that exposures to the dust generated by pulverizing tungsten in the tungsten carbide industry would be relatively, though not wholly, free from risk.

Schepers [34] also microscopically examined the effects of intratracheally injected tungsten carbide and carbon (94:6) on the respiratory systems of guinea pigs. The animals used in this investigation were those described by Delahant [35]. Schepers reported that the immediate response was a diffuse hyperemia and inflammation of the bronchial mucosa. After 1 month, much of the tungsten carbide and carbon mixture had been engulfed by multinucleated macrophages. While some of these cells penetrated the alveolar walls, most were still within the alveoli. Some of the dust particles observed had aggregated in the small vascular components.

The author [34] compared these findings with those from his investigations on tantalum and cobaltic oxides and noted that the tungsten carbide and carbon mixture did not readily reach the perivascular lymphatics. Marked lymphocytic hyperplasia with infiltration of the adjoining alveolar walls and some hyperemia were observed in these areas. Such alterations in the lymphoid tissues reportedly continued for several months after the acute reactions had subsided. Confluent interstitial pneumonitis in relation to trapped dust masses or isolated foci were observed 12 months after the exposure. Most massed granules in the mixture of tungsten carbide-carbon were engulfed by multinucleated, giant cells

within the relatively atrophic alveoli. Subpleural granulomata with a minor degree of fibrocyte formation were noted occasionally. Bronchiolitis and peribronchial and perivascular fibrosis were not persistent. Schepers concluded that the mixture of tungsten carbide and carbon was less harmful to lung tissue than tungsten metal.

Delahant [35] also reported the effects of intratracheal instillations of a 91:9 mixture of tungsten carbide and cobalt dusts and of inhalation exposures to a 3:1 mixture on the lungs of guinea pigs. Six guinea pigs were injected intratracheally with a total of 150 mg of the tungsten carbide and cobalt mixture, containing particles of 3  $\mu\text{m}$  or less in diameter, in three equal doses at weekly intervals. For inhalation exposures, Delahant exposed 20 guinea pigs to particles of the mixed tungsten carbide-cobalt dust, measuring 0.5-2.0  $\mu\text{m}$  in diameter. The total exposure period was 35 days, and the dust concentration range was 8,800-10,600 particles/cu cm for the first 20 days; after a 5-day recovery period, the animals were exposed to the dust mixture for an additional 15 days at a concentration of approximately 2,800 particles/cu cm. The age, weight, sex, and strain of the guinea pigs were not given, and no control group was described.

In the group injected intratracheally, none of the guinea pigs died during the experimental period [35]. Their lungs had well-circumscribed linear and diffuse patterns of black pigmentation. The time of this observation was not given. All lungs were reported to be normally soft with no observed progressive change in the character of the lesions during the study.

Animals that died during the inhalation experiment and two animals that were killed after 181 and 585 days of recovery in normal air following the second period of exposure were examined for gross changes in their lungs [35]. Five animals died during the first period of exposure, and three more died during the 5-day inter-exposure recovery period. During the second exposure, six more animals died, two each on days 10, 13, and 15 of exposure. Post-mortem examinations showed that some of the animals had acute pneumonitic consolidations of their lungs. The lungs of guinea pigs killed 181 and 585 days after the termination of the second exposure had faint, diffuse pigmentation by dust.

From the mortality rates and the nature of the gross pulmonary reactions, Delahant [35] concluded that particulate tungsten metal and a mixture of tungsten carbide and carbon were relatively inert and that, when cobalt was mixed with tungsten carbide, the toxic and lung irritant characteristics of the cobalt component were dominant.

In another study, Schepers [37] evaluated the effects of mixtures of tungsten carbide and cobalt on guinea pig lungs. The lung tissue used in these investigations was obtained from the animals Delahant [35] exposed to the dust mixtures by both inhalation and intratracheal injection. Guinea pig lungs were examined 1 or 12 months after the intratracheal instillation of tungsten carbide and cobalt in a ratio of 91:9. Lungs of animals exposed by inhalation to the dust of a 3:1 mixture of tungsten carbide and cobalt were removed for examination at the end of the first exposure, at the end of the second exposure, and 181 and 585 days after the second exposure.

One month after the intratracheal instillation, there were areas of dense fibrosis in the lungs wherever a massive deposition of the dust had occurred [37]. Infiltration of the alveolar walls by lymphocytes had produced almost confluent pneumonitic areas, and there was infiltration of lymphocytes into the perivascular areas. There was no involvement of the hilar lymphatics. While most of the bronchial epithelium was not damaged, in areas of massive dust deposition the bronchial mucosa showed a firm fibrous reaction with some development of bronchial crypts. One year after the injection, massed dust particles were still seen in the alveolar spaces. Other residual damage included patches of pneumonitis and mild cellular and fibrous reactions around the dust deposits. The hilar lymph nodes reportedly contained some particulate matter which could be subsequently deposited in the perilymphatic zones.

The exposure to a 3:1 mixture of tungsten carbide and cobalt provoked a diffuse inflammatory reaction at an unspecified time [37]. At the end of the first exposure, the lungs were hemorrhagic. Alveoli were infiltrated with erythrocytes, plasma cells, lymphocytes, polymorphonuclear leukocytes, and a fair number of macrophages containing ingested particles. "Prominent septal cells" lined the alveolar walls at numerous points. No changes were observed in the bronchi and larger blood vessels. Examination after the first exposure showed considerable recovery with mild residual atrophic vesicular emphysema and limited infiltration of polymorphonuclear leukocytes and macrophages into the alveoli. A considerable amount of dust was still retained, mainly within the pulmonary macrophages. The author attributed a specific lesion, namely, proliferation of the cortical reticuloendothelioid cells of the hilar lymph nodes, to the mixture of

tungsten carbide and cobalt. Neither tungsten carbide nor cobalt alone was capable of provoking a similar reaction.

After the second exposure, alveolar edema and diffuse hyperemia were once again evident [37]. Six months after the second exposure, metaplastic hyperplasia of the bronchial epithelium and the formation of villous papillomas were seen. Proliferating epithelial cells and papillomas occluded the bronchi, producing regional focal emphysema. Some undefined residual effects of tungsten carbide and cobalt were still noticeable 21 months after the exposure. Epithelial hyperplasia and metaplasia of the trachea and bronchi were present at this later examination. The characteristic foci of hyperplasia of epithelioid elements of the hilar lymph nodes persisted.

Schepers [37] commented that the pronounced proliferative and metaplastic epithelial changes may have reflected a sensitizing property of cobalt. The author also cautioned that, if observations in guinea pigs have any bearing on the way in which human lungs may react, extreme care should be exercised to control inhalation exposure to metal powders. Since the 91:9 ratio of tungsten carbide and cobalt used is comparable with those to which industrial workers might be exposed, these results have direct relevance to occupational exposure.

Kaplun and Mezentseva [38] conducted experiments on white rats in an attempt to establish the etiologic roles of tungsten, cobalt, and their mixtures in affecting workers' health in the hard-metal industry. In 4 series of experiments, 100 white rats of unspecified age, weight, sex, and strain were used. Groups of rats were given intratracheal injections of 10, 15, 25, or 50 mg of one of the following mixtures: (1) 8% cobalt and

92% tungsten, (2) 15% cobalt and 85% tungsten, and (3) 8% cobalt, 14% titanium, and 78% tungsten. The particle sizes of the dusts and the vehicle of administration were not reported, and no control group was described. Each dust mixture was administered to eight rats in the 50-mg series and to five rats in the 25-mg series; the number of rats used in the 15- and 10-mg series were not specified.

Injection of 50 mg of any of the three dust mixtures caused 100% mortality within 2 days after administration [38]. Within 5-7 days the rats that received 25 mg doses had 40, 100, and 60% mortalities in dust mixture groups 1, 2, and 3, respectively. At dosages of 15 mg, all the rats in group 2 died, and two each died in groups 1 and 3. At the 10-mg dose level, there were no deaths in groups 1 and 3, but 5 of 13 rats died within 2-3 days in group 2. The absolute lethal dose for the dust mixture of 15% cobalt and 85% tungsten was 15 mg (2.25 mg cobalt), while that of the other two groups was 50 mg (4 mg cobalt). The minimum lethal dose was 10 mg (1.5 mg cobalt) for the 15:85 mixture and 15 mg (1.2 mg cobalt) for the mixtures containing 8% cobalt. The authors, without presenting details, noted that the minimum and absolute lethal doses of pure metallic cobalt were 5 and 10 mg, respectively; however, the absolute lethal dose of the 15% cobalt dust mixture contained only 2.25 mg of cobalt. They concluded that the dust mixtures were more toxic than the separate dust components and that the toxicity of cobalt was increased in the presence of tungsten and titanium carbides. However, since the absolute lethal doses of the dust mixtures containing cobalt were 50-400% greater (15 and 50 mg) than that alleged for metallic cobalt alone (10 mg), it is probable that the dust mixture itself exerted some toxic effects.



Without making distinctions among the three dust groups, the authors [38] reported that animals dying after receiving doses of 25 mg or 50 mg of the dusts had thickened interalveolar septa that sometimes fused together to form solid areas of airless, homogeneous tissue containing many free-lying dust particles. The thickening of the septa was attributed to severe hyperemia and cellular infiltration by lymphocytes and macrophages. Large amounts of dust and secretions were observed in the bronchial lumina. In addition, marked hyperemia and cellular granulo-nodular degeneration were seen in the livers; and the kidneys of these animals showed granulomatous degeneration of the cells of the convoluted and descending tubules and obstruction in the glomeruli and proximal tubules.

Single doses of the three dust mixtures were administered intratracheally in the following amounts: 10 mg for the first mixture, 15 mg for the second mixture, and 10 mg for the third mixture [38]. The lungs, liver, and kidneys of animals given the first, second, and third mixtures were examined after 6, 4, and 6 months, respectively. The lungs of rats exposed to the first dust mixture showed extensive areas of dense tissue around the dust particles resulting from infiltration by lymphoid elements. Massive lymphocytic accumulations were observed around the small and medium-sized bronchi, and the mucosae of the bronchi were hypertrophic, with adenomatous proliferation and formation of papillomata. Changes in the walls of the blood vessels caused exudation of plasma, and there was excessive connective tissue around the small and medium-sized vessels. The liver and kidneys showed marked hyperemia. The lungs of rats exposed to the second dust mixture were similarly affected, but the adenomatous proliferation was more marked. Some hypertrophic adenomatous tissue was

overgrown by fine strands of connective tissue. In the third group, in addition to the lung changes seen with the first two dust mixtures, diffuse sclerosis of the lung tissue was observed. The effects of the second and third dust mixtures on the liver and kidneys were not described. The authors [38] concluded that the changes produced by the three dusts were similar and that they were more marked than those produced by metallic cobalt. These investigations indicated that the activity of cobalt is enhanced when tungsten and cobalt act together. The authors hypothesized that the solubility of cobalt might be increased in the presence of tungsten.

To test their hypothesis, Kaplun and Mezentseva [38] determined the solubilities of 100 mg of powdered metallic cobalt and of (the first two) dust mixtures containing 100 mg cobalt in unstated volumes of 0.3% hydrochloric acid, which has a pH equivalent to that of gastric juice. During a period of 24 hours, only 3 mg of cobalt dust was dissolved, while 12 and 15 mg of cobalt were dissolved from the 8:92 and 5:85 dust mixtures. Although the universality of this solubility behavior was not tested in other solvents, the authors believed that their hypothesis on the enhanced toxicity of cobalt in the presence of tungsten might still be acceptable. Although this conclusion was based on the cobalt content of the mixtures and on the assumption that dust mixtures containing tungsten or tungsten and titanium are more toxic than is pure cobalt, the data do not show clearly that the toxicity of cobalt was enhanced by the presence of tungsten or titanium.

Schepers [39] also assessed the biologic actions of a number of industrial chemicals that pose potential occupational hazards. Among these

were manganese tungstate, tungsten, and tungsten carbide. An unspecified number of guinea pigs were injected intratracheally with either a suspension or a solution of each of the above compounds, and the animals were examined after 12 months for pulmonary lesions, including epithelialization (proliferation of epithelial cells) and neoplasia. The experimental design was not described, and the nature of lung evaluation and rating was not stated.

Manganese tungstate was rated as slightly reactive in producing pulmonary lesions and moderately reactive in inducing epithelialization [39]. In comparison, both tungsten and tungsten carbide were graded as slightly reactive in inducing both pulmonary lesions and epithelialization. According to Schepers, none of the three materials induced tumors. Although the assessment of the reactivity of these substances included the number of animals affected, there was no report of the incidence rate of pulmonary lesions and the nature of their distribution.

Brakhnova and Samsonov [40] compared the toxicities of silicides of three transition metals, titanium, molybdenum, and tungsten. Albino rats of unspecified age, weight, sex, and strain were given tungsten silicide by inhalation or intratracheal injection for 1-6 months. The control group was not described, and the concentrations of tungsten silicide and the precise durations of exposure to it were not given. The toxicities of the silicides were evaluated (time not specified) qualitatively from microscopic examination of the lungs; the quantitative effects on the concentrations of collagen and ascorbic acid in the lungs served as estimates of the fibrogenic activity. The authors did not distinguish the effects of tungsten silicide administered by inhalation from those caused

by its intratracheal injection.

Pulmonary tissue obtained from rats exposed to tungsten silicide for 1-6 months exhibited hyperplastic lymph nodes, localized thickening of alveolar walls, perivascular infiltration by lymphocytes, and nodules composed of fibroblasts, lymphocytes, and macrophages [40]. Compared with the other silicides studied, tungsten silicide produced degenerative changes in various organs, including the liver, kidneys, and heart, that were considerably less pronounced. Vascular permeability was not affected by tungsten silicide. The concentrations of collagen in the lung were increased to 51.6 and 56.9% above the control levels at 1 and 6 months, respectively, after the exposure. Ascorbic acid synthesis in the lungs of animals exposed to tungsten silicide was elevated from 50 to 60% above that in the controls. The authors concluded that the results supported the maximum permissible concentration (MPC) of 6 mg/cu m for tungsten silicide established at that time in the USSR. However, it is not clear how the authors reached this conclusion.

Spiridonova and Suvorov [41] conducted several studies to assess the biologic effects of tungsten hexachloride and the products of its hydrolysis on rats and mice. In the authors' short-term experiments, rats were either given 1,800 mg/kg of tungsten hexachloride intragastrically or exposed to 0.043-0.14 mg of tungsten hexachloride/liter of air for an unspecified period. The age, weight, sex, and strain of the animals were not mentioned, and no control group seems to have been used.

The orally dosed animals had necrotic foci in the mucosa and submucosa of stomach and intestines and in the liver and kidneys [41]. Rats exposed to tungsten hexachloride at 0.043-0.14 mg/liter of air

lacrimated, had profuse blood-stained oral and nasal discharges followed by necrosis of the skin and mucosa, and died of pulmonary edema. No data were presented on the number of deaths. The authors, comparing the LD50's in mice of tungsten hexachloride (1,086.5 mg/kg of body weight by gavage) and of tungstic oxide (4,786 mg/kg of body weight by ip injection), suggested that the chloride form was more toxic than the oxide because the former probably was hydrolyzed in vivo, releasing a sufficient number of chloride ions to be toxic. Since the total buffer capacity of the blood is approximately 45-50 meq/liter and the stated LD50 dose of tungsten hexachloride corresponds to about 213 meq of HCl/liter of blood, this hypothesis seems tenable.

In a second series of experiments, the authors [41] used male white mice weighing 18-22 g to compare the toxicities of tungsten chloride in various stages of hydrolysis. Groups of 10 mice were given ip injections of 1 of 5 aqueous solutions made by starting with a sufficient dose of tungsten hexachloride to yield 3.3 meq/kg of Cl (216 mg/kg of tungsten hexachloride). This was administered immediately after preparation, after neutralization with sodium hydroxide, 30 minutes after preparation, on the following day, and on the following day after filtration. If the tungsten hexachloride were completely hydrolyzed, this dose would release about 44 meq of HCl/liter of blood. This would be sufficient to neutralize completely, or almost completely, the total buffer capacity of the blood. Neutralization reduced the mortality by 50%, while hydrolysis more than doubled the mortality. The toxic hydrolysis product was filterable. The investigators concluded that toxicity of the tungsten hexachloride was produced by its chloride ions and that the toxic effect of free ions in the

thoroughly dissociated hydrochloric acid was greater (100% mortality after 1 day) than that of the rare metal chlorides. They concluded further that the chloride ion plays a major role in the production of the acute toxic effects of tungsten hexachloride.

While these conclusions may be valid, it is not clear how the data relate to industrial exposure, since the route of administration in most studies was ip injection. Although inhalation exposures were conducted and deaths of animals from pulmonary edema were reported, the authors did not indicate the percentage of the experimental animals affected. They concluded that the chloride form of tungsten was more toxic than the oxide. However, their conclusion is not necessarily a valid one, because the LD50 comparisons cited were for different routes of exposure.

Gol'dman et al [42] reported the results of a study on the effects of a charge of calcium-magnesium-tungstate phosphor and finished calcium-magnesium-tungstate phosphor in rats. The chemical composition of the charge was: tungstic oxide, 75%; calcium oxide, 18%; magnesium oxide, 5%; and lead dioxide, up to 3%. The charge and the finished product (after heating of the charge) differed in physical structures (not specified). The particle size of neither product was described. Rats in 2 groups of 10 were given intratracheally single doses of 50 mg suspensions of either the charge or the finished calcium-magnesium-tungstate material brought to 0.5 ml in a sterile physiologic solution. The age, weight, sex, and strain of the rats were not presented, and no control group was described. Six months after dosing, rats from each group were killed and their liver, kidneys, spleen, and heart were examined for macroscopic changes. Changes in the lungs were evaluated by microscopic examination.

No gross changes were observed in the internal organs of either group, but the lungs, 6 months after the administration of the charge of calcium-magnesium-tungstate, showed evidence of cellular proliferation, mainly of lymphocytes and macrophages, with considerable thickening of the interalveolar septa [42]. Cellular infiltration was noted around the bronchi and vessels, and most dust particles had been engulfed by the infiltrated cells. In some areas, accumulations of the dust, with marked cellular proliferation around them, were seen. Some cellular foci were rich in collagen fibers. In the lungs from rats given the finished product, some interalveolar walls were thickened; in other places, the walls of the alveoli were thin, smooth, and torn. The dust was lying free, mostly as separate granules. The lungs showed some emphysematous areas and cellular proliferation around the bronchi and vessels. It was noted that, although the charge of calcium-magnesium-tungstate and the finished product caused similar lung changes, the former had the more severe effect.

Referring to the study of Mezentseva [28], Gol'dman and associates [42] commented that the changes in the lungs produced by the charge of calcium-magnesium-tungstate and those produced by tungsten trioxide were similar but that those caused by the former were the more marked. Since the phosphor contained other potentially toxic substances, such as lead dioxide, the comparison may not be a valid one.

Lauring and Wergeland [43] investigated the ocular toxicity of 12 industrial metals, including tungsten, in rabbits. Two rabbits with body weights of 1.5 and 2.0 kg were used in the experiments with tungsten. In one rabbit, the left eye, which was sham-operated, served as a control, while a particle of tungsten metal less than 1 mm in diameter was

introduced under general anesthesia into the midvitreous region of the right eye through a stab incision. In the second rabbit, one eye was left intact and untreated while the other received the metal in the midvitreous region by the same method. The age and sex of the rabbits were not mentioned. The rabbits were examined at weekly intervals for the first 4 postoperative weeks and then at monthly intervals for 1 year. The evaluated parameters included the size and shape of the pupils, the degree of anterior segment inflammation, the presence of cataracts, and the degree of vitreous inflammation. From these pathologic evaluations, the authors classified tungsten as "completely inert." No changes were noted in either the control or the untreated eyes of these rabbits.

Kinard and Van de Erve [44], in 1943, reported the effects of tungsten metal powder on the growth of rats. They used 38-day-old animals from mixed Wistar albino and Minnesota piebald strains. Groups of 10 rats, 5 of each sex, were caged separately and fed ad libitum with ground dog chow containing 2, 5, or 10% tungsten metal powder; controls were given the ground dog chow without tungsten. Food consumption was determined from the food remaining in the containers after each feeding. The weight gains of rats were recorded at 10-day intervals for 70 days. Animals were killed after 70 days, and their gastrointestinal tracts were examined grossly.

During the 70-day period, the weight gains of male rats fed diets containing 2, 5, or 10% tungsten were 94, 113, and 108% of those of male rats fed the control diet, while the weight gains of female rats fed the three experimental diets were 104, 97, and 85% of control weight gains [44]. During the dosing period, each male rat fed the 2% tungsten diet consumed an average of 21 g of tungsten, while those on 5 and 10% consumed



54.5 and 104 g, respectively. Female rats, in the same period, consumed an average of 14.8, 41.0, and 75.0 g of tungsten when fed the diets containing 2, 5, and 10% tungsten metal powder, respectively. The authors attributed the different weight gains by male and female rats fed diets containing 10% tungsten to the reduced food consumption of the female rats. They did not find any exudation of blood into the mucous membranes of the small or large intestines in rats fed tungsten for 70 days. They concluded that tungsten metal powder did not show marked toxic action at the levels used in their experiment.

In 1924, Karantassis [45] reported the toxic effects of sodium tungstate ( $\text{Na}_2\text{WO}_4 \cdot 2\text{H}_2\text{O}$ ) on guinea pigs. In one study, three guinea pigs with an average body weight of 616 g were each given a single dose of either 0.50 or 0.75 g of sodium tungstate by gastric intubation. In another study, five guinea pigs, weighing an average of 635 g, were given subcutaneous injections of sodium tungstate. Three guinea pigs received single doses of 0.50 g each, and the remaining two received daily doses of 0.10 g until their deaths at 16 and 17 days. The age, sex, and strain of the guinea pigs used were not reported. No control group was mentioned. In both studies, the author monitored the general visible signs before death, the time at which death occurred, the results of gross examination at autopsy, and the presence of tungsten in the gastrointestinal tract, lungs, liver, kidneys, bones, blood, and urine.

Guinea pigs from both experiments had anorexia, colic, uncoordinated movements, sudden jumps, trembling, and breathlessness [45]. Although the signs were similar in both groups, they were more pronounced and prolonged in the guinea pigs given sodium tungstate subcutaneously. Dose-related

differences were not reported.

