U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES ■ Public Health Service
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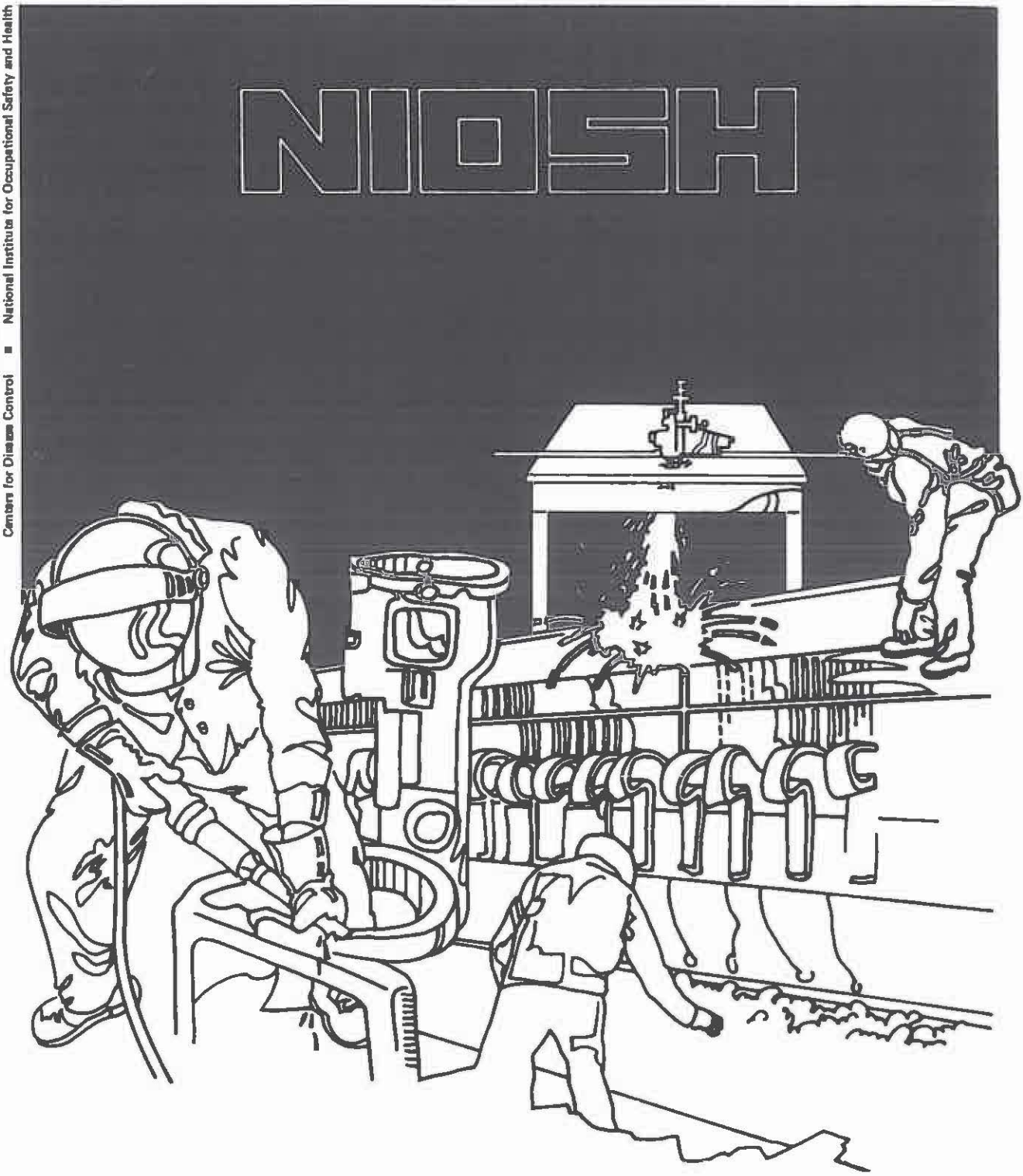

Health Hazard Evaluation Report

HETA 85-415-1806
ECCLES SAW AND TOOL COMPANY
CINCINNATI, OHIO

PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

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ECCLES SAW AND TOOL COMPANY
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I. SUMMARY

In August 1985, the National Institute for Occupational Safety and Health (NIOSH) received a request for a Health Hazard Evaluation from Eccles Saw and Tool Company, Cincinnati, Ohio, to evaluate metal dust exposures. The evaluation concerned exposures to tungsten carbide, cobalt and other metals generated by the process of sharpening saw blades and tools.

