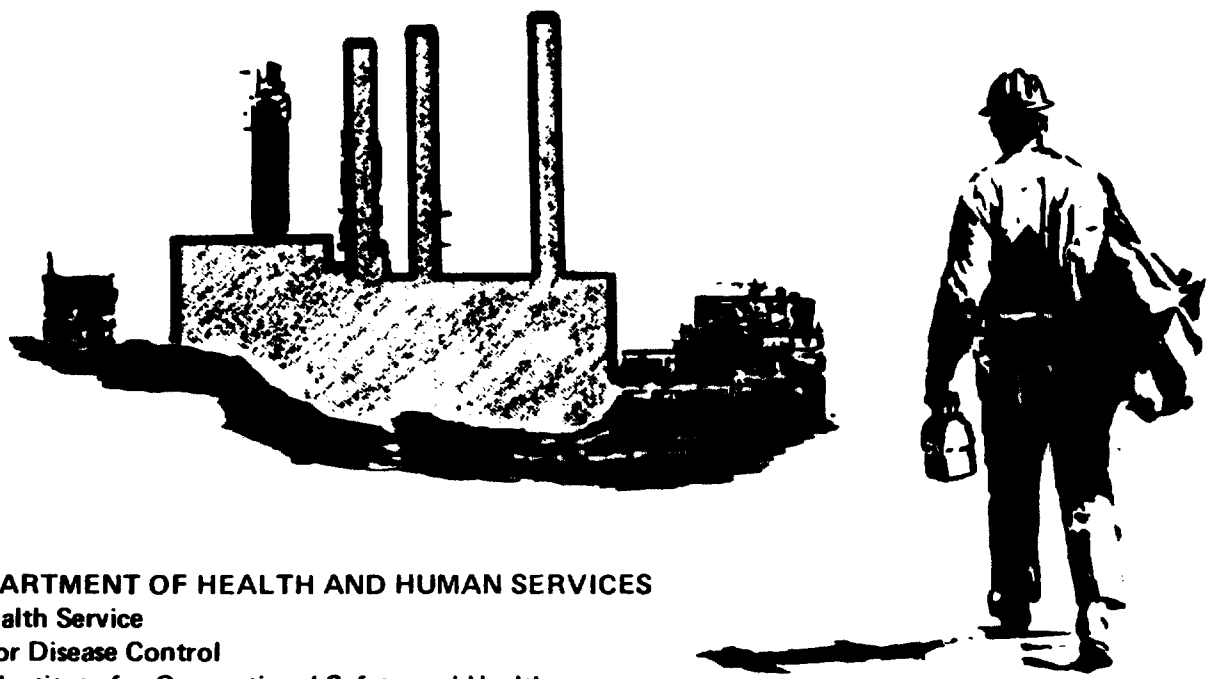
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NIOSH

OCCUPATIONAL HAZARD ASSESSMENT

Criteria for Controlling Occupational Exposure to Cobalt



U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health

CRITERIA FOR CONTROLLING

OCCUPATIONAL EXPOSURE TO COBALT

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health

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PREFACE

The National Institute for Occupational Safety and Health (NIOSH) has evaluated the information available on cobalt and concludes that a potentially serious hazard could exist in the US workforce from exposure to uncontrolled and excessive amounts of cobalt. For cobalt metal fume and dust, toxic effects have been observed in the lungs of workers and experimental animals exposed at or below the current Federal limit. Information on many of the cobalt compounds in commercial use is so limited that permissible exposure limits cannot be derived at this time.

NIOSH will periodically review the available data concerning cobalt and will make successive reports and revised recommendations as new research and epidemiologic studies are completed. If a previously unsuspected hazard becomes known, cobalt will be considered as a subject for recommending new standards.

Contributions to this document on cobalt by NIOSH staff are gratefully acknowledged as are the comments of other Federal agencies or departments, review consultants, and reviewers selected by the American Medical Association, the Society for Occupational and Environmental Health, and the Society of Toxicology, and Robert B. O'Connor, M.D., NIOSH consultant in occupational medicine. Most of these reviewers provided comments on an earlier draft criteria document on cobalt.

The views and conclusions expressed in this document are those of NIOSH. They are not necessarily those of the consultants, the reviewers selected by professional societies, or other Federal agencies. The review consultants and the Federal Agencies that received and commented on an earlier version of this document are listed on pages iv and v.

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CONTENTS

		Page
	PREFACE	iii
	REVIEW CONSULTANTS	iv
	FEDERAL AGENCIES	v
I.	SUMMARY AND CONCLUSIONS	1
II.	HEALTH EFFECTS	5
	Background	5
	Respiratory Effects	6
	Effects on the Skin	12
	Cardiac Effects	14
	Effects on Blood	16
	Effects on the Thyroid	18
	Carcinogenicity and Mutagenicity	19
	Effects on Reproduction	21
	Other Effects	22
	Distribution and Retention	24
III.	EXTENT AND MEASUREMENT OF EXPOSURE	26
	Uses of Cobalt	26
	Environmental Data	27
	Sampling Methods	31
	Analytical Methods	31
IV.	CONTROL OF EXPOSURE	34
	Engineering Controls	34
	Work Practices	36
	Work Clothing and Protective Equipment	38
	Effective Planning	39
	Worker Education and Monitoring	40
V.	BASIS FOR STANDARDS CONCERNING OCCUPATIONAL EXPOSURE TO COBALT	44
VI.	RESEARCH NEEDS	47
VII.	REFERENCES	48
VIII.	APPENDIX - Sampling and Analysis	71

CONTENTS (CONTINUED)

		Page
IX.	TABLES	80
X.	FIGURES	91

I. SUMMARY AND CONCLUSIONS

More than a million workers in the United States are potentially exposed to cobalt compounds in the course of their employment. By far, most are exposed to cobalt metal or cobalt oxides. Many are potentially exposed to oil-based paint driers containing cobalt, but their exposure is limited because these driers constitute only a small percentage of the ingredients in paint. Production of alloys and hard metal accounts for about 70% of cobalt use, but numerous other industries produce or use lesser amounts of cobalt. The degree of worker exposure in these industries depends on many factors, including the amount of cobalt used in the process, the number of engineering controls available, and the amount of cobalt-containing materials handled manually. Since workers are rarely exposed to only a single substance in the occupational environment, exposure to cobalt can be limited by the availability of engineering controls designed to reduce emission of other respirable and potentially harmful metal fumes and dusts.

The Occupational Safety and Health Administration (OSHA) standard for cobalt is 0.1 milligram of cobalt in a cubic meter of air (mg/cu m). OSHA has interpreted this standard to apply to all cobalt compounds. Of the workplaces inspected by OSHA for cobalt exposure, nearly 20% were not in compliance with this standard. Site visits revealed that plants using large quantities of cobalt have difficulties meeting this standard without extensive engineering controls.

Workers can be exposed to cobalt in many ways. Inhalation is a potential route of exposure occurring most commonly when material containing cobalt is heated, where cobalt powders are handled manually, or where cobalt materials are subjected to grinding. Dermal exposure to cobalt can occur in any operation where these materials are handled manually, but it is a special consideration where solutions containing cobalt are present. Ingestion of cobalt can also occur and is best prevented by good work practices, sanitation, and personal hygiene. The information on the health effects of cobalt is sufficient to conclude that exposure to it by any of these three routes can be a health hazard in the workplace.

Substantial information exists showing that workers who have inhaled cobalt can develop lung disease. Numerous case reports and medical and industry-wide surveys in the cemented carbide industry have demonstrated the persistent occurrence of diffuse interstitial fibrosis of the lung in exposed workers. Mild to fibrotic lung changes have been observed in workers exposed to cobalt at 0.1-0.2 mg/cu m, and airway obstruction has been observed at 0.06 mg/cu m. Although workers received exposures to cobalt mixed with other substances, animals inhaling cobalt metal have similarly developed lesions indicative of developing fibrosis at 0.1 mg/cu m. The adverse lung effects observed at or below the Federal permissible exposure limit (0.1 mg/cu m) pose the serious question of whether that limit is adequate. This is further aggravated by reports of pulmonary hypersensitivity developing in workers

exposed to cobalt. Such persons, once sensitized, could probably not tolerate inhalation of even small amounts of cobalt.

It is well known that cobalt is a skin sensitizer, and is commonly considered as a possible source of allergic dermatitis. In the workforce, persons who become sensitive to cobalt are usually transferred to other jobs, although these cases are rarely reported in the scientific literature. Since solutions of cobalt salts are used for patch testing (which itself can cause sensitization), it is reasonable to believe that these inorganic salts can also cause dermatitis and sensitization in the workplace. However, some workers may also develop skin diseases from the irritant effects of cobalt-containing dusts. These problems are best controlled through prevention of dust accumulation by engineering controls, prompt cleanup of spills, and clothing that prevents or limits excessive dermal contact with cobalt, either as dust or in solution.

Information on the toxic effects resulting from ingestion of cobalt comes from the nonoccupational environment. For example, the effects of cobalt on the heart have been studied because of an outbreak of acute cardiomyopathy in beer drinkers. The amount of cobalt consumed by these beer drinkers was substantially more than that inhaled by workers exposed at the current Federal limit. The effects of cobalt on the heart probably are less of a hazard than pulmonary fibrosis, although studies are needed to determine whether or not adverse effects on the heart become demonstrable following long-term exposures (such as in a stable workforce). Polycythemia and subsequent development of thyroid hyperplasia have also occurred following ingestion of cobalt salts. However, polycythemia does not appear to be a particularly toxic response, especially in workers exposed to cobalt at 0.1 mg/cu m or less. Experiments in animals suggest that some persons might develop changes in the thyroid even at these low concentrations, especially when individual susceptibility is considered. This evidence is sufficient to recommend palpation of the thyroid as part of the medical surveillance for workers exposed to cobalt.

Several other toxic effects can occur following exposure to cobalt. Degenerative changes in organs, including the liver, kidneys, and pancreas, can be demonstrated in animals. While few studies of workers have examined these effects, they do not appear to be severe in humans, even at doses that produce other toxic responses. Information concerning the ability of cobalt to alter blood clot formation indicates that this effect may occur at levels characteristic of the workplace. Cobalt administered orally at 1 mg daily for only 3 days resulted in a prolonged time for blood clot formation in humans. These changes are not easily discounted, having occurred at a low dose, but they need further documentation.

At least qualitatively, systemic effects, such as those on the heart, blood, and thyroid, do not appear to differ greatly between cobalt compounds tested or routes of administration used. Some quantitative differences, however, may be found as more information becomes available. A critical question is whether or not all cobalt compounds should be considered to be potentially capable of producing pulmonary fibrosis. Of the many cobalt

compounds in commercial use, only cobalt metal and cobaltous oxide have been tested by inhalation.

Both substances produced fibrotic or prefibrotic lesions and demonstrated a surprisingly high degree of solubility in lung tissue. This information, coupled with recent studies indicating that workers exposed to cobalt salts have developed adverse effects in the lungs, makes it prudent to conclude that all cobalt compounds may be capable of producing pulmonary fibrosis.

In reviewing the information available, NIOSH found that much research remains to be done before the toxic effects of the many cobalt compounds now in commercial use can be documented. One goal of this publication is to bring the pressing need for information that would permit a better analysis of occupational exposure limits for cobalt compounds to the attention of the research and occupational communities. In the interim, judgments must be made on how to control worker exposure and establish environmental and medical monitoring programs. A summary of these recommendations is presented below.

Engineering Controls

Well-maintained closed systems should be used whenever possible, and diligent attention paid to the prevention of dust accumulation. Properly-designed local ventilation should be utilized for specific operations such as grinding or buffing; particular care is necessary in designing ventilation for handling operations where cobalt-containing powders may be formed. Regular monitoring should be carried out for all ventilation systems by trained personnel.

Work Practices

Employers should institute and ensure the quality of programs that emphasize good personal hygiene. Physical hazards involved in handling cobalt-containing materials should be identified and measures taken to eliminate them. The combustion properties of individual cobalt compounds should be known by all who work with them. Proper methods of cleanup and plant maintenance should be established. Special precautions should be taken whenever a tank or vessel is entered.

Work Clothing and Protective Equipment

The use and proper cleaning of appropriate work clothing is required to prevent skin effects and sensitization. Protective equipment that is appropriate to their potential exposures to cobalt should be provided for all workers. If respirators must be used for specific situations, they should be of proper design, and well fitted and maintained.

Worker Education and Monitoring

Continuing education programs should be established that emphasize to workers proper handling and cleanup procedures for cobalt and its compounds. Instructional material should be available that covers, in written form, good work practices and known health effects, as well as other physical and chemical properties of the substances present.

Industrial hygiene surveys should be carried out to determine locations where workers might be exposed to cobalt. These surveys, carried out at 3-year intervals, should consist of personal monitoring procedures adequate to ensure that an understanding of worker exposure is obtained. All monitoring records should be maintained for 30 years following termination of employment.

Medical Surveillance

Because of the effects of the various forms of cobalt on the body, medical surveillance programs should be established that allow the responsible physician to ensure that workers are adequately surveyed. Primary emphasis should be placed on the occupational history and the surveillance of the lungs and skin. Depending on the specific work being done, the physician may wish to evaluate other organs such as the blood and heart. Medical records must also be kept for 30 years following termination of employment.

The following chapters present more detailed information on the toxic effects of cobalt and the extent and degree of worker exposure to substances containing cobalt. Methods for sampling and analysis of airborne cobalt, including a detailed procedure, are given. Engineering controls and work practices designed to control cobalt emissions into the workplace air are provided, as is a program recommended for medical and environmental surveillance and worker training.

II. HEALTH EFFECTS

Background

Cobalt occurs naturally and constitutes about 0.001% of the earth's crust [1]. It is an integral part of the cyanocobalamin molecule (vitamin B12), which is essential in the human diet to prevent the development of pernicious anemia [2,3]. The average daily intake of cobalt from food for adults in the United States has been estimated to be about 300 μg, with an additional 6 μg obtained from water and less than 0.1 μg from community air [4].

Even though cobalt salts had been used for centuries to color pottery and jewelry [1,5], the first major industrial application of cobalt metal did not occur until 1927 [6]. At that time it was discovered that tungsten carbide, often in combination with titanium carbide and other metals, could be heated in the presence of cobalt used as a binder. This process resulted in a new substance referred to as cemented tungsten carbide or hard metal.

The current OSHA standard for occupational exposure to cobalt is based on the Threshold Limit Value (TLV) of the American Conference for Governmental Industrial Hygienists (ACGIH) in effect in 1968 [7]. In 1975, the ACGIH Committee on Threshold Limit Values proposed lowering the TLV for cobalt metal fume and dust to 0.01 mg cobalt/cu m [8]. This proposed value was subsequently amended to 0.05 mg cobalt/cu m in 1976 [9], and it remained as an intended change in 1980 [10].

Data projected for the first quarter of 1980 from the National Occupational Hazard Survey, conducted by NIOSH in 1972-74, indicate widespread potential for exposure to cobalt. About 867,000 workers were estimated to be exposed to cobalt oxides, and 235,000 to cobalt metal. At least 300,000 were estimated to be exposed to what was identified only as cobalt drier, and 79,000 possibly exposed to one drier, cobalt naphthenate. Exposure estimates for various cobalt salts are much lower, ranging from 1,700 to 21,000 workers, but are still substantial. (Exact figures are given in Table IX-1.)

Much of the information on cobalt's effects on humans is not from the occupational setting. For example, at one time it was common to prescribe mixtures of cobalt and iron to anemic patients in order to stimulate red blood cell production. This practice led to the discovery that cobalt, in addition to producing polycythemia, could adversely affect the thyroid. Another example is the considerable research conducted on the heart effects of cobalt after an outbreak of acute cardiomyopathy in beer drinkers who had consumed cobalt as an additive in beer.

Almost all information on adverse effects in workers exposed to cobalt is from the hard metal industry. Pulmonary fibrosis was first described in hard metal workers in a 1940 report in Germany [11], which was soon followed by others. Such reports persist even though the current Federal standard (29 CFR

1910.1000) for cobalt of 0.1 mg/cu m was based, to a great extent, on prevention of hard metal disease.

Respiratory Effects

Exposure to a single substance in the occupational environment rarely occurs. For cobalt, almost all reported cases of respiratory effects in workers concerned mixed exposures in the cemented tungsten carbide industry. Other substances present were tungsten carbide, and sometimes other metal carbides or material from grinding wheels. Workers who manufacture alloys containing cobalt are potentially exposed to many other metals including nickel, chromium, iron, vanadium, and molybdenum. The effects of concomitant exposure to these materials is unknown.

(a) Hard Metal Disease

Numerous case reports [12-22], medical examinations [11,23-32], and industry-wide studies [33-38] demonstrate the presence of hard metal disease in the occupational environment (Table IX-2). Although this is by no means a recent discovery, pulmonary fibrosis remains a problem in the cemented carbide industry. A common pattern of the illness is described in these reports. First, the worker develops a cough, followed by labored breathing on exertion. The person may lose a substantial amount of weight and develop a progressive interstitial pulmonary fibrosis; in the final stages leading to death, cor pulmonale and cardiorespiratory collapse are usually experienced. Chest radiographs reveal increased linear striations and diffuse nodular opacities in the middle and lower zones of the lung [39]. The degree of abnormality evident in the radiographs becomes greater as the stage of the disease becomes more severe. In some cases, the disease has been reversible or has not progressed [13,23-28] if it was detected at an early stage and the worker received no further exposure to cobalt.

While chest radiographs can detect fibrotic lesions, diagnostic tools capable of measuring adverse effects at an earlier stage are clearly needed. Spirometric examination to detect restrictive ventilatory impairment is one possibility, but the results do not agree totally on the sensitivity of this test. Perhaps much of the conflicting evidence is the result of the less sophisticated equipment available to the earlier investigators. Miller et al [23], in 1953, observed that three tool grinders had reduced vital capacity, but all three also had reticulations clearly evident in chest radiographs. Barborik [33], in 1966, observed disturbances of pulmonary ventilation in 25 of 116 cemented carbide workers, but he also observed changes in the chest radiographs of 31 of 193 (not all received spirometric examinations). Turos et al [29], in 1969, actually found a greater number of abnormal radiographs than changes in vital capacity in a group of 62 cemented carbide workers. In contrast, a more recent study of 22 tool grinders by Lichtenstein et al [40] found no radiographic evidence of fibrotic lesions even though several workers had slight reductions in forced vital capacity.

A series of reports [41-43] described the results of lung function tests in 155 Swedish cemented carbide workers and 74 controls matched for sex, age, and smoking history. Tests conducted included spirometry and single breath nitrogen washout. Persons exposed at cobalt concentrations of 0.005-0.01 mg/cu m, persons dry polishing sintered material (average exposure of 0.01 mg/cu m), and persons wet polishing sintered material (average exposure of 0.008 mg/cu m) showed some changes in pulmonary function during the working week, but recovered over the weekend. Inspectors (average exposure of 0.002 mg/cu m) showed no changes. Persons who were heavily exposed (average cobalt concentration of 0.06 mg/cu m) showed changes throughout the week, which did not regress over the weekend or even over 1-month vacations. Smokers were more affected than nonsmokers. The authors interpreted the changes in pulmonary function tests as evidence of airway obstruction. They noted that they could not exclude the possibility of chronic reduction of lung function in the most exposed group.

Although diffuse interstitial lung disease has clearly been demonstrated as an occupational disease related to exposure in the hard metal industry, the dust concentrations that cause an effect and the role of cobalt must be considered. Exposure estimates were given in some cases, but the duration of exposure was unclear for persons who developed fibrosis. In some instances, fibrosis was reported after only a few years of exposure [11,13,29]; in others, some persons had been exposed more than 20 years [26,30,34]. Recent investigations [30,40] have shown localized clouds of dust in the breathing zones of grinding machine operators, a factor that may have been missed in earlier reports. All of these factors could account for discrepancies in the observed levels of airborne cobalt at which fibrosis has occurred. Nevertheless, some correlation between cobalt exposure and adverse effects in hard metal workers can be made. Because mixed exposures to cobalt and other substances occurred, the role of cobalt in producing the effects can be established only when supporting evidence in experimental animals becomes available. Such information exists now only for cobalt metal [44] and cobaltous oxide [45].

Exposures in the hard metal industry where the cobalt concentration in the air averaged several milligrams were clearly intolerable to workers. For example, Kaplun's study [35], reported in 1957, described pulmonary changes in 8 of 247 workers; exposure levels measured in the plant were 0.8-12 mg of cobalt/cu m of air. The workers complained of nausea, abdominal pains, loss of appetite, cough, and a deterioration of the sense of smell. Some also had decreased hemoglobin (Hb) levels and red blood cell (RBC) counts, liver and spleen enlargement, and dermatitis. After dust control measures were implemented, the levels of airborne cobalt decreased to 0.4-3.3 mg/cu m. Later, 117 workers were examined, and 35 (30%) had chronic bronchitis, 33 (29%) had early fibrotic changes evident in chest radiographs, and 52 (44%) had decreased blood pressure. Reasons for the increased incidence of pulmonary changes at the lower exposure level were not given. Any number of factors could have been involved, including the slowly progressive nature of fibrosis in an aging workforce.