In both studies, guinea pigs given 0.50 g of sodium tungstate died 16-23 hours later [45]. In the guinea pigs given sodium tungstate orally, autopsy revealed that the stomach contained a bloody, greenish substance, and the large intestine contained bloody, diarrheic fecal matter. The liver showed small, discolored lesions and the lungs contained small, hemorrhagic spots. The peritoneum and all other organs, including the small intestine, spleen, testes, brain, kidneys, and suprarenal capsules, appeared normal. In subcutaneously dosed animals, post mortem examination showed an intense congestion of the liver, large infarcts in both lungs, dark blood in the heart, bloody diarrhea, and an appearance of asphyxiation. The condition of the brain, testes, peritoneum, gastrointestinal tract, and suprarenal capsules was not reported. No distinction was made between the effects of 0.75- and 0.50-g doses in the orally dosed group. Subcutaneous injections of 0.10 g of sodium tungstate/day for 16-17 days caused necrotic and indurated patches in unspecified tissues and a 23% loss in body weight; detachment of a small portion of abdominal skin and yellow patches of degeneration in the liver and kidneys were also noted.

Analysis of the tissues of animals from both experiments showed that tungsten was present in the stomach, intestines, liver, kidneys, lungs, blood, and urine [45]. In the orally dosed group, tungsten was also present in the walls and in the contents of the gastrointestinal tract. In the subcutaneously injected group, tungsten was present in bones and in necrotic and indurated patches in unspecified tissues.

At the lower subcutaneous dose-level (0.10 g/day), the total lethal dose of sodium tungstate for each guinea pig was 1.65 g, while the lethal dose for a single administration was about 0.50 g [45]. Expressed as elemental tungsten, the single lethal doses for intragastric and subcutaneous administrations were estimated to be 0.55 and 0.45 g/kg, respectively. Because of the small sample size and lack of statistical analyses, the significance of these values is difficult to assess. However, this is the first published study attempting to distinguish between the oral and subcutaneous toxicities of sodium tungstate.

In a 1942 abstract, Selle [46] presented the results of a study of the effects of sodium tungstate on rats. Male and female rats were given 5 ml/kg daily of a 0.1 M aqueous solution of sodium tungstate (0.164 g/kg body weight) either subcutaneously or orally. The age, weight, and strain of the rats were not reported. Although controls were indicated, the treatment of the controls and their number were not described. The number of doses and the duration of sodium tungstate administration were not given. At an unspecified time after the administration of sodium tungstate, body weights were recorded, the rats were killed, and the weights of the kidneys and adrenal glands were determined. At an unspecified time, tungsten excretion in feces and urine was measured, and rectal temperature was monitored.

The subcutaneous injection of sodium tungstate resulted in body weights of male and female rats that were 26 and 11%, respectively, below those of controls [46]. The kidneys of these animals were 45 and 42% heavier in male and female rats, respectively, than in the controls, and the adrenal glands of the males were 43% heavier than those of controls.

The subcutaneous injection of sodium tungstate produced a severe drop in rectal temperature, about 8 F in 2 hours, with a return to normal in 8 hours. Most of the subcutaneously administered tungsten was eliminated in the urine within 12 hours, while that administered orally was excreted in urine and feces within 12-24 hours. When sodium tungstate was administered orally, no changes in kidney, adrenal, or body weights resulted; this may be a significant finding, since sodium tungstate administered subcutaneously was slightly more toxic than that given orally.

In 1940, Kinard and Van de Erve [47] compared the influence of age, sex, and postprandial conditions on the effects of subcutaneously injected sodium tungstate in rats. Rats, in equal numbers of each sex, were fed dog chow during the experiment. In the first experiment, aimed at determining the influence of postprandial conditions on the toxicity of sodium tungstate, 2 groups each containing 27 rats were fasted for 24 hours. One group was weighed after food had been withheld for a day, injected with a calculated dose of sodium tungstate, and then fed. A second group was weighed after fasting, fed, and then injected 1-3 hours later with sodium tungstate at a dose based on body weight before feeding. The age and weight of the rats and the amount of sodium tungstate administered were not reported. Mortality was recorded in both groups for an unspecified period.

Mortality was 40.7% in the first group and 14.8% in the second [47]. The authors concluded that rats were less susceptible to sodium tungstate administered during the periods of active absorption and metabolism that follow a meal than to administration following fasting. They conceded, however, that the data presented did not reveal whether the observed effect was caused by impairment in the direct absorption and utilization of

nutrients or by changes in the absorption of tungsten.

The second part of the study [47] was designed to evaluate the effects of age on the toxicity of subcutaneously injected sodium tungstate. Five groups of rats, ranging in age from 30 to 365 days, with an average of 23 rats per group, were fasted for 24 hours and weighed individually. Sodium tungstate was injected subcutaneously at a dose of 150 mg of tungsten/kg body weight. Mortality was recorded for an unspecified period. For rats 30, 44, 66, 170, 195, and 365 days of age, mortalities were 0, 30, 59, 100, 89.5, and 100%, respectively, ie, there was an increase in mortality with increased age. Analysis of deaths in the groups with 10-80% mortality revealed a 15:13 ratio of males to females, indicating that the male rats are slightly more susceptible to injected sodium tungstate than females.

In the third experiment, the dose-response relationship of sodium tungstate in 66-day-old rats was studied [47]. The sex and weight of the rats were not given. Rats were fasted for 24 hours, weighed, injected with sodium tungstate, and then fed. Nine groups of rats, with an average of 25 in a group, were given subcutaneous injections of sodium tungstate in doses equivalent to 100-250 mg of tungsten/kg of body weight. Mortality data were obtained during the 5 days after injection. The authors reported that the LD50 of subcutaneously injected sodium tungstate for 66-day-old rats was 223-255 mg/kg (140-160 mg/kg of tungsten). Their data did not indicate the times after the injection when the first and the most deaths occurred.

Kinard and Van de Erve [48] evaluated the comparative toxicities of tungstic oxide, sodium tungstate, and ammonium-p-tungstate. They used equal numbers of 37-day-old male and female rats, caged separately in

groups of five or six animals. Diets for experimental animals were prepared by incorporating 0.1, 0.5, or 2.0% tungsten equivalents of sodium tungstate, 0.1, 0.5, or 3.96% tungsten equivalents of tungsten trioxide, and 0.5, 2.0, or 5.0% tungsten equivalents of ammonium-p-tungstate into ground dog chow. Control animals were fed the dog chow only. The experiment continued for 70 days.

Rats had 100% mortality when fed diets containing ammonium-p-tungstate equivalent to 5.0% tungsten, tungstic oxide equivalent to 3.96% tungsten, or sodium tungstate equivalent to 2.0% tungsten [48]. However, ammonium-p-tungstate caused 80% mortality at a level equivalent to 2% tungsten. Tungstic oxide given at a level equivalent to 0.5% tungsten caused 80 and 66% mortalities in male and female rats, respectively, while sodium tungstate at the same level caused 50 and 66% mortalities. In comparison, 0.5% tungsten in the diet as ammonium-p-tungstate caused no deaths. Diets containing either sodium tungstate or tungstic oxide in a concentration equivalent to 0.1% tungsten caused no fatalities.

After 70 days, the male and female rats fed ammonium-p-tungstate equivalent to 0.5% tungsten weighed 3.9 and 5.3% less, respectively, than the controls [48]. Male and female rats receiving tungstic oxide at a tungsten equivalent of 0.1% weighed, respectively, 6.3 and 7.4% less than controls; for male and female rats given the same concentration of sodium tungstate, the weight decreases were 8.8 and 10.6%. Corresponding to the decreased weight gains in various groups, there were moderate-to-sharp declines in food intake which appeared to be dose-related. Thus, in male rats fed sodium tungstate at levels of 0.1, 0.5, and 2.0% of tungsten equivalents, the food intakes were 83, 47, and 20% of that of controls,

respectively, while in females they were 96, 72, and 31%. Similarly, the food intakes of tungstic oxide groups at levels of tungsten equivalent to 0.1, 0.5, and 3.96% were 91, 33, and 20% of control values for males and 100, 72, and 51% for females. With diets containing 0.5, 2.0, and 5.0% tungsten as ammonium-p-tungstate, the food intakes of male rats were 102, 27, and 46% of control values. Females fed ammonium-p-tungstate at levels of tungsten equivalent to 0.5 and 2.0% had food intakes 96 and 82% of that of the controls.

It is noteworthy that the proportional decreases in body weight in female rats were greater than those of males, although the tungsten compounds had a smaller effect on the intake of food by the females than on that by the males. This suggests that tungsten exerted some effect that was specific, or at least relatively specific, for females on either digestion and absorption of food stuffs or metabolic utilization of foodstuffs. Balance studies, especially of nitrogen, might be useful in deciding between these two possibilities. Johnson et al [49] found that tungsten could be incorporated into such enzymes as xanthine oxidase and sulfite oxidase in place of molybdeum to yield inactive proteins. This suggests that the second of the two general possibilities mentioned above may be the correct one. Johnson et al used only male rats and fairly high doses of tungsten (25 ppm in the drinking water) to obtain their results. If females were more sensitive to tungsten in this regard than were male rats, the effect on their metabolic activities catalyzed by these important enzymes would be much greater than those of males. Knowledge of the role of sex in this regard seems to be of some importance.

From the mortality and body weight data, the authors [48] concluded that ammonium-p-tungstate was the least toxic of the three compounds. Although the data were not analyzed statistically, this is the first report which reveals that tungstic oxide and ammonium-p-tungstate are less toxic than the soluble sodium tungstate.

Nadeenko [50] compared the acute toxicities of sodium phosphotungstate, sodium tungstate, and tungstic oxide by determining their oral lethal doses in 300 mice and 40 albino rats. The author did not present the experimental details. After brief excitation, all mice and rats given the three tungstate compounds sat with backs arched; their muscle tone was decreased; and the hind legs of some of the mice became slightly paralyzed. From these observations, Nadeenko concluded that the animals exhibited inhibited motor functions. The durations and doses at which these symptoms occurred were not reported. These experiments were evaluated statistically by probit analysis, with the results expressed as elemental tungsten.

The oral LD50 of sodium phosphotungstate was  $700 \pm 79$  mg/kg for mice and  $1,600 \pm 201$  mg/kg for rats [50]. The oral LD50 of sodium tungstate was  $240 \pm 13.5$  mg/kg for mice and  $1,190 \pm 129.5$  mg/kg for rats. Administration of tungstic oxide at 840 mg/kg killed half of the animals (species not stated); however, there was no further increase in the percentage of deaths with doses up to 2,500 mg/kg body. Nadeenko concluded that this compound was less toxic than either sodium tungstate or sodium phosphotungstate. He suggested that the lower toxicity of tungstic oxide was a result of its lower solubility.



Nadeenko [50] also studied the toxic effects of sodium tungstate on rabbits, guinea pigs, and rats. As in the above study, the experimental design was not presented. The oral LD0, LD50, and LD100 were reported as 500, 875, and 1,500 mg/kg respectively, for rabbits, 500, 1,152, and 2,000 mg/kg for guinea pigs, and 700, 1,190, and 2,000 mg/kg for rats. The author concluded that rabbits were more susceptible to the toxic action of sodium tungstate than were either rats or guinea pigs.

To study the selective effect of tungsten on individual physiologic functions and systems of animals, Nadeenko [50] conducted a set of subacute experiments for an unspecified period of time on albino rats and rabbits. The animals were given sodium tungstate orally at presumably daily doses of 100, 50, 25, or 10 mg/kg. The number, age, sex, weight, and strain of the animals were not reported, and no control group was described.

Nadeenko [50] reported that, at all doses, the growth of the rats was retarded and their blood cholinesterase activities were lowered; in rabbits, the sulfhydryl (SH) concentrations of whole blood and serum were decreased and synthesis of glycogen in the liver was disturbed. Stained sections of the gastrointestinal tract and kidneys showed signs of increased vascular permeability, hemorrhages, degenerative dystrophic changes, and moderate proliferative cellular reactions.

Nadeenko [50] also attempted to determine the noneffective and threshold chronic oral doses of tungsten in rats and rabbits. Sodium tungstate was administered to rabbits in doses of 5, 0.5, 0.05, or 0.005 mg/kg daily for 8 months and to rats in doses of 0.5, 0.05, or 0.005 mg/kg daily for 7 months. There were seven animals in each dosage group. Their sex, age, weight, and strain were not presented. Control groups of the

same size were used, but it was not clear how they were treated. Throughout the exposures, the general behavior and body weight of the animals were monitored. The blood SH content and cholinesterase and alkaline phosphatase activities were measured in the rabbits. Blood cholinesterase and alkaline phosphatase activities were determined at monthly intervals. The glycogen-forming function of the liver was measured in rabbits after loading them with galactose. In rats, the conditioned reflexes were monitored on animals trained to respond to the sound of a bell and to a light, and this test was used as a measure of the functional state of the CNS. The intervals at which such measurements were made were not given. At the end of the experimental period, the SH contents of brain and liver tissues of rats were determined. The author reported, without identifying the species, that, after the termination of chronic poisoning, he determined the tungsten concentrations in blood, liver, femur, intestines, and kidneys; he also conducted macroscopic and microscopic examinations of the gastrointestinal tract, lungs, liver, spleen, and brain of both species.

In rabbits, sodium tungstate at doses of 5 and 0.5 mg/kg of body weight decreased the availability of SH groups in serum during months 5-8 of the experiment and produced decreases of about 23 and 16%, respectively, in blood cholinesterase activity at the end of 8 months [50]. Sodium tungstate at doses of 0.05 and 0.005 mg/kg caused no changes in either the SH content or the blood cholinesterase activity of rabbits. Blood alkaline phosphatase activity was inhibited by as much as 18% ( $P < 0.01$ ) by sodium tungstate at a dose of 5.0 mg/kg during months 4-8 of the experiment. However, in the 0.5-mg/kg and 0.05-mg/kg groups, changes were sporadic and

inconsistent and, in the 0.005-mg/kg group, the blood alkaline phosphatase activity was not inhibited. Rabbits receiving sodium tungstate doses of 5.0 and 0.5 mg/kg of body weight had concentrations of glucose in their blood 20-25% higher than control levels 1 hour after iv loading with galactose. Nadeenko noted this change at months 6 and 7 of the experiment and interpreted it as an indication that the glycogen-forming function of the liver had decreased. No changes in the glycogen-forming function of the liver were observed in the 0.05 and 0.005 mg/kg groups. None of the rabbits showed pathologic changes in the esophagus, large intestines, lungs, liver, or spleen. In rabbits given tungsten at 5 mg/kg, some areas of the intestinal mucosae showed an increase in lymphoid infiltration of the villi and necrosis of their terminal portions.

In rats, sodium tungstate at doses of 0.5 and 0.05 mg/kg caused pronounced disturbances in conditioned reflexes [50]. The latent periods of sodium tungstate-treated animals were 1.6-1.7 seconds for the bell and 2.4-2.7 seconds for the light, compared to 0.9 and 2.0 seconds, respectively, for controls. Animals given the maximum doses of sodium tungstate exhibited a larger number of extinctions of the conditioned reflexes. Disturbances of conditioned reflexes were indicated by a statistically significant increase in the number of equalizing and paradoxical phase states (For explanation of terminology, see reference [51]). No data were presented on the statistical analyses. Nadeenko noted that study of extinction and recovery of the conditioned response to a bell revealed a pronounced decrease in the lability of nervous processes in the cerebral cortices of the 0.5 and 0.05 mg/kg dose-groups. No changes were noted in the conditioned reflex with a dose of 0.005 mg/kg. Necrotic

lesions and destruction of the apical portions of the intestinal villi were also evident in rats given sodium tungstate at 0.5 mg/kg. No animal had pathologic changes in the esophagus, large intestine, lungs, liver, or spleen.

Although he gave no values, Nadeenko [50] reported that analyses of the concentrations of tungsten in various tissues of experimental animals (species unspecified) revealed that the highest concentrations existed in groups receiving 5 and 0.5 mg/kg of sodium tungstate. A less pronounced but significant accumulation of tungsten was found in tissues of animals given sodium tungstate at 0.05 mg/kg, while a dose of 0.005 mg/kg produced increases only in the concentration of tungsten in the blood and intestines. From these findings, the investigator concluded that tungsten has cumulative toxicity. Nadeenko believed that the daily dose of 0.05 mg/kg was below the threshold level, since it did not cause appreciable biochemical changes in the animals, although it did affect the conditioned reflexes of rats.

In 1945, Kinard and Aull [52] described the distribution of tungsten in rat tissues after dietary feeding of tungsten or its compounds. Rats of an unspecified strain, 37 days old, were caged in groups of two males and two females. During the 100-day experimental period, the control group received ground dog chow, while the experimental groups were fed this basal diet with the incorporation of tungsten or tungsten compounds at one of the following levels: tungstic oxide and sodium tungstate at levels equivalent to 0.1% tungsten; ammonium-p-tungstate at 0.5% tungsten; tungsten metal at 2 and 10% tungsten; and purified tungsten metal (purified to remove any trace of oxide from the metal) at 10% tungsten. Diet and water were given

ad libitum to each group of rats.

Analysis of the tissues removed at the end of the 100-day period revealed that bone and spleen were the major sites of tungsten deposition [52]. The concentrations of tungsten ranged from 8 to 18 mg% in bone and from 2 to 14 mg% in the spleen, with averages of 11.5 and 7.5 mg%, respectively. Only traces of tungsten (less than 1 mg%) were present in skin, kidneys, and liver. The blood, lungs, testes, and muscles showed traces of tungsten only in some cases. Except for a single instance for each organ, the brain, heart, and uterus were free of tungsten. The investigators concluded that there were no marked differences among the distribution patterns of the various tungsten compounds tested. Since the doses of tungsten administered as various tungsten compounds were not comparable and since quantitative results were not presented for all the tissues that were analyzed, this conclusion may not be entirely valid.

Aamodt [53] measured the metabolism of inhaled tungstic oxide in dogs. Six purebred beagles, of unspecified sex and age, were anesthetized with pentobarbital at 27 mg/kg. They then inhaled a mist of radiolabeled <sup>181</sup>W-tungstic oxide of 98 microcuries/ml specific activity through a face-mask for 6 hours. The dogs were killed 165 days after exposure, and tissue samples were collected from all dogs.

Measurements of the radioactivity in inhaled and expired air showed that 1.9-8.0 microcuries of tungstic oxide were deposited in the respiratory tract [53]. Sixty percent of the inhaled activity was deposited in the lower part of the respiratory tract. In the partial body measurements made over the lung area, about 69% of the activity was lost with a biologic half life ( $t_{1/2}$ ) of 4 hours, the next 23% with a  $t_{1/2}$  of 20

hours, 4.6% more with a  $t_{1/2}$  of 6.3 days, and 3% with a  $t_{1/2}$  of 100 days. In contrast, measurements taken for the lower half of the body showed 94% with a  $t_{1/2}$  of 9 hours, whereas 4.1% had a  $t_{1/2}$  of 6.3 days. The remaining 1.6% of the radioactivity was removed with a  $t_{1/2}$  of 139 days. Without giving any actual experimental values, Aamodt indicated that the activity was lost rapidly from the blood, although there were some variations in the measured activity which, he explained, were caused by biologic variation in the clearance patterns in individual dogs. Of the organs tested, the lungs and kidneys were found to retain maximum radioactivity, 47.7 millionths and 41.3 millionths of the inhaled activity/g of tissue, respectively. Other tissues contained only about 10% as much activity as did the lungs and kidneys. In terms of total burden of radioactivity, most of the activity was found in the skeleton (37%), lungs (31%), kidneys (15%), liver (9.7%), and skeletal muscle (5.7%).

The reported effects of insoluble tungsten or cemented tungsten carbide administered intratracheally or by inhalation were chiefly respiratory in nature. No studies are available of the effects of soluble tungsten compounds given by similar routes of exposure. Moreover, the oral administration studies did not clearly distinguish the principal organs affected by soluble tungsten. Few reports are available on the dermal effects of tungsten compounds or cemented tungsten carbide in experimental animals.

#### Correlation of Exposure and Effect

Effects of both short-term and long-term occupational exposures to tungsten and its compounds have been identified among employees in the

cemented tungsten carbide industry (Table III-1). The only work areas in this industry that permit specific evaluation of the effects of tungsten and its compounds are those processing stages that precede the incorporation of other toxic metals into the final products. Only two human studies meeting this criterion were obtainable [27,28]. These studies showed that the effects of inhaled tungsten and tungsten compounds are exerted chiefly on the respiratory system. Radiologic signs of pulmonary fibrosis were reported by Mezentseva [28] and by Kaplun and Mezentseva [27] in 9-11% of the hard-metal workers who were exposed to dusts of tungsten and its compounds.

In both short- and long-term animal experiments, the major effects of inhalational or intratracheal exposure to tungsten and its compounds were similarly limited to the respiratory system, while the effects of ingestion were not so clearly apparent in any organs (Table III-2). As in the reports of the human studies, no dermatologic effects were described in these animal reports. Mezentseva [28] reported that lungs of rats exposed by inhalation to tungsten carbide at 600 mg/cu m, 1 hour/day, for 5 months showed proliferative reactions of the lymphoid histiocytic elements and uniform thickening of the alveolar walls followed by mild fibrosis. Mezentseva [28] also stated that rats given single intratracheal doses of 50 mg of either metallic tungsten, tungsten carbide, or tungsten trioxide showed no severe pulmonary changes upon microscopic examination.

Dusts of metallic tungsten or tungsten carbide, given intratracheally to guinea pigs by Delahant [35] as a total dose of 150 mg in three equal weekly doses, did not irritate the lung tissue. Similarly, 1 ml of a 10% suspension of tungsten was given intratracheally to rats by Miller et al

[16], who found that the only changes in the animals' lungs, such as mobilization of septal cells, engulfment of pigment, and accumulation of particles in the air sacs, lymphoid tissue, and alveolar walls, were those typically produced by an inert dust.

However, Schepers [34] found that the intratracheal injection of 3 weekly doses of 50 mg of a 94:6 mixture of tungsten carbide and carbon caused acute hyperemia and bronchial inflammation in guinea pigs. Minor residual changes, such as the development of subpleural fibrocellular granulomata, were also noted in their lungs. Brakhnova and Samsonov [40] reported that inhalational and intratracheal exposure of rats to tungsten silicide for 1-6 months caused hyperplasia of the lymph nodes, sporadic thickening of the alveolar walls, and increased collagen in the lungs. These results suggest that tungsten and some of its compounds, such as those most frequently encountered in the cemented tungsten carbide industry, have distinct toxicities.

While opportunities for occupational exposures to soluble tungsten compounds are limited, a number of animal studies are available delineating their toxicities. In 1924, Karantassis [45] administered single intragastric and subcutaneous doses of sodium tungstate to guinea pigs and observed anorexia, colic, incoordination of movement, trembling, dyspnea, and loss of weight prior to death. Rats injected subcutaneously with sodium tungstate also had congested livers and large infarcts in both lungs. Smaller subcutaneous doses of sodium tungstate also produced degenerative patches in liver and kidneys. Selle [46] also noticed that rats given subcutaneous doses of sodium tungstate had increased kidney weights.