During the environmental evaluation personal and area air samples were collected for determination of cobalt, tungsten, cadmium, nickel and silver concentrations. Cobalt concentrations on the two personal samples were 26 ug/M^3 and 63 ug/M^3. The ACGIH Threshold Limit Value (TLV) for cobalt is 50 ug/M^3. Tungsten concentrations on these samples were 365 ug/M^3 and 925 ug/M^3. Nickel concentrations were 3.9 ug/M^3 and 9.3 ug/M^3. The NIOSH recommended exposure level (REL) for nickel is 15 ug/M^3. Cadmium and silver concentrations were low (<2 ug/M^3). All area concentrations were lower than those measured for the personal samples. A sample for cobalt and nickel determination was also collected on the exhaust side of the ventilation system. This was done to determine if metals were being entrained in the local exhaust ventilation air from the grinders, which was then recirculated back into the work area at a central location. The results indicated a cobalt concentration of 30 ug/M^3 and a nickel level of 7 ug/M^3 in the air being discharged back into the work area.

Three workers participated in the medical evaluation, which consisted of a questionnaire, pre- and post-shift pulmonary function tests, and determination of pre- and post-shift urine cobalt concentration. All three participants reported coughing 4-6 times per day, four or more days per week, and occasional episodes of wheezing. There were no significant changes over the workshift in pulmonary function parameters. The worker whose air cobalt level was 63 ug/m^3 had pre- and post-shift urine cobalt concentrations appreciably higher than those of 8 office workers. The other 2 study participants had urine cobalt concentrations that were mostly within the corresponding pre- or post-sift range found in the office workers.

Based on the data collected during this study, at least one worker was exposed to a excessive cobalt concentration. The study could not determine whether the chronic respiratory symptoms reported by the study participants were related to occupational cobalt exposure. Recommendations to reduce exposure, particularly from the practice of recirculating contaminated air from the local exhaust system are included in Section VIII of this report.

KEYWORDS: SIC 3541 (Machine Tools, Metal Cutting Types); tungsten carbide, cobalt, tungsten, nickel

II. INTRODUCTION

On August 15, 1985, the National Institute for Occupational Safety and Health received a confidential employee request to evaluate metal dust exposures at the Eccles Saw and Tool Company in Cincinnati, Ohio. The metal dusts of concern were tungsten carbide, cobalt and other metals generated by the process of sharpening saw blades and tools, an estimated 20-40% of which is tungsten carbide.

On August 29, 1985, NIOSH personnel conducted a walk-through inspection of the plant. Arrangements were made to conduct a follow-up medical and industrial hygiene survey September 19, 1985, during which two workers wore personal air sampling pumps during their workshifts to measure total breathing zone exposures to cobalt, tungsten carbide, cadmium, nickel and silver. Several air area samples and bulk samples were also obtained. Three workers volunteered to participate in the medical evaluation, which included a questionnaire, pre- and post-shift pulmonary function tests, and collection of pre- and post-shift urine specimens for determination of cobalt concentration.

III. BACKGROUND

Eccles Saw and Tool Company, which consists of four machining departments (metal circular saws, carbide circular saws, tool and cutter grinders, and knife departments), sharpens, repairs, and cleans high speed carbon steel and tungsten carbide tools. Sharpening is predominantly by wet grinding, although dry grinding of some tungsten carbide tools (milling saws) is performed. Repairs consist of retooling (replacement of tungsten carbide cutting edges by silver soldering) of circular saw blades and other cutting tools. The cobalt content of retooled parts ranges from five to eight percent. Cleaning, using silica sand, is performed in an abrasive blasting glove box located in the tool and cutter grinder department.