Barborik [33] described an investigation of 193 hard metal workers (104 men and 89 women) who had been employed for 1-13 years. Dust concentrations in the plant were 13-100 mg/cu m, and the dust contained 5-25% metallic cobalt. Of the 193 workers, 47% suffered from cough; numerous workers in the preparatory area and the forming area also complained of labored breathing, a burning sensation in the throat, and loss of the sense of smell. In 31 workers, distinct changes were observed in radiographic films of the lungs, and 25 of the 116 workers examined showed moderately severe to severe disturbances of pulmonary ventilation.

Salikhodzhayev and Vengerskaya [36] reported that 73 of 178 hard metal workers (41%) had chronic rhinitis and rhinopharyngitis. Cobalt concentrations in the air ranged from 0.4 mg/cu m where the distillers were unloaded to 2.9 mg/cu m at the sifting site. In a subsequent report [46] that appears to be on the same workers, 50% of those examined reported toxic signs or symptoms. The most frequent worker complaints included labored breathing, coughing, pounding of the heart, headache, dizziness, nausea, loss of appetite, and olefactory disorders. Medical tests revealed evidence of altered kidney and liver function.

In several studies, airborne dust measurements were made, but the information is inadequate to provide a meaningful relationship between cobalt exposure and fibrosis. For example, Moschinski et al [34] examined 282 male and 23 female workers at four plants, 56 of whom had radiographic evidence of fibrosis, but found no relationship between duration of exposure and onset of symptoms. A similar difficulty exists in analysis of the report of Reber and Burckhardt [13]. Nine workers showed signs of pneumoconiosis, but one of two exposure estimates varied from 0.06 to 0.3 mg cobalt/cu m. Tolot et al [24] and Dorsit et al [25] observed 3 cases of fibrosis and 26 cases of lesser pulmonary involvement in workers employed in a plant that manufactured alloys of sintered metals. Since dust concentrations were 2.3-10.6 mg/cu m and contained 0.01-0.3% cobalt, this might demonstrate a fibrotic response at very low concentrations of cobalt. However, the major components of the dust were not identified. Jirkova [30], in 1971, described an examination of 61 grinders employed in the production of sintered carbides. Concentrations of airborne cobalt near grinding machines with cooling or dust-collecting equipment averaged 0.043 mg/cu m (range, 0.006-0.09). Near grinding machines without safeguards, the concentration was 1.2 mg/cu m (range, 0.84-1.3). Unfortunately, the report did not state whether all 6 workers with fibrosis or the 23 with lesser signs were exposed at the higher dust levels and whether all of the workers used both types of equipment.

Few reports described toxic effects in hard metal workers at relatively low and constant concentrations of cobalt. Fairhall et al [37] found little evidence of fibrosis in a group of 1,802 workers, some of whom were probably exposed to cobalt at measured levels of 0.05-0.14 mg/cu m. Because this cross-sectional industry survey was reported only 20 years after the process was first developed, many persons in the group had probably been exposed to cobalt for only a short time. Miller et al [23], also in an older study, described three cases of fibrosis in tool grinders. The concentrations of cobalt in the breathing zone of grinding machine operators were 0.1-0.2

mg/cu m; tungsten was not found in general air samples. These three workers had been exposed for 6-8.5 years, and had used silicon carbide and aluminum oxide wheels, although a diamond wheel was used for most grinding. In 1975, Lichtenstein et al [40] found some cases of early signs of restrictive ventilatory impairment, but no radiographic evidence of fibrosis, in 22 grinding machine operators using diamond wheels and exposed to cemented carbide dust for 1-30 years. The mean TWA concentration of cobalt in the breathing zones of seven individuals was 0.18 mg/cu m (range, 0.03-0.43); four concentrations exceeded 0.1 mg/cu m. The latter two studies [23,40] support a tentative conclusion that hard metal workers can develop fibrosis or prefibrotic changes when exposure to cobalt is in the range of 0.1-0.2 mg/cu m. The Swedish studies [41-43] suggest possible chronic lung obstruction at average concentrations of 0.06 mg/cu m.

(b) Other Respiratory Diseases in Hard Metal Workers

Several reports have also described evidence of bronchitis [11,13,29,34], emphysema [13,14,16,30], or asthma [13,14,24-26,47] in workers exposed to cobalt in the hard metal industry. Bronchitis was said to be more prevalent in workers exposed to what were described only as large dust particles; pneumoconiosis was said to have occurred more often after exposure to dusts of smaller size [11].

The asthmatic responses seem to represent a true sensitization to cobalt. These persons developed a hacking [26] or wheezy [14,26] cough within 1 month [26] to 1.7 years [24,25] of initial exposure to cobalt. One person also had eczema [13]. Recovery occurred when the workers left the work environment [13,14,24-26,47]. One worker remained on his job as a tool grinder, and he developed nodules visible on chest radiographs 2.5 years later [26]. The others had no signs of fibrosis.

A recent study at a Swedish hard metal plant attributed four cases of allergic alveolitis to exposure to soluble cobalt dissolved in the coolant used for wet grinding [48]. One worker showed only signs and symptoms of asthma, but the other three showed evidence of mild pulmonary fibrosis as well. All four had contact eczema and were sensitive to cobalt. Even though they ceased all exposure to cobalt, the four persons have continued to show abnormalities on their chest radiographs.

(c) Other Reports of Respiratory Disease in Workers Exposed to Cobalt

Studies of the effects of cobalt exposure on workers not employed in the manufacture or use of hard metal are rare, but some indicate pulmonary disease in these workers. At a Finnish metal refinery, one or two new cases of bronchial asthma had been diagnosed annually in a group of 230-240 cobalt workers [49]. A cross-sectional study demonstrated a highly significant correlation between exposure to cobalt sulfate and asthma. In contrast, nonasthmatic cobalt workers at the same plant had no evidence of any excess of chronic bronchitis. Kochetkova [50] described the death of a woman with a history of 7 years of exposure to metallic cobalt dust. The cause of death was cardiopulmonary insufficiency resulting from massive fibrosis. Levels of

cobalt in the lung, liver, heart, and kidney tissue were described as markedly elevated. Her heart was enlarged, the liver and spleen were congested, and the cortical layer of the kidneys was swollen. In contrast, Cau et al [51] did not find radiographic evidence of fibrotic damage to the lungs of seven workers employed as sifters of cobalt powder. The workers had been exposed from 2 to 5 years, and they complained of cough, exertional dyspnea, nasopharyngeal irritation, and digestive disorders; two subjects had polycythemia. The authors concluded that exposure to finely divided cobalt metal dust did not induce pulmonary damage. In light of the toxic signs described by the workers and the short duration of exposure, this conclusion seems inappropriate.

An extensive study of workers exposed to cobalt was reported by Verhamme [52] in 1973. Medical examinations had been given to workers at a plant where cobalt oxides, powders, and salts were produced. Measurements of airborne cobalt were not reported, but the author believed that workers in the hydrometallurgic area had little or no dust exposure and that workers in the salts, oxides, and finished powders production areas were the most exposed groups. The finished powders had an average diameter of less than 1.4 μm, and oxides averaged 3 μm in diameter. Radiographic examination of 120 workers revealed no changes in 112 persons, accentuated pulmonary reticulations in 1 worker, fibrotic changes in 4 individuals known to have had tuberculosis, and evidence of pneumoconiosis in 3 workers with extensive coal mining experience. Several workers employed 15-20 years in the production of cobalt oxides showed symptoms of chronic bronchitis that improved or disappeared with job transfer. The plant had assigned workers to areas on the basis of their medical histories.

(d) Evidence in Animals

Several investigative groups have administered cobalt intratracheally in animals and reported adverse effects on the lung. Harding [53] found marked edema and hemorrhages in the lungs of rats shortly after they were administered large doses of cobalt metal. Guinea pigs given 10 or 25 mg of cobalt metal developed acute pneumonitis [54]. The lungs of guinea pigs that survived a 50-mg injection of cobalt metal had perivascular diffuse cellular infiltration and many eosinophils within the alveoli [55]. Regional obliterative bronchiolitis, with peribronchiolar fibrosis and arteriolar spasm, was also present. After 12 months, there was fibrocellular infiltration and there were regions of adenomatosis where cobalt metal was deposited. Those that survived a 25-mg dose had similar but less intense evidence of damage to the lungs, and a 10-mg dose produced no long-term tissue damage. The effects in guinea pigs [55] were consistent with those described by Schiller [56], who observed that as little as 1 mg of cobalt produced intra-alveolar pulmonary edema and inflammation of the bronchioles of rats after 6 hours; no tissue damage was observed 3 months later. These studies clearly demonstrate adverse reactions of both a short- and long-term nature.

Although tungsten carbide was nontoxic when administered intratracheally to animals, different results occurred when this substance was mixed with cobalt. Delahant [54] noted that tungsten metal dust, tungsten carbide, and a

91:9 mixture of tungsten carbide and cobalt administered in 150-mg doses produced no deaths in guinea pigs. Elaborating on this study, Schepers [57] reported that the lungs of guinea pigs administered the 91:9 mixture contained focal accumulation of massed particles in the alveolar spaces surrounded by fibrous tissue. This study [57] supports the previous one [55] suggesting a fibrogenic potential for cobalt, but it does not clarify whether the reaction is intensified by the presence of tungsten carbide.

Studies that have examined subchronic or chronic effects of inhalation of cobalt compounds are limited. In one such experiment, Kerfoot et al [44] exposed miniature swine to aerosols of cobalt metal. Two experimental groups, exposed to cobalt at 0.1 mg/cu m or 1 mg/cu m, and one control group of five animals were used. To investigate the suggestion of Bruckner [58] that pulmonary reactions to cobalt may be mediated through a hypersensitivity mechanism, Kerfoot et al first exposed the experimental animals 6 hours a day for 5 days. After a 10-day lapse, the animals were reexposed 6 hours daily, 5 days a week for 3 months. All animals appeared normal until the 4th week of exposure, after which time the animals from both cobalt groups became lethargic. Some animals appeared to be wheezing during exposure; this was confirmed by auscultation.

Postexposure pulmonary function tests showed a significant reduction in mean tidal volume, mean total compliance, and mean specific compliance in both cobalt-exposed groups [44]. The authors interpreted change in compliance as demonstrating functional lung impairment. This change was reversed in animals examined 2 months after the cobalt exposure ended.

Serial radiographs conducted on all animals showed no evidence of diffuse pulmonary disease [44]. The animals were examined grossly and by light microscopy at necropsy, and the lungs, heart, liver, and kidneys did not show any persistent or significant abnormalities. There was no evidence of cobalt in any lung tissue examined. Electron microscopic examination of lung biopsy tissue taken at the end of exposure revealed masses of collagen, elastic tissue, and fibroblasts in some of the tissues. However, no quantitative analysis was presented, nor was there mention of the methodology used in selecting tissue sites for examinations. The greatest changes were observed in the high-concentration cobalt exposure group, with the fewest changes being observed in controls.

In a Soviet study, male albino rats received exposures to aerosols of metallic cobalt continuously (24 hours a day, 7 days a week) for 3 months [59,60]. There apparently was a concurrent control group, but the reports did not contain important details such as the number of animals exposed or tables of results presenting actual data. Chronic inhalation of cobalt at 0.5 mg/cu m was said to be irritating to the lungs [59]. The animals exposed to cobalt at 0.05 and 0.005 mg/cu m were described as having changes similar to, but less pronounced than, the changes in animals exposed at 0.5 mg/cu m. The lowest concentration (0.001 mg/cu m) produced what was described only as a weak general toxic effect.

Microscopic examination of lung tissue from animals exposed at the highest concentration of 0.5 mg/cu m revealed accumulations of macrophages in the alveoli [60]. Interalveolar septa were consolidated in several areas of the lung. The severity of these changes reportedly was related to the exposure concentration. The lungs of animals exposed at 0.001 mg/cu m were described as similar to those of controls. While information in this report is not completely satisfactory, effects such as consolidation of the alveolar septa are indicative of a prefibrotic condition.

Wehner et al [45] exposed 2-month-old Syrian golden hamsters to cobaltous oxide aerosols at 10 mg/cu m for their lifespans. The animals were exposed for 7 hours daily for 5 days a week to aerosols with a median diameter of 0.45 μm. Particulate material accumulated in alveolar macrophages and became denser as exposure continued. Emphysema became apparent. The extent of the changes became more severe as exposure time increased, and hyperplasia and hypertrophy of alveolar lining cells also appeared. Macrophages, frequently containing particulate matter, increased in number throughout the lung, and numerous focal accumulations of these cells could be found. Proliferative changes involving the epithelial components of bronchi and bronchioles became detectable early during exposure and increased in severity with exposure time. Laryngeal lesions occurred in five animals (10%) during the study. The lifetime exposure of hamsters to cobaltous oxide clearly resulted in pneumoconiosis, but it did not significantly shorten their lives.

From the experimental studies in animals, the ability of some cobalt compounds to cause pulmonary fibrosis is evident. However, the information is insufficient to determine what effect, if any, exposure to other substances often present in cemented tungsten carbide may have on the development of hard metal disease. Thus, the possibility must be considered that adverse effects in workers exposed to cobalt at the 0.06-0.2 mg/cu m range were aggravated by exposure to mixtures. However, the degree of worker exposure to cobalt is clearly related to the number of signs and symptoms observed in workers. Thus, decreasing exposure to cobalt should reduce the risk of adverse health effects. For cobalt metal, results in animals indicate that worker exposure should be limited as much as possible, and that in no case should the present Federal limit be exceeded. Similar information is not available for any other cobalt compound, including those in commercial use.

Effects on the Skin

The incidence of sensitivity to cobalt in the general population appears to be low; in a small group (41 persons), none showed a positive response [61], whereas 0.8% and 1.8% did in two larger studies [62,63]. In persons with dermatosis, the observed incidence of sensitivity to cobalt, although somewhat dependent on experimental design, has been about 5-10% [62,64-66]. Intradermal testing results were more easily correlated to occupation than the results from patch tests [67]. Some cobalt-sensitive persons react to extremely low concentrations; in one study, 14 of 60 responded to topical application of 0.16% cobaltous chloride [68]. In another group, several of the 18 cobalt-sensitive individuals tested responded to 0.000001% cobaltous

chloride given intradermally [69]. Of 379 persons with hand dermatitis, 17 who had not been cobalt-sensitive responded to cobaltous chloride when given a second patch test 6-21 months later [65]. These results suggest that the patch test itself induced sensitivity to cobalt in some cases. A single report [70] demonstrated that most persons who are sensitive to cobalt sulfate respond to cobaltic trisethylenediamine chloride, chloropentamine cobaltic chloride, and sodium cobaltinitrite as well. Thus, sensitization to cobalt, regardless of the compound involved, seems probable.

A number of studies [67,70-80] have considered sensitivity to nickel and chromium as well as to cobalt in persons with dermatitis or eczema. The populations studied ranged from 9 [74] to 5,416 persons [75]. The percentage showing a positive response to cobalt ranged from 1.6 [67] to 100 [76]. Cobalt-sensitive individuals showed an incidence of positive responses to nickel, or chromium, or both substances that ranged from 18.2% [77] to 100% [74,76,78-80]. When all available data are combined, approximately 5% of the persons studied were cobalt-sensitive; 82% of these had combined metal sensitivities. All three metals are prevalent in the general environment, which suggests that individuals with cobalt sensitivity also have had contact with nickel and chromium. Their responses thus could represent independent sensitization reactions, so that cross-sensitivity is not necessarily implied.

Very few reports on skin diseases in workers who have contacted cobalt have been published. Skog [81] evaluated the reaction of 14 hard metal workers with eczema to patch tests with a 2% cobaltous chloride solution. Only three were sensitive to cobalt. In the other cases, eczema was believed to be due to the irritant effect of hard metal dust. In another study [29], 6 of 68 hard metal workers had dermatitis or eczema at the time of a medical examination. Four persons, including one with no skin disorders, responded to topical applications of 2% cobaltous chloride. Two remained sensitive to cobalt when tested 3 years later even though they had transferred to work environments free of hard metal dust. Among 436 pottery workers, 12 responded to the topical application of a 5% solution of cobaltous nitrate [82]. In workers with dermatitis, only 1 of 18 (5.6%) who had this condition for a month or less was sensitive to cobalt, compared with 9 of 28 (32.1%) workers with dermatitis for more than a month. In a survey of 1,004 persons with eczema from occupational and nonoccupational causes, 41 of 246 (16.7%) building trades workers with cement eczema were sensitive to a 5% solution of cobaltous chloride [83]. Twenty-six of 293 (8.9%) other individuals with occupationally-related eczema were sensitive to cobalt, as opposed to 77 of 296 (26%) with nonoccupational dermatoses. Considerably more information is needed before a realistic estimate can be made of the extent to which cobalt-induced dermatosis affects the workforce. Nevertheless, sensitization to cobalt does occur, and persons once sensitized would have difficulty continuing to work with cobalt. It is probable that many of these persons are transferred to other jobs.

The mechanism of the allergic response to cobalt after dermal contact is unknown. Haxthausen [84] contended that about 90% of the cobalt is absorbed from the skin following intradermal administration before any allergic reaction becomes visible. Norgaard [85], however, reported that cobalt is not

absorbed through intact skin when applied to the forearm as a 0.3% or 5% solution. This same study reported systemic absorption of cobalt in solutions applied to the depilated skin of rabbits and guinea pigs [85]. Another investigative group also observed systemic absorption when solutions of cobaltous chloride were placed on the clipped backs of guinea pigs [86]. From this information, NIOSH concludes that the risk of systemic poisoning from dermal absorption of soluble salts of cobalt is minimal for skin contact under normal working conditions. Abraded skin should be adequately protected to prevent contamination with cobalt compounds so that systemic absorption will not occur.

No information is available to judge the effects of many cobalt compounds. Some organocobalt complexes are uncharged and relatively apolar, which would indicate that they probably can be absorbed dermally at least as effectively as the inorganic salts. At least some of these complexes may be stable compounds in body fluids, so that their toxic effects could differ from the simple salts.

Cardiac Effects

Some cobaltous salts have been implicated as a causative agent in certain forms of cardiac disease. Cobalt, in the form of cobaltous sulfate or cobaltous chloride, was used as a foam stabilizer in beer during the mid-1960's in various countries, including the United States. Between 1964 and 1966, American breweries reportedly added 1-1.5 ppm of cobaltous chloride to 20-25% of all beer sold in the United States [87]. At the same time, several epidemics of a peculiar form of cardiomyopathy occurred among heavy beer drinkers in Quebec, Belgium, Omaha, Minneapolis, and New York [87-92]. All patients were heavy beer drinkers. For example, the Quebec group consumed daily from 2 to more than 6 liters of beer containing 0.8 to more than 1.6 mg cobalt/liter [93-95]. The signs and symptoms of illness in the beer-drinking patients included gastrointestinal problems, labored breathing, abdominal pain, cyanosis, lowered blood pressure, heart enlargement, pericardial effusion, rapid heart rate, and electrocardiographic (ECG) abnormalities. In one group, azotemia or oliguria, or both, were frequently noted, especially in fatal cases [90]. Sullivan et al [96] found cobalt concentrations 10 times normal in heart tissue of several persons who had died from beer drinkers' cardiomyopathy. In 20 who survived, 11 had ECG abnormalities persisting for up to 24 months [97].

The extensive literature on beer drinkers' cardiomyopathy suggests that factors in addition to cobalt may have contributed to the effects seen. Several authors suggested that cobalt may have increased existing cardiac impairment induced by thiamine deficiency [90,93] or excessive ethanol consumption [87,90]. Nutritional deficiencies other than lack of thiamine also may have contributed to the cardiomyopathy. The diet of a group of Belgian beer drinking patients was nutritionally inadequate, especially in regard to protein [91]. In contrast, chronic beer drinkers who did not develop marked cardiac abnormalities had nutritionally adequate diets. They

were brewery workers who had consumed a daily average of 16 glasses of beer containing 1.2 ppm cobalt.

The effects of cobalt on cardiac function have been examined in controlled experiments in humans. Garello et al [98] hypothesized that cobalt exerts a direct effect on the biochemical phenomena responsible for cardiac contraction. This hypothesis was based on the observation that a daily intramuscular (im) injection of 20 mg cobalt benzenesulfonate (equivalent to 3.2 mg cobalt) for 5 consecutive days resulted in slight increases in heart rate, atrioventricular conduction time, and duration of systole. Similar ECG changes had been reported [99] after im injection of mixtures containing 20 mg cobaltous chloride and 10 mg cobaltous benzenesulfonate. These experiments, of short duration, in humans do not account for the severe effects observed in the beer drinkers.