Two studies attempted to differentiate between the toxicities of the soluble and the insoluble tungsten compounds. Kinard and Van de Erve [48] fed young male and female rats diets containing 0.1-5% tungsten equivalents of tungstic oxide, sodium tungstate, and ammonium-p-tungstate. At these levels, 100% mortality was caused by a 2% tungsten equivalent of sodium tungstate, by a 3.96% tungsten equivalent of tungstic oxide, and by a 5% tungsten equivalent of ammonium-p-tungstate. Tungsten equivalents of 0.5% in the diet caused no deaths in the ammonium-p-tungstate group; tungstic oxide, at the same concentration of the metal caused 88 and 66% deaths in males and females, respectively, and sodium tungstate caused 50 and 60% deaths. Sodium tungstate was concluded to be the most toxic of the three compounds.

Nadeenko [50] compared the oral toxicities of tungstic oxide, sodium tungstate, and sodium phosphotungstate in rats and mice. After brief periods of excitability, all mice and rats given the three tungsten compounds sat with backs arched; their muscle tone was decreased, and the hind legs of some mice became slightly paralyzed. In mice, the oral LD50 of tungstic oxide was 840 mg/kg, while those for sodium phosphotungstate and sodium tungstate were 700 and 240 mg/kg, respectively. Nadeenko concluded that tungstic oxide was the least toxic of the three and suggested that its lower solubility, compared with those of the other two compounds, was the cause. Thus, soluble tungsten compounds were considerably more acutely toxic in experimental animals than the insoluble compounds.

Most reports of occupational exposure to tungsten and its compounds, with the exception of the two studies already described, deal with the

effects of mixed dusts containing cobalt [27,28]. The effects of such mixed dusts were chiefly respiratory in nature, although some dermal effects were evident. The pulmonary involvement reported in a number of these studies was characterized by exertional dyspnea, coughing, and weight loss [15,17-19,25]. These clinical signs sometimes progressed to extrinsic asthma [23], diffuse interstitial pneumonitis [18], or fibrosis [17,20,22]. The type of pneumoconiosis seen in the cemented tungsten carbide industry is referred to as "hard-metal disease." While the total dust levels and cobalt concentrations were reported in most studies, tungsten concentrations were documented in only a few cases. Most dust particles generated in various operations in which tungsten is processed and used are less than 5  $\mu\text{m}$  in diameter and hence are in the respirable size range [17,25,28,42,54].

Some authors described the pulmonary responses of cemented tungsten carbide workers as hypersensitivity [17,20]. This response was so described because of the reversibility of some clinical symptoms, the occasional radiologic improvement on withdrawal from exposure, and the recurrence of symptoms on reexposure. Bruckner [23] diagnosed extrinsic asthma in a cemented tungsten carbide worker and attributed it to a hypersensitivity mechanism. This worker experienced asthmatic symptoms 1-3 minutes after beginning work, even though he wore a respirator designed to remove particles of 0.6  $\mu\text{m}$  diameter or larger.

Two studies [14,24] described the dermatologic effects of occupational exposures to unspecified levels of dusts in cemented tungsten carbide industries. Schwartz et al [14] stated that 20 workers employed 1 month or more in this industry developed erythematous, papular dermatitis,

mainly on the sides of the neck, the eyelids, and the forearms. While the abrasiveness of the dust reportedly contributed to the sensitization process, cobalt sensitization was concluded to be the cause of the dermatitis. Skog [24] reported skin effects including contact eczema, pruritus, folliculitis, and neurodermatitis, in 34 (9.4%) of the 361 workers in the cemented tungsten carbide industry. Cobalt sensitization was detected by patch tests in 3 of the 14 workers with contact eczema, found mainly on the eyelids and between the fingers. This author [24] concluded that the primary irritant effect of the combined metal dusts produced most of the contact eczema.

Although these studies are well documented in terms of the observed effects, it is difficult to distinguish the effects of tungsten and its compounds from those produced by cobalt and perhaps other metals and compounds. However, most of the authors attributed the effects of these mixed exposures primarily to the presence of cobalt.

The effects of exposures of experimental animals to tungsten-cobalt mixtures on the respiratory system are well documented, but dermatologic effects were not described in laboratory animals. In a collaborative experiment with Delahant [35], Schepers [37] noted that guinea pigs, exposed by inhalation to a 3:1 mixture of tungsten carbide and cobalt for 20 days at 8,800-10,000 particles/cu cm and then at 2,800 particles/cu cm for 15 days developed acute inflammation of the respiratory tract, followed by focal pneumonitis and residual bronchial epithelial hyperplasia and metaplasia. He emphasized that the unusual epithelial reaction was the characteristic feature of exposure to a mixture of tungsten carbide and cobalt. Delahant [35] noted that five animals died during the 20-day

exposure and that an additional six died during the following 15-day exposure. Delahant [35] and Schepers [37] also examined the responses of guinea pigs to intratracheal administration of 3 weekly 50-mg doses of a 91:9 mixture of tungsten carbide and cobalt. They reported pneumonitis and mild cellular and fibrotic reactions around the dust deposits in the lungs of these guinea pigs. Delahant [35] and Schepers [37] concluded that the intense irritant property of cobalt was dominant when cobalt was combined with tungsten carbide. Kaplun and Mezentseva [38] injected rats intratracheally with the following dust mixtures: (1) 15% cobalt and 85% tungsten, (2) 8% cobalt and 92% tungsten, or (3) 8% cobalt, 14% titanium, and 78% tungsten. The rats that received 25 mg of dust had 100, 40, and 60% mortalities in groups 1, 2, and 3, respectively, within 5-7 days. At the 10 mg-dose level, 5 of the 13 rats in group 1 died within 2-3 days, but none of the rats in groups 2 and 3 died.

The absolute lethal dose for the dust mixture containing 15% cobalt was 15 mg (2.25 mg cobalt)/rat, while that for the other two mixtures was 50 mg (4 mg cobalt) [38]. Comparing these values with the absolute lethal dose of metallic cobalt alone (10 mg), the authors suggested that the toxicity of cobalt was enhanced in the presence of tungsten because the latter increased the solubility of the former. However, as the absolute lethal doses of the three dust mixtures were 50-400% greater than that of metallic cobalt, the results do not necessarily support the hypothesis of enhanced toxicity of cobalt; cobalt and tungsten may, however, have synergic actions that are additive to some degree.

## Carcinogenicity, Mutagenicity, Teratogenicity, and Effects on Reproduction

In 1961, Bech et al [25] reported a case of cancer in a 63-year-old man who had been exposed to hard-metal dusts containing tungsten for 17 years and who had previously worked as a core maker in various iron foundries for 32 years. Ten years after beginning employment in the hard-metal industry, he developed a persistent dry cough, dyspnea, and pain and tightness in the chest. Radiologic chest examinations revealed hilar shadows, linear markings, and micro-nodular opacities. Four years later, there were signs of progressive deterioration with increased shadows above the right portion of the diaphragm. The subject died 3 years later with an anaplastic adenocarcinoma of the right lower lobe of the bronchus. The hilar and paraaortic glands were also invaded by the tumor, and a neoplastic invasion of the right renal capsule was noted at autopsy. Since the worker had been exposed to a number of industrial dusts of unknown composition and amounts, the tumor cannot be attributed specifically to exposure to tungsten.

Collet and coworkers [26], in 1963, described a case of pulmonary fibroadenomatosis in a 57-year-old worker employed for 14 years in the manufacture of hard-metal tools. During this period, he was known to have been exposed to dusts of tungsten carbide, metallic cobalt, tantalum, titanium, and cobalt oxalate, and to trichloroethylene vapor. After he had worked in this environment for 8 years, radiologic examination of his chest revealed reticulation at the bases and in the left central portion of the lungs. After 2 more years, fever and breathing difficulties were reported; a year later, exertional dyspnea had become more severe. During the next three years, clinical observations and pulmonary function tests indicated

that his condition had deteriorated further. At the end of this period, he died of respiratory failure and cardiovascular collapse. A biopsy of lung tissue performed 7 months before his death led to the diagnosis of pulmonary fibroadenomatosis; spectrographic analysis of the tissue taken for biopsy demonstrated what were described as large amounts of tungsten, nickel, and titanium in his lungs, although he had no known occupational exposure to nickel. In this case, as in the case reported by Bech et al [25], the worker had been exposed to a mixture of metal dusts and to the vapor of trichloroethylene at unknown concentrations [26], The precise role of tungsten in the development of the fibroadenoma cannot therefore be determined.

The aforementioned reports indicate that tungsten and its compounds are neither confirmed nor suspected as carcinogens, mutagens, or teratogens.

TABLE III-1

EFFECTS OF OCCUPATIONAL EXPOSURE  
TO TUNGSTEN-CONTAINING SUBSTANCES

Composition of Substances	Exposure Concentration and Duration	Number of Workers	Effects	Reference
Tungsten carbide	8.6-106.6 mg/cu m	36	Diffuse pulmonary fibrosis in 4	27
"	8.3-83 mg/cu m 7-10 yr	54	Diffuse pulmonary fibrosis in 5	28
Cemented tungsten carbide 90% tungsten, 6% cobalt, 4% titanium, silica, aluminum, magnesium, iron, etc	195-1,230 particles/ ml	255	Hard-metal disease in 1; in 12 given pulmonary function tests, reduced ventilatory capacity and rise in airway resistance in 2, rise in respiratory airway resistance in 2, wheezing in 4; slight change in chest X-rays of several	25
Cemented tungsten carbide 70-90% tungsten carbide, 8-78% titanium carbide, 5-25% cobalt	13-100 mg/cu m	193	Coughing in 47%, dyspnea in 35%, disturbance of pulmonary ventilation in 21.5% of 116 examined by spirometry; reticulation in chest X-rays in 16% of 93 workers	31
Cemented tungsten carbide 0.27-1.75 mg/cu m of cobalt	3-186 mg/cu m	247	Damage to upper respiratory tract in 117; chronic bronchitis in 35; incipient pulmonary fibrosis in 33	27
Cemented tungsten carbide 2.4-4.1% cobalt	2.1-3.5 mg/cu m 6 yr	1,802	Inflammation of conjunctivae, upper respiratory tract, and mucous membranes; chest X-ray abnormalities in 36; cobalt sensitivity	15
Cemented tungsten carbide 0.6-3.2 mg/cu m of cobalt	0.75-6.1 mg/cu m up to 3 yr	178	Dyspnea, coughing, impaired sense of smell in 88; some impairment of liver function	32
Cemented tungsten carbide 0.04-0.93 mg/cu m of cobalt 0.28 mg/cu m mean	0.2-12.8 mg/cu m as tungsten; 5.1 mg/cu m mean 1-30 yr	22	Reduced FVC and elevated FEV <sub>1</sub> /FVC ratio; exertional dyspnea in 3; productive cough in 8	29
Cemented tungsten carbide 80-90% tungsten carbide, 8-18% titanium carbide, 5-25% cobalt	-	375	Allergic asthma and exertional dyspnea in 1, reversed on removal from work environment; coughing; dyspnea, restrictive ventilatory defect, and micronodular or linear opacities on chest X-rays in 12, with death of 8 from bronchopneumonia or cardiorespiratory failure	19
Cemented tungsten carbide	2.3-62.3 mg/cu m up to 17 yr	29	Dry cough, exertional dyspnea in 9; allergic asthma in 3; pulmonary fibrosis in 1	17
Cemented tungsten carbide pure metals, carbides, and oxides of tungsten, titanium, tantalum, and niobium, and cobalt metal	-	100	Irritation of upper respiratory tract in 15, diminishing on removal and recurring on reexposure; exertional dyspnea (not reversible), rapid weight loss in 5	20
Cemented tungsten carbide	-	5	Episodic wheezing and coughing, relieved by removal from work environment and recurring upon return; no chest X-ray abnormalities	18

TABLE III-1 (CONTINUED)

EFFECTS OF OCCUPATIONAL EXPOSURE  
TO TUNGSTEN-CONTAINING SUBSTANCES

Composition of Substances	Exposure Concentration and Duration	Number of Workers	Effects	Reference
Cemented tungsten carbide 75% tungsten carbide, 3-25% cobalt, small amounts of titanium carbide, chromium carbide, and nickel	- 12-28 yr	12	Cough, exertional dyspnea, progressive interstitial fibrosis; cor pulmonale leading to cardio-respiratory arrest in 8	18
Cemented tungsten carbide 67.8%-76.1% tungsten, 7.6-21.2% cobalt, 0-1.7% titanium, 0.3-2.3% iron, 7.0%-16% volatiles (paraffin)	277-4,064 particles/ cu m	208	Asthmatic symptoms, bronchitis, emphysema, pulmonary fibrosis	33
Cemented tungsten carbide tungsten, cobalt, titanium, tantalum, niobium, or their oxides and carbides	- -	1	Coughing, expectoration, exertional dyspnea, diffuse bilateral clouding, nonresonant wet rales; restrictive ventilatory impairment; improved on removal from work environment	21
Cemented tungsten carbide tungsten, cobalt, or tantalum	- 1 yr	1	Exertional dyspnea, dry irritated cough, fever, cyanosis, rales, restrictive ventilatory, impairment of lung function; small lungs with striated or honeycomb pattern; no improvement on removal from work environment	21
Cemented tungsten carbide tungsten carbide, cobalt, sometimes titanium and tantalum carbide	- 7 yr	1	Diffuse bilateral densities from chest X-rays; reduced vital capacity; lung biopsy showed multifocal pulmonary scarring with patchy, interstitial fibrosis	30 29
High speed steel containing cobalt				
Cemented tungsten carbide tungsten, tantalum, and titanium carbides, cobalt	- 1 mon or more	20 of 1,200	Erythematous, papular eruptions on neck, eyelids, forearms, backs of hands and ankles; patch tests on 6 showed sensitivity to cobalt powder	14
Cemented tungsten carbide 0-95% tungsten carbide, 0-40% titanium carbide, 0-20% tantalum-niobium carbide, 5-25% cobalt	- -	34 of 361	Contact eczema in 16; pruritus without skin lesions in 8; folliculitis in 6; neurodermatitis in 4; patch testing in 14 who had contact eczema revealed that 3 reacted to cobalt chloride and 2 to hard-metal powder as well	24
Cemented tungsten carbide tungsten carbide, metallic cobalt, tantalum, cobalt oxalate	- 14 yr	1	Fever, exertional dyspnea, reticulonodular opacities on chest X-rays; reduced vital capacity; death from respiratory failure and cardiovascular collapse; diagnosis of pulmonary fibroadenomatosis	26
Vapors of trichloroethylene				



TABLE III-1 (CONTINUED)  
EFFECTS OF OCCUPATIONAL EXPOSURE  
TO TUNGSTEN-CONTAINING SUBSTANCES

Composition of Substances	Exposure Concentration and Duration	Number of Workers	Effects	Ref- erence
Cemented tungsten carbide 90% tungsten, 6% cobalt, 4% titanium, alumi- num, magnesium, iron, etc	195-505 particles/ml  22 mon-22 yr	6	Persistent dry cough; dyspnea, and pain and tightness in chest; hilar shadows, linear markings, and micro- nodular opacities in chest X-rays; one died from anaplastic adenoma of lower lobe of bronchus	25
Cemented tungsten carbide primarily tung- sten carbide	34 mon -	1	Wheezing, respiratory distress, 2 asthma attacks requiring hospital- ization; asthmatic symptoms re- curred on reexposure to work en- vironment; diagnosis of extrinsic asthma	23

TABLE III-2

EFFECTS ON ANIMALS OF INHALATION EXPOSURE AND INTRATRACHEAL INJECTION OF  
TUNGSTEN-CONTAINING SUBSTANCES

Composition of Substance	Route of Exposure	Species	Exposure Concentration, Duration	Effects	Reference
Tungsten hexachloride	Inhalation	Rats	43 - 140 mg/cu m	Lacrimation, profuse blood-stained oral and nasal discharges; death	41
Tungsten carbide: cobalt 75:25	"	Guinea pigs	8,800 - 10,600 particles/cu m x 20 d, then 2,900 particles/cu m x 15 d	Lung inflammation, dilation of alveolar-wall capillaries, infiltration by lymphocytes and plasma cells	37
Tungsten carbide: cobalt 10:1	Intratracheal	"	50 mg/dose 1 dose/wk x 3 wk	Pneumonitic areas in lungs, lymphocytic infiltration of perivascular areas, bronchial crypts	37
Tungsten carbide: cobalt 94:6	"	"	50 mg/dose 1 dose/wk x 3 wk	Hyperemia and bronchial catarrh, minor interstitial pneumonitis, lymphoid hyperplasia, trapped dust masses in lungs, pleural fibrocellular granulomata	34
Tungsten dust	"	"	50 mg/dose 1 dose/wk x 3 wk	Interstitial pneumonitis and bronchiolitis	36

TABLE III-3

## EFFECTS ON ANIMALS OF ORAL DOSES OF TUNGSTEN-CONTAINING SUBSTANCES

Composition	Route of Exposure	Species	Exposure Concentration Duration	Effects	Reference
Powdered tungsten	Oral	Rats	10% in diet	Weight loss in females 15.4%; no effects in males	44
Sodium tungstate	"	Guinea pigs	2,000 mg/kg	LD50	50
"	"	"	1,152 mg/kg	"	50
"	"	"	500 mg/kg	LD0	50
"	"	Mice	240 mg/kg	LD50; arched backs, decreased muscle tone, slight paralysis of hindlegs	50
"	"	Rats	2,000 mg/kg	LD100	50
"	"	"	1,190 mg/kg	LD50	50
"	"	"	700 mg/kg	LD0	50
"	"	"	2% in diet for 70 d	Death of 100%	48
"	"	"	0.5% in diet for 70 d	Death of 80%	48
"	"	Rabbits	1,500 mg/kg	LD100	50
"	"	"	875 mg/kg	LD50	50
"	"	"	500 mg/kg	LD0	50
"	"	Guinea pigs	0.50 - 0.75 g/ 600 g	Anorexia, colic, uncoordinated movements, trembling, dyspnea; death at 16-23 hr	45

TABLE III-3 (CONTINUED)

## EFFECTS ON ANIMALS OF ORAL DOSES OF TUNGSTEN-CONTAINING SUBSTANCES

Composition	Route of Exposure	Species	Exposure Concentration Duration	Effects	Reference
Sodium tungstate	Oral	Guinea pigs	0.55 g/kg	Single lethal dose	45
Sodium phospho-tungstate	"	Mice	700 mg/kg	LD50; arched backs, decreased muscle tone, slight paralysis of hindlegs	50
Tungstic oxide	"	"	840 mg/kg	"	50
"	"	Rats	0.5% in diet for 70 d	LD50	48
Ammonium-p-tungstate	"	"	2% in diet for 70 d	Death of 80%	48
"	"	"	0.5% in diet for 70 d	No deaths	

#### IV. ENVIRONMENTAL DATA AND ENGINEERING CONTROLS

##### Environmental Concentrations

Most of the environmental data available on tungsten and its compounds are derived from cemented tungsten carbide (hard-metal) industries (Table IV-1) [29,55-60]. Depending on the stage and type of the process, the proportions of tungsten, cobalt, and other components in the airborne dusts vary considerably [59]. The initial stages of processing the ore involve exposure to primary ore dusts, namely those of scheelite and wolframite. In the course of production and use of tungsten, aerosols of sodium tungstate ( $\text{Na}_2\text{WO}_4 \cdot 2\text{H}_2\text{O}$ ), ammonium-p-tungstate [ $5(\text{NH}_4)_2\text{O} \cdot 12\text{WO}_3 \cdot 11\text{H}_2\text{O}$ ], tungstic oxide ( $\text{WO}_3$ ), tungsten pentoxide ( $\text{W}_2\text{O}_5$ ), tungsten dioxide ( $\text{WO}_2$ ), metallic tungsten (W), tungsten carbide (WC), and their mixtures may be generated. In the stages following the preparation of tungsten or tungsten carbide, exposures may occur also to aerosols of such binder metals as cobalt and nickel and to other commonly added metals and their derivatives (Figure XII-1).

Except for the studies by Lichtenstein et al [29], Fairhall et al [56], and McDermott [60], very little information is available on the environmental concentrations of tungsten, cobalt, or total dust in the cemented tungsten carbide industry in the United States. Because of differences in work practices and engineering controls, it is not possible to extrapolate directly from the observations of foreign investigators on environmental concentrations in industry to occupational exposures in the United States. However, the basic steps in manufacturing cemented tungsten carbide are quite similar throughout the world, so that a review of foreign

reports would provide a valuable comparative assessment of potential exposure sites in the industry. A number of factors might contribute to the observed variations in the environmental concentrations of tungsten, cobalt, or total dust among the various studies. Values might be affected by the sampling zone selected, ie, breathing or general atmosphere, and by the sampling and analytical methods. In addition, Fairhall et al [56] suggested that individual plant structure, ventilation, layout of machines and processes, and the segregation of the dustiest operations from the others were factors influencing the environmental concentrations and operator exposures.

Fairhall et al [56] determined the average dust and cobalt content of air in powder processing and tool and die operations in three cemented tungsten carbide plants (Table IV-1). The total dust concentrations in the general atmosphere were 20% and 50% lower than the operators' exposures in the powder processing and tool and die operations, respectively. The cobalt concentrations in the powder processing and tool and die operations were, respectively, 10% and 5% of the total dust concentrations. The authors emphasized that the total dust values are given as averages and that there are operations, such as calcination of cobalt nitrate, which produced total dust concentrations as high as 123 mg/cu m with cobalt concentrations as high as 79 mg/cu m. Operations such as screening powders, weighing and milling, and cleaning the mills were also reported to produce excessive exposures to dust and cobalt. Powder processing was the dustiest operation surveyed. Fairhall and his colleagues also noted the presence of cobalt in all areas of the plants, including those where no manufacturing was done, a significant finding.

To determine typical dust exposures, McDermott [60] studied the atmospheric cobalt levels of various operations in seven cemented tungsten carbide plants. The cobalt concentrations in the 173 samples ranged from undetected to 0.8 mg/cu m. Basing his conclusions on the present United States Federal occupational environmental limit for cobalt (0.1 mg/cu m), he found that 70% of the samples represented acceptable working conditions.