One local exhaust ventilation (LEV) system services the wet and dry grinders and the abrasive blasting operation; however, the exhausted air is recirculated back into the work area. Standard fiberglass filters (AmerKleen filters, manufactured by American Air Filter) were in place during this evaluation. High-efficiency particulate air (HEPA) filters were not used in the LEV system.

IV. EVALUATION DESIGN AND METHODS

A. Environmental

On September 19, 1985, air samples were collected to establish the grinders' exposure to cobalt and tungsten carbide. Personal breathing zone air samples for determination of total cobalt and tungsten exposures were collected on AA filters using flow rates of 1-1.5 liters per minute. These samples were also analyzed for cadmium, nickel and silver fumes that may have been generated from the brazing operation.

Area air samples were also collected during the survey. Area air samples for total dust concentrations of cobalt and tungsten carbide were obtained in the southeast corner of the carbide circular saw room and by the Hanchet knife grinder in the knife department. The exhaust side of the ventilation system was monitored for cobalt dust that may have become entrained in the local exhaust ventilation system and recirculated into the work area.

Six settled dust samples were obtained from the work areas; these were analyzed for cobalt. Three bulk liquid samples from several of the carbide grinders were collected for analysis of suspended cobalt.

B. Medical

Employees were asked to volunteer for a study which included a self-administered questionnaire, pre- and post-shift pulmonary function tests, a chest X-ray, and analysis of pre- and post-shift urine samples for cobalt and creatinine concentration.

The questionnaire addressed symptoms, smoking history, and occupational history.

Four valid pre- and post-shift spirometric curves were obtained from each participant. Forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1), and the forced expiratory flow rate between 25 and 75% of FVC (FEF_{25-75}) were measured with an Ohio Medical Model 822 dry rolling seal spirometer. Equipment and test procedures conformed to the American Thoracic Society's criteria for screening spirometry.[1] Predicted values for FEV_1, FVC, and FEF_{25-75} were calculated using the equations of Knudson.[2] For the determination of FEV_1, FVC, and FEV_1/FVC%, the largest FEV_1 and FVC from each set of 4 valid spirograms were used, regardless of the curve(s) on which they occurred. The FEF_{25-75} was calculated from the best curve, the one which gave the largest sum of FEV_1 and FVC.

Arrangements were made for each of the study participants to have a chest X-ray several months after the initial study, but none of them had this done.

Participants were requested to collect a morning urine sample at home (before they dressed for work, after a shower, and with their hair wrapped in a clean towel). When they arrived at work they placed surgical hats over their hair and a covering over beards, removed their work-shirt, and washed their hands and arms before

providing a second urine sample. Each worker repeated this procedure at the end of his shift. He also poured deionized water from a common, closed bottle into a urine collection container after he had given a urine sample. This served as a field blank. The workers then went home, showered, and collected an additional urine specimen approximately 1 hour after the end of the workshift.

All urine samples were processed within 2-3 hours of collection by the method specified by the laboratory at the Centers for Disease Control, Center of Environmental Health (CEH). This method specifies that 0.1 ml concentrated, ultra-pure nitric acid be added to a 15 ml plastic tube which had undergone a 1% ultra-pure nitric rinse to reduce metal contamination. Ten milliters $\pm$ 1.0 ml of urine was then added to the tube. This procedure was done under a NIOSH laboratory hood to reduce the possibility of airborne contamination. Also under the laboratory hood, deionized water was poured into the 15 ml tube to serve as a laboratory blank. The samples were frozen immediately and shipped frozen to the CEH laboratory. The urine samples were then analyzed by CEH's modified Zeeman Effect Graphite Furnace Atomic Absorption Method.[3] On August 29, 1985, similarly processed pre- and post-shift urine samples from 5 men and 3 women, all non-smokers, who worked in a Cincinnati office building with no known source of cobalt exposure served as controls. Two area air samples from this workplace had no detectable cobalt.