Animal experiments, often conducted for longer periods, present a different picture. In 20 male guinea pigs fed cobaltous sulfate for 5 weeks at high levels (stated to be 20 mg cobalt/kg daily), heart weights and ratios of heart weight to body weight were increased, and pericardial effusion was observed in 9 (45%) animals; myocardial degenerative changes were present in 15 (75%) [100]. Swigart [101] also found a significant increase in mean heart weights as well as right ventricular hypertrophy in rats administered 2.5 mg cobalt/kg (as cobaltous chloride) intraperitoneally (ip) for 63 days. Although microscopic changes were not reported in Swigart's experiment, Lin and Duffy [102] found cardiac lesions in rats following ip injection of 5 mg/kg cobalt (as cobaltous nitrate) daily for up to 7 days. The lesions resembled those observed in cardiomyopathy due to alcoholism, thiamine deficiency, and chemical toxins, and similar observations in rats [103] and in rabbits [104] were made by other investigators.

Kucharin and Sinitsin [105], in 1976, reported on a condition characterized as allergic myocarditis in five workers employed in the cobalt shop of a foundry for 2-19 years. Cardiac enlargement was the principal clinical finding. All five persons had abnormalities in ECG patterns that improved or disappeared in 1-2 years. At the height of their illness, heart volume was increased 51-110%. Blood counts reported for one subject indicated elevated Hb and RBC levels. The authors considered the disease to be of occupational origin and attributable to cobalt. However, data to support this conclusion were not provided in the report.

Alexandersson and Atterhog [106] examined dry and wet grinders exposed to cobalt at 0.01 mg/cu m and powder handlers exposed at 0.06 mg/cu m. No ECG changes were observed in dry grinders or powder handlers, even though the powder handlers showed evidence of impaired pulmonary function. The ECG's of wet grinders were abnormal and showed a high incidence of ectopic (premature) heartbeats. However, the authors believed that the ECG changes were related to exposure to cutting oils rather than cobalt.

Only one study in animals has examined the effects on the heart of cobalt exposure similar to that encountered in the workplace. In the study on miniature swine exposed to cobalt metal dust at 0.1 or 1 mg/cu m, Kerfoot and

coworkers [44] observed abnormalities in ECG's taken at the end of the 3-month exposure period. These alterations were interpreted by the authors to indicate a decrease in the strength of ventricular contraction and repolarization abnormalities.

Sufficient evidence exists to suggest that both cobalt metal and cobalt salts can produce cardiac changes, but apparently only at high doses. It should be noted that the amount of cobalt ingested daily by persons who consumed 6 liters a day of beer was about 5-10 mg, much higher than the amount inhaled by a worker breathing 10-15 cu m of air in a day at the current Federal limit of 0.1 mg/cu m. The question of whether changes such as those seen in the miniature swine could result in significant problems over a long period of exposure in especially susceptible individuals, such as those with existing heart disease and, possibly, persons who consume alcohol on a regular basis, needs to be examined.

Effects on Blood

Cobalt-induced polycythemia is a well known effect. This property of cobalt has been exploited therapeutically in the treatment of refractory anemia [107-115]. The information collected for this review, however, revealed that other lesser known effects may occur at concentrations below those needed to produce polycythemia. The information available, although often incomplete, is presented below.

The polycythemic effects of cobalt have been observed not only in patients but also in controlled experiments in persons with normal blood counts and in animals. Volunteers receiving oral doses of cobaltous chloride ranging from 100 to 1,200 mg daily for 1-12 weeks [107] or 120-150 mg daily for 1-3 weeks [116] experienced an increase in RBC and Hb levels. Numerous studies [117-125] in animals administered cobaltous chloride at high dosage levels for as long as 16 weeks either orally, or by subcutaneous (sc) or ip injection, also reported slight to substantial increases in RBC or Hb. One study in mice demonstrated an increase in RBC's subsequent to ip injection of cobaltous nitrate [126]. However, inhalation studies in animals exposed to cobalt metal [44,59] provide little evidence to support the contention that polycythemia would be expected in workers exposed to cobalt at low concentrations. Similar studies on cobalt salts have not been conducted. Since the effects of cobalt on the blood would be expected to depend on its ability to concentrate at certain critical sites, cobalt salts might demonstrate effects at levels differing from cobalt metal. However, the mechanisms responsible for the changes in blood that are induced by cobalt are not known.

While a polycythemic effect is clearly demonstrated in the reports described above, cobalt may also interfere with iron absorption from the gastrointestinal tract, indirectly affecting Hb formation. Rats administered 0.3-3 mg of iron by stomach tube had a significant decrease in iron absorption with the addition of 3 mg cobalt as the chloride salt [127,128]; this finding was confirmed in a subsequent study [129].

Several surveys have examined the possibility that polycythemia might result from industrial exposure to cobalt. Fairhall and coworkers [37] found no evidence of polycythemia in a survey of 1,802 cemented carbide workers. Cobalt exposures ranged from 0.05 to 0.14 mg/cu m. Verhamme [52] also found no evidence of polycythemia in 83 workers at a plant producing cobalt oxides, powder, and salts. However, Barborik [130] and Kaplun [35] independently found evidence of polycythemia in hard metal workers. Kaplun reported that RBC's were increased in workers described as having many years of service; the levels of airborne cobalt were 0.4-3.3 mg/cu m. Barborik found a higher mean Hb level in 88 male and 70 female workers than in a control group of 149 men and 70 women. RBC's, measured in 41 men and 34 women, were also elevated. Sixteen workers had Hb levels at least 19% above those found in controls, and seven had RBC increases in excess of 12.5%. All but two workers remained at their jobs, and a subsequent survey failed to duplicate these results. In contrast to the well known polycythemic effect, Sozieva [131] found a reduced Hb level in 22.8% of a group of hard metal workers exposed to cobalt. Hypotension was also said to be present in most of the exposed workers. Dust levels ranged from less than 10.6 to 62.6 mg/cu m, but the cobalt content was not reported. Kaplun [35] also found decreased Hb levels and RBC's in 247 hard metal workers exposed to cobalt at 0.8 to 12 mg/cu m. Although limited, this information suggests complex and possibly conflicting mechanisms of action of cobalt on blood components.

Even though experiments in animals suggest the possibility that cobalt could affect the white blood cell (WBC) count, this effect has apparently not been examined in workers. Miniature swine exposed to cobalt metal dust showed an increase in WBC's during the 3rd week of exposure [44]. Other investigators have examined the effect of cobaltous chloride [122,132-136] or cobaltous nitrate [126] on the WBC's of animals. Some reported a slight to significant increase [122,133-136], one reported a decrease [132], and one reported no significant change [126]. Since WBC's would be affected by infection, NIOSH concludes that this limited information is insufficient to demonstrate a causal relationship with exposure to cobalt.

Effects on blood components other than the cells may occur at exposure levels not producing polycythemia. A number of animal studies demonstrated certain nonspecific changes in blood protein levels. Significant increases in total protein and globulin levels were found in rabbits receiving im injections of cobaltous chloride [135]. Similar changes in serum globulin levels, but not in total protein, were found in a second study [137]. Cobaltous chloride administered to dogs by sc injection and to rabbits by ip injection resulted in significant increases in alpha-globulin levels even though RBC and Hb levels were unaffected [138]. Similar results were obtained when rabbits were exposed to aerosols of cobalt metal. Miniature swine exposed to cobalt metal dust at 0.1 or 1 mg/cu m had slight increases in alpha-globulin levels [44]. In humans, the mean erythropoietin level was 58% above that of controls in 21 persons employed in the hydrometallurgical production of cobalt even though no changes in RBC, hematocrit, or Hb levels were found [139]. Concentrations of airborne cobalt ranged from 0.43 to 1.6 mg/cu m. Increased serum globulin levels cannot be related readily to any specific disease state, but the increases in erythropoietin, a substance that

migrates electrophoretically with the alpha-globulins, may be highly relevant to the polycythemic effects of cobalt. Erythropoietin is involved in the regulation of erythrocyte production.

A second effect that could be relevant to persons exposed to cobalt in the occupational environment is the apparent ability of cobalt to prolong the time needed for blood clot formation. Ternovi and Mosketi [140] reported significant changes in blood clotting time (35% increase), thromboplastic activity (28% decrease), and clot retraction time (20% increase) in 15 persons administered cobalt orally as a solution of cobaltous chloride at 1 mg a day for 3 days. These findings seem to be supported by results of animal studies [133,136,141-143] and from patients administered cobaltous chloride for treatment of anemia. In the patients, infants receiving 120-200 mg cobaltous chloride for up to 100 days had increased leukocyte and thrombocyte counts [112], but these findings were not confirmed in those that received cobalt for 1-3 weeks [116].

The information available suggests to NIOSH that the effects of cobalt on the blood are multiple and poorly understood. Polycythemia is clearly one manifestation of exposure to cobalt metal or cobalt salts, and increases in erythropoietin could be a factor in producing this effect. The airborne concentrations of cobalt at which this polycythemic effect would be significant are poorly documented, but they appear to be well above the Federal standard for cobalt metal. Information is not adequate to judge whether this airborne concentration, 0.1 mg/cu m, would be adequate to prevent polycythemia from other cobalt compounds. Because RBC's have been found to decrease in exposed workers only at very high airborne concentrations of cobalt, this effect is judged insignificant if exposure is through inhalation. The role of cobalt in retarding blood clot formation is not well documented, but preliminary evidence suggests that it could be highly significant.

Effects on the Thyroid

The goitrogenic effects of cobaltous chloride were first noted in 1954 in individuals being treated for sickle cell anemia [144]. More detailed descriptions later reported that two children given approximately 3 mg/kg daily of cobalt preparation developed visible goiters [145,146]. One child also had signs of hypothyroidism. A third patient who developed a goiter also had a significant reduction in radioiodine uptake. Placebo tablets were substituted for the cobalt preparation, and thyroid function returned to normal by 12 weeks. Numerous additional reports that followed now show that 2-10 mg cobalt (as chloride)/kg given orally each day for 2-4 months will induce goiter formation in a small percentage of persons [147-154]. In all cases, the thyroid hyperplasia has been reduced or reversed after cobalt administration was stopped.

Controlled clinical studies have also examined the effects of cobalt on the thyroid gland. In one study, 12 adults with normal thyroids were given 50 mg of cobaltous chloride orally three times a day for 2 weeks [155]. Radioactive iodine uptake was reduced in all but one person after the 1st

week; by 2 weeks all uptake levels were near zero. The effect was reversible after cobalt administration ceased. In a separate study, the majority of individuals given cobaltous chloride either orally or by iv infusion also had decreased iodine uptake in the thyroid [156]. In fact, cobaltous chloride has been tested as a treatment for hyperthyroidism. Pimentel-Malaussena et al [157] observed that four of eight patients receiving 150-600 mg a day for up to 117 days responded with clinical improvement as manifested by reductions in tachycardia, metabolic rate, and uptake of radioactive iodine. Three of four, however, had thyroids of increased size, and the authors concluded that the action of cobalt was unpredictable.

The results of studies in guinea pigs [158] and rats [159,160] support the evidence in humans that cobaltous chloride can affect thyroid function and cause microscopically observable changes. In addition, a moderate reduction of iodine uptake was demonstrated in rats that received 20 mg/kg of cobaltous oxide or cobaltic oxide sc daily for 45 days [161]. These studies, like those in humans, were all conducted at high doses and by routes of administration not typically found in the workplace.

The most conclusive evidence that cobalt can affect the thyroid at low doses is the study of Popov et al [60]. In this experiment, rats exposed continuously to aerosols of cobalt metal were killed for examination after 1.5 or 3 months of exposure or after 3 months of recovery. The thyroid glands of animals exposed at the highest concentration, 0.5 mg/cu m, showed microscopic evidence of follicles containing foci of epithelial hyperplasia. Changes in thyroid function were noted in animals exposed at 0.05 mg/cu m but not at 0.005 and 0.001 mg/cu m. This continuous exposure at 0.05 mg/cu m would correspond to an 8-hour exposure of about 0.2 mg/cu m.

Cobalt metal, cobalt oxides, and cobalt salts have caused alterations of the thyroid glands of humans or animals. These effects represent both functional and morphologic alteration of the gland. The only experiment using low doses was conducted by Popov and coworkers [60], and it suggests that slight changes in thyroid function would be expected in workers exposed at 0.2 mg/cu m. This value is near the permissible exposure limit for cobalt metal fume and dust, and long-term effects at low levels have not been studied in humans for any cobalt compounds. This information suggests that alterations of thyroid function should be considered as a possible effect of workplace exposure to cobalt.

Carcinogenicity and Mutagenicity

Fibrosis, and not carcinogenicity, has received most of the attention of the occupational health community concerned with cobalt. The many reports available concerning fibrosis would not be adequate to demonstrate any carcinogenic effect, because none are epidemiologic studies concerned with incidence rates. The three case reports describing the development of benign or malignant tumors in hard metal workers [17,32,38] and two epidemiologic studies describing an increased risk of lung cancer in cobalt recovery areas of nickel refineries [162,163] are not useful in demonstrating a possible

carcinogenic effect of cobalt. Arsenic was also present in the air of some plants examined in the epidemiologic studies. Mixed exposures to substances known to be or suspected of carcinogenicity were involved in all cases. The case reports cannot distinguish between the role of occupational exposure and other factors, including normal incidence. Thus, information from human studies does not provide any answers about the possible carcinogenic potential of cobalt.

The two studies conducted in animals by routes of administration most applicable to workplace exposure did not demonstrate a carcinogenic effect for cobalt [45,164]. The incidence of tumor development was low and did not differ significantly from controls in male Syrian golden hamsters exposed for their life spans to aerosols of cobaltous oxide at 10 mg/cu m, 7 hours daily, 5 days a week [45]. Many of these animals did develop fibrotic changes. Hamsters injected intratracheally once a week for 30 weeks with 4 mg of cobaltous cobaltic oxide (Co_3O_4) failed to develop a statistically significant excess of tumors compared with control animals [164]. It should be noted, however, that the two tumors that developed in the 50 animals exposed to cobalt were alveolar in origin while the four tumors in controls were at sites unrelated to exposure. These studies provide little evidence to suggest that cobalt oxides are carcinogens, but no similar studies have been conducted in animals other than hamsters.

The results of studies of the carcinogenic potential of cobaltous chloride are conflicting. Gunn et al [165] reported that no tumors were observed for 10-16 months in Wistar rats given four simultaneous injections of cobaltous chloride (0.18 mg cobalt/site) into a vital organ (liver or kidney), a gland (salivary or ventral prostate), and two mesenchymal mesodermal structures (chest, interscapular area, thigh, or femur). Similarly, Shelley [166] reported that no tumors were observed in 10 ICR mice given cobaltous chloride (3.5 mg cobalt) in the dorsal earlobe, although the 2- to 5-month observation period was relatively short. On the other hand, Shabaan et al [167] found subcutaneous fibrosarcomas in 14 of 27 rats surviving 8-12 months following 10 injections of cobaltous chloride at 40 mg/kg into the central abdominal wall. Two tumors were at the injection site, and the remainder were at various distances from the abdominal region. Metastases were not found in any tumor-bearing animal. This study provides evidence that cobaltous chloride could be a carcinogen, and it argues for the need to perform chronic testing of cobalt salts in animals by routes of exposure more applicable to the workplace.

Several investigators have examined the carcinogenic potential of cobalt given parenterally. In one study, neither metallic cobalt nor cobalt sulfide induced tumors in rats when these substances were injected into the poles of the right kidney [168]. Heath and associates [169-174], in a series of papers from 1954 to 1972, examined the effects of cobalt metal powder or alloys injected into rats. Many animals developed injection site sarcomas 3-12 months after im administration of the powder in fowl serum. Sarcomas were also induced in cardiac muscle following the administration of cobalt metal powder by intrathoracic injection [175,176]. Gilman [177] and Gilman and Ruckerbauer [178] were also able to induce injection site sarcomas in rats following im injection of cobaltous oxide or cobaltous sulfide, but they did

not observe tumors in mice similarly injected with cobaltous oxide. In experiments with cobaltous naphthenate, five rabbits injected im developed rhabdomyomas at the injection site after 2-6 months [179]; similarly, one of three injected iv developed an osteochondroma at the site. A rabbit given cobaltous naphthenate by intrahepatic injection and another animal receiving the compound intrapleurally also showed tumor formation. In a similar study in mice, injection site tumors developed after what was apparently an im injection of cobaltous naphthenate [180].

Mutagenicity tests in Allium cepa cells [181], Vinca faba roots [182], bacteriophage T4 [183], and yeast [184,185] have indicated that cobalt interferes with mitosis and can cause cell death in sufficient concentrations. Four cobalt compounds ($CoCl_2$, $Co(OH)_3$, $CoSO_4$, and $2CoCO_3.3Co(OH)_2$) were weakly positive in rec assays, but cobalt chloride and cobalt hydroxide were negative in spot mutation induction tests with two strains of E coli and five strains of Salmonella [186]. Cobalt nitrate had no effect on the chromosomes of human leukocytes [187], but cobalt chloride has decreased the fidelity of DNA synthesis [188]. Cobalt salts have also enhanced transformation of hamster embryo cells exposed to simian adenovirus SAT [189]. These studies are insufficient to serve as any indicator of possible carcinogenicity.

Information on cobalt is inadequate to conclude that cobalt is a carcinogen. The information is also inadequate to conclude that cobalt is noncarcinogenic. In fact, limited data [167,175,176] provide suggestive evidence that at least some cobalt compounds may prove carcinogenic when subjected to long-term testing by currently accepted protocols. Until such testing is performed, no definitive guidelines can be given. Tumor induction at the injection site, however, would argue for the need to adequately clean any wound contaminated with cobalt.

Effects on Reproduction

Whether exposure to a substance in the workplace can result in adverse effects on reproductive capacity or harm to the developing fetus is of grave concern to workers and to all persons responsible for the health of workers. The information available on cobalt is conflicting and insufficient to draw definite conclusions on the effects of cobalt on reproduction. Although this information does not substantiate any effects, it is presented below.

Studies in humans [190-192] and animals [193,194] demonstrate that cobalt can cross the placenta. This effect does not appear related to the essentiality of vitamin B12 [193]. Whether or not cobalt compounds possess any teratogenic activity is not established. Only 1 of 41 hamster embryos exposed to cobaltous acetate through the iv injection of 5 mg/kg in the dam on the 8th day of gestation was resorbed [195]. No gross abnormalities were evident. This experiment, however, would only have detected teratogenic effects under very limited conditions because of the single exposure and the lack of any microscopic examination. The most frequent malformations observed in chick embryos exposed to cobaltous chloride through injection of 0.4 or 0.5 mg per egg were in the skeletal system and eyes [196]. No embryo exposed

through injection of 0.1-0.3 mg was malformed. The mortality of the embryos was high, 75.7% in those injected with cobaltous chloride and 54.3% in control embryos. This experiment is of questionable relevance because of the nonmammalian test system used.

The literature describing the effects of cobalt on testicular function is characterized by disagreement among investigators. Hoey [197] reported marked but reversible changes (including suppression of spermatogenesis, abnormal sperm, deformation of the epididymal tubules, and necrosis of the duct system) in rats administered cobaltous chloride by sc injection. Kamboj and Kar [198] injected cobaltous nitrate sc in mice or intratesticularly in rats, and they found no microscopic damage to the testes or effects on spermatozoa. Niebroj [199] injected cobaltous chloride ip, and considered the effects on the testes of these mice to be generally positive.

Other Effects

There are numerous studies of additional toxic effects attributed to cobalt. Some, such as the acute lethality studies in animals, have little impact on the occupational environment since such exposure conditions are rarely, if ever, encountered. Some effects, such as increased levels of plasma lipids, have been observed in humans, but only rarely in anemic patients receiving cobalt [145,150,200]. Some, such as experimental induction of epilepsy by implantation of cobalt in the brain of animals, appear irrelevant. Other effects, such as kidney and liver changes, may be relevant; but information on occupational exposure is quite limited.

(a) Kidney Effects and Hyperglycemia

Workers exposed for no more than 3 years to cobalt at 0.6-3.2 mg/cu m in a Soviet plant manufacturing tungsten bars and hard metal alloys showed evidence of disturbed kidney function and hyperglycemia [46]. About half of the 178 workers complained of some symptom, including labored breathing, coughing, pounding of the heart, headache, dizziness, nausea, loss of appetite, and olefactory disorders. Blood glucose, measured in 37 subjects, was elevated in 8 (22%). Glucose tolerance tests produced prolonged elevations of blood glucose in 8 of 14 (57%) individuals. Blood chloride levels were increased in press operators but decreased in reducers; both groups showed a reduced chloride concentration in the urine. In a separate study, a worker apparently acutely poisoned by exposure to cobalt acetate suffered from albuminuria [201]. A similar effect was observed in guinea pigs administered large doses of cobaltous chloride for 6-7 days [202]. Both the cortex and the glomeruli were damaged, and pronounced changes occurred in nearly all tubules of the kidney. This study [202] and another using cobalt oxides [161] were consistent with the limited information provided by Popov et al [60]. In this study [60], rats exposed to cobalt dust continuously at 0.5 mg/cu m for 3 months reportedly had slight degenerative changes in the convoluted tubules. Except for the statement that this effect was not seen at 0.001 mg/cu m, no additional information was provided.