Lichtenstein et al [29] reported the airborne tungsten and cobalt concentrations in operations involving wet-grinding of tool bits and inserts made of two commercial grades of cemented tungsten carbide: one contained 72% tungsten carbide, 8% titanium carbide, 11.5% tantalum carbide, and 8.5% cobalt; the other contained 94% tungsten carbide and 6% cobalt. About 70% of the tool-grinding activity involved the latter grade. Approximately 75% of the tool-work involved regrinding old carbide tips, and 25% involved grinding new carbide tips. The air was sampled with filters in the workers' breathing zones, and the filters were analyzed for tungsten and cobalt by atomic absorption spectrometry. The concentrations of tungsten ranged between 0.2 and 12.8 mg/cu m, while those of cobalt were 0.04-0.93 mg/cu m. Mean concentrations were 5.16 mg/cu m for tungsten and 0.28 mg/cu m for cobalt. Of the 25 samples taken, 40% exceeded 5 mg/cu m for tungsten and 60% exceeded 0.1 mg/cu m for cobalt. Since such airborne levels of tungsten and cobalt could not be effectively controlled by an existing exhaust ventilation system, Lichtenstein et al designed an improved exhaust ventilation system which is described in the Engineering Controls section. A follow-up study done after the installation of the improved system reportedly showed cobalt concentrations well below the detection limit of an atomic absorption spectrophotometer and TWA exposures

one order of magnitude below the TLV for cobalt.

Reber and Burckhardt [57] evaluated the potential exposures of workers to airborne total dust, tungsten, and cobalt in operations such as mixing, die casting, forming, and grinding of sintered pieces in five Swiss hard-metal plants. The results showed that the cobalt limit of 0.5 mg/cu m was exceeded by 13% of the measurements in the mixing division and by 25% of the values in the forming area. However, cobalt concentrations in the die casting division never reached the limit. In comparison, the limit of 6 mg/cu m for tungsten was rarely exceeded in these operations. The authors attributed the high dust and cobalt concentrations in mixing and forming divisions to the machining of unfinished pieces with large diameters, a process which generates sufficiently large amounts of the dusts to exceed the capacity of the exhausts. In the grinding of sintered pieces, Reber and Burckhardt found that dry grinding produced high total dust and cobalt concentrations, and they suggested that wet grinding gave concentrations far below the appropriate limits.

Salikhodzhaev and Vengerskaya [59] examined the airborne concentrations of total dust, tungsten, and cobalt generated in various processing stages of hard-metal production in the USSR. During the powder processing stage, the dust levels were between 10 and 693 mg/cu m with averages of 3-130 mg/cu m for various operations. The dust levels were between 3.3 and 153 mg/cu m (averages 5.5-48 mg/cu m for various operations) during casting and between 3.3 and 32.5 mg/cu m (average 13 mg/cu m) during forming operations. Thus it appears that high levels of dust are generated during all stages of processing, although powder processing generated the highest levels. However, in all of the



operations, the average tungsten concentrations were well below the limit for tungsten. In the powder processing operation, the 0.5 mg/cu m limit for cobalt was exceeded. The authors did not explain why the tungsten concentrations were well below the limit when the dust levels were quite high in many of these operations.

In general, dust concentrations generated in hard-metal production were higher during the powder processing and forming stages than during either die casting or grinding of sintered products. The dust levels presented in Table IV-1 indicate that the highest exposures might occur in the powder processing and forming operations.

#### Engineering Controls

To decrease the concentration of tungsten to the recommended limit or below it, engineering controls must be instituted where the airborne concentrations of tungsten exceed the TWA concentration limits. Industrial experience indicates that closed-system operations are commonly used in manufacturing processes. Such systems must be used whenever feasible to control tungsten and its compounds wherever they are manufactured, processed, packaged, or used. Closed systems should operate under negative pressure whenever possible so that, if leaks develop, the flow of air will be inward. Closed-system operations are effective only when the integrity of the system is maintained. The system should be inspected frequently and leaks should be repaired promptly.

A ventilation system may be required if a closed system proves to be impractical, and is desirable as a standby if the closed system should

TABLE IV-1  
 CONDITIONS OF TUNGSTEN-COBALT EXPOSURES  
 IN THE HARD-METAL INDUSTRY

Type of Operation	Total Dust Concentration (mg/cu m)	Tungsten (mg/cu m)	Cobalt (mg/cu m)	Sample Type*	Location	Reference
Powder processing	14.9	7.7	0.95	G	Austria	55
(Stages from	10. - 14	-	1.05 - 1.67	B	United States	56
Ammonium paratungstate	2.0 - 3.3	-	0.12 - 0.22	G	"	56
to tungsten carbide are	0.3 - 9.8	1.8 - 8.24	0 - 0.55	B	Switzerland	57
shown on Figure XII-1)	1.1 - 32.0	0.88 - 25.6	0.09 - 2.72	"	Sweden	58
	3.1 - 130.3	2.2 - 3.5	1.8 - 2.0	G	USSR	59
	-	-	0.01 - 0.8	B	United States	60
	-	-	0 - 0.14	G	"	60
105 Tool and die operations	1.0 - 5.0	-	0.05 - 0.23	B	"	56
	0.5 - 2.2	-	0.01 - 0.07	G	"	56
Casting	0.22 - 7.5	0.52 - 4.56	0 - 0.41	B	Switzerland	57
	15	12	0.21	"	Sweden	58
	0.7 - 3.0	0.6 - 2.6	0.04 - 0.19	G	"	58
	5.5 - 47.7	1.4 - 2.1	6.8	"	USSR	59
	21.5	17.6	1.14	"	Austria	55
	-	-	0 - 0.08	B	United States	60
Forming	0.5 - 24.6	0.97 - 26.7	0.03 - 1.75	"	Switzerland	57
	0.1 - 7.5	0.08 - 5.9	0.008 - 0.56	"	Sweden	58
	0.2 - 0.7	0.17 - 0.58	0.01 - 0.04	G	"	58
		3.3 - 32.5	- -	"	USSR	59
	11.1	8.8	1.12	B	Austria	55
	-	-	0.001 - 0.8	"	United States	60
	-	-	0.01 - 0.15	G	"	60
Grinding of sintered pieces						
Dry grinding without exhaust	6.6 - 44	-	0.04 - 2.44	B	Switzerland	57
Wet grinding without exhaust	-	0.2 - 12.8	0.04 - 0.93	"	United States	29

\* B = breathing zone; G = general air

fail. The principles set forth in Industrial Ventilation--A Manual of Recommended Practice [61] published by the American Conference of Governmental Industrial Hygienists, Fundamentals Governing the Design and Operation of Local Exhaust Systems, ANSI Z9.2-1971 [62], published by the American National Standards Institute, and NIOSH's Recommended Industrial Ventilation Guidelines [63] should be applied to control workplace atmospheric concentrations of tungsten. These principles and guidelines have been used to control the concentrations of aerosols in the cemented tungsten carbide industry [29,57,60]. Because large accumulations of fine dust in dust collectors may pose a fire hazard, such systems should be located away from the other structures and areas where employees may be exposed. Internal sprinkler systems with fusible-link activation should also be considered.

From the data collected during a survey of five Swiss cemented tungsten carbide plants, Reber and Burckhardt [57] found that wet-grinding of sintered pieces produced cobalt concentrations far below the recommended limit of 0.5 mg/cu m. Although no exhaust was required for automatic machinery, grinding machines with manual adjustment reportedly required an efficient localized exhaust system. The authors concluded that individual exhaust pipes, with suction systems similar to vacuum cleaners which are attached directly to the lathe or grinding machine, are best for dust removal. They also recommended daily clean-up of the system to ensure its continuing efficiency. Reber and Burckhardt noted that the use of an exhaust ventilation system in an operation liberating total dust concentrations of 6 mg/cu m (of which 10% was cobalt) reduced the dust levels to 0.3-0.6 mg/cu m. They also mentioned that too small exhaust pipe

inlets, improper adjustment of exhaust pipe inlets, cut grinding, and machining of large pieces contributed to the buildup of high dust concentrations. Although Salikhodzhaerv and Vengerskaya [59] gave no details of the ventilation system, they stated that, in the work places equipped with a local dust extraction system, the dust concentration was reduced from 153 mg/cu m to 28 mg/cu m and the tungsten level decreased from 3 to 0.3 mg/cu m. They recommended mechanization of the processes, hermetic sealing of the sifting devices, and use of local dust extraction systems as measures to control dust concentrations.

Lichtenstein et al [29] successfully used a local exhaust ventilation system to control worker exposures to tungsten and cobalt during wet grinding. Well-fitted hoods, equipped with sliding doors controlling the designed air quantity of 400 cu ft/min, were installed around tool grinding wheels. Each hood was connected to a bench plenum by short flexible ducting with a smooth-walled interior, and each outlet was provided with an individual blast gate. All branch plena were connected to a main plenum. The measured air transport velocities of both the main and branch plena exceeded 4,500 ft/min. To allow for the draining of the coolant used in grinding and to reclaim the diamond dust from grinding wheel breakdown, swing check valves were provided in the plena. The plena also had provisions for cleanout. The authors recommended that regular maintenance and cleaning schedules be adopted for the exhaust system to prevent its degradation.

McDermott [60] recommended an efficient low-volume-high-velocity ventilation system (Figure XII-2) to reduce worker exposures to total dust and cobalt in the cemented tungsten carbide industry. In this system, the

exhaust fittings, because of their small duct size, are mounted near the dust source. According to McDermott, the system has significant advantages: an improved recovery rate for valuable material which would reduce the additional cost of such equipment; and, in climates where compensating makeup air must be tempered, a smaller volume of makeup air units to control specific operations, thus resulting in greater conservation of energy. The latter advantage may take on added importance, since many jurisdictions do not permit recirculation of toxic dusts.

Ventilation systems of this type will require regular inspection and maintenance to ensure effective operation. A program of scheduled inspection should be established in which the ventilation systems are checked routinely. These routine checks should include face velocity measurements of the collecting hood, inspection of the air mover and collector, and measurements of workroom airborne concentrations. Any changes which may affect the ventilation system or the operations being ventilated must be assessed promptly to ensure that engineering controls continue to provide adequate protection of employees.

### Sampling and Analysis

In general, two types of sampling methods, electrostatic precipitation and filtration, have been reported for collecting dusts in industrial atmospheres that contained tungsten and its compounds. Electrostatic precipitators have been used to collect general room dust samples and samples taken near the breathing areas of employees [16,56,60]. Total air volumes of 45-180 cu ft were sampled within 15-60 minutes (85 liters/min) [60]. Although some tasks would allow positioning of the

sampler in the general breathing area of an employee, electrostatic precipitators are generally used for area monitoring.

Filter sampling, using glass fiber [64], paper [54,58], and membrane filters [10,29], has been applied to the collection of area air samples and to personal monitoring. In one study [54], an average of 3 cu m of air/hr (50 liters/min) was drawn through 9-cm filter papers, except in areas where dust concentrations were low, where flowrates up to 6 cu m of air/hr (100 liters/min) were used. In another study, Lundgren and Ohman [58] collected general air samples and samples close to the breathing areas of employees for several hours through filter paper at a flowrate of 25 liters/min. The specific sampling time and collection efficiency data were not provided. However, these high flowrates are not suitable for personal monitoring.

Measurements of general airborne dust concentrations only indicate possible employee exposure. The concentrations of tungsten dust close to a machine or process may be quite different from that in the breathing zone of an employee. Therefore, collection of breathing zone samples is essential if one is to determine employee exposure. A breathing zone sampling device should be small enough to be conveniently attached to an employee's clothing without interfering with that person's normal activities and should ideally contain no fragile glassware or liquids. Membrane filter collection of dust using a battery-operated personal sampling pump satisfies these conditions.

Personal sampling has been performed using membrane filters at flowrates of 2-2.5 liters/minute for 2-6 hours [11]. Lichtenstein et al [29] reported using Millipore Type AA membrane filters at a flowrate of 4.3 liters/minute to sample the general room air and at a flowrate of 2.5

liters/minute for 2-4 hours to obtain breathing zone samples.

Airborne tungsten present in work areas is predominantly in the form of highly dispersed aerosols of negligible volatility. For the collection of these aerosols, personal monitoring by the membrane filter method (described in NIOSH Method No. P & CAM 173 [65]) is recommended. This method for the collection of aerosols containing tungsten and its compounds consists of drawing a known volume of air through a membrane filter as described in Appendix I. Although this sampling method has not been evaluated by NIOSH for this specific application, it is recommended for metals in general.

The analytical chemistry of tungsten has been reviewed, including detailed discussions on the precipitation, gravimetric, colorimetric, polarographic, amperometric, emission spectroscopic, atomic absorption, X-ray fluorescence, and neutron activation methods [66,67]. Of the methods available for determining tungsten, colorimetric [68-70], emission spectrographic [71], atomic absorption [10,29], neutron activation [72-77], and X-ray fluorescence [78] analyses have been used to determine its concentrations in air. Most of these methods were applied to multi-elemental analysis with no special modification for tungsten determination.

The colorimetric method is based on the principle that, in hydrochloric acid or in a sulfuric/hydrochloric acid solution, tungsten(VI) forms a yellow complex with thiocyanate in the presence of a reducing agent such as stannous or titanous chloride. The complex contains tungsten(V) and has a maximum absorption at about 400 nm [66]. Salyamon and Krashenitsyna [68] studied the usefulness of two thiocyanate methods, using either titanium trichloride or stannous chloride as a reducing agent, to

analyze tungsten and its compounds in air. In the method which used titanium trichloride, the color was stable for 4 hours with a minimum detection limit of 0.5  $\mu\text{g}$ . The color developed by stannous chloride reduction was stable for only 1.5 hours with a minimum detection limit of 0.1  $\mu\text{g}$ .

Urusova [70] reported that when stannous chloride was used, although most of the tungsten was converted to the colored complex after 1 hour, the intensity of the color continued to increase even after 2 hours. However, when titanium(III) was used as the reducing agent, the color developed more rapidly and stabilized sooner. Urusova stated that the sensitivity of the method was 5  $\mu\text{g}$  of tungsten in the colorimetric volume. Peregud and Gernet [69] reported the sensitivity of the method to be 0.1 mg/cu m, using stannous chloride as the reducing agent, and they found no interferences in the method from titanium, cobalt, iron, vanadium, molybdenum, manganese, chromium, and thorium. Fogg et al [79] concluded from a review of literature that vanadium, iron, and molybdenum interfered with the method. Sample preparation for the method generally involved ashing the filter containing the sample in a muffle furnace at 500-600 C until tungstic oxide was formed [68,69]. Urusova [70] believed that, in samples containing mixtures of cobalt, titanium, and tungsten, protective coatings might be formed when they were ashed. Hence, the author recommended that, for a complete decomposition of such samples, concentrated sulfuric acid and ammonium sulfate be used.

Seeley and Skogerboe [71] described the use of an emission spectrographic method to determine atmospheric concentrations of several elements, including tungsten. Porous cup graphite spectrographic



electrodes were used as filters for atmospheric particles, collected at 1 liter/minute for 30 minutes, to determine concentrations of less than 0.1  $\mu\text{g}/\text{cu m}$ . The samples were analyzed directly by a spectrograph (with an absolute detection limit of 5 ng) at 400.8 nm for tungsten and with a precision and an accuracy of 10-20% of the amount present. The authors found that, for the spectrographic determination, vaporization of refractory elements such as tungsten was not completely effective because the bottom of the electrode did not reach a sufficiently high temperature. This problem was corrected by forming metal chlorides with the vapor of hydrochloric acid in the sample chamber.

A nondestructive neutron activation analytical procedure for the determination of 33 elements, including tungsten, in air polluting particles was reported by Dams et al [73] and Linch et al [76]. Aerosols were sampled by using a polystyrene filter with a flowrate of 12 liters/minute/sq cm. The samples were irradiated for 5 minutes at a flux of  $2 \times 10^{12}$  neutrons/sq cm/second (\* means "to the power of") and counted in a Ge(Li) detector. Tungsten in the irradiated samples was measured at photopeaks of 479.3 and 685.7 Kev as  $^{187}\text{W}$  having a half-life of 24 hours. The detection limit for tungsten was 0.005  $\mu\text{g}$ , and the minimum concentration detected in a 24-hour urban air sample was 0.0005  $\mu\text{g}/\text{cu m}$ . These reports [73,76] indicated that the standard deviations of the analysis were greater than 40% when the elements were in concentrations near the limit of detection. However, the authors did not report specific deviations for tungsten analysis.

Peirson and associates [77] similarly analyzed atmospheric trace elements by neutron activation analysis and found that the concentrations

of tungsten ranged from less than 0.1 to 0.3 ng/kg of air (0.12-0.36 ng/cu m). There were no data on the sensitivity or precision of the method in determining tungsten. Pszeny and Russell [75] also determined environmental levels of various elements using neutron activation analysis. However, they ashed the filter paper containing the sample in a furnace at 450 C before irradiating for 8 hours in a flux of  $2 \times 10^{13}$  neutrons/sq cm/sec. The elements, including tungsten, were assayed by a high-resolution gamma-ray spectroscope using a Ge(Li) detector. The concentrations of airborne tungsten were between 19 and 25 ng/cu m with a standard deviation of greater than 50%. Because of the high resolution of the method, Mamuro and his colleagues [72] recognized the usefulness of neutron activation analysis in the determination of tungsten in airborne dust, yet they believed that it was not suited for routine evaluation of air contaminants.

Dzubay and Stevens [78] and Roberts [74] discussed the usefulness of X-ray fluorescence analysis for determining trace elements in aerosols. Although the authors stated that it was one of the most sensitive available methods, its application for determining tungsten was not discussed in either report. The method requires a homogeneous sample for better reproducibility and hence a special sampling device that separates the dust particles by size [11,78].

In general, neutron activation, emission spectrographic and X-ray fluorescence analyses are sensitive methods. However, no reports were found in which any of these methods were examined for their ability to determine tungsten in samples of air from industrial environments. In addition, the methods were used primarily for general analysis of trace

elements, so any interferences in determining tungsten alone, accuracy for tungsten, or details of specific sample preparation for tungsten were not discussed. Such information is required to assess the suitability of these methods for determining environmental concentrations of tungsten in industrial atmospheres.

Although a colorimetric method using thiocyanate was successfully used by Russian investigators [68-70] to determine tungsten in air, there are potential interferences in the method if the sample contains vanadium, iron, or molybdenum. Other variables, such as the type of reducing agent used in developing the colored complex and sample preparation, have substantial influence on the tungsten values and have not been adequately investigated. Thus, although the colorimetric method may be useful for determining tungsten at the recommended TWA concentration, interferences and lack of standardized procedures would make this method unacceptable unless improvements were made and procedures were standardized.

Of all the analytical methods discussed in the recent literature, atomic absorption stands out as the most acceptable, particularly because it is simple, rapid, sensitive, selective, and suited for use with generally available laboratory equipment and personnel. Lichtenstein et al [29] successfully analyzed hard-metal workplace breathing zone air samples by atomic absorption. The membrane filter containing tungsten carbide and cobalt was divided into two portions which were separately analyzed for tungsten and cobalt. For analyzing tungsten, the filter and sample were digested in aqua regia and brought up to 10-ml volume by adding deionized water. The solution was analyzed by atomic absorption spectrophotometry against known concentrations of each metal. The accuracy of the method was

noted to be  $\pm 0.1$  mg in the total sample. In the cemented tungsten carbide industry, the authors found that tungsten concentrations were 0.2-12.8 mg/cu m.

In two other plants producing cemented tungsten carbide, atomic absorption was used to determine levels of airborne tungsten [11]. The levels of tungsten found in these plants were 2.6-36.3 mg/cu m and 0.13-1.3 mg/cu m. The details of analysis were not available for one plant, but at the other the filter was dissolved in a suitable acid and tungsten was determined by using a reducing nitrous oxide-acetylene flame. Husler [80] recommended dissolution of the tungsten sample in a mixture of nitric and hydrofluoric acids as particularly advantageous since this reagent keeps tungstic oxide from precipitating out of solution. Edgar [81] showed that tungsten samples contaminated with molybdenum, chromium, manganese, vanadium, and nickel gave 50-60% higher absorbance readings and that the addition of 2% of sodium sulfate eliminated such interference and enhanced the sensitivity of tungsten absorbance. In most of these studies, the fuel was acetylene and the oxidant was nitrous oxide.

Since analysis by atomic absorption is generally available to industry, possesses the required precision and sensitivity, and has been successfully used by several companies in the cemented tungsten carbide industry to determine concentrations of airborne tungsten, the method outlined in Appendix II is recommended for use in determining adherence to the recommended standard. The recommended procedure is based on Method No. P & CAM 173 of the NIOSH Manual of Analytical Methods [65] with suggested modifications derived from other appropriate literature [80-84]. The recommended procedure is a synthesis of the best available methods, but it

has not been validated by NIOSH. The disadvantages of this method include its inability to determine tungstic oxide, an acid-insoluble compound, and to distinguish insoluble compounds from water soluble ones. Methods shown to be at least equivalent in precision and sensitivity to the recommended method may also be used.

## V. WORK PRACTICES

Most exposures to tungsten compounds in the occupational environment occur during the production of tungsten metal from the ore and in the preparation of tungsten carbide powders. Exposures to cemented tungsten carbide occur in the manufacturing and grinding of cemented tungsten carbide (hard-metal) parts [9]. Dusts and mists of tungsten and its compounds or cemented tungsten carbide are produced during crushing, mixing, ball milling, loading and unloading, sintering, and grinding operations. Because of the high melting points of tungsten compounds and of cobalt, exposure to their vapors or fumes is negligible. Exposures to sodium tungstate, a soluble compound, are limited and occur primarily during the first few stages of processing the ore (see Figure XII-1). Although NIOSH is aware that exposures also occur during filament manufacture and that processes such as high-voltage smelting and wire-drawing pose specific safety problems, it is not possible to elaborate on control measures for these operations because of lack of access to this portion of the industry.

To control the exposure of employees to tungsten compounds and cemented tungsten carbide in these industries, engineering controls and work practices should be designed and applied to minimize or prevent inhalation or ingestion of, or skin contact with, the aerosols.

Properly designed and maintained ventilation systems will minimize dispersal of tungsten compounds and cemented tungsten carbide in the work atmosphere, not only controlling exposure to the airborne compounds but also reducing their accumulation on surfaces. These systems will minimize

maintenance problems and the chance of redispersal during cleanup. Ventilation practices and principles to control workplace air concentrations of tungsten should be based on those set forth in the ACGIH manual Industrial Ventilation: A Manual of Recommended Practice [61], Fundamentals Governing the Design and Operation of Local Exhaust Systems (ANSI Z9.2-1971) [62], and NIOSH's Recommended Industrial Ventilation Guidelines [63]. Exhaust air should be adequately cleaned before it is discharged outside the plant to ensure that it meets applicable federal, state, and local air standards. The airflow, static pressure, and leakage of the ventilation system should be monitored frequently by trained personnel to ensure proper functioning of the system. A logbook showing the airflow design and the results of periodic ventilation monitoring measurements should be maintained. In areas where crushing, mixing, ball milling, loading and unloading, sintering, and grinding operations are performed, local exhaust ventilation should be located as close to the operation as feasible and in accordance with good industrial hygiene engineering practices [61-63]. Alternate means of control for certain processes may include the reduction in density of production equipment and a decrease in equipment speed. Mechanization and enclosure of processes offer additional engineering controls which may be effectively used under some circumstances [11].

In addition to using sound engineering controls, employers should institute a program of work practices which emphasizes good sanitation and personal hygiene. These practices are important in preventing skin and respiratory irritation caused by tungsten compounds or cemented tungsten carbide. Skin irritation has not been found to be a problem with insoluble

tungsten compounds in the occupational environment, but the dermatologic problems observed in cemented tungsten carbide workers were attributed in part to the abrasiveness of the powder. In experimental animals, tungsten hexachloride, a soluble tungsten compound, caused necrosis of the skin and membranes [41]. Workers handling soluble tungsten compounds or cemented tungsten carbide must thoroughly wash their hands and face before drinking, eating, or smoking. Employees must not store and use food, beverages, tobacco, or other materials that may be placed in the mouth, in these exposure areas. Rest areas, eating facilities, and smoking areas should be physically separated from these work areas. In addition, workers should be advised to shower or bathe after the workday. If skin irritation is observed, the employee should be referred to a physician for appropriate protective measures.

Respirators should not be used as a substitute for proper engineering controls in normal operations. However, during emergencies and during nonroutine repair and maintenance activities, exposures to airborne dusts or mists of tungsten compounds or cemented tungsten carbide might not be reduced either by engineering controls or by administrative measures to the levels specified in Chapter I, Section 1(a). If this occurs, then respiratory protection may be used only: during the time necessary to install or test the required engineering controls; for operations such as maintenance and repair activities causing brief exposure at concentrations above the TWA concentration limits; and during emergencies when airborne concentrations may exceed the TWA concentration limits. Respirators conforming to the Respirator Selection Guides in Tables I-1, I-2, and I-3 should be provided to employees, and a respiratory protective program



meeting the requirements of 29 CFR 1910.134 should be established. The Respiratory Protective Devices Manual [85] should be consulted for further information on respirators. Emphasis should be placed on providing clean, well-maintained, well-fitted respirators for use in unusual circumstances and emergencies. Contamination of the interior of the facepiece should be guarded against.

Eye irritation has rarely been a problem for employees involved in handling tungsten compounds or cemented tungsten carbide [15], but operations, such as grinding, which produce and scatter fine particles into the air, require eye protection in accordance with 29 CFR 1910.133. Both individual sensitivity to cobalt and the abrasiveness of powdered metals may contribute to skin irritation [14,24]. Gloves or other protective clothing may be helpful, but employees experiencing skin irritation should be referred to a physician to ensure effectiveness of the protective measures.

Spills of tungsten compounds or cemented tungsten carbide should be promptly cleaned up to minimize inhalation or contact. Liquid material spills should be copiously flushed with water and channeled to a treatment system or a holding tank for reclamation or proper disposal. Spills of dry material should be removed by vacuuming, wet mopping, or hosing, first with a mist of water that dampens the spilled material and then with a more forceful stream that flushes it into a holding tank or other facility for handling contaminated water. No dry sweeping or blowing should be permitted. All waste material generated in the handling of tungsten compounds or cemented tungsten carbide should be disposed of in compliance with local, state, and federal regulations.

Most tungsten compounds pose no fire or explosive hazards. However, powders of submicron-size tungsten metal or tungsten-aluminum-titanium alloys, tungsten metal powders of less than 100 mesh, and ferrotungsten powders of less than 200 mesh are classified as actively combustible [86] and should be handled with caution. Lamprey and Ripley [87] reported that tungsten powder of submicron size which came in contact with hot chlorine gas in a large glass receiver reacted violently and shattered the equipment. These materials should be stored in tight drums under argon or helium gases. The drums should never be vented in air. Great care must also be exercised in handling hexamethyl tungsten, since it is potentially explosive [88,89]. Similarly, tungsten carbonyls were reported to be moderate fire hazards when exposed to flame [90].

Warning labels indicating the potential fire or explosion hazards, skin and respiratory effects, and ventilation requirements should be placed on containers used to transport tungsten compounds or cemented tungsten carbide. In all industries where soluble tungsten compounds or cemented tungsten carbide are handled, written instructions informing employees of the particular hazards of these materials, the methods of handling, procedures for cleaning up spilled material, and the use of personal protective equipment must be on file and available to employees. Employers may use the Material Safety Data Sheet in Appendix III as a guide in providing the necessary information.

## VI. DEVELOPMENT OF STANDARD

### Basis for Previous Standards

In 1967, the Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists proposed a threshold limit value (TLV) of 5 mg of tungsten/cu m for tungsten and its insoluble compounds and a TLV of 1 mg of tungsten/cu m for soluble tungsten compounds [91]. These proposed limits were adopted by the ACGIH in 1969 [92]. The 1971 Documentation of the Threshold Limit Values for Substances in Workroom Air [93] cites a number of reports which were used to support the need for separate limits for soluble and insoluble tungsten compounds. Karantassis [45] found that the toxic single doses of sodium tungstate, administered intragastrically and subcutaneously to guinea pigs, were 0.55 and 0.45 g of tungsten/kg, respectively. These guinea pigs suffered from anorexia, colic, incoordination of movement, trembling, dyspnea, and weight loss before a delayed death. Kinard and Van de Erve [47] reported the LD50 of subcutaneously injected sodium tungstate for rats to be 0.14-0.16 g/kg and ascribed death to generalized cellular asphyxiation. Kinard and Van de Erve [48] suggested that, when administered orally to rats, sodium tungstate was more toxic than the insoluble compounds, tungsten trioxide and ammonium-p-tungstate.

Emphasizing the relatively low toxicity of the insoluble compounds, the documentation [93] also cited the studies of the following investigators to support separate standards for soluble and insoluble tungsten compounds: Kinard and Van de Erve [44] stated that female weanling rats fed a diet containing 2-10% of tungsten metal powder gained

15.4% less weight than did either female controls or male weanling rats on a tungsten-free diet. Frederick and Bradley [94] found that the LD50 of tungsten metal powder injected intraperitoneally in rats was 5 g/kg and that it did not produce a distinct fibrosis. Delahant [35] administered 150 mg of tungsten metal powder or tungsten carbide intratracheally to guinea pigs in three weekly doses and concluded that it did not irritate lung tissue. Schepers [36] examined the lungs of these guinea pigs microscopically and found that tungsten metal caused moderate interstitial cellular proliferation. He concluded that tungsten metal dust was relatively inert and that exposures of human beings to it would be relatively safe, though not wholly free from risk. Harding [95] reported no "unexpected" effects of intratracheal injections of tungsten metal and tungsten carbide powders on guinea pig lungs. Mezentseva [28] showed that, while rats given tungsten intratracheally had a proliferation of the intra-alveolar septa, those exposed by inhalation to dusts of tungsten, tungsten dioxide, and tungsten carbide had only minor changes in the lungs.

In 1976, the ACGIH also recommended short term exposure limits (STEL's) of 10 mg and 3 mg of tungsten/cu m of air for insoluble and soluble compounds, respectively [96]. The STEL was defined by the ACGIH as a maximum allowable concentration, or absolute ceiling, not to be exceeded at any time during a 15-minute excursion period.

The 1975 USSR maximum workplace concentrations were 6 mg/cu m for tungsten, tungsten carbide, and tungsten silicide, and 2 mg/cu m for tungsten carbonyl [97]. In 1970, the maximum allowable concentration (MAC) in Rumania for tungsten and tungsten carbide was 6 mg/cu m [98]. The 1973 standard for tungsten, tungsten carbide, and tungsten oxide in the German

Democratic Republic was an MAC of 6 mg/cu m with a 30-minute excursion limit of 12 mg/cu m [99]. No bases for these foreign standards have been found. Brakhnova [97] recommended standards for tungsten sulfide (6 mg/cu m), tungsten selenide (2 mg/cu m), and tungsten telluride (0.01 mg/cu m), but they seem not to have been approved yet by the USSR State Sanitary Inspection Office.

There is currently no US Federal Standard for tungsten or its compounds.

#### Basis for the Recommended Standard

##### (a) Permissible Exposure Limits

Almost all reports of human exposure to tungsten and its compounds deal with the effects observed after exposure to mixtures of dusts encountered in the hard-metal (cemented tungsten carbide) industry. The components of hard metal include tungsten carbide, cobalt, and sometimes tantalum, titanium, niobium, chromium, nickel, iron, or derivatives of these metals. Only two studies [27,28] were found which described the effects of occupational exposure to tungsten and its compounds without concurrent exposure to other toxic metals.

In 1960, Mezentseva [28] reported that radiologic examination of 54 workers engaged in the production of malleable tungsten or in the early process stages of hard-metal production, where dust exposure was limited to tungsten trioxide, metallic tungsten, and tungsten carbide, showed early signs of diffuse pulmonary fibrosis in 9%. Total dust levels measured ranged from 1.3 mg/cu m in the breathing zone of workers loading furnaces for malleable tungsten production to 83 mg/cu m during loading and

unloading of mills in the production of hard metal. The diameters of 72-82% of the particles were less than 4  $\mu\text{m}$ .

Kaplun and Mezentseva [27] compared results of periodic medical examinations of 36 workers in contact only with tungsten and its compounds with those of 247 workers exposed to dusts of mixed compounds (cobalt and tungsten carbide) in hard-metal production. Radiologic signs of early diffuse pulmonary fibrosis were found in 11% of the first group, while almost 50% of the second group showed damage to the upper respiratory tract in addition to a 13% incidence of pulmonary fibrosis. Subjective symptoms, such as loss of appetite, nausea, coughing, and olfactory disorders, were also reported for the second group, along with abnormalities, such as hypotension and blood changes, which were not limited to the respiratory system. Total concentrations of dust measured in areas where only tungsten compounds were released were 8.6-106 mg/cu m. The total concentrations of the mixed dusts in other parts of the plants were 3-186 mg/cu m, with cobalt content contributing 0.3-1.75 mg/cu m.

Studies with animals disclosed the effects of single and multiple exposures to tungsten and its compounds by inhalation, intratracheal administration, and ingestion. The major effects of inhalation and intratracheal administration were limited to the respiratory system, while the effects of ingestion were not so clearly apparent on any organ of the body. Although no report was found on the dermal effects of tungsten and its compounds in humans, exposure of rats to aerosols of tungsten hexachloride reportedly [41] caused irritation of the skin and mucosa.

In the inhalation study conducted by Mezentseva [28], rats were exposed to tungsten carbide at a concentration of 600 mg/cu m (77% of the

particles were less than 5  $\mu\text{m}$  in diameter) for 1 hour/day for 5 months. Microscopic examination of the lungs showed proliferative reactions of the lymphoid histiocytic elements, particularly at sites of dust accumulation, and subsequent mild fibrosis. Thickening and homogeneity of the pulmonary walls were also noted. Mezentseva also reported that rats intratracheally exposed to a single 50-mg dose of metallic tungsten, tungsten carbide, or tungsten trioxide similarly showed no severe pulmonary changes upon microscopic examination. Totals of 150 mg of metallic tungsten dust or tungsten carbide dust introduced intratracheally in three equal doses at weekly intervals by Delahant [35] did not irritate the lungs of experimental animals. Miller et al [16] observed that 1 ml of a 10% suspension of tungsten carbide administered intratracheally to rats produced no changes in the lungs other than those typical of an inert dust after 18 weeks. Schepers [34] stated that intratracheal administration of tungsten carbide and carbon (in a 94:6 ratio), in three equal weekly doses totaling 150 mg, caused acute hyperemia, bronchial inflammation, and minor residual changes in the lungs, such as the development of subpleural fibrocellular granulomata. These reports suggest that tungsten and its compounds, when encountered without concomitant cobalt exposure, have a distinct but less severe toxicity than tungsten-cobalt mixtures.

The effects of exposure to mixed tungsten-cobalt dusts, reported almost exclusively from the cemented tungsten carbide industry, also relate to the respiratory system, with some effects seen on the skin. A number of reports of such studies identified pulmonary involvement as the outstanding result of the exposures [15,18,25,100]. The signs and symptoms included weight loss, exertional dyspnea, and cough which might progress to

extrinsic asthma [23], diffuse interstitial pneumonitis [18], or fibrosis [17,20,22]. The type of pneumoconiosis seen in the cemented tungsten carbide industry is referred to as "hard-metal disease" [21,25,101].

Skog [24] reported skin changes, including contact eczema, pruritus, folliculitis, and neurodermatitis, that affected 34 (9.4%) of 361 workers involved in production of hard metal. Contact eczema was observed mainly on the eyelids and between the fingers. Of the workers with contact eczema, 3 of 14 patch tested were allergic to cobalt. Although cobalt sensitization was apparent, Skog concluded that most of the cases of contact eczema were caused by the primary irritant effect of the combined metal dust.

Similar dermatologic effects have not been reported from experiments on animals, but the effects of tungsten-cobalt mixtures on the respiratory system have been well documented. In animals injected intratracheally with suspensions of tungsten carbide and cobalt (in a 10:1 ratio), Schepers [37] noted a transient inflammatory response with residual fibrosis. The inhalation of tungsten carbide and cobalt in a 3:1 ratio produced acute inflammation, followed by focal pneumonitis with residual hyperplasia and metaplasia of the bronchial epithelium. The author emphasized that the unusual epithelial reaction was a unique feature of exposure to the tungsten carbide-cobalt mixture. Both Delahant [35] and Schepers [37] found that the intense irritant property of cobalt was dominant when cobalt was combined with tungsten carbide.

Since the use of tungsten in soluble form constitutes only 3% of the total usage of tungsten and its compounds [8], occupational exposures to soluble tungsten compounds are limited except in the early stages of



production of tungsten from its ores. Occupational exposure effects from the soluble compounds of tungsten are virtually unknown. However, comparisons have been made of the toxicity of soluble and insoluble tungsten compounds when administered to experimental animals. Karantassis [45] found that the lethal single doses of sodium tungstate, administered intragastrically and subcutaneously to guinea pigs, were 0.55 and 0.45 g of tungsten/kg of body weight, respectively. Guinea pigs given the toxic dose had anorexia, colic, incoordination of movement, trembling, dyspnea, and loss of weight before a delayed death. Death was attributed to generalized cellular asphyxiation. Kinard and Van de Erve [47] found that the LD50 of subcutaneously injected sodium tungstate in rats was 0.14-0.16 g/kg. Kinard and Van de Erve [48] concluded that, when administered orally to rats, tungsten trioxide and ammonium-p-tungstate were less toxic than soluble sodium tungstate. Comparing the LD50 values of orally administered sodium tungstate, sodium phosphotungstate, and tungstic oxide (W03), Nadeenko [50] concluded that tungstic oxide was the least toxic of the three compounds. He suggested that the lower toxicity of W03 was caused by its lower solubility.

Insoluble tungsten, soluble tungsten, tungsten carbide-cobalt mixtures, and the much less common tungsten carbide-nickel mixtures constitute four different potential exposures with different types and degrees of hazard for employees. NIOSH, therefore, recommends separate environmental limits on the following bases:

- (1) Dusts of insoluble tungsten compounds pose a hazard considered to be somewhat greater than that of nuisance dust. Generally accepted characteristics of lung-tissue reaction to nuisance aerosols are:

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. The 9-11% incidence of pulmonary fibrosis in two studies [27,28] in which employees were exposed to tungsten without concomitant cobalt exposure indicates that the respirable fraction of insoluble tungsten compounds should be limited to below the respirable nuisance dust standard of 5 mg/cu m. Dust of insoluble tungsten compounds has been found to contain a high percentage of respirable particles (approximately 72-90% less than 10 $\mu$ m) [28,42,54]. In addition, collection of total dust is less difficult than size selective sampling. Therefore, NIOSH recommends that dust containing insoluble tungsten be limited to 5 mg/cu m measured as tungsten, which allows both a margin of safety and ease of sampling.

(2) Soluble tungsten compounds are considered to be potentially more toxic than insoluble compounds, although the magnitude of the difference is not precisely known. Comparison of acute toxicity of tungstic oxide and sodium tungstate in mice (oral LD50's of 840 mg/kg and 240 mg/kg, respectively) [50] gives a 3.5-fold difference. A calculated limit of 1.4 mg/cu m for soluble tungsten compounds is suggested when the ratio of the LD50's is applied to the recommended limit for insoluble compounds. In addition, the effects of insoluble tungsten compounds are confined to the respiratory system, while soluble sodium tungstate has been found to cause systemic effects involving the gastrointestinal tract and CNS in guinea pigs. [45]. NIOSH considers the broader range of toxic effects an indication of the need for a somewhat increased margin of safety beyond the 3.5-fold difference and therefore recommends a limit of 1

mg/cu m for soluble tungsten compounds.

(3) The majority of industrial exposures to tungsten and its compounds occur along with exposure to cobalt, used in cemented tungsten carbide in percentages ranging from 3-25%. Whenever employees are involved in the manufacture, use, storage, or handling of cemented tungsten carbide containing more than 2% cobalt, such exposures shall be limited to the current US federal standard for cobalt. If a future NIOSH recommendation for an occupational exposure limit for cobalt differs from the US federal standard for cobalt, this new recommendation should be considered to replace the current recommendation for an occupational exposure limit for dusts of cemented tungsten carbide containing more than 2% cobalt.

(4) Some cemented tungsten carbide is made with nickel or iron as the cementing substance. Iron dust constitutes no great hazard and can be controlled adequately by adherence to the occupational exposure limit for tungsten. Nickel presents a different sort of situation, however, because it seems to be able to induce pulmonary cancers. When nickel is used as a binder rather than cobalt and the nickel content of the mixture exceeds 0.3%, then the NIOSH recommended standard for nickel of 15  $\mu\text{g}/\text{cu m}$  should apply [1].

(b) Sampling and Analysis

Personal sampling with a membrane filter is recommended, since most of the compounds in the industrial environment exist as aerosols. Analysis by atomic absorption spectrophotometry has been successfully used in monitoring air for tungsten and cobalt at the necessary sensitivity ranges. The method is relatively simple, quick, and highly sensitive. It has few

interferences, and they can be corrected. The method has not been tested by NIOSH, and it does distinguish insoluble from soluble tungsten compounds. In the absence of differential sampling and analysis, application of the environmental limits for insoluble or soluble tungsten compounds depends on knowledge of the processes within a building or independent structure. Plant observations [11] indicate that only insoluble compounds exist in some operations, and these will be adequately controlled by adherence to the recommended standard for insoluble tungsten compounds. However, when soluble compounds are final, intermediate, or starting products and sampling and analysis can be performed only for total tungsten, concern for worker health and safety dictates that the recommended standard for soluble tungsten compounds should apply throughout that building.

(c) Medical Surveillance and Recordkeeping

Preplacement medical screening is recommended to identify any preexisting pulmonary conditions that might make a worker more susceptible to exposures in the work environment. Periodic medical examinations will aid in early detection of any occupationally related illnesses which might otherwise go undetected because of either delayed toxic effects or subtle changes. Maintenance of medical records for a period of 30 years is recommended.

(d) Personal Protective Equipment and Clothing

Since much of the dust and mist generated in the tungsten industries falls within the range of particle sizes generally considered to include the respirable fraction, respiratory protective equipment may be used when necessary under the following conditions:

(1) During the time necessary to install or test the required engineering controls.

(2) For operations, such as maintenance and repair activities, that cause brief exposure at concentrations above the TWA concentration limits.

(3) During emergencies when airborne concentrations may exceed the TWA concentration limits.

Eye protection is required for operations which produce and scatter fine particles into the air, such as grinding, in accordance with 29 CFR 1910.133. Skin irritation may occur, especially in individuals sensitive to cobalt or to the abrasive action of hard-metal dust, even at environmental concentrations below the action level. Appropriate protective and therapeutic measures should be recommended by a physician. These may include fingerless gloves, protective sleeves, or creams.

(e) Informing Employees of Hazards

Employee awareness is important in an overall effort to reduce occupational injuries and illness. Therefore, employees should be informed through discussion and the Material Safety Data Sheet (see Appendix III) of the possible effects of exposure to tungsten compounds and to dust from cemented tungsten carbide and of the measures being taken to protect the workers against such exposures. In addition, labels and posters should be readily visible to employees.

(f) Work Practices

Exposures to tungsten and its compounds in occupational environments can best be prevented by engineering controls and good work practices. Since tungsten compounds and dusts from cemented tungsten carbide affect

chiefly the respiratory system, measures are recommended that will reduce the atmospheric concentrations of tungsten in the work atmosphere. Adoption of these measures during normal operations will also minimize the possibility of skin contact or accidental ingestion.

(g) Monitoring and Recordkeeping Requirements

It is recognized that many workers are exposed to tungsten compounds or cemented tungsten carbide at concentrations considerably below the recommended TWA limits. Under these conditions, it should not be necessary to comply with many of the provisions of this recommended standard. However, concern for worker health requires that protective measures be instituted below the enforceable limits to ensure that exposures do not exceed the standards. For this reason, "Occupational exposure to tungsten and cemented tungsten carbide" has been defined as exposure above half the recommended TWAs, thereby delineating those work situations which do not require the expenditure of health resources for compliance with such provisions as performance of frequent environmental monitoring and associated recordkeeping. For cemented tungsten carbide products in which nickel is the binder, the NIOSH definition of occupational exposure to nickel shall apply [1].

To relate the employee's known occupational exposure to possible chronic sequelae which do not appear during the period of employment, records of environmental monitoring should be kept for the same 30-year period as the medical records.

## VII. RESEARCH NEEDS

Proper assessment of the toxicity of tungsten and evaluation of its potential hazard to the working population require further animal and human study. The following aspects of epidemiologic and toxicologic research are especially important.

### (a) Epidemiologic Studies

Further research is desirable to assess the effects of long-term occupational exposure to tungsten. Therefore, detailed long-term epidemiologic studies, retrospective and prospective, of worker populations exposed at or below the recommended environmental limits should be conducted. Such studies should consider the pulmonary, dermal, ocular, and metabolic effects of tungsten and should distinguish exposures to tungsten and its compounds from mixed exposures to tungsten and to compounds of tungsten and cobalt, nickel, vanadium, chromium, or other metals and compounds of these metals. As a minimum, these studies should include environmental air measurements, medical histories, pulmonary function studies, histories of known or suspected acute exposures to tungsten, and comparisons with morbidity and mortality information for the general population.