Cobaltous chloride therapy for anemia has reportedly led to hyperglycemic reactions in patients [203]. This response is more clearly demonstrated in experiments with animals. Parenterally administered cobalt salts and trisethylenediamine cobaltic chloride have produced time- and dose-related rises in blood glucose levels (hyperglycemia). This effect was noted in rats [204-207], guinea pigs [208], rabbits [209], and dogs [210]. The typical increase in blood glucose level usually peaked after 1-2 hours and lasted up to 10 hours depending on the dose. The hyperglycemia induced by cobalt appeared to be independent of its effects in alpha cells of the pancreas (see section (c)), but the mechanism of action is not yet understood.

NIOSH considers the information available sufficient to conclude that cobalt metal and salts, and probably cobalt oxides and a number of organocobalt complexes, can damage the kidneys. These effects are thought to occur only at levels that also produce other toxic effects.

(b) Liver Effects

Hard metal workers exposed to cobalt at 0.6-3.2 mg/cu m had slight to moderate changes in liver function tests in 19 of 34 (56%) individuals examined [46]. Other workers, exposed at 0.8-12 mg/cu m, had enlarged livers [35], and a woman who died from massive fibrosis had liver congestion [50]. These reports would indicate that cobalt can affect the liver, either directly or indirectly. However, the lack of similar information at lower doses suggests that the liver is not the critical organ for cobalt toxicity.

Exposure to cobalt has produced liver damage in laboratory animals. Popov et al [60] noted that the hepatocytes of liver tissue of rats exposed continuously to cobalt dust at 0.5 mg/cu m had necrotic changes. This effect was reported to be dose-dependent, with no effect at 0.001 mg/cu m. At higher doses (10-18 mg/kg of cobaltous chloride in a single iv injection), rabbits examined for up to 7 days developed a transient but marked glycogen depletion, necrotic changes in a few liver cells, and moderate to marked fatty degeneration [209]. In a study on rabbits given sodium cobaltic nitrate or cobaltic oxide im at relatively high doses for up to 219 days, areas of focal necrosis and liver cell degeneration were observed [211]. This information, in experimental animals, supports the contention derived from human data that cobalt can adversely affect the liver but only at high doses.

(c) Pancreatic Effects

Parenteral administration of cobalt salts in high doses has induced degenerative changes in pancreatic tissue of experimental animals. The guinea pig pancreas appears to be very sensitive to the effects of cobalt, resulting in it often being used in research [212-223]. The characteristic effect noted after administration of cobalt salts at 10-80 mg/kg was alpha cell degranulation and necrosis. Beta cells were usually not affected. Changes reached a peak 24-48 hours after a single dose. Animals that survived regenerated pancreatic tissue. This phenomenon may not be occupationally relevant because of the high doses used. In addition, no human data are

available that indicate a potential problem with pancreatic function after cobalt exposure.

(d) Additional Studies

Cobalt solutions, unless they are strongly acidic or basic, probably do not pose a serious risk of damage to the eye from splashes or spills. Subconjunctival administration of 1 ml of a 0.1-0.2% cobaltous sulfate solution to three rabbits produced no irritation of the mucosa [224]. However, the greatest risk of damage to the eyes from cobalt is probably a physical effect caused by flying chips of cobalt or hard metal.

Kaplun [35] studied olfactory acuity in 37 workers employed in the manufacture of hard-metal alloys. Twenty-eight subjects worked with tungsten carbide and cobalt and tungsten, and the rest worked with tungsten carbide alone. The author concluded that 25 of 28 persons working with tungsten carbide and cobalt had impaired senses of smell when compared with individuals working with tungsten carbide alone. However, details of the experimental design were not provided.

Distribution and Retention

Since it is a constituent of vitamin B12, cobalt should be present in small amounts in the normal human body. In 1962, Yamagata et al [225] used neutron activation analysis (NAA) to calculate the whole body content of cobalt (1.1 mg in humans). Approximately 44% was stored in muscle, 32% in bone, and the remainder in soft tissue. In workers who died of pulmonary fibrosis, however, the few studies available present contradictory evidence of cobalt accumulation in the lung; most found little or no cobalt [12,15,19,22,26], but others found large amounts [16,50,226]. Two groups of investigators examined the urine of workers exposed to cobalt compounds, and found a substantial elevation in the amount of urinary cobalt [226,227].

Accidental inhalation of radioactive cobalt as the metal or the oxides has provided some data on the kinetics of cobalt elimination after entry into the lungs. Several reports [228-231] have shown that lung clearance occurs in at least three stages, a rapid component having a half-life of 0.5-2 days, an intermediate component of 3-42 days, and a long-term component of 60-120 days. The International Commission on Radiological Protection, in a 1959 report, estimated the biologic half-life of inhaled cobalt-60 to be 9.5 days [232], but investigators have since reported data indicating that some inhaled cobalt can be retained in the body for many years [233,234].

Controlled experiments on the distribution of cobalt in humans have considered only cobaltous chloride. When administered orally, most was eliminated in the feces [156]. Absorption through the gastrointestinal tract appeared to be reduced if cobalt was given after a meal, in an albumin complex, or as a carrier-free solution [235]. Cobaltous chloride, given iv, was rapidly cleared from the blood [236], but a substantial amount (5-16%) was retained in the body for a year or longer [236,237].

The results of animal studies confirm and supplement the reports in humans. Numerous studies in animals also demonstrate that most cobaltous chloride administered orally is eliminated in the feces [238-241]. When cobaltous chloride was administered repeatedly in the drinking water of rats [242] or mice [243] (an exposure more typical of general human consumption), cobalt accumulated to increasingly higher tissue levels until a steady state was reached after 30 days. Cobaltous oxide was poorly absorbed in hamsters; when administered by gavage, less than 0.5% accumulated in body tissues [244]. Cyanocobalamin, in contrast, showed a much greater absorption and retention than cobaltous chloride [245]. Several investigators [242,243] have noted the tendency of cobalt to accumulate in the liver following administration of cobaltous chloride, and two [238,242] also described its long-term accumulation in bone. Considering these sites of deposition, it is not at all surprising that a fraction of the administered dose is retained for a long time.

Retention of cobalt oxides after inhalation has been examined in animals. Wehner and Craig [244] reported that hamsters eliminated 90% of the total dose of cobaltous oxide, inhaled at 15.6 mg/cu m 7 hours a day for 2 days, within the subsequent 3 days (day 5 of experiment); however, the remainder was retained tenaciously in the body of the animal. Barnes et al [246] administered respirable-sized particles of cobalt oxides by inhalation to dogs. In 10 days, only 10% of the cobaltous oxide was retained, compared with 60% of the cobaltous cobaltic oxide. Cobaltous cobaltic oxide showed accumulation in the lymph nodes of the lung, a typical reaction of tissue-insoluble substances, while substantially more cobaltous oxide was found in the tissues and blood.

The information on distribution and retention of cobalt is typical of substances showing moderate to substantial tissue solubility. Information on the chloride salt is particularly revealing. It appears that enough of the salts can be absorbed through the gastrointestinal tract to warrant good sanitation and personal hygiene by workers so that they can avoid absorbing significant amounts of cobalt through ingestion. The long-term accumulation of cobalt in the liver may also be relevant.

Uses of Cobalt

Cobalt (atomic number 27, atomic weight 58.93) is a transition element located between iron and nickel in group VIII of the periodic table. The metallic form is silver gray to bluish white and is magnetic. There are three oxides of cobalt: cobaltous oxide, CoO; cobaltic oxide, Co_2O_3; and cobaltous cobaltic oxide, Co_3O_4. Ionic cobalt can exist in either a divalent or a trivalent form. The cobaltous ion can form numerous inorganic and organic salts [247], and cobalt can form complexes with amines, nitrites, and cyanides [248]. Divalent cobalt can have a coordination number of either 4 or 6, whereas trivalent cobalt has a coordination number of 6 [2]. The physical and chemical properties of some cobalt compounds are given in Table IX-3, and substances containing cobalt that are listed in the Toxic Substances Control Act Chemical Substance Inventory [249] are listed in Tables IX-4 to IX-6.

Cobalt is a byproduct or coproduct of the refining of other mined metals such as copper and nickel. Some of the commercially mined ores are carrollite, $CuCo_2S_4$; smaltite, $CoAs_2$; cobaltite, CoAsS; siegenite, $(Co, Ni)_3S_4$; and sphaerocobaltite, $CoCO_3$ [1,5]. The amount of cobalt mined is relatively small in comparison with copper or nickel, and the cobalt supply depends to a large extent on the demand for the latter two metals.

According to the Bureau of Mines (RA Markle, written communication, September 1978), there has been no domestic mine production of cobalt for several years, and there is only one refinery that processes cobalt in the United States. Most of the cobalt imported, nearly 77%, comes from Zaire and Zambia. Imports were up 6%, to 17.5 million pounds, in 1977. Cobalt consumption in the United States for 1977 was about 16.6 million pounds, up 0.6% from 1976. Much of that increase was due to demand in the aircraft industry.

About 70% of the cobalt used in the United States goes into the alloy and hard metal industries, mostly to produce magnetic and super-hard alloys [250]. The aluminum-nickel-iron-cobalt alloys are widely used in permanent magnets [39]. Vanadium-iron-cobalt alloys are used for ductile permanent telephone diaphragms. Vitallium, an alloy of cobalt, chromium, nickel, and molybdenum, has been used for prostheses in bone replacement surgery [39,250]. Cobalt is used extensively in the tungsten carbide or hard metal industry as a binder of ingredients in these heat- and wear-resistant metals [39,250-252]. Cobalt-bearing high-speed steel and cemented carbides have excellent cutting properties. Major uses include various chemical, cutting tool, and hard facing applications. Cobalt alloyed with chromium and nickel is used in heat-resistant gas turbine blades and jet engine parts. Hard metal is found in high-speed cutting tools, armaments, masonry drills and cutters, high-speed dental drills, and tire studs.

Cobalt compounds have important uses as catalysts [39,250,253-255]. Cobalt naphthenate, octanoate, oleate, resinate, tallate, and linoleate are used as drying agents for oil-based paints, varnishes, and printing inks. The cobalt compounds act as oxidizing catalysts for the polymerization of unsaturated glycerides. Cobalt is an important catalyst in oxidation and in desulfurization, a process used in refining crude oils. Cobalt acetate is used in the manufacture of dimethyl terephthalate and terephthalic acid. Cobalt catalysts are also used in the production of several primary alcohols and aldehydes.

Cobalt salts have a variety of uses [39,250,255]. Cobalt sulfate and chloride are used in electroplating. Cobalt carbonate, chloride, and sulfate are incorporated as additives in animal feeds. Cobalt chloride is a dessicant indicator, the hydrated form being pink and the anhydrous form blue. This property also leads to its somewhat unusual use as "invisible" ink. Cobalt pigments are very important in the ceramic and pottery industry. A wide variety of pigments in differing shades and colors contain cobalt, often as mixtures with other metal salts. Black cobalt oxide paint is of particular value because of its heat and wear resistance. A mixture of cobalt oxide and cobalt aluminate forms a blue color. Cobalt blue is sometimes added to glass to detint the yellowish color that occurs if iron is present. Small amounts of cobalt salts are also used as boiler water scavengers, in magnetic tapes, and in steel-belted tires.

Table III-1 lists some occupations in which there is potential exposure to cobalt compounds [256]. The amount of exposure to cobalt that workers actually receive in these occupations probably varies widely. Large amounts of cobalt are used in some industries, such as cobalt salts manufacturing, the cemented carbide industry, and the manufacture of cobalt-containing alloys. Many users of these products handle extremely small quantities of cobalt. Others perform cutting, grinding, or welding operations on products, and the potential for worker exposure to cobalt is significant. Exposure of painters to cobalt driers is probably minimal; exposure of spray painters to cobalt pigments is not.

Environmental Data

Cobalt is present in low concentrations in ambient air. Dams et al [257] found mean concentrations of 2.6 ng/cu m (1 mg = 10 exp +6 ng) in an urban area (East Chicago, Indiana) and 0.95 ng/cu m in a rural area (Niles, Michigan). Rancitelli et al [258] reported cobalt concentrations in North and South America ranging from 0.07 ng/cu m (Thule, Greenland) to 3.6 ng/cu m (Santiago, Chile). Hewitt [259] found a concentration of 5 ng/cu m in urban air in England. Also, Janssens et al [260] reported that air samples collected in Belgium, apparently near a cobalt-manufacturing plant, showed levels of 0.4-7.3 ng/cu m (mean, 2.8). All of these levels in ambient air are far below the Federal standard of 0.1 mg/cu m for cobalt metal fume and dust.

TABLE III-1

OCCUPATIONS WITH POTENTIAL EXPOSURE TO COBALT

Acetic acid makers	Gasoline blenders
Alloy makers	Glass colorers
Alnico magnet makers	Glaze workers
Ammonia mask makers	High-speed tool steel workers
Barometer makers	Hygrometer makers
Bright platers	Ink makers
Catalyst workers	Iron-cobalt platers
Cement makers	Lamp filament makers
Cemented carbide workers	Magnet steel workers
Ceramic workers	Metal smelters
Cermet makers	Nickel byproduct workers
Cobalt soapmakers	Paint drier makers
Cosmetic makers	Painters
Dye workers	Porcelain workers
Drug makers	Protective-coating makers
Electroplaters	Refractory brick makers
Enamelers	Rubber makers
Ethyl acrylate makers	Silicate paint makers
Fertilizer workers	Stone preserver makers
Frit workers	Weatherproof cement makers
Gas mask makers	Welders

Adapted from reference [256]

Much of the data on occupational exposure to cobalt have come from the tungsten carbide industry. McDermott [261], in 1971, found the average concentration of cobalt in 40 general air samples of seven tungsten carbide facilities to be below 0.1 mg/cu m (range, 0.005-0.15). About 70% of the 133 breathing zone samples were also less than 0.1 mg/cu m. Based on the data provided, however, mean concentrations in breathing zones of press operators and machine tool operators averaged 0.16 and 0.21 mg/cu m, respectively.

A tungsten carbide facility reported that during 1977-78 the concentration of cobalt in worker breathing zones averaged 0.048 mg/cu m in the powder area, 0.033 mg/cu m in the pressing area, 0.019 mg/cu m in grinding, and 0.025 for general maintenance [255]. The highest concentration measured was 0.17 mg/cu m and the lowest was 0.004 mg/cu m.

A study of eight cemented carbide plants in Sweden revealed that exposure to cobalt varies considerably with job classification [262]. Groups handling powder were most exposed, while press operators, shapers, dry grinders, wet grinders, face grinders, and inspection personnel received decreasing levels of exposure to cobalt (in that order). Some workers handling powder probably received short-term exposures in excess of 0.1 mg cobalt/cu m.

At an operation where tungsten carbide tools containing 6.0-8.5% cobalt were sharpened by wet grinding, general air samples were well below 0.1 mg/cu m [40]. Two-thirds of the breathing zone samples, however, were above 0.1 mg/cu m of cobalt (range, 0.04-0.93 mg/cu m). Full-shift TWA exposures were measured on a number of the grinders. Two-thirds of those values were above 0.1 mg/cu m with a range of 0.03-0.56 mg/cu m and a mean of 0.24 mg/cu m.

A US nickel refinery provided cobalt dust exposure data collected in June 1978 (B Roy, written communication, September 1978). Average concentrations of cobalt were generally low, ranging from less than 0.002 to 0.099 mg/cu m, except for two tower cleanup workers whose exposures averaged 0.156 mg/cu m. Within the occupational groups, the concentration ranges were sometimes quite variable; for example, the exposure of one cobalt operator was 0.313 mg/cu m, and for a supervisor it was 0.148 mg/cu m, even though averages for all 13 workers in these groups were substantially lower.

In a plant manufacturing cobalt salts, concentrations of cobalt were measured at work stations where the salts were handled manually [255]. Workers in dusty areas were required to wear respirators. Cobalt concentrations where fine powders were dried were low, ranging from 1 to 2 μg/cu m. Where the dried cakes were broken into fine powders, cobalt concentrations varied, ranging from not detectable to 0.2 mg/cu m. On the average, the drum-loading operations were the dustiest, but the cobalt concentrations, 0.04-0.5 mg/cu m, varied widely. Other operations in the plant were contained and well ventilated so that the concentration of cobalt in those areas would be expected to be low.

An environmental survey conducted in May 1978 at a US jet aircraft engine assembly plant provided exposure data for three occupational groups (Table III-2) [255]. Workers processed metal alloy parts containing 1-15% cobalt and varying percentages of nickel and chromium. For each location, personal and area samples were collected.

A US manufacturer of cobalt-containing alloys provided data on concentrations of airborne cobalt near the furnaces (most were in the melt shops) and other operations [255]. These data covered 1970 to 1974. Exposures to cobalt on the average were substantially less than 0.1 mg/cu m, but they were quite variable, even for the same areas. For example, exposure to cobalt in the breathing zones of workers in the melt shops were 0.001-1.43 mg/cu m. In the powder products area, six samples ranged from 0.09 to 9.7 mg/cu m. General area samples tended to show a lower cobalt concentration than breathing zone samples.

Almost no published data on exposure to cobalt in welding and thermal cutting operations were found. Hewitt [259] measured cobalt and other metallic contaminants in a number of welding operations in well ventilated areas. Airborne cobalt concentrations were all 0.1-0.4 μg/cu m. Cobalt-containing welding rods can contain other metals such as nickel or chromium that can also be hazardous if they become airborne.

TABLE III-2

ENVIRONMENTAL DATA FOR COBALT ALLOY OPERATIONS

Occupation, Area	Alloy Composition (% cobalt)	Cobalt Concentration (mg/cu m)
Parts Polisher	1 (maximum)	<0.001
"	"	<0.001
"	15	0.008
Polishing Area	--	<0.001
Bench Operator	1 (maximum)	0.001
"	12-15	0.001
Bench Area	--	<0.001
Cutter-Grinder	5-8	0.006
"	7.75-8.25	0.005
"	7.75-8.25	0.001
Cutting-Grinding Area	--	0.003-0.006

Although small amounts of cobalt driers are commonly added to paints and varnishes, suggesting that worker exposure is minimal, almost no information was found to confirm this suggestion. In a survey of a boat manufacturer, none of 13 lamination workers was exposed to detectable amounts of cobalt [263]. Cobalt naphthenate was present in small amounts in the resin used for lamination.

From July 1972 to January 1978, OSHA collected and measured samples in 79 different workplaces for cobalt dust concentrations; 15 had concentrations exceeding the Federal limit of 0.1 mg/cu m (29 CFR 1910.1000). The majority of inspections were conducted in various metal manufacturing industries, with the remainder in inorganic chemical manufacturing and glass container, abrasive products, ceramics, and other industries [264].

Eight plant site visits were conducted to collect information for this report [255]. The number of production workers ranged from less than 50 to 25,000. The three largest plants (at least 800 production workers) all had formal plans for sampling and inhouse capabilities for analysis. Two other plants sampled for cobalt on an irregular basis, and one other sampled for nuisance dust only. The two plants with less than 50 production workers had no program for air monitoring. These plants had relied on corporate headquarters, insurance carriers, and contractors for air monitoring where in-house capability was unavailable.

Sampling Methods

Air sampling for particulate cobalt can be performed by methods recommended for metals in general [258,265,266] (see the Appendix). Collecting media include cellulose ester membrane filters, polycarbonate membrane filters [267], cellulose fiber filters [259,260], and polystyrene filters [257]. Cellulose ester membrane filters are widely used and recommended [268,269]. They have high sampling efficiency, relatively low background contamination from cobalt and other trace metals, ease of ashing, and convenience for personal monitoring [40,269,270]. Glass fiber filters, which are widely available, are less suitable for metals because of high and variable background contamination [257] and incompatibility with many routine analytical procedures.

Particle sizing can be performed by using an optical or electron microscope or by light-scattering techniques. Cascade impactors or cyclone separators can be used to separate particles according to size as they are collected [271]. Impaction devices have been used to determine the size distribution of cobalt-containing aerosols [259,270,272]. Since the Federal standard for cobalt requires measurement of total cobalt and not the respirable component, these devices should not be used for compliance purposes. However, they could have use in conducting basic research on the effects of exposure to cobalt.