### (b) Acute and Chronic Animal Studies

No definitive acute or chronic inhalation studies have been found for many of the tungsten compounds currently in use. There is some indication of the skin and eye irritation potential of tungsten hexachloride and of tungstic acid, respectively [41,102]. Hence, dermal and eye irritation studies should be undertaken to aid in evaluating the toxic effects of

tungsten and its compounds with and without concurrent exposure to cobalt or other metals commonly used in the production of hard metal. Some studies are available which indicate the possible effects of tungsten and its compounds on the liver, kidneys, adrenals [38,46], and central nervous system [50]; however, these studies are not conclusive. Hence, additional studies are needed to assess the toxic effects of tungsten and its compounds, especially on the liver, kidneys, adrenals, lungs, and central nervous system of various species. Studies should use exposure schedules simulating occupational exposure and should involve routes of exposure which are likely to occur in occupational contact with tungsten (ie, inhalation, ingestion, and absorption through the skin). These results may then provide insight into probable effects of tungsten on human health. Similar studies should also be planned to determine the synergistic or potentiating effects of other metals and compounds commonly found where tungsten and/or its compounds is used.

In experimental animals, high doses of tungsten have been incorporated into such enzymes as xanthine oxidase and sulfite oxidase by competing with molybdenum and producing inactive proteins [49,103-106]. Since xanthine oxidase affects purine and pyrimidine metabolism, a study of its effect through alterations in nitrogen metabolism may reveal some new information about the toxicity of tungsten.

(c) Studies on Carcinogenicity, Mutagenicity, Teratogenicity, and Effects on Reproduction

At present the available literature [25,26] does not supply the information necessary for an evaluation of the carcinogenic, mutagenic, or teratogenic potential of tungsten and its products. No studies were found



which addressed effects of tungsten on reproduction. Hence, further research concerning these possible effects of tungsten should be conducted. These studies should include microbial tests and extensive long-term and multigeneration experiments to evaluate the synergistic and carcinogenic actions, if any, of tungsten carbide. Properly designed and performed experiments can furnish information on all three types of effects resulting from reactions with nucleic acids.

(d) Sampling and Analysis

Investigations of sampling and analytical techniques are encouraged, especially those concerned with developing an analytical method which distinguishes aerosols of soluble tungsten compounds from those which are insoluble in water. NIOSH is currently conducting an analytical methods development project which will distinguish tungsten carbide and tungsten trioxide from sodium tungstate as examples of acid soluble, base soluble, and water soluble tungsten compounds.

## VIII. REFERENCES

1. National Institute for Occupational Safety and Health: Criteria for a Recommended Standard...Occupational Exposure to Inorganic Nickel, HEW publication No. (NIOSH) 77-164. Rockville, Md, US Dept Health, Education, and Welfare, Public Health Service, Center for Disease Control, NIOSH 1977, 282 pp
2. Weast RC (ed): CRC Handbook of Chemistry and Physics, ed 53. Cleveland, The Chemical Rubber Co, 1972, pp B76,B79,B81,86,B90,B100,-B104,B107,B110,B118,B127,B135,B141,B143,B150-51,C715
3. Meyer RJ (ed): [Tungsten, in Gmelins Handbook of Inorganic Chemistry ed 8 rev.] Berlin, Verlag Chemie, 1935, System-Number 54, pp 126,138-41,148-9,152,165-6,179-80,209,216,224-25,228,231,245,248-49,255,258, 265,269,328,342,361 (Ger)
4. Linke WF: Solubilities--Inorganic and Metal-organic Compounds--A Compilation of Solubility Data from the Periodical Literature, ed 4 rev. Princeton, NJ, D Van Norstrand Co Inc, 1958, vol 1, pp 400,693,780-81,903
5. Linke WF: Solubilities--Inorganic and Metal-organic Compounds--A Compilation of Solubility Data from the Periodical Literature, ed 4. Washington, DC, American Chemical Society, 1965, vol 2, pp 340,361,441,784, 1178-79,1195,1337,1346,1381,1456,1462,1525,1631
6. Bokii GB, Anikin IN: [The determination of the solubility of scheelite (CaWO<sub>4</sub>) in water and in aqueous solutions of NaCl and LiCl by the radiochemical method.] Zh Neorg Khim 1:1926-1928, 1956 (Rus)
7. Zadra JB: Milling and Processing Tungsten. Springfield, Va, US Department of Commerce, National Technical Information Service, 1959, 120 pp (NTIS PB 242 218)
8. Tungsten in April 1976, in Mineral Industry Surveys. US Dept of the Interior, Bureau of Mines, Division of Ferrous Metals, 1976, 13 pp
9. Materials Survey--Tungsten. Springfield, Va, US Dept of Commerce, National Technical Information Service, 1956, 126 pp (NTIS PB 242 220)
10. Plant observation reports and evaluation. Menlo Park, Calif, SRI International, March 1977, 150 pp (submitted to NIOSH under contract No. CDC-99-74-31)
11. Tungsten, in Fairhall LT: Industrial Toxicology. Baltimore, Williams and Wilkins Co, 1957, pp 128-29

12. Kopelman B, Smith JS: Wolfram, in Kirk-Othmer Encyclopedia of Chemical Technology, ed 2 rev. New York, Interscience Publishers, 1971, vol 22, pp 344-46
13. Jobs H, Ballhausen C: [Powder metallurgy as a source of dust from the medical and technical standpoint.] *Vertrauenarzt* 5:142-48, 1940 (Ger)
14. Schwartz L, Peck SM, Blair KE, Markuson KE: Allergic dermatitis due to metallic cobalt. *J Allergy* 16:51-53, 1945
15. Fairhall LT, Castberg HT, Carrozzo NJ, Brinton HP: Industrial hygiene aspects of the cemented tungsten carbide industry. *Occup Med* 4:371, 1947
16. Miller CW, Davis MW, Goldman A, Wyatt JP: Pneumoconiosis in the tungsten carbide tool industry. *AMA Arch Ind Hyg Occup Med* 8:453-65, 1953
17. Dorsit G, Girard R, Rousset H, Brune J, Wiesendanger T, Tolot F, Bourret J, Galy P: [Pulmonary fibrosis in three individuals working in the same factory and exposed to cobalt and tungsten carbide dusts--Pulmonary problems in the hard metal industry.] *Sem Hop* 46:3363-76, 1970 (Fre)
18. Coates EO, Watson JHL: Diffuse interstitial lung disease in tungsten carbide workers. *Ann Intern Med* 75:709-16, 1971
19. Bech AO: Hard metal disease and tool room grinding. *J Soc Occup Med* 24:11-16, 1974
20. Baudouin J, Jobard P, Moline J, Labandier M, Roullier A, Homasson JP: [Diffuse interstitial pulmonary fibrosis--Responsibility of hard metals.] *Nouv Presse Med* 4:1353-55, 1975 (Fre)
21. Scherrer M: [Three cases of hard metal pneumoconiosis.] *J Suisse Med* 100:2251-55, 1970 (Ger)
22. Rochemaure J, Anola M, Trinquet G, Meyer A: [A case of pulmonary fibrosis--Possible role of exposure to tungsten dust.] *J Fr Med Chir Thorac* 26:305-12, 1972 (Fre)
23. Bruckner HC: Extrinsic asthma in a tungsten carbide worker. *J Occup Med* 9:518-19, 1967
24. Skog E: Skin affections caused by hard metal dust. *Ind Med Surg* 32:266-68, 1963
25. Bech AO, Kipling MD, Heather JC: Hard metal disease. *Br J Ind Med* 19:239-52, 1962

26. Collet A, Liot F, Gallouedec C, Roussel G, Martin J, Reuet C, Brouet G: [Electron microscopy study of various cellular aspects of a pulmonary fibroadenomatoses with diffusion disorders.--Discussion on the etiological role of cobalt and tungsten carbide] Rev Tuberc Pneumol 27:357-81, 1963 (Fre)
27. Kaplun ZS, Mezentseva NV: [Hygienic evaluation of aerosols formed in the manufacture of hard alloys.] Gig Sanit 24:16-22, 1959 (Rus)
28. Mezentseva NV: Tungsten, in Izrael'son ZI (ed): Toxicology of the Rare Metals. Springfield, Va, US Dept of Commerce, National Technical Information Service, 1967, pp 28-35 (NTIS AEC-tr-6710)
29. Lichtenstein ME, Bartl F, Pierce RT: Control of cobalt exposures during wet process tungsten carbide grinding. Am Ind Hyg Assoc J 36:879-85, 1975
30. Bartl MD, Lichtenstein CIH: Tungsten carbide pulmonary fibrosis--A case report. Am Ind Hyg Assoc J 37:668-70, 1976
31. Barborik M: [Hard metal disease in workers employed in production of tungsten carbide.] Prac Lek 18:241-47, 1966 (Cze)
32. Vengerskaya KY, Salikhodzhaev SS: [Some problems relating to the effects of tungsten powder on humans.] Gig Tr Prof Zabol 6:27-29, 1962 (Rus)
33. Heuer W: [Pulmonary fibroses among hard metal production workers.] Arch Gewerbepath Gewerbehyg 19:613-32, 1962 (Ger)
34. Schepers GWH: Biologic action of tungsten carbide and carbon--Experimental pulmonary histopathology. AMA Arch Ind Health 12:137-39, 1955
35. Delahant AB: An experimental study of the effects of rare metals on animal lungs. Arch Ind Health 12:116-20, 1955
36. Schepers GWH: The biological action of particulate tungsten metal. AMA Arch Ind Health 12:134-36, 1955
37. Schepers GWH: The biological action of tungsten carbide and cobalt--Studies on experimental pulmonary histopathology. AMA Arch Ind Health 12:140-46, 1955
38. Kaplun ZS, Mezentseva NV: Industrial dusts encountered in powder metallurgy (hard alloys), in Izrael'son ZI (ed): Toxicology of the Rare Metals. Spingfield, Va, US Dept of Commerce, National Technical Information Service, 1967, pp 155-63 (NTIS AEC-tr-6710)
39. Schepers GWH: Lung tumors of primates and rodents. Ind Med 40:48-53, 1971

40. Brakhnova IT, Samsonov GV: [Comparative assessment of the effect on the organism of silicides of the transitional metals.] Gig Sanit 35:42-45, 1970 (Rus)
41. Spiridonova VS, Suvorov SV: [On the mechanism of the acute toxic action of chlorides of rare metals.] Gig Sanit 32:79-82, 1967 (Rus)
42. Gol'dman EI, Mezentseva NV, Mogilerskaya OY: Industrial dust of luminophores, in Izrael'son ZI (ed): Toxicology of the Rare Metals. US Dept of Commerce, 1967, pp 170-81
43. Lauring L, Wergeland FL Jr: Ocular toxicity of newer industrial metals. Mil Med 135:1171-74, 1970
44. Kinard FW, Van de Erve J: Effect of tungsten metal diets in the rat. J Lab Clin Med 28:1541-43, 1943
45. Karantassis T: [On the toxicity of compounds of tungsten and molybdenum.] Ann Med Leg 5:44-50, 1924 (Fre)
46. Selle SR: Effects of subcutaneous injections of sodium tungstate on the rat. Fed Proc Fed Am Soc Exp Biol 1:165, 1942
47. Kinard FW, Van de Erve J: Rat mortality following sodium tungstate injection. Am J Med Sci 199:668-70, 1940
48. Kinard FW, Van de Erve J: The toxicity of orally ingested tungsten compounds in the rat. J Pharmacol Exp Ther 72:196-201, 1941
49. Johnson JL, Rajagopalan KV, Cohen HJ: Molecular basis of the biological function of molybdenum--Effect of tungsten on xanthine oxidase and sulfite oxidase in the rat. J Biol Chem 249:859-66, 1974
50. Nadeenko VG: Maximum permissible concentrations of tungsten in water basins. Hyg Sanit 31:197-203, 1966
51. Beard RR: Early development of behavioral toxicology in the US in Xintaras C, Johnson BL de Groot I (eds): Behavioral toxicology--Early detection of occupational hazards. US Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1974, p 437
52. Kinard FW, Aull JC: Distribution of tungsten in the rat following ingestion of tungsten compounds. J Pharmacol Exp Ther 83:53-55, 1945
53. Aamodt RL: Inhalation of tungsten-181 labeled tungstic oxide by six beagle dogs. Health Phys 28:733-42, 1975
54. Reber E: [Investigations on dust hazards accompanying the production and machining of hard metals.] Staub-Reinhalt Luft 29:57-62, 1969 (Ger)

55. Kitzler W: [Dust measurements in Austrian hard metal works.] Staub 21:530-32, 1961 (Ger)
56. Fairhall LT, Keenan RG, Brinton HP: Cobalt and the dust environment of the cemented tungsten carbide industry. Public Health Rep 64:485-90, 1949
57. Reber E, Burckhardt P: [Hard-metal pneumoconiosis in Switzerland.] Respiration 27:120-42, 1970 (Ger)
58. Lundgren KD, Ohman H: [Pneumoconiosis in the hard metal industry-- Technical and medical study.] Arch Pathol Anat Physiol Klin Med 325:259-84, 1954 (Ger)
59. Salikhodzhaev SS, Vengerskaya KY: [Some industrial hygiene problems in the production of hard alloys.] Poroshk Metall 2:106-10, 1962 (Rus)
60. McDermott FT: Dust in the cemented carbide industry. Am Ind Hyg Assoc J 32:188-93, 1971
61. American Conference of Governmental Industrial Hygienists, Committee on Industrial Ventilation: Industrial Ventilation--A Manual of Recommended Practice, ed 14. Lansing, Mich, ACGIH, 1976, pp 1-1 to 14-8
62. American National Standards Institute Inc: Fundamentals Governing the Design and Operation of Local Exhaust Systems, ANSI Z9.2-1971. New York, ANSI, 1971, 63 pp
63. Hagopian JH, Bastress EK: Recommended Industrial Ventilation Guidelines, HEW publication No. (NIOSH) 76-162. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health Division of Physical Science and Engineering, 1976, 330 pp
64. Moschinski G, Jurisch A, Reinl W: [Pulmonary changes in sintered hard metal workers.] Arch Gewerbepathol Gewerbehyg 16:697-720, 1959 (Ger)
65. National Institute for Occupational Safety and Health, Division of Laboratories and Criteria Development: General Procedures for Metals-- Physical and Chemical Analysis Branch Method 173, in NIOSH Manual of Analytical Methods, HEW publication No (NIOSH) 75-121. Cincinnati, US Dept of Health, Education, and Welfare, Public Health Service, Center for Disease Control, NIOSH, 1974, pp 173-1 to 174-8
66. Elwell WT, Wood DF: Analytical Chemistry of Molybdenum and Tungsten--Including the analysis of the metals and their alloys (Belcher R, Freiser H, eds). New York, Pergamon Press, 1971, 277 pp

67. Butler CC, Kniseley RN: Nonferrous metallurgy--II. Zirconium, hafnium, vanadium, niobium, tantalum, chromium, molybdenum, and tungsten. Anal Chem 45:129R-50R, 1973
68. Salyamon GS, Krashenitsyna LA: [The determination of tungsten and its compounds in air.] Gig Sanit 37:78-79, 1972 (Rus)
69. Peregud YA, Gernet YV: [Chemical Analysis of Air in Industrial Enterprises, ed 3 rev.] Leningrad, Khimiya Press, 1973, pp 22-23,358-61 (Rus)
70. Urusova TM: [On the method of determining tungsten, cobalt and titanium in their mixtures.] Gig Sanit 34:72-74, 1969 (Rus)
71. Seeley JL, Skogerboe RK: Combined sampling-analysis method for the determination of trace elements in atmospheric particulates. Anal Chem 46:415-21, 1974
72. Mamuro T, Fujita A, Matsuda Y: Activation analysis of airborne dust. Annu Rep Radiat Cent Osaka Prefect 10:9-20, 1969
73. Dams R, Robbins JA, Rahn KA, Winchester JW: Nondestructive neutron activation analysis for air pollution particulates. Anal Chem 42:861-67, 1970
74. Roberts NJ: Aerosol Trace Element Analysis Using Neutron Activation and X-ray Fluorescence. Springfield, Va, US Dept of Commerce, National Technical Information Service, 1974, 117 pp (NTIS UCRL 51499)
75. Pszenny AAP, Russell IJ: An Analysis of Air Particulates Generated in the Boston Sumner Tunnel and Environs, report No. COO 3015-10. Unpublished report submitted to Atomic Energy Commission, Chicago Operations Office by Boston College, Dept of Chemistry, Chestnut Hill, Mass, June 1974, 14 pp
76. Linch AL, Hendrickson ER, Katz M, Martin RJ, Nelson GO, Pattison JN, Kolk ALV, Weidner, RB, Harman JN: Nondestructive neutron activation analysis of air pollution particles. Health Lab Sci 10:251-62, 1973
77. Peirson DH, Cawse PA, Salmon L, Cambray RS: Trace elements in the atmospheric environment. Nature 241:252-56, 1973
78. Dzubay TG, Stevens RK: Ambient air analysis with dichotomous sampler and X-ray fluorescence spectrometer. Environ Sci Technol 9:663-68, 1975
79. Fogg AG, Marriott DR, Burns DT: The spectrophotometric determination of tungsten with thiocyanate--A review of procedures. Analyst 95: 848-53, 1970

80. Husler J: Determination of chromium, manganese, molybdenum, tungsten, and vanadium in tool and alloy steels by atomic absorption spectrometry. At Absorpt Newsl 10:60-62, 1971
81. Edgar RM: Application of sodium sulfate in eliminating interferences in determination of tungsten by atomic absorption spectrometry. Anal Chem 48:1653-54, 1976
82. Rao PD: Solvent extraction procedure for the determination of tungsten in ores. At Absorpt Newsl 9:131-32, 1970
83. Welcher GG, Kriege OH: The determination of major alloying elements in high temperature cobalt-base alloys by atomic absorption spectrophotometry. At Absorpt Newsl 9:61-64, 1970
84. Manning DC: The nitrous oxide-acetylene flame in atomic absorption spectroscopy. At Absorpt Newsl 5:127-34, 1966
85. Joint AIHA-ACGIH Respiratory Protective Devices Committee: Respiratory Protective Devices Manual. Ann Arbor, Mich, American Industrial Hygiene Association and American Conference of Governmental Industrial Hygienists, 1963, 162 pp
86. Roberts WC: The Ferroalloy Industry--Hazards of the Alloys and Semimetals: Part II. J Occup Med 7:71-77, 1965
87. Lamprey H, Ripley RL: Ultrafine tungsten and molybdenum powders. J Electrochem Soc 109:713-16, 1962
88. Mertis K, Galyer L, Wilkinson G: Permethyls of tantalum, tungsten, and rhenium--Warning. J Organomet Chem 97:C65, 1975
89. Galyer LM, Wilkinson G: New synthesis of hexamethyltungsten (VI)--Hexamethylrhenium (VI) and dioxotrimethylrhenium (VII). J Organomet Chem 85:C37-C38, 1975
90. Lee S, Kye RK: Tungsten, alloys, compounds, in Encyclopaedia of Occupational Health and Safety. Geneva, International Labour Office, 1972, Vol II, pp 1143-44
91. American Conference of Governmental Industrial Hygienists: Threshold Limit Values for 1967--Recommended and Intended Values. Cincinnati, ACGIH, 1967, pp 16-17
92. American Conference of Governmental Industrial Hygienists: Threshold Limit Values for Airborne Contaminants Adopted by the ACGIH for 1969. Cincinnati, ACGIH, 1969, pp 14-15
93. American Conference of Governmental Industrial Hygienists, Committee on Threshold Limit Values: Documentation of Threshold Limit Values for Substances in Workroom Air, ed 3, 1971. Cincinnati, ACGIH, 2nd printing, 1974, pp 272-74



94. Frederick WG, Bradley WR: Toxicity of some materials used in the manufacture of cemented tungsten carbide tools. *Ind Med* 15:482-83, 1946
95. Harding HE: Notes on the toxicology of cobalt metal. *Br J Ind Med* 7:76-78, 1950
96. American Conference of Governmental Industrial Hygienists: TLV's--Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1976. Cincinnati, ACGIH, 1976, p 30
97. Toxic effect of refractory compounds, in Brakhnova IT: *Studies in Soviet Science--Environmental Hazards of Metals--Toxicity of Powdered Metals and Metal Compounds* (Slep JH transl). New York, Consultants Bureau, 1975, pp 115-249
98. Permissible Levels of Toxic Substances in the Working Environment--Sixth Session of the Joint ILO/WHO Committee on Occupational Health, Geneva, 4-10 June 1968. Geneva, International Labour Office, 1970, pp 240,343
99. [Industrial Hygiene--Allowable Concentrations of Harmful Substances in the Atmosphere of Workplaces--Definitions and Limit Values, Industrial Standard No. 22310.] Democratic Republic of Germany, East German Ministry of Health, 1972, 3 pp (Ger)
100. Sivjakov KI, Braun HA: The treatment of acute selenium, cadmium, and tungsten intoxication in rats with calcium disodium ethylenediaminetetracetate. *Toxicol Appl Pharmacol* 1:602-08, 1959
101. Joseph M: Hard metal pneumoconiosis. *Aust Radiol* 12:92-95, 1968
102. Hughes WF Jr: The tolerance of rabbit cornea for various chemical substances, in Friedenwald JS, Woods AC (eds): *The Physiology, Biochemistry and Cytopathology of the Cornea in Relation to Injury by Mustard Gas and Allied Toxic Agents*. Baltimore, Johns Hopkins University, 1944, pp 338-49
103. Higgins ES, Richert DA, Westerfeld WW: Molybdenum deficiency and tungstate inhibition studies. *J Nutr* 59:539-59, 1956
104. Johnson JL, Cohen HJ, Rajagopalan KV: Molecular basis of the biological function of molybdenum--Molbdenum-free sulfite oxidase from livers of tungsten-treated rats. *J Biol Chem* 249:5046-55, 1974
105. Johnson JL, Wand WR, Cohen HJ, Rajagopalan KV: Molecular basis of the biological function of molybdenum--Molybdenum-free xanthine oxidase from livers of tungsten-treated rats. *J Biol Med* 249:5056-61, 1974

106. Owen EC, Proudfoot R: The effect of tungstate ingestion on xanthine oxidase in milk and liver. Br J Nutr 22:331-40, 1968
107. Thomerson DR, Price WJ: A comprehensive scheme for the analysis of a wide range of steels by atomic-absorption spectrophotometry. Analyst 96:825-34, 1971
108. Keller E, Parsons ML: Determination of tungsten in silicate ores. At Absorpt News1 9:92-93, 1970

## IX. APPENDIX I

### METHOD FOR SAMPLING TUNGSTEN AND ITS PRODUCTS IN AIR

This sampling method is adapted from NIOSH Method No. P & CAM 173 [65]. Although this method of sampling is recommended, other methods shown to be at least as efficient may be used.

#### General Requirements

Collect personal samples in the breathing zone of individual employees without interfering with the employees' freedom of movement. Enough samples should be obtained to permit calculation of a TWA concentration and to evaluate the exposure of each employee at every operation or location in which there is occupational exposure to tungsten. Record the sampling locations and conditions, including ambient temperature and pressure, equipment used, time and rate of sampling, and any other pertinent information.

#### Equipment for Air Sampling

(a) Filter: Cellulose ester membrane filter with a pore size of 0.8  $\mu\text{m}$  mounted with backup pad in a 2- or 3-piece closed-face cassette.

(b) Battery-operated personal sampling pump: The pump should have a means for attachment, such as a clip, to the employee. All pumps and flowmeters must be calibrated using a calibrated test meter or other reference, as described in the Section on Calibration of Equipment.

Battery-operated pumps should be capable of operating at flowrates of 1.5-2.5 liters/minute and must be capable of at least 4 and preferably 8 hours of continuous operation without recharging.

### Calibration of Equipment

Since the accuracy of an analysis can be no greater than the accuracy with which the volume of air is measured, the accurate calibration of the sampling pump is essential. The frequency of calibration required depends upon the use, care, and handling to which the pump is subjected. Pumps should be recalibrated if they have been abused or if they have just been repaired or received from the manufacturer. Maintenance and calibration should be performed on a routine schedule, and records of these should be maintained.

Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples. If extensive field sampling is performed, calibration may also be performed periodically during sampling to ensure the continuous satisfactory operation of the pump and sampler. The accuracy of calibration depends on the type of instrument used as a reference. The choice of calibration instrument will depend largely upon where the calibration is to be performed. For laboratory testing, a spirometer or soapbubble meter is recommended, although other calibration instruments, such as a wet test meter, dry gas meter, or rotameter can be used. The calibration instrument should be calibrated to  $\pm 5\%$ . The actual setups will be similar for all instruments.

The calibration setup for a personal sampling pump with a membrane

filter is shown in Figure XII-3. Since the flowrate given by a pump depends on the pressure drop across the sampling device, the pump must be calibrated while operating with a representative filter in line. Instructions for calibration with the soapbubble meter follow. If another calibration device is selected, equivalent procedures should be used.

(a) Check the voltage of the pump battery with a voltmeter to ensure adequate voltage for calibration. Charge the battery if necessary.

(b) Place a membrane filter in the holder.

(c) Assemble the sampling train as shown in Figure XII-2.

(d) Turn on the pump and moisten the inside of the soapbubble meter by immersing the buret in the soap solution and drawing bubbles up the inside until they are able to travel the entire length of the buret without bursting.

(e) Adjust the pump flow controller to provide the desired flowrate.

(f) Check the water manometer to ensure that the pressure drop across the sampling train does not exceed 13 inches of water (approximately 1 inch of mercury).

(g) Start a soapbubble up the buret and measure with a stopwatch the time the bubble takes to move from one calibration mark to another.

(h) Repeat the procedure in (f) at least 3 times, average the results, and calculate the flowrate by dividing the volume between the preselected marks by the time required for the soapbubble to traverse the distance.

(i) Record the data for the calibration including volume measured, elapsed time, pressure drop, air temperature, atmospheric pressure, serial

number of the pump, date, and the name of the person performing the calibration.

#### Collection of Samples

(a) Assemble a sampling train consisting of a 0.8- $\mu$ m cellulose ester membrane filter with a portable, battery-operated personal sampling pump.

(b) The recommended sampling flowrate is 2-2.5 liters/minute. Establish the calibrated flowrate as accurately as possible, using the manufacturer's directions.

(c) Measure and record the temperature and pressure of the atmosphere being sampled.

(d) Record the elapsed time. The sample volume is obtained by multiplying the flowrate by the elapsed time.

(e) Immediately after sampling, seal the filter container.

(f) Treat at least one filter in the same manner as the sample (open and reclose cassette, seal, and ship), but do not draw air through it. This filter will serve as a blank.

## X. APPENDIX II

### ANALYTICAL METHOD FOR TUNGSTEN

The use of an atomic absorption spectrophotometer probably provides the most economical method for the analysis of tungsten. This method is recommended by NIOSH for determining compliance with the recommended tungsten standard. The procedure is based on Method No. P & CAM 173 of the NIOSH Manual of Analytical Methods [65]. Other modifications of the method in extraction [82], dissolution [80], and elimination of interferences [81] may be beneficial in improving the sensitivity. Other methods of analysis of tungsten may be used, provided their precision and sensitivity are determined to be at least equivalent to this method.

#### Principle of the Method

The sample, collected on a cellulose membrane filter, is washed using a mixture of nitric acid and hydrofluoric acid [80] to destroy the organic matter and to bring tungsten into solution. Samples, blanks, and standards are aspirated into the nitrous oxide-acetylene flame of the atomic absorption spectrophotometer. A hollow cathode lamp for tungsten provides the characteristic lines at 255 [80,81,107] or 401 nm [82-84,108]. The former of these two wavelengths is preferred because of its greater sensitivity. However, the 255 nm line is more susceptible to interferences, especially from molybdenum, manganese, and chromium [81]. The absorption of these lines by the ground-state atoms in the flame is

proportional to the tungsten concentration in the aspirated sample.

### Range and Sensitivity

For tungsten, the optimum working concentration is 500  $\mu\text{g/ml}$  [83], and the lowest detectable concentration is 3  $\mu\text{g/ml}$  [84]. The method can be extended to higher concentrations by dilution of the sample. The sensitivity is 35  $\mu\text{g}$  of tungsten/ml/1% absorbance [84]. This value will vary somewhat with the instrument used.

### Interferences

Edgar [81] reported enhancement of tungsten absorption caused by the presence of contaminant metals, particularly by vanadium, molybdenum, manganese, chromium, and nickel. He recommended a 2% addition of sodium sulfate to eliminate interference by these other metals.

### Precision and Accuracy

In general, this analytical method will provide a coefficient of variation of approximately 2%, depending to an extent upon the instrument used. Keller and Parsons [108] estimated a standard deviation of 0.06% based on their analyses of tungsten in silicate ores. Data on the accuracy and precision of the method are not yet available.



### Advantages and Disadvantages

The method is rapid because there is little sample preparation involved. It can be performed with generally available laboratory equipment and by general laboratory personnel. Although interferences by other metals were recognized [81], these interferences were not reported [10,29] in the determination of environmental concentrations of tungsten. However, since the method measures total tungsten, it is not capable of distinguishing insoluble from soluble tungsten compounds. Furthermore, the proposed method will not determine tungstic oxide, an acid-insoluble compound.

### Apparatus and Equipment

- (a) Hollow cathode lamp for tungsten.
- (b) Atomic absorption spectrophotometer having a monochromator with a reciprocal linear dispersion of about 6.5 Angstroms/mm in the ultraviolet region and equipped with a burner head for nitrous oxide-acetylene flame.
- (c) Oxidant: Nitrous oxide is required as an oxidant since higher temperatures are needed in the analysis of tungsten.
- (d) Fuel: Acetylene, commercially available for atomic absorption use.
- (e) Pressure-reducing valves: A 2-gauge, 2-stage pressure-reducing valve and appropriate hose connections are needed for each compressed gas tank used.
- (f) Glassware:
  - 15-ml graduated tubes

10-ml and 100-ml volumetric flasks

(g) Plastic Laboratory Ware:

125-ml polytetrafluoroethylene (PTFE) beakers with  
PTFE lids

125-ml polyethylene bottles

(h) Hotplates capable of reaching 250 C

Reagents

(a) Doubly distilled or deionized water

(b) Redistilled concentrated nitric acid

(c) Concentrated hydrofluoric acid

(d) 10% aqueous sodium sulfate

(e) Aqueous stock standards of tungsten or cobalt of 2,000  $\mu\text{g}/\text{ml}$

Procedure

(a) Cleaning of Equipment:

Before use, plastic and glassware should be washed with a laboratory glassware detergent, rinsed with tap water, then soaked for 30 minutes with 10% nitric acid. Several rinses with distilled or deionized water should follow.

(b) Analysis of Samples:

Samples are transferred to clean 125-ml PTFE beakers (glassware should not be used at this stage) and several milliliters of concentrated nitric acid and hydrofluoric acid are added to each. Each beaker is covered loosely with a PTFE lid and heated on a hotplate (140 C) in a fume

hood until the sample chars or until a slightly yellow solution remains. Several additions of nitric acid and hydrofluoric acid may be needed to completely ash and destroy the organic material.

Once the ashing is complete, as indicated by a whitish residue in the beaker and following several minutes on the high temperature hotplate (200-250 C), the ash is then dissolved with deionized water and quantitatively transferred to a 15-ml graduated tube. If the sample is expected to be heavily contaminated with such metals as vanadium, molybdenum, manganese, chromium, and nickel, it may be useful to add 2% of sodium sulfate as recommended by Edgar [81] to reduce interferences. Aliquots of this can be diluted if necessary, or the volume can be reduced by evaporation to get the tungsten concentration within the working range of the method.

The sample is then aspirated into a reducing nitrous oxide-acetylene flame and measured at 255 nm (alternatively 401 nm). The other operating parameters are set according to the instrument instructions from the manufacturer. When very low tungsten concentrations are found in the sample, scale expansion can be used to increase instrument response.

#### Calibration and Standards

From 2,000  $\mu\text{g}$  of tungsten/ml of stock standard solutions, prepare working standards to cover the range between 3 and 1,000  $\mu\text{g}/\text{ml}$ . The standard solutions are made in deionized water, with 2% sodium sulfate added if the samples were similarly treated, and are stored in polyethylene bottles. The low concentration standards may deteriorate and should be made on the day they are to be used.

Aspirate the series of standards and record the percentage of absorption.

Prepare calibration curves by plotting on linear graph paper the absorbance versus the concentration of each standard in  $\mu\text{g/ml}$ . It is advisable to run a set of standards both before and after a sample run to ensure that conditions have not changed.

### Calculations

From the calibration curve, read the concentration in  $\mu\text{g/ml}$  in the analysis sample.

Blank values, if any, are subtracted from each sample. The concentration of tungsten in air can be expressed as milligrams of tungsten/cubic meter of air, which is numerically equal to micrograms of tungsten/liter of air:

$$\text{mg tungsten/cu m} = \mu\text{g tungsten/V} \times \text{D}$$

where:

$\mu\text{g tungsten}$  = micrograms of tungsten from the  
calibration curve

V = volume of air sampled (in liters) at 25 C  
and 760 mmHg

D = dilution, if any

## XI. APPENDIX III

### MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material shall be provided in the appropriate block of the Material Safety Data Sheet (MSDS).

The product designation is inserted in the block in the upper left corner of the first page to facilitate filing and retrieval. Print in upper case letters as large as possible. It should be printed to read upright with the sheet turned sideways. The product designation is that name or code designation which appears on the label, or by which the product is sold or known by employees. The relative numerical hazard ratings and key statements are those determined by the rules in Chapter V, Part B, of the NIOSH publication, An Identification System for Occupationally Hazardous Materials. The company identification may be printed in the upper right corner if desired.

#### (a) Section I. Product Identification

The manufacturer's name, address, and regular and emergency telephone numbers (including area code) are inserted in the appropriate blocks of Section I. The company listed should be a source of detailed backup information on the hazards of the material(s) covered by the MSDS. The listing of suppliers or wholesale distributors is discouraged. The trade name should be the product designation or common name associated with the material. The synonyms are those commonly used for the product, especially formal chemical nomenclature. Every known chemical designation or

competitor's trade name need not be listed.

(b) Section II. Hazardous Ingredients

The "materials" listed in Section II shall be those substances which are part of the hazardous product covered by the MSDS and individually meet any of the criteria defining a hazardous material. Thus, one component of a multicomponent product might be listed because of its toxicity, another component because of its flammability, while a third component could be included both for its toxicity and its reactivity. Note that a MSDS for a single component product must have the name of the material repeated in this section to avoid giving the impression that there are no hazardous ingredients.

Chemical substances should be listed according to their complete name derived from a recognized system of nomenclature. Where possible, avoid using common names and general class names such as "aromatic amine," "safety solvent," or "aliphatic hydrocarbon" when the specific name is known.

The "%" may be the approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range or maximum amount, ie, "10-40% vol" or "10% max wt" to avoid disclosure of trade secrets.

Toxic hazard data shall be stated in terms of concentration, mode of exposure or test, and animal used, eg, "100 ppm LC50-rat," "25 mg/kg LD50-skin-rabbit," "75 ppm LC man," or "permissible exposure from 29 CFR 1910.1000," or, if not available, from other sources of publications such as the American Conference of Governmental Industrial Hygienists or the American National Standards Institute Inc. Flashpoint, shock sensitivity

or similar descriptive data may be used to indicate flammability, reactivity, or similar hazardous properties of the material.

(c) Section III. Physical Data

The data in Section III should be for the total mixture and should include the boiling point and melting point in degrees Fahrenheit (Celsius in parentheses); vapor pressure, in conventional millimeters of mercury (mmHg); vapor density of gas or vapor (air = 1); solubility in water, in parts/hundred parts of water by weight; specific gravity (water = 1); percent volatiles (indicated if by weight or volume) at 70 degrees Fahrenheit (21.1 degrees Celsius); evaporation rate for liquids or sublimable solids, relative to butyl acetate; and appearance and odor. These data are useful for the control of toxic substances. Boiling point, vapor density, percent volatiles, vapor pressure, and evaporation are useful for designing proper ventilation equipment. This information is also useful for design and deployment of adequate fire and spill containment equipment. The appearance and odor may facilitate identification of substances stored in improperly marked containers, or when spilled.

(d) Section IV. Fire and Explosion Data

Section IV should contain complete fire and explosion data for the product, including flashpoint and autoignition temperature in degrees Fahrenheit (Celsius in parentheses); flammable limits, in percent by volume in air; suitable extinguishing media or materials; special firefighting procedures; and unusual fire and explosion hazard information. If the product presents no fire hazard, insert "NO FIRE HAZARD" on the line labeled "Extinguishing Media."

(e) Section V. Health Hazard Information

The "Health Hazard Data" should be a combined estimate of the hazard of the total product. This can be expressed as a TWA concentration, as a permissible exposure, or by some other indication of an acceptable standard. Other data are acceptable, such as lowest LD50 if multiple components are involved.

Under "Routes of Exposure," comments in each category should reflect the potential hazard from absorption by the route in question. Comments should indicate the severity of the effect and the basis for the statement if possible. The basis might be animal studies, analogy with similar products, or human experiences. Comments such as "yes" or "possible" are not helpful. Typical comments might be:

Skin Contact--single short contact, no adverse effects likely; prolonged or repeated contact, possibly mild irritation.

Eye Contact--some pain and mild transient irritation; no corneal scarring.

"Emergency and First Aid Procedures" should be written in lay language and should primarily represent first-aid treatment that could be provided by paramedical personnel or individuals trained in first aid.

Information in the "Notes to Physician" section should include any special medical information which would be of assistance to an attending physician including required or recommended preplacement and periodic medical examinations, diagnostic procedures, and medical management of overexposed employees.



(f) Section VI. Reactivity Data

The comments in Section VI relate to safe storage and handling of hazardous, unstable substances. It is particularly important to highlight instability or incompatibility to common substances or circumstances, such as water, direct sunlight, steel or copper piping, acids, alkalies, etc. "Hazardous Decomposition Products" shall include those products released under fire conditions. It must also include dangerous products produced by aging, such as peroxides in the case of some ethers. Where applicable, shelf life should also be indicated.

(g) Section VII. Spill or Leak Procedures

Detailed procedures for cleanup and disposal should be listed with emphasis on precautions to be taken to protect employees assigned to cleanup detail. Specific neutralizing chemicals or procedures should be described in detail. Disposal methods should be explicit including proper labeling of containers holding residues and ultimate disposal methods such as "sanitary landfill," or "incineration." Warnings such as "comply with local, state, and federal antipollution ordinances" are proper but not sufficient. Specific procedures shall be identified.

(h) Section VIII. Special Protection Information

Section VIII requires specific information. Statements such as "Yes," "No," or "If necessary" are not informative. Ventilation requirements should be specific as to type and preferred methods. Respirators shall be specified as to type and NIOSH or US Bureau of Mines approval class, ie, "Supplied air," "Organic vapor canister," etc. Protective equipment must be specified as to type and materials of construction.

(i) Section IX. Special Precautions

"Precautionary Statements" shall consist of the label statements selected for use on the container or placard. Additional information on any aspect of safety or health not covered in other sections should be inserted in Section IX. The lower block can contain references to published guides or in-house procedures for handling and storage. Department of Transportation markings and classifications and other freight, handling, or storage requirements and environmental controls can be noted.

(j) Signature and Filing

Finally, the name and address of the responsible person who completed the MSDS and the date of completion are entered. This will facilitate correction of errors and identify a source of additional information.

The MSDS shall be filed in a location readily accessible to employees exposed to the hazardous substance. The MSDS can be used as a training aid and basis for discussion during safety meetings and training of new employees. It should assist management by directing attention to the need for specific control engineering, work practices, and protective measures to ensure safe handling and use of the material. It will aid the safety and health staff in planning a safe and healthful work environment and in suggesting appropriate emergency procedures and sources of help in the event of harmful exposure of employees.

--


## MATERIAL SAFETY DATA SHEET

I PRODUCT IDENTIFICATION		
MANUFACTURER'S NAME	REGULAR TELEPHONE NO EMERGENCY TELEPHONE NO	
ADDRESS		
<b>TRADE NAME</b>		
<b>SYNONYMS</b>		
II HAZARDOUS INGREDIENTS		
MATERIAL OR COMPONENT	%	HAZARD DATA
III PHYSICAL DATA		
BOILING POINT (760 MM HG)		MELTING POINT
SPECIFIC GRAVITY (H <sub>2</sub> O=1)		VAPOR PRESSURE
VAPOR DENSITY (AIR=1)		SOLUBILITY IN H <sub>2</sub> O, % BY WT
% VOLATILES BY VOL		EVAPORATION RATE (BUTYL ACETATE=1)
APPEARANCE AND ODOR		

<b>IV FIRE AND EXPLOSION DATA</b>				
FLASH POINT (TEST METHOD)		AUTOIGNITION TEMPERATURE		
FLAMMABLE LIMITS IN AIR, % BY VOL.		LOWER		UPPER
EXTINGUISHING MEDIA				
SPECIAL FIRE FIGHTING PROCEDURES				
UNUSUAL FIRE AND EXPLOSION HAZARD				
<b>V HEALTH HAZARD INFORMATION</b>				
HEALTH HAZARD DATA				
ROUTES OF EXPOSURE				
INHALATION				
SKIN CONTACT				
SKIN ABSORPTION				
EYE CONTACT				
INGESTION				
EFFECTS OF OVEREXPOSURE				
ACUTE OVEREXPOSURE				
CHRONIC OVEREXPOSURE				
EMERGENCY AND FIRST AID PROCEDURES				
EYES				
SKIN				
INHALATION:				
INGESTION				
NOTES TO PHYSICIAN				

<b>VI REACTIVITY DATA</b>
CONDITIONS CONTRIBUTING TO INSTABILITY
INCOMPATIBILITY
HAZARDOUS DECOMPOSITION PRODUCTS
CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION
<b>VII SPILL OR LEAK PROCEDURES</b>
STEPS TO BE TAKEN IF MATERIAL IS RELEASED OR SPILLED
NEUTRALIZING CHEMICALS
WASTE DISPOSAL METHOD
<b>VIII SPECIAL PROTECTION INFORMATION</b>
VENTILATION REQUIREMENTS
SPECIFIC PERSONAL PROTECTIVE EQUIPMENT
RESPIRATORY (SPECIFY IN DETAIL)
EYE
GLOVES
OTHER CLOTHING AND EQUIPMENT

**IX SPECIAL PRECAUTIONS**

PRECAUTIONARY  
STATEMENTS

OTHER HANDLING AND  
STORAGE REQUIREMENTS

PREPARED BY \_\_\_\_\_

ADDRESS \_\_\_\_\_

DATE \_\_\_\_\_

TABLE XII-1

CHEMICAL AND PHYSICAL PROPERTIES OF  
TUNGSTEN AND SELECTED TUNGSTEN COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (c)	Density	Solubility g/100cc	
					H <sub>2</sub> O*	Other
Ammonium metatungstate	(NH <sub>4</sub> ) <sub>6</sub> H <sub>2</sub> W <sub>12</sub> O <sub>40</sub>	—	Decomposes 200	4.0	Very soluble	—
Ammonium paratungstate	(NH <sub>4</sub> ) <sub>10</sub> (H <sub>10</sub> W <sub>12</sub> O <sub>46</sub> )	—	—	—	Insoluble	Decomposes in acid or alkali
Ammonium phosphotungstate	(NH <sub>4</sub> ) <sub>3</sub> P(W <sub>3</sub> O <sub>10</sub> ) <sub>4</sub>	2,931.27	—	—	Slightly soluble	Soluble in alkali, insoluble in acid
Cadmium tungstate	CdWO <sub>4</sub>	360.25	—	—	0.05	Soluble in NH <sub>4</sub> OH
Calcium tungstate	CaWO <sub>4</sub>	287.93	—	6.062	0.00064 (15 C)	—
"	"	"	—	—	0.00323 (25 C)	Insoluble in alcohol, acid; soluble in NH <sub>4</sub> Cl
Calcium tungstate (Scheelite)	"	"	—	—	0.0013 (20 C)	—
"	"	"	—	—	0.0002 (90 C)	—
Calcium metatungstate	Ca <sub>3</sub> H <sub>4</sub> [H <sub>2</sub> (W <sub>2</sub> O <sub>7</sub> ) <sub>6</sub> ] 27H <sub>2</sub> O	3,500.96	-7H <sub>2</sub> O 105	—	—	Decomposes in acid
Cerium (III) tungstate	Ce <sub>2</sub> (WO <sub>4</sub> ) <sub>3</sub>	1,023.78	1,089	6.77	—	—
Cesium tungstate	Cs <sub>2</sub> WO <sub>4</sub>	380.76	—	—	85.6 (17 C)	—
Cobalt tungstate	CoWO <sub>4</sub>	306.78	—	8.42	Insoluble	Soluble in hot concentrated acid
Copper (II) tungstate	CuWO <sub>4</sub> ·2H <sub>2</sub> O	347.42	Red heat	—	0.1 (15 C)	Soluble in NH <sub>4</sub> OH; insoluble in alcohol; decomposes in mineral acid
Cyclopentadienylbitungsten hexacarbonyl	(C <sub>5</sub> H <sub>5</sub> ) <sub>2</sub> W <sub>2</sub> (CO) <sub>6</sub>	665.95	240-2 decomposes	—	—	Soluble in chloroform, CCl <sub>4</sub> , CS <sub>2</sub>
Iron (II) tungstate	FeWO <sub>4</sub>	303.69	—	6.64	—	—
Lead tungstate	PbWO <sub>4</sub>	455.04	—	—	0.00159 (25 C)	—
Lithium tungstate	Li <sub>2</sub> WO <sub>4</sub>	261.73	742	3.71	Very soluble	Decomposes in acid; insoluble in alcohol
Magnesium tungstate	MgWO <sub>4</sub>	272.73	—	5.66	Insoluble	"