Electrostatic precipitators were once widely used to sample air for cobalt and other metal aerosols [261], but they have been replaced, for the most part, by some of the more convenient sampling methods discussed above. Electrostatic precipitators have the advantage of being free of filter clogging and problems with flowrate or humidity. They are also effective in collecting particles of all sizes. One disadvantage of electrostatic precipitators is that actual worker exposure may not be estimated as closely as is possible with personal sampling.

Analytical Methods

Many methods have been developed for the analysis of cobalt samples collected from air. Among these, colorimetric procedures can be satisfactory provided that other metals do not interfere with the results. Color reagents that have been used include nitroso R salt (sodium nitroso-2-naphthol-3,6-disulfonate) and sodium thiocyanate [273-275]. Although these methods are tedious and require skill, they can give satisfactory results.

Emission spectrometric methods have been used frequently to analyze airborne particulate material. Because of its sensitivity, the emission spectrograph has been used to measure pollutants in the community atmosphere [276], but this technique is also useful to determine concentrations of airborne cobalt in the workplace. One investigator, using a specialized electrode for air sampling, was able to perform direct analysis of samples using the emission spectrograph [277]. Equipment cost and limited

availability are important factors to consider in recommending the use of the emission spectrograph in routine analysis of samples.

A widespread method used for determination of airborne cobalt levels is analysis by atomic absorption spectrophotometry (AAS) [266,267,278,279]. The advantage of the atomic absorption method is that it can be made both highly specific and sensitive for the detection of cobalt. The NIOSH P&CAM method 173 recommends AAS for detection of cobalt [268]. This method is sufficiently sensitive to detect cobalt in the concentration ranges normally found in the air of plants manufacturing or using cobalt products if sampling is carried out at a flowrate of 1.5 liters per minute for up to 8 hours using a 37-mm cellulose ester filter with a pore size of 0.8 μm. The method will not distinguish between individual cobalt compounds. In a collaborative test of P&CAM 173, the mean cobalt concentration detected by 17 laboratories was 11.9% below a reference value, suggesting some loss of sample. The coefficient of variation was 45.5% for all of the laboratories, but only 2-10% for single laboratories on replicate samples [266]. With the use of graphite electrodes, flameless AAS methods have extended detection limits for cobalt to ambient air levels [267].

Atomic absorption spectrophotometry has been used extensively to analyze biologic samples for cobalt [280-282]. Because of the extremely low concentrations of many metals (including cobalt) normally in such samples, innovative methods to enhance sensitivity have been developed. An example is the formation of an organometallic complex, such as with a dithiocarbamate, which is readily extracted into an organic solvent, eg, methyl isobutyl ketone [283,284]. This procedure increases sensitivity, not only because it concentrates the cobalt in the sample, but also because of enhanced sensitivity of the AAS obtained from the use of an organic solvent. Using ion exchange separation and electrothermal atomic absorption, Alexandersson and Lidums [282] found reasonably good correlation between blood and urine concentrations of cobalt in workers and airborne levels in cemented carbide plants.

Neutron activation analysis (NAA) can analyze a single small sample for a number of trace metals. Dams et al [257], using NAA, reported a detection limit for cobalt of 2 ng. The minimum concentration of cobalt reported to be detectable in a 24-hour sample of urban air was 25 pg/cu m (1 mg = 10 exp +9 pg) in the presence of 32 other trace elements that were similarly determined. Although this method is sensitive and does not destroy the sample, the requirements for elaborate equipment, the safety precautions necessary for use of an irradiating beam or radioactive sources, and the relatively small thermal neutron cross section of cobalt make NAA less practical than AAS for routine use.

Another technique for analysis of airborne cobalt use is the x-ray fluorescence method [258,266,272]. The fluorescence method resembles the neutron activation procedure in specificity, sensitivity, and ability to analyze a single sample for a number of elements. It is nondestructive and requires a minimum of sample handling. Results can be obtained quite rapidly for cobalt as well as for a number of other metals [266,272], and the x-ray

fluorescence method can be an acceptable alternative to AAS when analyzing cobalt.

In summary, the collection of air samples on membrane filters and the analysis of those samples by AAS, as described in the NIOSH P&CAM 173, are the methods of choice for monitoring of workers' exposure to cobalt (see the Appendix). Other methods, at least as sensitive and precise, are also acceptable.

IV. CONTROL OF EXPOSURE

Over a million workers have some potential for exposure to at least one cobalt-containing compound in the course of their employment. Many receive little exposure because they work with very small amounts of cobalt on an intermittent basis. Others, however, work with large amounts of cobalt regularly. These workers can become exposed through inhalation, dermal contact, or ingestion. In some operations, exposure to cobalt is limited because of the use of extensive engineering controls. Other operations, however, require manual handling of materials containing cobalt. Local exhaust ventilation [261,285] and good work practices can minimize exposure to cobalt in such circumstances. In all cases, ingestion of cobalt is easily prevented by the use of good personal hygiene in combination with the maintenance of a clean worksite. Workers who maintain or repair equipment, enter confined spaces such as tanks or vessels, or are involved in emergencies or other nonroutine situations have an especially high risk of exposure to cobalt. These workers should be trained to recognize hazards, and in some circumstances they may require special protective equipment or clothing to decrease exposure.

Information on typical industrial practices for the control of cobalt came from several site visits to plants manufacturing or using cobalt-containing products. These plants included a refinery, an alloy producer, cemented tungsten carbide producers, and cobalt salt manufacturers. Other sites visited included a welding-rod maker and an aircraft engine manufacturing facility. There is some published information on control of cobalt [261,285], but much of the following information came from plant visits [255].

Engineering Controls

Well-maintained closed systems and the prevention of dust generation, when compatible with the operation involved, are the most effective methods of limiting worker exposure. When a closed system is used, it must prevent or minimize the release of materials. When closed systems are not practical, worker exposure can often be reduced by process equipment modification, the use of control rooms, or local exhaust ventilation.

Properly designed and maintained ventilation systems, including local exhaust systems, can prevent the accumulation of airborne cobalt-containing dusts and fumes. Local exhaust systems were in frequent use in the plants visited. Examples include the use of glove boxes for welding, exhaust hoods provided to individuals using hand-held buffers and grinders, local exhaust on the other grinding machines and on press machines, and dust-collecting hoses for packaging operations [255]. In addition, a ventilation system is desirable as a standby, should a closed system fail. Where exhaust ventilation is required, adequate makeup air, conditioned as necessary for worker comfort, must be provided.

Ventilation requirements for grinding, buffing, and polishing operations were described in a NIOSH research report [286]. Dry grinding equipment was classified as pedestal type, abrasive cutoff tools, surface-type disc grinders, portable grinders and cutoff machines, and other grinding machines according to ventilation system design requirements. Polishing (coated abrasive) equipment was classified as wheel and drum type, disc type, belt polishers, and complex machines. Buffing (loose abrasive) equipment was classified as pedestal type, portable, or multiple buffer machines. For grinding and abrasive polishing, most of the equipment-generated particulate material was thought to be from the workplace. This would be so even for hard metal if a diamond wheel is used. Much more dust was contributed by the abrasive and the abrasive support materials in buffing. The report, in noting that the TLV for cobalt is very much less than for nuisance dust, considered that adequate control of cobalt is not likely to be possible with standard ventilation systems. Totally enclosed ventilation systems and respiratory protection for personnel within the enclosure were recommended. However, many alloys containing only a small percentage of cobalt are used, and restrictions could be less severe in such cases. Engineering controls for most buffing and grinding machines are built to conform to the configuration of the equipment and the materials being handled. Specific guidelines, therefore, are difficult to propose. Figure X-1 shows a diagram of typical local exhaust ventilation for hand held grinders [287]. Workers doing grinding or buffing should be adequately trained to recognize the proper configuration of ventilation equipment for each shape being handled.

Several potentially dusty situations exist in the manufacture or use of cobalt salts, especially if powders rather than crystals are formed. While filter presses are not considered especially dust-producing, they are readily enclosed by local exhaust ventilation as shown in Figure X-2 [288]. Powders formed through filtration, however, present a significant dust problem both in their packaging and in subsequent repackaging and use. The material removed from the filter press is dried, and this dry cake can cause substantial amounts of airborne dust as it is being placed in the pulverizer. One possible type of local exhaust ventilation that would greatly reduce worker exposure is shown in Figure X-3 [288]. Solutions, powders, and crystals of cobalt compounds and fine powders of cobalt metal or mixtures of tungsten carbide and cobalt should be packaged while using local exhaust ventilation. Two typical arrangements are shown in Figure X-4 [288]; many other configurations are also possible. When material is transferred to or from a container to obtain the nominal weight of the package or to use the product, material is often scooped from one container to another. This can be exceedingly dusty, especially with fine powders. Hoods that prevent the powder from dispersing into the work area and do not interfere with the weighing procedure are difficult, but not impossible, to design. Design requirements, however, are quite dependent on the size and shape of the container.

In most of the plants visited, engineering controls in use were generally not specific to cobalt alone, but rather to the entire process [255]. For example, emissions from furnaces preparing specialty steels must be controlled for all the metals in the melt. Cobalt may not be the limiting factor in

control of exposure in such situations. Ball mills used in the cemented carbide industry use solvents, such as acetone, and these emissions must be controlled. Several plants used cobalt in foundry operations where silica and other substances used in the molds dictated many of the control measures used. In milling, hot and cold rolling, and other operations, noise was often a factor in the use of control booths and of other means of control that often helped to lower exposure to cobalt. In buffing, exposure to the abrasive support material must be controlled as must oil mists generated in wet grinding. In manufacture of salts, extensive use of strong acids led to requirements for emission control and cleanup procedures that also limited exposure to cobalt. These overall requirements must be considered in designing engineering controls for cobalt.

To ensure effective operation of ventilation systems, trained personnel should conduct a regular monitoring program. Routine inspection should include face velocity measurements of the collecting hood, examination of the air mover and collection or dispersion system, and measurements of atmospheric concentrations of cobalt in the work environment. Where appropriate, the use of continuous airflow indicators, such as water or oil manometers properly mounted at strategic locations and marked to indicate acceptable air flow, should be considered. Any changes in the work operation, process, or equipment that may affect the ventilation system must be evaluated promptly to ensure that control measures adequately protect workers. All exhaust emissions from ventilation systems should be passed through a system designed to minimize the release or recirculation of raw materials, cobalt, and wastes into the occupational and community environments.

Work Practices

Ultimately, any program of worker protection must consider the prevention of pulmonary fibrosis, which has been clearly demonstrated to result from excessive exposure to cobalt metal and oxides. Dermatitis and an occasional case of extreme pulmonary hypersensitivity to cobalt, however, are much more frequently encountered problems in the day-to-day operation of health care for cobalt workers. These problems cannot be neglected in establishing an effective program for worker protection.

Employers should institute programs that emphasize good personal hygiene to prevent skin and respiratory irritation caused by cobalt-containing dusts. After working with cobalt products, workers should thoroughly wash their hands and face before drinking, eating, or smoking. If skin contact with cobalt solutions occurs, the worker should wash the affected skin promptly. The employer should provide showers if workers have substantial contact with cobalt. These workers should be encouraged to wash or shower after each workshift. Employers should prohibit smoking or carrying of tobacco products in work areas because of possible cobalt contamination. For the same reason, employers should prohibit eating, food handling, or food storage within the work area.

To maintain a safe workplace, employers must identify the many physical hazards involved in handling cobalt and take measures to eliminate them. In particular, several potentially hazardous situations were observed in plant site visits [255]. These included sharp edges on some rolled alloys and rolled-alloy scraps, the need to lift heavy containers of cobalt, and press operators who placed their fingers under moving machinery to remove parts. Rolled alloy workers can avoid finger cuts by wearing gloves. Trained workers who follow proper lifting techniques could avoid back injuries when they lift dense material such as cobalt metal. Protective clothing must not increase the potential for injury of workers who handle cobalt when working with machinery with moving parts.

In storage, cleanup of spills, and emergencies involving fires, employers must be aware of the properties of individual cobalt compounds to ensure proper planning. Finely divided cobalt will ignite, but its fire and explosion hazard is very weak [289]. Certain cobalt compounds used in paints, including cobalt linoresinate, cobalt tallate, and cobalt 2-ethyl hexanoate, are often mixed with kerosene or mineral spirits as solvents. These materials present a moderate fire hazard when exposed to heat or flame [255]. Cobaltous acetylacetonate and cobaltous nitrate are incompatible with easily oxidized material. Cobaltous nitrate can react to cause ignition, violent combustion, or explosion. Cobaltous acetylacetonate flashes at its sublimation point. Other substances can emit highly toxic or acrid fumes when heated to decomposition; these substances include cobaltous nitrate, cobalt benzoate, and cobaltous phosphate.

General plant maintenance must be conducted regularly to prevent cobalt-containing dusts from accumulating in work areas. Cleaning should be performed with vacuum pickup or wet mopping to minimize the amount of dust dispersed into the air. A decontamination room should be available for cleaning equipment that is to receive major overhaul or maintenance. Spills of cobalt-containing material should be promptly cleaned up to minimize inhalation or dermal contact. Liquid material spills can be copiously flushed with water and channeled to a treatment system or holding tank for reclamation or proper disposal. Spills of dry material can be removed by vacuuming or wet mopping. Some spills can be removed by hosing, first with a mist of water to dampen the spilled material and then with a more forceful stream that flushes it into a holding tank or other facility for handling contaminated water. Work surfaces or contaminated clothing should never be cleaned by dry sweeping or blowing with pressurized hoses. Recovery systems used to reclaim waste metals should comply with Federal, state, and local regulations. All waste material generated in the handling of cobalt-containing substances should be disposed of in compliance with Federal, state, and local regulations.

Maintenance and repair workers face special problems regarding their potential exposure. The very circumstances that require the maintenance or repair work and dictate the work conditions will often preclude use of some control procedures. Consequently, very careful supervisory control must be exercised over such activities. These workers must wear appropriate protective equipment and clothing, and they must be trained to recognize and control the hazards they face.

Special precautions are necessary when workers must enter tanks or vessels, such as reaction vessels containing cobalt catalysts or vessels used to prepare cobalt salts [290]. Before any worker enters a vessel, all sources for transferring cobalt and other materials into or out of the vessel must be blanked to prevent their entry. The vessel interior must then be washed with water and purged with air. After purging the vessel's interior, trained personnel should test the vessel's atmosphere with suitable instruments to ensure that no hazards from fire, explosion, oxygen deficiency, or dust inhalation exist. No one should enter a tank or vessel without first being equipped with an appropriate respirator and a secured lifeline or harness. Mechanical ventilation should be provided continuously when workers are inside the tank. At least one other worker similarly equipped with respiratory protection, lifeline, and harness should watch at all times from outside the vessel. Workers inside the tank must be able to communicate with those persons outside. Other workers must be available to assist in an emergency. Flame- or spark-generating operations, such as welding or cutting, should be performed only when an authorized representative of the employer has signed a permit based on a finding that all necessary safety precautions have been taken.

Work Clothing and Protective Equipment

Cobalt causes skin sensitization, and some substances, such as cemented carbides, are abrasive to the skin as well [81]. The use of coveralls or similar work clothes can minimize the possibility of skin irritation, especially if the clothing is changed daily or more promptly in the event of gross contamination from a spill or leak. Workers in flame- or spark-generating operations should, in addition, wear work clothes made of fire-retardant materials. Gloves and protective sleeves should be used in manual transfer operations, together with a face shield (if respiratory protection is not needed) to keep soiled fingers from touching the face. Employers should provide change rooms and dual lockers so workers can remove soiled coveralls during breaks. Work clothing should be worn for only a single day work period, then collected directly into a designated and labeled container, and laundered by a management-selected laundry service. This is encouraged to prevent the spread of contamination into the homes of workers.

Eye irritation has apparently not been a problem for workers handling cobalt products; however, in operations that do scatter fine particles in the air, such as grinding, eye protection must be used that complies with 29 CFR 1910.133. Workers who are especially sensitive to cobalt or who handle solutions of cobalt in such a manner that cobalt can splash on the skin or into the eyes must wear protective suits, or face shields with goggles as appropriate, to prevent appreciable skin and eye contact. Emergency eyewashing facilities, where needed, must be readily available to affected workers. Workers experiencing skin irritation should see a physician promptly.

Respirators are not a substitute for proper engineering controls. Respirators may be needed, however, for nonroutine maintenance work, entry

into confined spaces, pending installation of adequate controls, and in emergencies where the concentration of cobalt could be unknown or excessive. In these situations, employers must provide workers with respirators, and establish a respiratory protective program meeting the requirements of 29 CFR 1910.134. These programs should emphasize the importance of having clean, well-maintained, well-fitted respirators for use in unusual circumstances and emergencies. Workers must be aware of the need to guard against contamination of the interior of the facepiece. Table IV-1 is a respirator selection guide for cobalt developed under the joint NIOSH/OSHA Standards Completion Program [291]. It is based on the Federal standard for cobalt.

Effective Planning

No program to minimize worker exposure to cobalt can be effective unless adequate attention has been given to problems before they arise. When a new facility is to be constructed, persons knowledgeable in occupational safety and health and industrial hygiene engineering should review plans both in the design phase and during construction. They should ensure that working areas and engineering controls are designed so that spills and airborne cobalt will be minimized when the plant is in operation.

For plants already in operation, management should review material-handling operations, maintenance and repair procedures, and process operations periodically. This review should identify areas and job locations where workers might be exposed to cobalt or cobalt-containing waste products, either through inhalation or direct contact with the skin or eyes. If work practices or engineering controls in any area are no longer adequate, management should modify them promptly.

Contingency planning for emergencies, inadvertent release of materials, and breakdown of facilities is vital. These plans should be developed for each department as well as plantwide. They should outline where appropriate equipment and trained personnel are located, and should be written, well understood by workers, and updated as required. Each plant should also be prepared to assess the impact or hazards of a specific spill or release of material should it reach a waterway or create a cloud. Supervisors should have information available, including lists of appropriate names and telephone numbers, for reporting emergencies. In addition to internal reporting procedures, plant management should clearly understand what situations require recordkeeping or external reporting to OSHA, the Environmental Protection Agency (EPA), and any appropriate state agencies.

TABLE IV-1

RESPIRATOR SELECTION GUIDE FOR COBALT

Concentration (mg/cu m, as cobalt)	Respirator Type Approved under Provisions of 30 CFR 11*
Less than or equal to 0.5	(1) Dust and mist respirator, except single-use respirators
Less than or equal to 1.0	(1) Dust and mist respirator, except single-use and quarter-mask respirators (2) Fume or high-efficiency particulate respirator
Less than or equal to 5	(1) High-efficiency particulate respirator with a full facepiece (2) Supplied-air respirator with full facepiece, helmet, or hood (3) Self-contained breathing apparatus with full facepiece
Less than or equal to 20	(1) Powered air-purifying respirator with a high-efficency filter and a full facepiece** (2) Type C supplied-air respirator with a full facepiece operated in pressure-demand or other positive pressure or continuous-flow mode
Escape	(1) High-efficiency particulate respirator with full facepiece (2) Self-contained breathing apparatus

*Respirators specified for use in higher concentrations of cobalt may be used in atmospheres of lower concentrations.

**A high-efficiency filter is defined as one having a penetration of <0.03% when tested against a 0.3-μm dioctyl phthalate (DOP) or equivalent aerosol.

Worker Education and Monitoring

A fundamental part of any program designed to provide a more healthful workplace involves education and monitoring of workers. Workers who understand the reasons for rules concerning hygiene and work practices are

more likely to help keep their work area clean. For cobalt, medical and environmental monitoring are especially important since the potential effects of exposure are poorly understood. Employers must follow the results of medical examinations and correlate them with exposure measurements to ensure that workers are adequately protected. Workers should understand the importance of the medical and environmental monitoring to ensure that they will cooperate and report any possible adverse reaction to cobalt promptly.

(a) Training Programs

Workers must know how to handle cobalt safely. Instructions concerning proper handling methods, cleanup procedures, personal protective equipment, and emergency procedures should be part of a continuing education program presented in training sessions and in written form. The NIOSH publication An Identification System for Occupationally Hazardous Materials [292] describes the types of information on physical, chemical, and toxicologic properties of materials that should be kept in written form. This information should be readily available to persons who handle cobalt-containing substances, and each cobalt compound used should have a corresponding material safety data sheet.

(b) Workplace Monitoring

Manufacturers of all cobalt compounds and users (unless trivial amounts are involved) should conduct an industrial hygiene survey at each location where cobalt is used to determine where workers are likely to be exposed to cobalt. Surveys should be repeated at least once every 3 years, preferrably annually, and as soon as possible after any process change that is likely to increase the concentration of airborne cobalt.

For workers in areas where the concentration of airborne cobalt is likely to exceed 0.05 mg cobalt/cu m (one-half the Federal limit), the employer should establish the following program.

1. Personal monitoring should be conducted to identify and measure, or permit calculation of, the exposure of each worker. Appropriate methods for sampling and analysis are listed in the Appendix.