TABLE XII-1 (Continued)

CHEMICAL AND PHYSICAL PROPERTIES OF  
TUNGSTEN AND SELECTED TUNGSTEN COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (c)	Density	Solubility g/100cc	
					H <sub>2</sub> O *	Other
Mercury (I) tungstate	Hg <sub>2</sub> WO <sub>4</sub>	649.03	Decomposes	—	Insoluble	Decomposes in acid; insoluble in alcohol
Mercury (II) tungstate	HgWO <sub>4</sub>	448.44	"	—	"	"
Mesitylene-tungsten tricarbonyl	(CH <sub>3</sub> ) <sub>3</sub> C <sub>6</sub> H <sub>3</sub> W(CO) <sub>3</sub>	388.08	160-165 decomposes	—	—	—
Metatungstic acid	H <sub>2</sub> W <sub>4</sub> O <sub>13</sub> ·9H <sub>2</sub> O	1,107.4	—	2.5239 3.6503	88.57 (22 C) 111.87 (435C)	—
Phospho-tungstic acid	H <sub>3</sub> [P(W <sub>3</sub> O <sub>10</sub> ) <sub>4</sub> ] · 14H <sub>2</sub> O Tungstophosphoric acid	3,132.39	160-165 decomposes	—	Soluble	Soluble in alcohol and ether
Phospho-tungstic acid	H <sub>3</sub> [P(W <sub>3</sub> O <sub>10</sub> ) <sub>4</sub> ] · 24H <sub>2</sub> O Dodecatungstophosphoric acid	3,312.54	89	—	"	—
Potassium tungstate	K <sub>2</sub> WO <sub>4</sub> · 2H <sub>2</sub> O	362.08	921	3.113	51.5	Decomposes in acid; insoluble in alcohol
Potassium metatungstate	K <sub>6</sub> [H <sub>2</sub> W <sub>12</sub> O <sub>40</sub> ] · 18H <sub>2</sub> O	3,407.08	930	—	Soluble	Decomposes in acid
Silicotungstic acid	H <sub>8</sub> SiW <sub>12</sub> O <sub>42</sub>	2,914.2	—	—	961.5 (18 C)	—
Silver tungstate	Ag <sub>2</sub> WO <sub>4</sub>	463.59	—	—	0.05 (15 C)	—
Sodium tungstate	Na <sub>2</sub> WO <sub>4</sub>	293.83	698	4.179	57.5 (0 C) 73.2 (21 C)	—
Sodium tungstate, dihydrate	Na <sub>2</sub> WO <sub>4</sub> · 2H <sub>2</sub> O	329.86	698 Anhydrous	3.23-3.25	41.0 (0 C)	Slightly soluble in NH <sub>3</sub> ; insoluble in alcohol, acid
Sodium metatungstate	Na <sub>2</sub> O · WO <sub>2</sub> · 10H <sub>2</sub> O	1,169.53	706.6	—	Soluble	Insoluble in acid
Sodium paratungstate	Na <sub>6</sub> W <sub>7</sub> O <sub>24</sub> · 16H <sub>2</sub> O	2,097.12	-12H <sub>2</sub> O -16H <sub>2</sub> O	3.987	8	—
Strontium tungstate	SrWO <sub>4</sub>	335.47	Decomposes	6.187	0.14 (15 C)	Insoluble in dilute acid, alcohol
Tungsten (Wolfram)	W	183.85	3,410±20	19.35	Insoluble	Slightly soluble in HNO <sub>3</sub> , H <sub>2</sub> SO <sub>4</sub> , aqua regia; soluble in HNO <sub>3</sub> +HF, fused in NaOH+NaNO <sub>3</sub> ; insoluble in HF, KOH



TABLE XII-1 (Continued)  
CHEMICAL AND PHYSICAL PROPERTIES OF  
TUNGSTEN AND SELECTED TUNGSTEN COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (c)	Density	Solubility g/100cc	
					H <sub>2</sub> O	Other
Tungsten arsenide	WAs <sub>2</sub>	333.69	Decomposes in red heat	6.9	Insoluble	Decomposes in hot HNO <sub>3</sub> , hot H <sub>2</sub> SO <sub>4</sub>
Tungsten diboride	WB <sub>2</sub>	205.47	2,900	10.77	"	Soluble in aqua regia
Tungsten dibromide	WBr <sub>2</sub>	343.67	Decomposes 4,400	—	Decomposes	—
Tungsten pentabromide	WBr <sub>5</sub>	583.40	276	—	"	Soluble, in absolute alcohol, chloroform, ether, alkali
Tungsten hexabromide	WBr <sub>6</sub>	663.30	232	6.9	Insoluble	Soluble in absolute alcohol, ether CS <sub>2</sub> , NH <sub>4</sub> OH
Tungsten carbide	WC	195.86	2870+50	15.63	"	Soluble in HNO <sub>3</sub> +HF, aqua regia
Tungsten dicarbide	WC <sub>2</sub>	379.71	2860	17.15	"	Soluble in HNO <sub>3</sub> +HCl
Tungsten carbonyl	W(CO) <sub>6</sub>	351.91	Decomposes N 150	2.65	"	Soluble in fuming HNO <sub>3</sub> , slightly soluble in alcohol, ether
Tungsten dichloride	WCl <sub>2</sub>	254.76	—	5.436	—	—
Tungsten tetrachloride	WCl <sub>4</sub>	325.66	Decomposes	4.624	—	—
Tungsten pentachloride	WCl <sub>5</sub>	361.12	248	3.875	—	Slightly soluble in CS <sub>2</sub>
Tungsten hexachloride	WCl <sub>6</sub>	396.57	275	3.52	Decomposes (60 C)	Soluble in alcohol, ether, benzene, CCl <sub>4</sub> , very soluble in CS <sub>2</sub> , POCl Impure WCl <sub>6</sub> decomposes at even lower temperatures
Tungsten hexafluoride	WF <sub>6</sub>	297.84	2.5	3.44 (Liquid) 12.9 g/l (Gas)	Decomposes	Soluble in alkali

TABLE XII-1 (Continued)  
CHEMICAL AND PHYSICAL PROPERTIES OF  
TUNGSTEN AND SELECTED TUNGSTEN COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (c)	Density	Solubility g/100cc	
					H <sub>2</sub> O*	Other
Tungsten diiodide	WI <sub>2</sub>	437.66	Decomposes	6.799	Insoluble	Soluble in alkali; insoluble in alcohol, CS <sub>2</sub>
Tungsten tetraiodide	WI <sub>4</sub>	691.47	"	5.2	"	Soluble in absolute alcohol insoluble in ether, chloroform, turpentine
Tungsten dinitride	WN <sub>2</sub>	211.86	Above 400(vacuum)	—	Decomposes	—
Tungsten dioxide	WO <sub>2</sub>	215.85	1,500 (in N <sub>2</sub> )	12.1	Insoluble	Soluble in acid, KOH
Tungsten trioxide (Wolframite)	WO <sub>3</sub>	231.85	1,473	7.16	"	Soluble in hot alkali; insoluble in acid
Tungsten pentoxide	Mineral blue W <sub>2</sub> O <sub>5</sub> or W <sub>4</sub> O <sub>11</sub>	447.70 or 911.39	800-900 Sublimes	—	Insoluble	Insoluble in acid
Tungsten dioxydibromide	WO <sub>2</sub> Br <sub>2</sub>	375.67	Decomposes	—	—	—
Tungsten oxytetrabromide	WOB <sub>4</sub>	519.49	277	—	Decomposes	—
Tungsten oxytetrachloride	WOCl <sub>4</sub>	341.66	211	—	"	Soluble in CS <sub>2</sub> , S <sub>2</sub> Cl <sub>2</sub> and benzene
Tungsten dioxydichloride	WO <sub>2</sub> Cl <sub>2</sub>	286.75	266	—	Soluble	Insoluble in alcohol; soluble in NH <sub>4</sub> OH, alkali
Tungsten oxytetrafluoride	WOF <sub>4</sub>	275.84	110	—	Decomposes	Slightly soluble in CS <sub>2</sub> ; insoluble in CCl <sub>4</sub>
Tungsten phosphide	WP	214.82	—	8.5	Insoluble	Soluble in HNO <sub>3</sub> +HF; insoluble in alkali, HCl
Tungsten phosphide	WP <sub>2</sub>	245.80	Decomposes	5.8	"	Soluble in HNO <sub>3</sub> +HF; insoluble in alkali, HCl; soluble in aqua regia
Tungsten phosphide	W <sub>2</sub> P	398.67	"	5.21	—	Soluble in fused Na <sub>2</sub> CO <sub>3</sub> ; insoluble in acid, aqua regia

TABLE XII-1 (Continued)

CHEMICAL AND PHYSICAL PROPERTIES OF  
TUNGSTEN AND SELECTED TUNGSTEN COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (c)	Density	Solubility g/100cc	
					H <sub>2</sub> O*	Other
Tungsten silicide	WSi <sub>2</sub>	240.02	Above 900	9.4	Insoluble	Soluble in HNO <sub>3</sub> +HF; insoluble in aqua regia
Tungsten disulfide (Tungstenite)	WS <sub>2</sub>	247.98	Decomposes	7.5	"	Soluble in HNO <sub>3</sub> +HF, fused alkali; insoluble in alcohol
12-Tungstophosphoric acid	P <sub>2</sub> O <sub>5</sub> ·24WO <sub>3</sub> ·45H <sub>2</sub> O	6,516.35	—	—	86.75 (92 C)	—

\* For the purpose of compliance with the recommended standards, insoluble tungsten compounds include all those for which water solubility is listed as insoluble or less than 0.01 g/100 cc. Soluble tungsten compounds are those listed as very soluble, soluble, slightly soluble, equal to or greater than 0.01 g/100 cc, or decomposes. Those compounds for which no solubility information is listed should be considered soluble unless it can be demonstrated that they are insoluble in water.

Adapted from references 2-6

TABLE XII-2

## OCCUPATIONS WITH POTENTIAL TUNGSTEN EXPOSURE

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Alloy makers	Melting, pouring, casting workers
Carbonyl workers	Metal sprayers
Ceramic workers	Ore-refining and foundry workers
Cemented tungsten carbide workers	Paint and pigment makers
Cement makers	Papermakers
Dyemakers	Penpoint makers
Dyers	Petroleum refinery workers
Flameproofers	Photographic developers
High-speed tool steelworkers	Spark-plug makers
Incandescent-lamp makers	Textile dryers
Industrial chemical synthesizers	Tool grinders
Inkmakers	Tungsten and molybdenum miners
Lamp-filament makers	Waterproofing makers
Lubricant makers	Welders

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Adapted from reference 12

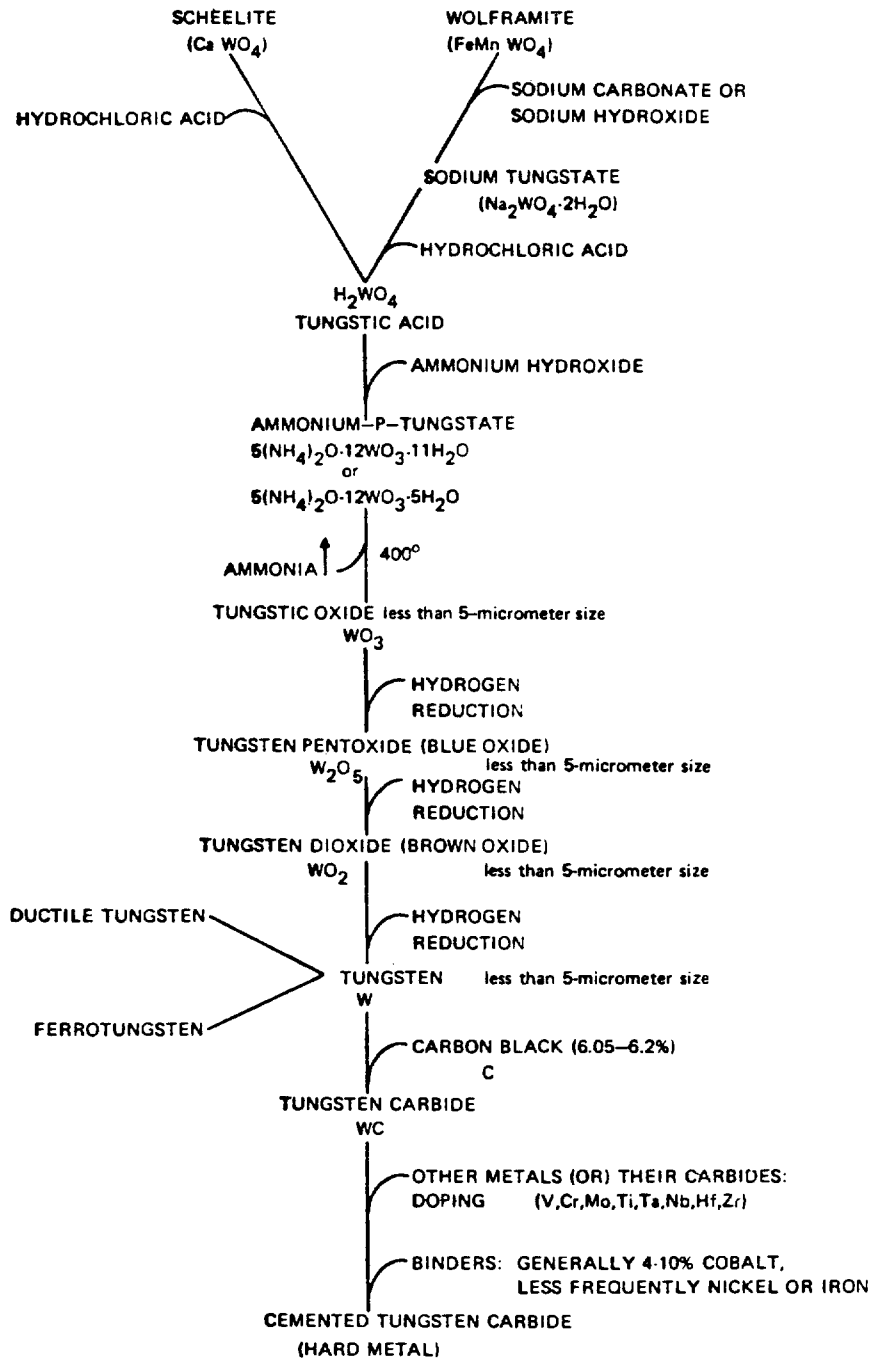


FIGURE XII-1

PRODUCTION OF COMMERCIAL TUNGSTEN COMPOUNDS AND  
CEMENTED TUNGSTEN CARBIDE

From references 10 and 11

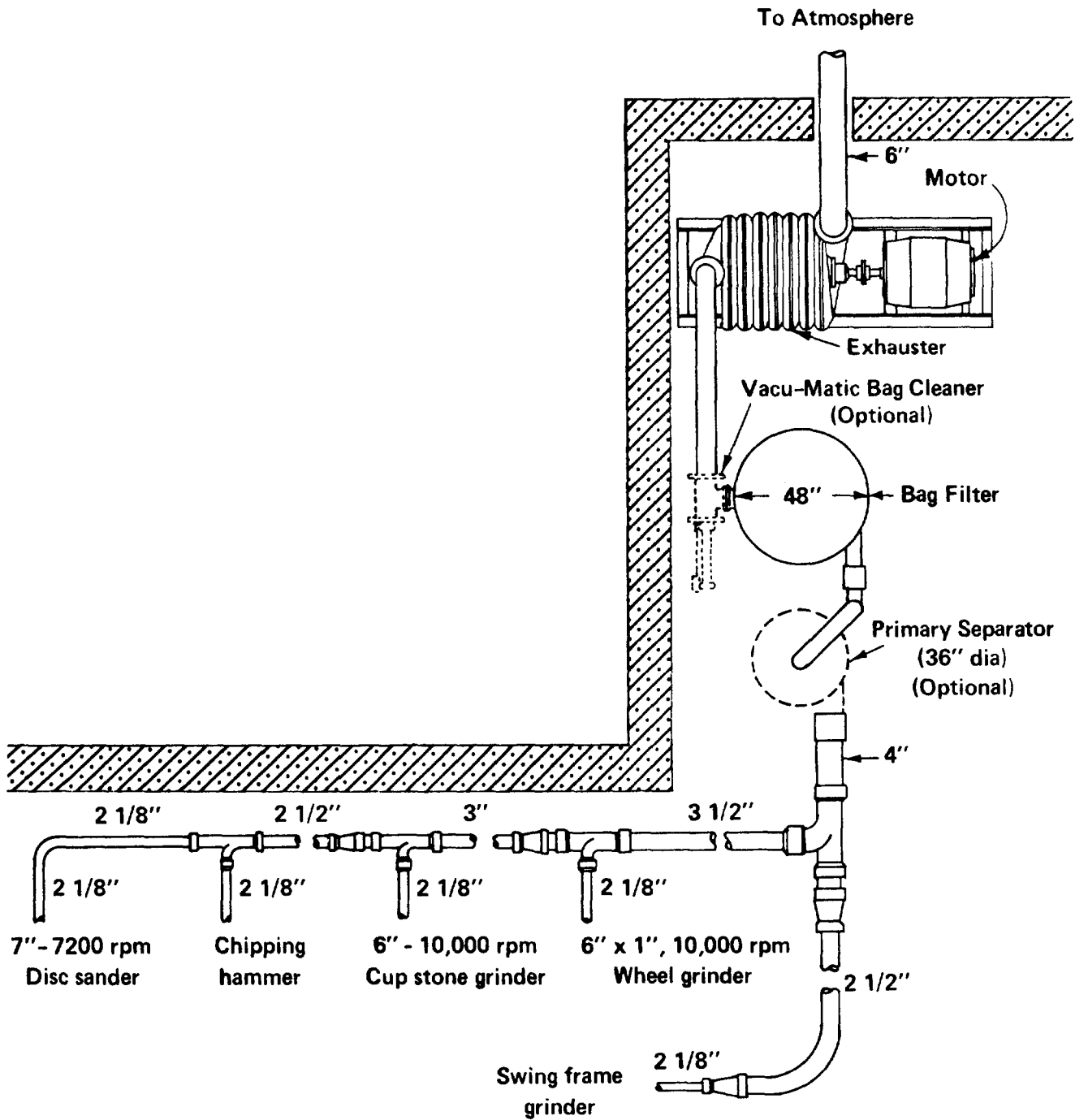


FIGURE XII-2

**TYPICAL LOW VOLUME HIGH VELOCITY VENTILATION SYSTEM\***

\*Bell and socket, smooth-flow type tubing and fittings should be used throughout the system. When system will be used for vacuum cleaning of abrasive materials, Schedule No. 40 and C.I. drainage fittings, or heavier, should be used in place of tubing.

Adapted from reference 61

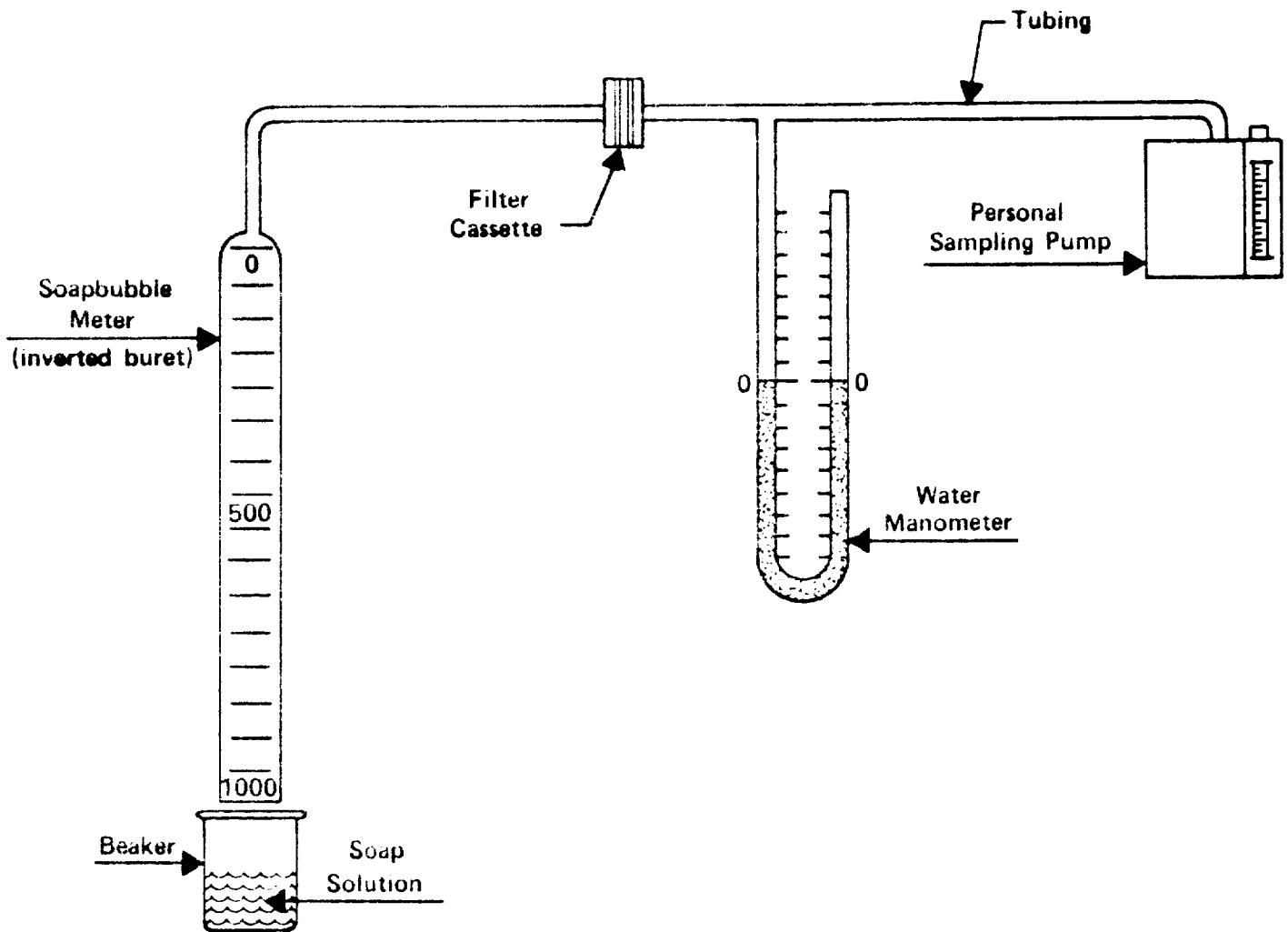


FIGURE XII-3  
 CALIBRATION SETUP FOR PERSONAL SAMPLING PUMP  
 WITH FILTER CASSETTE