2. Samples should be representative of the breathing zone air of workers, but source and area monitoring can be a useful supplement for identifying leaks or other sources of emissions.

3. While all workers do not have to be monitored, sufficient samples should be collected to characterize the exposure of all workers. Variations in exposure during different shifts, because of location or job function and of changes in production schedule, should be considered in deciding the number of samples to be collected.

4. If a worker is exposed to cobalt at concentrations exceeding the Federal standard, improved measures to control exposure should be taken immediately. Once the control measures are in effect, exposure monitoring should be repeated. If two consecutive measurements, taken at least 1 week

apart, are below the Federal standard, the worker's exposure may be considered no longer excessive.

Personal monitoring records should be kept for 30 years to ensure compliance with new OSHA regulations concerning worker rights to information about their exposure (29 CFR 1910.20).

(c) Medical Surveillance

NIOSH encourages the provision of medical surveillance programs for workers. Some companies already maintain high-quality programs that allow absorption of hazardous chemicals to be detected at the earliest possible time. This allows early intervention by surveying the workplace and initiating work practice and engineering changes necessary to protect workers.

Medical surveillance programs should ideally include preplacement examinations and periodic reevaluations that will, based on the characteristics of the individual substance or agent, allow detection of absorption before onset of perceptible damage. In most programs, periodic examinations should be carried out on an annual basis. Since cobalt has been implicated to lesser or greater degrees with many effects on the body, these examinations should include not only the points discussed below but also be thorough enough to give reasonable assurance that effects have not occurred elsewhere in the body. The circumstances of each workplace and the types of cobalt compounds present will of course influence the program chosen by the responsible physician.

The most dramatic effect of cobalt is on the lungs, involving development of pulmonary fibrosis. As a result, a medical history is important to determine the presence of factors that would argue against placement in a job requiring exposure to cobalt. The physical examination should give special attention to the chest and lungs. Because pulmonary function studies are sensitive indicators of early changes in lung tissues, NIOSH recommends that they be conducted yearly. The forced expiratory volume in 1 second (FEV 1) and forced vital capacity (FVC) tests are sensitive and easy to administer. Chest x-rays can be useful but are somewhat less able to detect early changes as compared to pulmonary function tests, so 3 years is probably a suitable time between radiologic examinations.

The skin should receive attention because cobalt compounds can cause allergic responses and sensitization. Once sensitized, a worker can probably not tolerate any additional exposure. As a result, any worker with a previous history of allergic skin disease should be carefully counselled and schooled in techniques that will minimize contact with cobalt. In addition, skin protection should be stressed in the workplace to keep the number of new cases of skin sensitization at a minimum.

Cobalt affects the thyroid gland, and results in production of goiter. While the strength of this association is not clear, it seems that palpation of the thyroid for enlargement would be a prudent and simple step to include.

Thyroid function studies do not seem necessary unless enlargement or some other sign or symptom of thyroid dysfunction is present.

Present information suggests the possibility of polycythemia in the blood and electrocardiographic changes in the heart being produced by exposure to cobalt. The information is very unclear and appears to show effects only at a very high dosage. Therefore, no specific tests are suggested for these organ systems unless there is a potential for exposure to high levels of cobalt in the workplace.

The medical surveillance program should be reevaluated frequently and changed to reflect current working conditions and knowledge of health effects.

Medical records should be kept for 30 years after the worker's last exposure to cobalt. This will ensure compliance with OSHA's (29 CFR 1910.20) standard concerning worker's rights to his or her own medical records.

V. BASIS FOR STANDARDS CONCERNING OCCUPATIONAL EXPOSURE TO COBALT

A TLV for workplace exposure to cobalt of 0.5 mg/cu m was proposed by the ACGIH in 1962 [293] and adopted in 1963 [294]. ACGIH's 1966 Documentation of Threshold Limit Values [295] supported the TLV by citing the development of pulmonary changes in workers in the tungsten carbide industry. Reports by Miller et al [23] and Lundgren and Swenssen [296] were mentioned as evidence that cobalt was the etiologic agent involved. Work by Fairhall and colleagues [37,297] was cited as showing the potential for serious and occasionally fatal responses to cobalt at exposure concentrations of 1-2 mg/cu m or less in the tungsten carbide industry. These reports indicated that lung impairment often diminished when workers were removed from the workplace environment and that a hypersensitivity reaction appeared to be involved. A study by Stokinger and Wagner [138] was cited as evidence for the involvement of hypersensitivity in cobalt-exposed animals. A description by Schwartz et al [298] of an allergic dermatitis observed after exposure to cobalt was also noted. Irritation and itching had also been cited by ACGIH in its 1966 documentation [295].

A change in the TLV for cobalt from 0.5 to 0.1 mg/cu m as cobalt metal dust and fume was recommended by the ACGIH in 1966 [299] and adopted in 1968 [7]. The 1971 documentation (1976 addendum) [300] supported this lower TLV and cited additional information including a 1955 report by Schepers [55], which showed that intratracheal administration of cobalt metal led to chronic pneumonitis in guinea pigs. The results of surveys in Michigan and Pennsylvania were also cited. These showed that workplace concentrations of cobalt could be reduced to 0.1 mg/cu m or less. A concomitant reduction in cases of pneumonitis or dermatitis was observed in the Michigan survey over 18 years when cobalt levels were reduced from 14.42 mg/cu m to below 0.1 mg/cu m.

A reduction of the TLV from 0.1 to 0.01 mg/cu m was tentatively recommended in 1975 by the ACGIH Committee on Threshold Limits [8]. This recommendation was subsequently revised, and a limit of 0.05 mg/cu m was proposed by the TLV committee in 1976 [9]. The supporting documentation for both recommendations was the same and can be found in the 1974-75 and 1976 supplements to the Documentation of Threshold Limit Values for Substances in Workroom Air [300] or in the Transactions of the 38th Annual ACGIH Meeting [301]. Information cited to support the change included reports by Siegesmund et al [22] and Kerfoot et al [44]. The reason for lowering the TLV from 0.1 mg/cu m was not clear from the documentation, but it probably was based on the finding of Kerfoot et al [44] that swine exposed at 0.1 mg/cu m developed pulmonary changes.

Standards for occupational exposure to cobalt in other countries are listed in Table V-1. Since 1971, a commission of the German Research Association of the Federal Republic of Germany has included cobalt in the form of respirable dusts of metallic cobalt and sparingly soluble cobalt salts on its list of animal carcinogens (D Henschler, written communication, November 1978). Documents considered in this decision included papers on carcinogenicity [169,170,174,177,178,302-304] and other studies [305,306].

The present US limit (29 CFR 1910.1000) for workplace exposure to cobalt metal, fume, and dust measured as cobalt is an 8-hour TWA concentration limit of 0.1 mg/cu m. This standard is based on the TLV adopted by the ACGIH in 1968.

TABLE V-1

OCCUPATIONAL ENVIRONMENTAL LIMITS FOR COBALT IN FOREIGN COUNTRIES

Country	Compound	Concentration (mg/cu m)	Reference No.
Australia	Cobalt and cobaltous oxide	0.1	307
Belgium	"	0.01	307
Bulgaria	"	0.5	307
Czechoslovakia	Cobalt metal, fume, and dust	0.1	308
Finland	Cobalt and cobaltous oxide	0.1	307
Germany (Federal Republic)	Cobalt metal, fume, and dust	0.5	308, 309
Germany (Democratic Republic)	"	0.1	308
Italy	Cobalt and cobaltous oxide	0.1	307
Netherlands	"	0.1	307
Poland	Cobalt	0.5	310
Romania	Cobalt and cobaltous oxide	0.2 (av.) 0.5 (max)	310
Sweden	Cobalt metal, fume, and dust	0.1	308
Switzerland	Cobalt and cobaltous oxide	0.1	307
USSR	Cobalt metal, dust, and fume	0.5	308
Yugoslavia	Cobalt	0.1	310

VI. RESEARCH NEEDS

Much research conducted on cobalt compounds has served a very specific purpose, and the investigations have not examined overall toxicity. Very few reports except for those concerning pulmonary fibrosis in the hard metal industry have examined the effects of cobalt exposure in the workplace. Considerably more research needs to be done on cobalt, but the following is particularly needed.

The possibility that cobalt compounds in general are fibrogenic should be considered. Inhalation studies in animals should include as a minimum a cobalt salt. Workers in industries other than those manufacturing or using cemented carbide should be examined for adverse lung effects. For both animals and humans, an attempt should be made to obtain dose-response data if fibrosis or prefibrotic changes are found.

The possibility that certain specialty steels cannot release cobalt in the lung following inhalation should be examined. Some of these substances are used because of their extreme heat and corrosion resistance. It is possible that their toxic effects are unrelated to their cobalt content.

An effort should be made to determine the prevalence of dermatitis caused by sensitization to cobalt in various US industries. Information should be collected for different types of cobalt and should include cobalt metal, cemented carbide, and cobalt salts. Work practices and types of protective clothing should be considered in order to determine the protection needed by workers to minimize their chances of becoming sensitized.

The issue of the possible carcinogenicity of cobalt is very much unresolved. Inhalation studies need to be conducted in animals, and such studies could be performed in conjunction with examination of possible fibrotic effects. In humans, epidemiologic studies should be conducted. The cemented carbide industry, in particular, should be examined since the process is sufficiently old that an appropriate cohort should be available. Any epidemiologic study should also collect information on heart disease. The possibility that cobalt exposure at fairly low levels over a considerable period of time could lead to heart disease cannot be ruled out from information now available.

Finally, many cobalt compounds in commercial use have not been examined for toxicity, or very little information of limited use is reported. For example, some cobalt pigments and cobalt driers for oil-based paints have widespread use. Compounds such as these should be subjected to thorough testing for toxicity.

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VIII. APPENDIX

SAMPLING AND ANALYSIS

The sampling and analytical procedures are based on Method No. P&CAM 173 of the NIOSH Manual of Analytical Methods [268], the NIOSH Manual of Sampling Data Sheets [269], the Complete Testing of NIOSH Method for the Determination of Trace Metals by Atomic Absorption Spectrophotometry [266], and updates by NIOSH, Inorganic Methods Development Section.

Sample analysis by atomic absorption spectrophotometry (AAS) is probably the most economical method for the measurement of cobalt. NIOSH recommends this method for determining compliance with the Federal standard for cobalt.

General Sampling Requirements

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

Equipment for Air Sampling

(a) Filter: 37-mm cellulose ester membrane filter with a pore size of 0.8 μm should be mounted with backup pad in a two- or three-piece closed-face cassette.

(b) Battery-operated personal sampling pump: The pump should have a device, such as a clip, for attachment to the worker's clothing. All pumps and flowmeters must be calibrated using a calibrated test meter or other reference, as described in Calibration of Equipment. Battery-operated pumps should be capable of operating at flowrates of 1-2 liters/min to maintain a face velocity of 2.6 cm/s and must be capable of up to 8 hours of continuous operation without recharging.

(c) Thermometer.

(d) Manometer.

(e) Stopwatch.

(f) Various clips, tubing, spring connectors, or belt sufficient to connect sampling apparatus to worker being sampled.

Calibration of Equipment

Since the accuracy of an analysis can be no greater than the accuracy with which the volume of air is measured, accurate calibration of the sampling pump is essential. The frequency of calibration required depends on the use, care, and handling that the pump receives. 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, to collect a large number of field samples, pumps should be calibrated in the laboratory both before and after they have been used. If extensive field sampling is performed, the pumps should be calibrated periodically during sampling to ensure 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 on 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 calibrated 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 X-5. 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.

(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 33 cm of water (approximately 2.54 cm 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 (g) at least three 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, data, and the name of the person performing the calibration.

Collection Samples

(a) Assemble a sampling train consisting of the recommended size cellulose ester membrane filter with a portable, battery-operated personal sampling pump.

(b) Establish the calibrated flowrate as accurately as possible, using the manufacturer's directions. The recommended sampling flowrate is 1.5 liter/min, corresponding to a sampling volume of 0.72 cu m for an 8-hour sample or about 0.02 cu m for a 15-minute sample.

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

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

(e) Immediately after sampling, replace the plugs in the cassette.

(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.

Principle of the Analytical Method

The sample, collected on a cellulose membrane filter, is ashed using a mixture of nitric acid and perchloric acid [266] to destroy the organic matter and to bring cobalt into solution. Samples, blanks, and standards are aspirated into the air-acetylene flame of the AAS. A hollow cathode lamp for cobalt provides a characteristic line at 240.7 nm [268]. The absorption of this line by the ground-state atoms in the flame is proportional to the cobalt concentration in the aspirated sample.

Range and Sensitivity

For cobalt, the absolute detection limit is approximately 0.01 μg cobalt/ml, but the lower limit for quantitative determination is 0.2 μg cobalt/ml. The upper limit for quantitative determination is 5.0 μg cobalt/ml, but the method can be extended to a higher concentration by dilution of the sample. For a 10-ml solution and 0.72 cu m of sampled air,

this corresponds to a range of 2.8-70 μg/cu m. For a 0.02-cu m sample, the lower limit corresponds to 100 μg/cu m.

Interferences

Physical interferences may result if the physical properties of the samples vary significantly. Changes in viscosity and surface tension can affect the sample aspiration rate and thus cause erroneous results. Sample dilution or the method of standard additions are used to correct such interferences. High concentrations of silicates in the sample can cause an interference and may cause aspiration problems. If large amounts of silicates are extracted from the samples, the samples should be allowed to stand for several hours and be centrifuged or filtered to remove the silicates.

Background or nonspecific absorption can occur from particles produced in the flame, which can scatter the incident radiation, causing an apparent absorption signal. Light-scattering problems may be encountered when solutions of high salt content are being analyzed. Light-scattering problems are most severe when measurements are made at the lower wavelengths, ie, below about 250 nm. Background absorption may also occur as the result of the formation of various molecular species that can absorb light. The background absorption should be accounted for by the use of background correction techniques, preferably by the use of a nonabsorbing wavelength, and, if necessary, by the use of D2 or H2 continuum.

Precision and Accuracy

For analysis only, the relative standard deviation (RSD) is 54% at 0.05 μg cobalt/ml, compared with a RSD of 9% at 0.2 μg cobalt/ml. From 0.5 to 5 μg cobalt/ml, the RSD of the analytical measurement is approximately 3% [268].

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, and it is sufficiently sensitive. However, the method measures total cobalt and is not capable of distinguishing individual cobalt compounds.

Apparatus and Equipment

(a) Hollow cathode lamp for cobalt.

(b) Atomic absorption spectrophotometer equipped with a burner head for air-acetylene flame.

(c) Oxidant: Air supply, with a minimum pressure of 40 pounds per square inch, filtered to remove oil and water.

(d) Fuel: Acetylene, commercially available for atomic absorption use.

(e) Pressure-reducing valves: A two-gauge, two-stage pressure-reducing valve and appropriate hose connections for each compressed gas tank used.

(f) Glassware, borosilicate:

(1) 125-ml Phillips or Griffin beakers with watchglass covers.

(2) 15-ml graduated centrifuge tubes.

(3) 10-ml volumetric flasks.

(4) 100-ml volumetric flasks.

(5) 1-liter volumetric flasks.

(6) 125-ml polyethylene bottles.

Additional auxiliary glassware such as pipets and different size volumetric glassware will be required depending on the elements being determined and the dilutions required to have sample concentrations above the detection limit and in the linear response range. All pipets and volumetric flasks required in this procedure should be calibrated class A volumetric glassware.

(g) Hotplate (suitable for operation at 140°C).

Reagents

(a) Purity: ACS analytical reagent grade chemicals or equivalent should be used in all tests. References to water shall be understood to mean double distilled water or equivalent. Care in selection of reagents and in following the listed precautions is essential if low blank values are to be obtained.

(b) Concentrated nitric acid (68-71%), redistilled, specific gravity 1.42.

(c) Standard stock solutions (1,000 µg/ml for cobalt), commercially prepared or prepared per instrument manufacturer's recommendations.

(d) Perchloric acid, reagent grade.

Procedure

(a) Cleaning of Equipment

Before initial use, clean glassware with saturated solution of sodium dichromate in concentrated sulfuric acid and then rinse thoroughly with warm tapwater, concentrated nitric acid, tapwater, and deionized water, in that order, and then dry. Soak all glassware in a mild detergent solution immediately after use to remove any residual grease or chemicals. For glassware that has previously been subjected to the entire cleaning procedure, it is not necessary to use the chromic acid cleaning solution. This glassware should be cleaned in 1:1 diluted nitric acid and rinsed several times with distilled water.

(b) Preparation of Samples

Transfer samples and blanks (a minimum of 1 filter blank for every 10 filter samples) to clean 125-ml beakers, and add 1 ml of concentrated $HClO_4$ and 6 ml of concentrated HNO_3 to each. Cover each beaker with a watchglass, and heat on a hot plate (140°C) in a fume hood. Completion of digestion is indicated by a colorless solution or a white residue in the beaker. Once the ashing is complete, remove the watchglass, and allow the sample to evaporate to near dryness (approximately 0.5 ml). If charring occurs, add HNO_3 (1 ml) and evaporate to near dryness (0.5 ml) again. Repeat, if necessary.

Remove the beaker from the hotplate, cool, and add 1 ml HNO_3 and 2-3 ml of distilled water. Transfer the solution quantitatively with distilled water to a 10-ml volumetric flask, and then dilute the samples to volume (10 ml) with water.

The 10-ml solution may be analyzed directly for any element of very low concentration in the sample that was dissolved by this ashing procedure. Aliquots of this solution may then be diluted to an appropriate volume for cobalt present at higher concentrations.

(c) Analysis of Samples

Set the instrument operating conditions as recommended by the manufacturer. The instrument should be set at 240.7 nm for cobalt.

Match standard solutions with the sample matrix as closely as possible, and run in duplicate. Aspirate working standard solutions, freshly prepared each day, into the flame, and record the absorbance. Prepare a calibration graph as described in a later section. (Note: All combustion products from the atomic absorption flame must be removed by direct exhaustion through the use of a good separate flame ventilation system.) Blank filters must be carried through the entire procedure each time samples are analyzed.

Aspirate the appropriately diluted samples directly into the instrument, and record the absorbance for comparison with standards. Should the absorbance be above the calibration range, dilute an appropriate aliquot to 10

ml. Aspirate water after each sample. As a minimum, a midrange standard must be aspirated with sufficient frequency, ie, once every five samples, to ensure the accuracy of the sample determinations. It is best to run a standard with a concentration close to that of a sample after each sample is run. To the extent possible, base all determinations on replicate analyses.

Calibration and Standards

(a) Dilute standards (100 μg metal/ml). Pipet 10 ml of the stock (1,000 μg cobalt/ml) into a 100-ml volumetric flask, add 10 ml HNO_3, and dilute to volume with distilled water. Prepare fresh standards at least weekly, preferably with each use, and store in polyethylene bottles. Polyethylene containers are suitable for storage because of their low metallic content and nonpolar surfaces.

(b) Working standards. Prepare working standards by dilution of the dilute standards or the stock standards so that the final acid concentration is the same for the samples and standards, ie, 10% V/V HNO_3, 5% V/V $HClO_4$. Concentrations of the working standards should cover the range 0.5-5.0 μg/ml. Prepare fresh solutions daily.

(c) Standard solutions. Aspirate standard solutions into the flame, and record the absorbance (or concentration). If the instrument used displays transmittance, convert these values to absorbance. Prepare a calibration curve by plotting absorbance (units) vs metal concentration. The best fit curve (calculated by linear least square regression analysis) is fitted to the data points. This line, or the equation describing the line, is used to obtain the cobalt concentration in the samples being analyzed.

(d) Preparation procedure. To ensure that the preparation procedure is being properly followed, spike clean membrane filters with known amounts of cobalt by adding appropriate amounts of the previously described standards, and carry them through the entire procedure. Determine the amount of cobalt, and calculate the percent recovery. These tests will provide recovery and precision data for the procedure as it is carried out in the laboratory for the soluble cobalt compounds being determined.

(e) Analysis by the method of standard additions. In order to check for interferences, analyze samples initially and periodically by the method of standard additions, and compare the results with those obtained by the conventional analytical determination. For this method, divide the sample into three 2-ml aliquots. To one of the aliquots, add an amount of cobalt approximately equal to that in the sample. To another aliquot add twice this amount. (Note: Additions should be made by micropipetting techniques so that the volume does not exceed 1% of the original aliquot volume, ie, 10-μl and 20-μl additions to a 2-ml aliquot. This method should be applied only if the concentration of cobalt in the solutions is sufficiently low that absorbance is linearly related to concentration.) Then analyze the solutions, and plot the absorbance readings against metal added to the original sample. The line obtained from such a plot is extrapolated to zero absorbance, and the

intercept on the concentration axis is taken as the amount of metal in the original sample. If the result of this determination does not agree to within 10% of the values obtained with the procedure described in (c), an interference is indicated and standard addition techniques should be utilized for sample analysis.

Calculations

(a) The estimated air volume may need to be corrected for elevation and temperature. For personal sampling pumps with rotameters only, the following correction for air volume should be made:

$$V = \frac{f \times t}{1{,}000} \left(\sqrt{\frac{P1}{P2} \times \frac{T2}{T1}} \right)$$

where:

V = sample volume (cu m)
f = sample flowrate (liters/min)
t = sampling time (minutes)
P1 = pressure during calibration of sampling pump (in mmHg)
P2 = pressure air samples (in mmHg)
T1 = temperature during calibration of sampling pump (K)
T2 = temperature of air sampled (K)

(b) The uncorrected volume collected by the filter is calculated by averaging the beginning and ending sample flowrates, converting to cubic meters, and multiplying by the sample collection time. The formula for this calculation is:

$$V = \frac{(fb + fe)t}{2{,}000}$$

where:

V = sample volume (cu m)
fb = sample flowrate at beginning of sample collection (liter/min)
fe = sample flowrate at end of sample collection (liter/min)
t = sample collection time (minutes)

(c) Cobalt concentrations are calculated by multiplying the micrograms of cobalt/ml in the sample aliquot by the aliquot volume and dividing by the volume of air sampled by the filter:

$$\mu g \text{ cobalt/cu m} = \frac{(C \times Va) - B}{V}$$

where:

C = concentration (μg cobalt/ml) in the aliquot
Va = volume of aliquot (ml)
B = total μg of cobalt in the blank
V = volume of air sampled (cu m)

IX. TABLES

TABLE IX-1

ESTIMATED WORKER EXPOSURE TO COBALT

Substance	No. of Workers Exposed*
Cobalt metal	235,000
Cobalt oxides	867,000
Cobalt drier**	301,000
Cobalt paste drier**	8,300
Cobalt naphthenate	79,000
Cobalt neodecanoate	1,300
Cobalt octanoate	8,100
Cobalt tallate	19,000
Cobalt titanate	900
Cobaltous acetate	21,000
Cobaltous carbonate	5,100
Cobaltous chloride	10,000
Cobaltous nitrate	8,600
Cobaltous oxalate	1,700
Cobaltous sulfate	8,300
Cobalt cyanide	7,200
Cobalt hydroxide	2,500
Cobalt 2-ethylhexoate	3,900

*Many of these workers would be potentially exposed to only small amounts of cobalt through inhalation, ingestion, or dermal contact. The exposure estimates are not additive, since some workers would be exposed to more than one compound.

**Substance(s) comprising the drier were not identified.

From the NIOSH National Occupational Hazard Survey, 1972-74 [313]

TABLE IX-2

EFFECTS OF WORKPLACE EXPOSURE TO COBALT-CONTAINING SUBSTANCES

Substance(s) and Composition (if known)	Concentration (mg cobalt/cu m)	Duration, Years	No. of Workers	Effect	Reference No.
Cobalt metal	*	2-5	7	Respiratory irritation; no evidence of fibrosis	51
"	*	7	1	Marked pulmonary fibrosis resulting in death; elevated cobalt levels in lungs	50
"	*	12.5 (av.)	120	1 case of increased pulmonary reticulations	52
"	*	4	1	Death from cardiac insufficiency; elevated cardiac levels of cobalt	311
"	*	*	6	No evidence of pulmonary damage	34
Cobalt oxide	*	15-20	*	Chronic bronchitis and respiratory insufficiency	52
Cobaltous oxide	1-303 (av. 106)				
or		*	247	Pulmonary changes in 8 (3.2%)	35
Tungsten carbide, cobalt	0.8-12				
Tungsten carbide 95.9-97.6%, cobalt 2.4-4.1%	0.05-0.14	4.4 (av.)	1,802	Abnormal lung radiographic findings in 13 (0.8%)	37
Tungsten carbide 75-96%, cobalt 4-25%	0.6-25	1-13 (av. 6)	193	Lung radiographic changes in 31 (16%); spirometric abnormalities in 25 of 116 (22%)	33
Tungsten carbide, cobalt 6-11%	*	61.3% for 1-5, 37.1% for 5	62	Chronic bronchitis in 5 (8.1%); decreased vital capacity in 9 (14.5%)	29
Tungsten carbide, titanium carbide, cobalt 4.3-8.4%	0.043-0.57 (av. 0.21)	3-26	305	Lung radiographic changes in 56 (18.4%)	34

TABLE IX-2 (CONTINUED)

EFFECTS OF WORKPLACE EXPOSURE TO COBALT-CONTAINING SUBSTANCES

Substance(s) and Composition (if known)	Concentration (mg cobalt/cu m)	Duration, Years	No. of Workers	Effect	Reference No.
Tungsten carbide, titanium carbide, cobalt 10%	*	0.5-4	27	Bronchitis in 11 (41%), pneumoconiosis in 9 (33%), radiographic lung changes in 8 (30%)	11
Tungsten carbide, cobalt	0.007-0.19	5-16	29	Well-established fibrosis in 3, labored breathing in 11	24 25
"	0.1-0.2	(av. 6)	200-300	Pneumoconiosis in 3	23
"	*	14	1	Pulmonary fibrosis; tungsten, nickel, and titanium found in lungs	17
"	*	17	1	Well-established pulmonary fibrosis	21
"	0.045-0.47 (av. 0.22)	1-30 (av. 11)	22	No radiographic evidence of pulmonary fibrosis; possible reduction of vital capacity	40
"	0.03-0.56 (av. 0.24)	6	1	Well-established pulmonary fibrosis	20
"	*	9	1	"	15
"	0.6-3.2	(up to 3)	178	Pulmonary irritation in 50%	46
"	*	5	1	Well-established pulmonary fibrosis; titanium found in lungs	19
"	0.4-3.3	*	117	Chronic bronchitis in 35 (30%); mild fibrosis in 33 (28.2%)	35
"	*	2-8	3	Pulmonary fibrosis in one lung biopsy specimen	12
"	*	0.08-28 (av. 12.6)	1,500	Pulmonary fibrosis in 9 (0.6%)	26 27
"	0.03-0.3	1-14 (av. 5.8)	10	Mild fibrosis	13

TABLE IX-2 (CONTINUED)

EFFECTS OF WORKPLACE EXPOSURE TO COBALT-CONTAINING SUBSTANCES

Substance(s) and Composition (if known)	Concentration (mg cobalt/cu m)	Duration, Years	No. of Workers	Effect	Reference No.
"	0.006-1.3	30% for <10, 44% for 10-20, 26% for >20	61	Pulmonary changes in 30	30
"	*	*	1	Asthma-like labored breathing	47
"	*	9	1	Marked fibrosis and emphysema; cobalt and other metals found in lung	15
"	*	8	1	"	16
"	*	*	5	Asthma-like labored breathing	26 27
"	*	1.5	1	"	14
Tungsten carbide/oxide, titanium, cobalt	0.4-2.9	*	178	Respiratory irritation in 73 (41%); bronchitis with labored breathing in 2	36
Tungsten carbide, titanium carbide, niobium, cobalt	*	0.08-9	100	Pulmonary irritation in 15 subjects; progressing to fibrosis in 5	28
Tungsten carbide, titanium, cobalt	*	6	1	Well-established pulmonary fibrosis	18
Tungsten carbide, titanium carbide, cobalt	*	*	200-250	6-10% with labored breathing; radiographic changes in 4%	38
Hard-metal tool manufacturing	* *	16-50 (av. 36)	12	Well-established pulmonary fibrosis	14
Welding fumes	*	15 (2 h/d)	1	Well-established pulmonary fibrosis; cobalt, chromium, nickel, and iron found in lung tissue	22

*Data not reported

TABLE IX-3

CHEMICAL AND PHYSICAL PROPERTIES OF COBALT AND SELECTED COBALT COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (C)	Density	Solubility (g/100 cc) H2O	Solubility (g/100 cc) Other Solvents
Cobalt	Co	58.933	1,495	8.9	--	Acid
Cobaltous acetate tetrahydrate	Co(C2H3O2)2.4H2O	249.08 (-4H2O,177.03)	-4H2O, 140	1.705 (19 C)	--	Dilute acids, alcohol pentyl acetate
Cobaltic acetyl-acetonate	Co(HC(COCH3)2)3	356.26	241	1.43 (15 C)	--	--
Cobaltous carbonate (spherocobaltite)	CoCO3	118.94	Decomposes	4.13	--	Acid
Basic cobaltous carbonate	2CoCO3.3Co(OH)2.H2O	534.74	--	--	Decomposes in hot	Acid, (NH4)2CO3
Cobaltous chromate	CoCrO4	174.93	--	--	Decomposes in hot	Mineral acids, NH4OH
Cobaltous citrate	Co3(C6H5O7)2.2H2O	591.04	-2H2O, 150	--	0.8 in cold	Dilute acids
Cobaltous cyanide dihydrate	Co(CN)2.2H2O	147.00	-2H2O, 280 (decomposes at 300)	Anhydrous 1.872 (25 C)	0.00418 in cold	KCN, HCl, NH4OH
Cobaltous formate	Co(CHO2)2.2H2O	185.00	-2H2O, 140 (decomposes at 175)	2.129 (22 C)	5.03 in cold	--
Cobalt halides:						
Cobaltous bromide	CoBr2	218.75	678 (under HBr and N2)	4.909	66.7 (59 C), 68.1 (97 C)	77.1 alcohol, 58.6 methanol; acetone, ether, methyl acetate
Cobaltous chloride	CoCl2	129.84	735	3.356 (25 C)	45 (7 C), 105 (96 C)	54.4 alcohol, 8.6 acetone, 38.5 methanol, slightly soluble, ether, glycerol, pyridine

TABLE IX-3 (CONTINUED)

CHEMICAL AND PHYSICAL PROPERTIES OF COBALT AND SELECTED COBALT COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (C)	Density	Solubility (g/100 cc)	
					H2O	Other Solvents
Cobaltous fluoride	CoF2	96.93	ca 1,200	4.43 (25 C)	1.5 (25 C) (decomposes in hot)	Warm mineral acids
Cobaltic fluoride	CoF3	115.93	--	3.88	Decomposes to Co(OH)3	--
Cobaltous iodide	CoI2	312.74	515 (in a vacuum)	5.68	159 (0 C) 420 (100 C)	Alcohol, acetone
Cobaltous hydroxide	Co(OH)2	92.95	Decomposes	3.597 (15 C)	--	Acids, NH4 salts
Cobaltic hydroxide	Co(OH)3 (Co2O3.3H2O)	109.96 (219.91)	-- Decomposes	-- 4.46	--	Acids
Cobaltous linoleate	Co(C18H31O2)2	617.83	--	--	--	Alcohol, ether, acetone
Cobaltous naphthenate	A mixture of many cobalt naphthenates that have varying chain lengths	--	--	--	--	Alcohol, ether, oil
Cobaltous nitrate	Co(NO3)2	182.96	Decomposes 100-105	2.49	--	Most organic solvents
Cobaltous oleate	Co(C18H33O2)2	621.86	235	--	--	Alcohol, ether, oils, benzene
Cobaltous oxalate	CoC2O4	146.95	Decomposes 250	3.02 (25 C)	--	Acid, NH4OH
Cobalt oxides:						
Cobaltous oxide	CoO	74.93	1,935	6.45	--	Acid, alkali
Cobaltic oxide	Co2O3	165.86	Decomposes 895	4.81-5.60	--	Acid

TABLE IX-3 (CONTINUED)

CHEMICAL AND PHYSICAL PROPERTIES OF COBALT AND SELECTED COBALT COMPOUNDS

Compound	Molecular Formula	Formula Weight	Melting Point (C)	Density	Solubility (g/100 cc) H2O	Solubility (g/100 cc) Other Solvents
Cobaltous cobaltic oxide	Co3O4	240.80	Transition point to CoO 900-950	6.07	--	Alkalis, acids
Cobaltous phosphate	Co3(PO4)2	366.74	--	2.587 (25 C)	--	H3PO4, NH4OH
Cobalt monosilicide	CoSi	87.03	1,395	--	--	HCl
Cobalt disilicide	CoSi2	115.11	1,277	5.3	--	--
Dicobalt monosilicide	Co2Si	145.95	1,327	7.28 (0 C)	--	Decomposes in acid
Cobaltous sulfate	CoSO4	155.00	735	3.472	36.2 (20 C) 83 (100 C)	1.04 (18 C) methanol
Cobaltous sulfide	CoS	91.00	1,116	5.45 (18 C)	0.00038 (18 C)	Slightly soluble acids
Cobalt disulfide	CoS2	123.06	--	4.269	--	HNO3, aqua regia
Cobaltic sulfide	Co2S3	214.06	--	4.8	--	Decomposes in acid, aqua regia
Cobalt tallate	Cobalt derivative of refined tall oil - of varying composition	--	--	--	--	--
Cobalt thiocyanate	Co(SCN)2	175.10	-3H2O, 105	--	--	Alcohols, ether, acetone, CHCl3
Cobaltous tungstate	CoWO4	306.78	--	8.42	--	Hot concentrated slightly soluble dilute acid

Adapted from references 2,247,312

TABLE IX-4

SIMPLE COBALT SALTS, ORGANIC AND INORGANIC

CAS NO.	NAME	CAS NO.	NAME	CAS NO.	NAME
71-48-7	cobaltous acetate	11114-55-9	alloy;Al, Co	14640-56-3	cobalt pyrophosphate
136-52-7	cobalt 2-ethylhexanoate	12006-78-9	cobalt boride	14666-94-5	cobalt oleate
513-79-1	cobaltous carbonate	12013-10-4	cobalt sulfide (CoS2)	14666-96-7	cobalt linoleate
542-84-7	cobaltous cyanide	12016-80-7	cobalt hydroxide oxide	14965-99-2	cobaltic cyanide
544-18-3	cobaltous formate	12017-01-5	Co, Ti oxide (CoTi03)	15238-00-3	cobaltous iodide
814-89-1	cobaltous oxalate	12017-12-8	cobalt silicide	18718-10-0	cobaltous phosphate (2:1)
866-81-9	cobaltous citrate	12017-13-9	cobalt telluride	21041-93-0	cobaltous hydroxide
932-69-4	cobalt benzoate	12017-38-8	Co, Ti oxide (Co2Ti04)	26490-63-1	cobalt tetrafluoro-borate
1307-96-6	cobaltous oxide	12017-68-7	Co; cmpd. with Sm (5:1)	27016-73-1	cobalt arsenide
1307-99-9	cobalt selenide	12044-42-7	cobaltous arsenide	27253-31-2	cobalt neodecanoate *
1308-04-9	cobaltic oxide	12045-01-1	cobaltous boride	34262-88-9	cobalt terephthalate
1308-06-1	cobaltous cobaltic oxide	12052-28-7	Co, Fe oxide	37261-99-7	alloy; WC 88, Co 12
1317-42-6	cobalt sulfide (CoS)	12052-42-5	cobalt antimonide	38582-17-1	cobalt cyclohexanebutanoate
1333-88-6	Al, Co oxide	12134-02-0	cobalt phosphide	42978-77-8	cobalt methylbenzoate
1345-19-3	cobalt tin oxide	12214-13-0	Ce, cmpd. with Co (1:5)	49676-83-7	cobalt; 3,5,5-trimethyl-hexanoate
3017-60-5	cobalt thiocyanate	12256-04-7	cobaltic arsenide	53219-02-6	alloy; Co 9.9-90, Al 9.8-90
6700-85-2	cobalt octanoate	12263-08-0	cobalt molybdophosphate	58197-53-8	cobalt, 2-propionate
7440-48-4	cobalt	12774-15-1	alloy; WC 94, Co 6	58591-45-0	co, Ni oxide
7646-79-9	cobaltous chloride	13455-25-9	cobaltous chromate	67801-57-4	cobalt; 1,2,4-benzenetricarboxy-late
7789-43-7	cobaltous bromide	13455-31-7	cobaltous perchlorate	67952-53-8	cobalt; 2-methyl 2-propionate
				68016-03-5	molybdic acid; Co, Ni salt (2:1:1)
10026-17-2	cobaltous fluoride	13455-36-2	cobaltous phosphate	68123-03-5	cobalt, 4-aminobenzoate
10026-18-3	cobaltic fluoride	13596-21-9	cobaltous phosphate	68647-47-2	cobalt molybdate (1:3)
10101-58-3	cobaltous tungstate	13762-14-6	cobalt molybdate (1:1)	69011-09-2	Co, Zr oxide
10124-43-3	cobaltous sulfate	14017-41-5	cobalt sulfamate	12190-79-3	Co, Li oxide
10141-05-6	cobaltous nitrate	14590-13-7	cobaltous ammonium phosphate	13586-84-6	cobalt octadecanoate

*Alternate CAS No. of 52270-44-7

TABLE IX-5

ORGANOCOBALT COMPLEXES AND COMPLEX COBALT SALTS

CAS No.	Molecular Formula	CAS No.	Molecular Formula
3252-99-1	C8-H14-Co-N4-04	49651-10-7	C42-Br2-Co-P2
3317-67-7	C32-H16-Co-N8	51084-32-3	C9-H8-04.1/2Co
10210-68-1	C8-Co2-08	51839-24-8	C2-H6-Co5-012.H20
10534-89-1	C1.1/2C0-H18-N6	52729-67-6	C32-H15-Co-N8-03-S.Na
12602-23-2	C2-H6-Co-012	62207-76-5	C16-H12-Co-F2-N2-02
12715-61-6	C32-H30-Co-N8-010-S2	67875-38-1	C32-H15-C1-Co-N8-02-S
13408-73-6	C6-H24-Co-N6.3C1	67906-18-7	C6-H20-Co-N4.2C2-Au-N2
13586-82-8	C8-H16-02.xCo	68133-85-7	C4-H5-Co-N-04
13859-51-3	C1-Co-H15-N5.2C1	68189-40-2	C32-H14-C12-Co-N8-04-Se
13869-30-2	C4-H5-Co-N-04	68239-56-5	(C10-H18-Co-N2-04)x
14024-48-7	C10-H14-Co-04	68239-58-7	(C6-H12-C12-Co-N2)x
14123-08-1	C6-Co-N6.3/2Co	68475-45-6	C14-H26-Co-016
14126-32-0	C36-H30-Br2-Co-P2	68958-90-7	C4-H16-Co-N4.2C2-Au-N2
14931-83-0	C10-H12-Co-N2-08	69140-59-6	C2-H8-07-P2.Co.2K
21679-46-9	C15-H21-Co-06	69140-60-9	C2-H8-07-P2.Co.2Na
25971-15-7	C26-H38-Co2-N10-08	69178-34-3	C2-H8-07-P2.Co.2H3-N
29383-29-7	C32-H14-Co-N8-06-S2.2H	69178-42-3	C34-H32-Co-N8-010-S2
30638-08-5	C32-H15-Co-N8-03-S.H	69198-43-2	C48-H54-Br2-Co-P2
40621-10-1	C12-H23-P-S2.1/2Co		

TABLE IX-6

SUBSTANCES OF UNIDENTIFIED STRUCTURE

CAS No.	Description
1345-16-0	Cl pigment blue 28
5931-89-5	cobalt acetate
8011-87-8	Cl pigment green 19
11104-61-3	cobalt oxide
12653-56-4	cobalt sulfide
12672-27-4	aluminum cobalt oxide
12737-30-3	cobalt nickel oxide
37367-90-1	cobalt borate
37382-24-4	chromium cobalt oxide
29261-75-6	cobalt zirconium oxide
61789-51-3	cobalt naphthenate
61789-52-4	fatty acids, tall-oil, Co salts
63497-09-6	chromium cobalt iron oxide
68130-37-0	Co, moly-denum hydroxide oxide phosphate
68152-91-0	tall oils, cobalt salt
68186-85-6	spinels; Co, Ti green
68186-86-7	spinels; Al, Co blue
68186-87-8	spinels; Al, Co, Zn blue
68186-89-0	periclase; Co, Ni gray
68186-97-0	spinels; Cr, Co, Fe black
68187-05-3	spinels; Co, Sn gray
68187-11-1	spinels; Al, Cr, Co blue green
68187-40-6	olivine, cobalt blue
68187-49-7	spinels; Cr, Co green
68187-50-8	spinels; Co, Fe black
68409-81-4	fatty acids, C6-19-branched, Co salts
68411-08-5	Co, dextrin complexes
68412-74-8	phenakite; Co, Zn blue
68442-96-6	hydrofluoric acid, reaction products with alumina and cobalt chloride
68457-13-6	Co, borate neodecanoate complexes
68457-90-9	zirconium, carbonate dipropylene glycol 2-ethylhexanoate isobutyl alcohol oxopropionate cobalt complexes
68478-57-9	Co, 2-ethylhexanoate isononate complexes
68478-58-0	Co, 2-ethylhexanoate 3,5,5-trimethylhexanoate complexes
68512-31-2	periclase, Co blue gray
68553-15-1	linseed oil; Co, Mn, salt
68584-96-3	2-naphthalene sulfonic acid, 6-hydroxy-5-nitroso; Co, Na salt; complex with vinylpyrrolidone polymer

TABLE IX-6 (CONTINUED)

SUBSTANCES OF UNIDENTIFIED STRUCTURE

CAS No.	Description
68608-09-3	spinels; Al, Co, Sn
68608-93-5	boric acid; Co, Mg salt; red-blue
68609-02-9	Co, borate isononoate neodecanoate complexes
68610-13-9	phosphoric acid; Co, Li salt; violet
68784-10-1	CdS; solid soln. with ZnS, Al, Cu, and Co; Ag-doped
68855-86-7	fatty acids, tallow, hydrogenated, Co salts
68955-83-9	fatty acids, C9-13-neo-, Co salts
68956-82-1	resin acids and rosin acids, Co salts
68988-10-3	zirconium, dipropylene glycol, isobutyl alcohol, neodecanoate, propionate Co complexes
69012-37-9	slimes and sludges, Co terephthalate
69012-71-1	leach residues, Zn ore-calcine, Co repulp
69012-72-2	leach residues; Zn ore-calcine; Zn, Co
70131-61-2	cobalt salt of mixed polymer

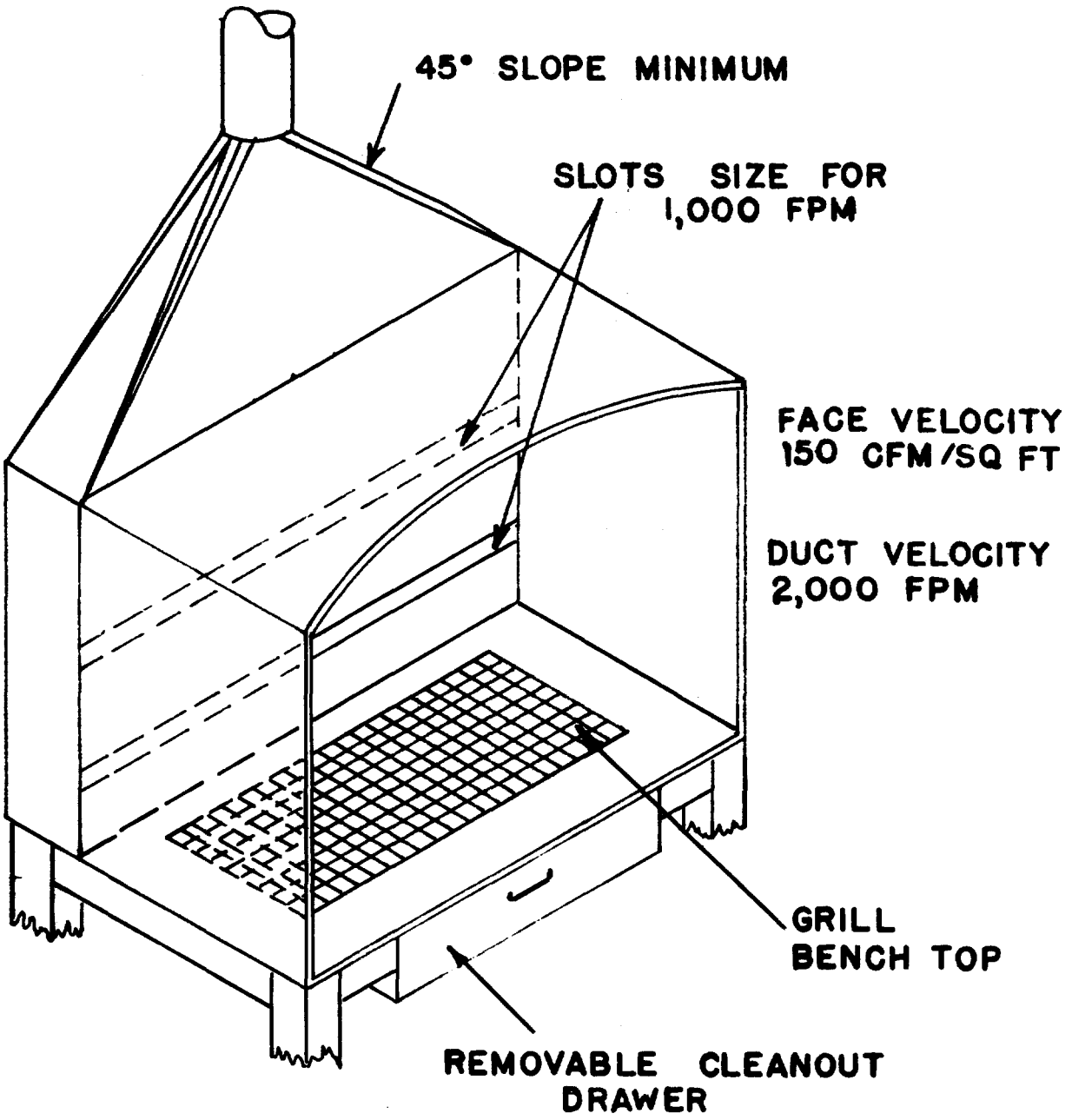


FIGURE X-1. EXHAUST BOOTH FOR PORTABLE GRINDIMG

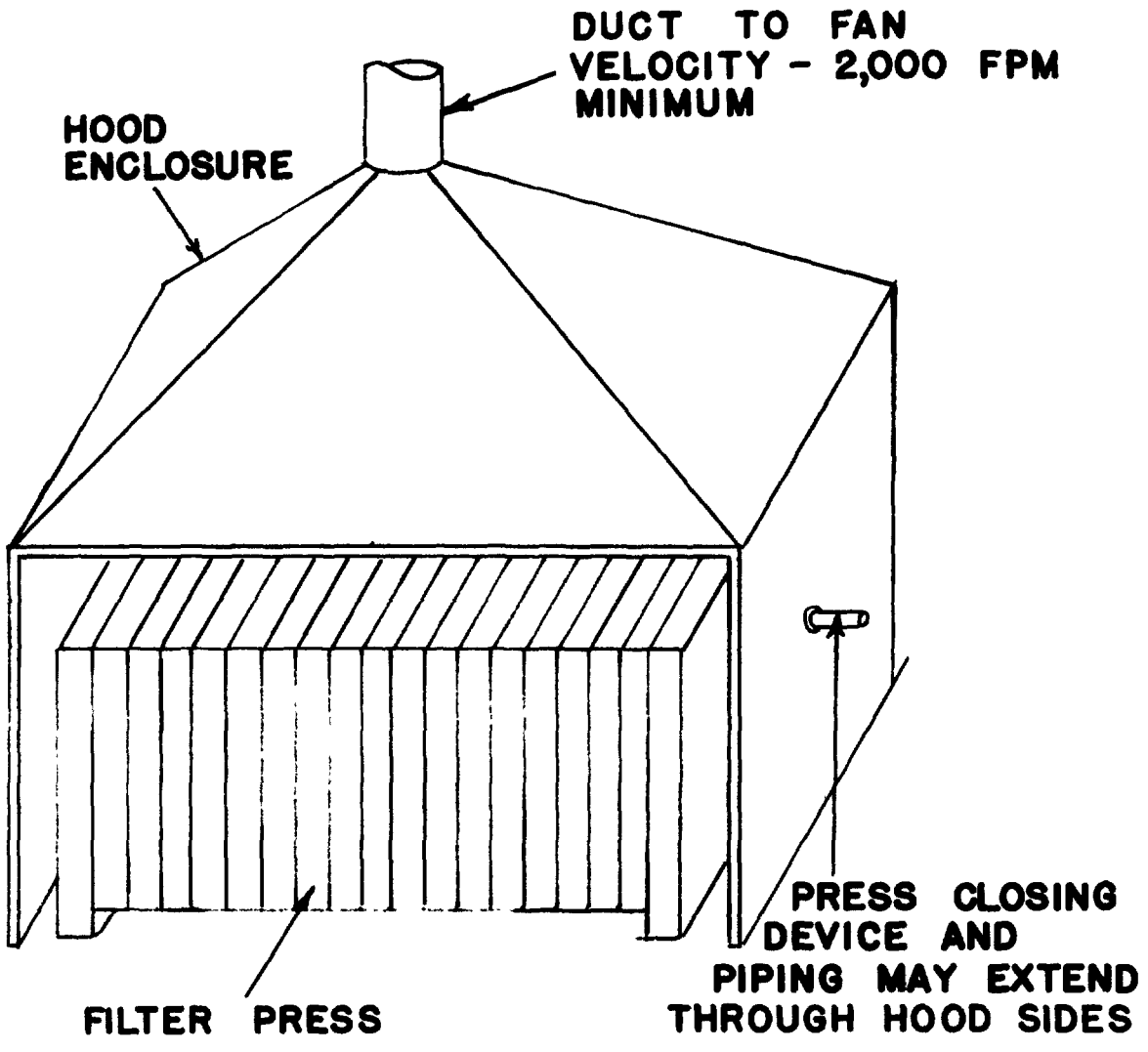


FIGURE X-2. ENCLOSURE FOR FILTER PRESSES

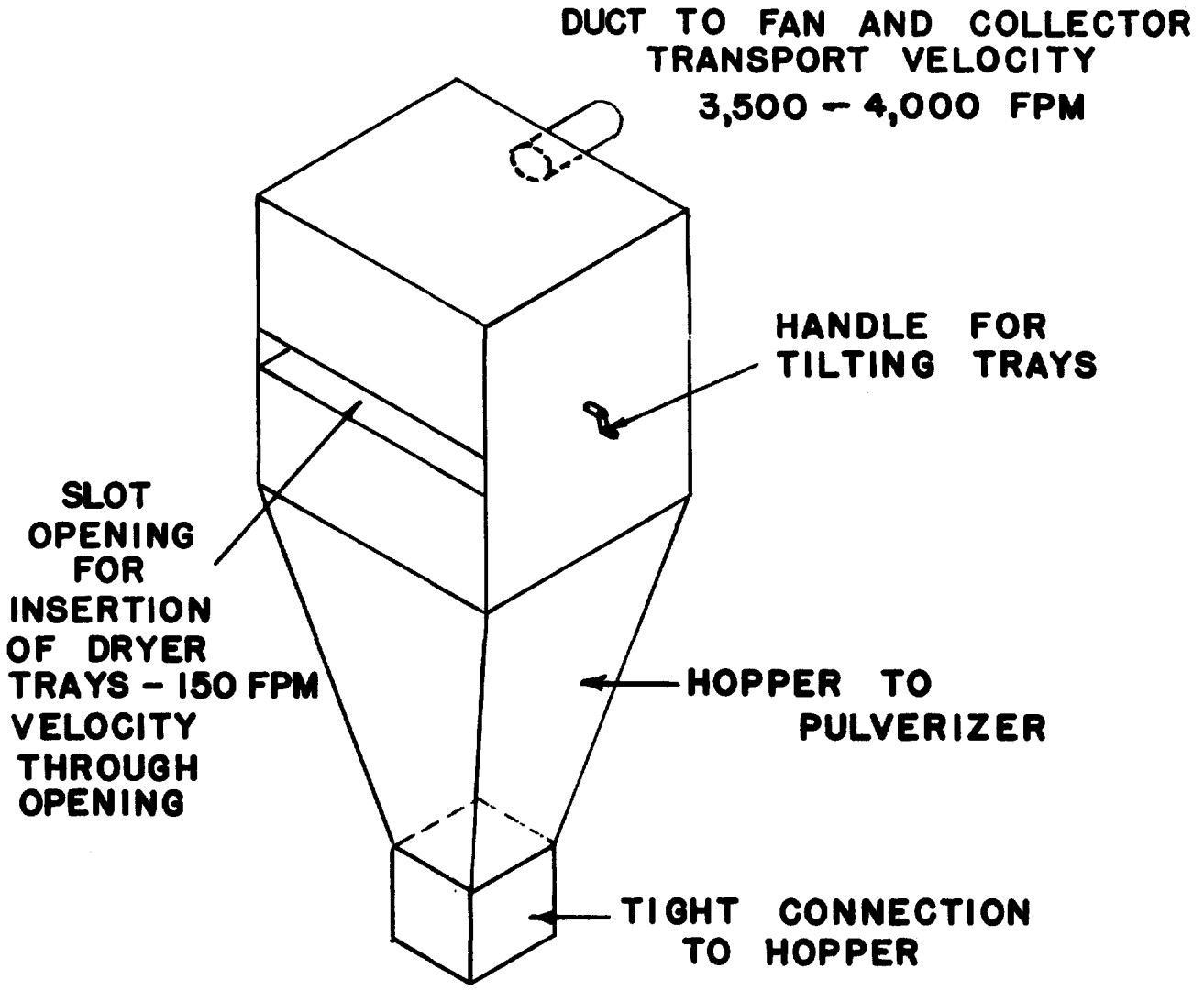


FIGURE X-3. ENCLOSURE FOR EMPTYING TRAYS OF CAKED POWDERS

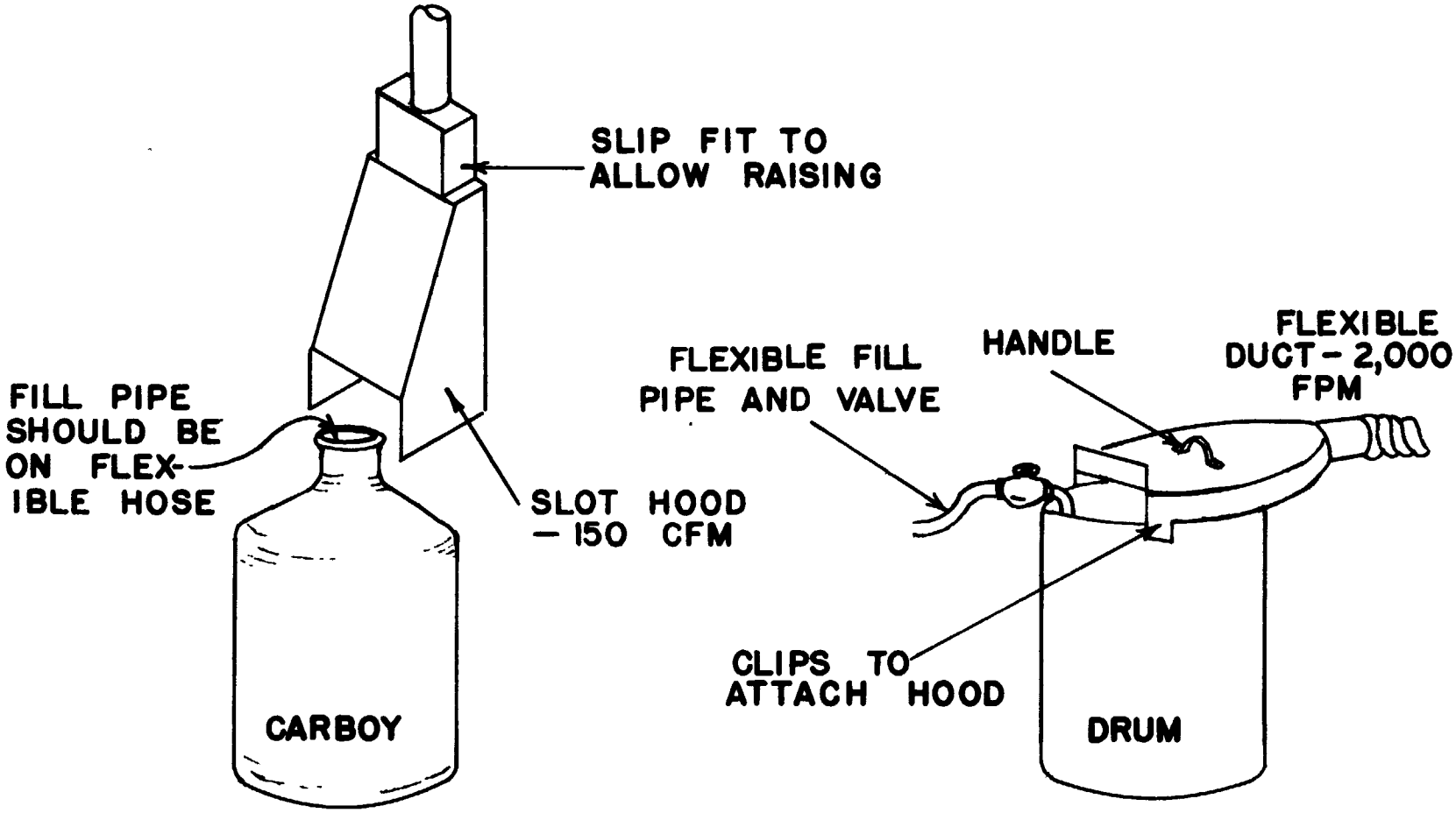


FIGURE X-4. TYPICAL ARRANGEMENTS FOR FILLING CONTAINERS

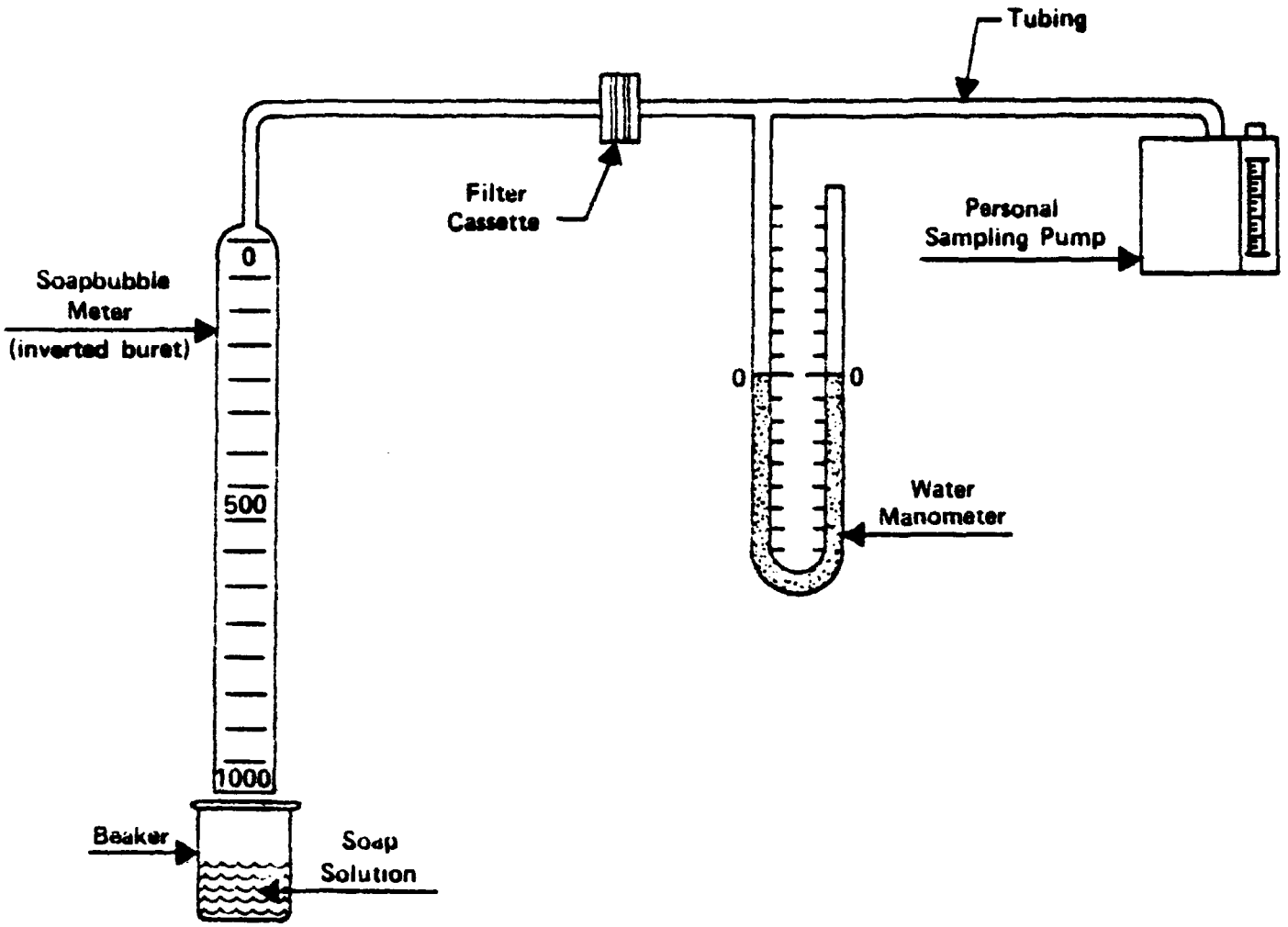


FIGURE X-5. CALIBRATION SETUP FOR PERSONAL SAMPLING PUMP WITH FILTER CASSETTE

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