Urine was analyzed for creatinine by the Jaffe reaction method.[4] In order to standardize urinary cobalt to the degree of urine concentration, all urinary cobalt levels were corrected to a urine containing a creatinine concentration of 1 gram per liter (g/l).

V. EVALUATION CRITERIA

As a guide to the evaluation of the hazards posed by workplace exposures, NIOSH field staff employ environmental evaluation criteria for assessment of a number of chemical and physical agents. These criteria are intended to suggest levels of exposure to which most workers may be exposed up to 10 hours per day, 40 hours per week for a working lifetime without experiencing adverse health effects. It is, however, important to note that not all workers will be protected from adverse health effects if their exposures are maintained below these levels. A small percentage may experience adverse health effects because of individual susceptibility, a pre-existing medical condition, and/or a hypersensitivity (allergy).

In addition, some hazardous substances may act in combination with other workplace exposures, the general environment, or with medications or personal habits of the worker to produce health effects even if the occupational exposures are controlled at the level set by the evaluation criterion. These combined effects are often not considered in the evaluation criteria. Also, some substances are absorbed by direct contact with the skin and mucous membranes, and thus, potentially increase the overall exposure. Finally, evaluation criteria may change over the years as new information on the toxic effects of an agent become available.

The primary sources of environmental evaluation criteria for the workplace are: 1) NIOSH Criteria Documents and recommendations, 2) the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values (TLVs), and 3) the U.S. Department of Labor (OSHA) occupational health standards. Often, the NIOSH recommendations and ACGIH TLVs are lower than the corresponding OSHA standards. Both NIOSH recommendations and ACGIH TLVs usually are based on more recent information than are the OSHA standards. The OSHA standards also may be required to take into account the feasibility of controlling exposures in various industries where the agents are used; the NIOSH-recommended exposure limits, by contrast, are based primarily on concerns relating to the prevention of occupational disease. In evaluating the exposure levels and the recommendations for reducing these levels found in this report, it should be noted that industry is legally required to meet those levels specified by an OSHA standard.

A time-weighted average (TWA) exposure refers to the average airborne concentration of a substance during a normal 8- to 10-hour workday. Some substances have recommended short-term exposure limits or ceiling values which are intended to supplement the TWA where there are recognized toxic effects from high short-term exposures.

Tungsten Carbide

Cemented tungsten carbide is a unique metal commonly used in saw blades or cutting tools because its hardness approaches that of a diamond. Tungsten and carbon powders are first blended and then heated to form tungsten carbide. Cobalt (Co) in various amounts (3% to 25%) is added to tungsten carbide powder as a binding agent. Depending on the desired properties of the final product, other metal powders such as titanium carbide, tantalum carbide, chromium carbide, and nickel[6] may be added. Exposures to cobalt and other metal constituents occur during grinding, milling, and cutting of new tools, or during the resharpening and repair of old tungsten carbide tools.

Cobalt

Cobalt occurs naturally and is an integral part of the cyanocobalanium molecule (vitamin B_{12}) which is essential to the human diet to prevent the development of pernicious anemia.[7] The average U.S. daily cobalt intake from food, water and community air have been estimated to be 0.3 mg (milligrams), 0.006 mg and 0.0001 mg respectively.[8] Higher cobalt exposure can cause toxicity in humans.

Fibrotic lung changes have been observed in workers exposed to airborne cobalt concentrations of 0.1 - 0.2 mg cu m[6,9-14]. A common pattern of illness is described in these reports. The worker may first develop a cough, followed by labored breathing on exertion. There may be a substantial weight loss, and the individual may go on to develop a progressive interstitial pulmonary fibrosis (scar tissue in the lung) which may be accompanied by cor pulmonale (hypertension in the lungs), leading ultimately to cardiorespiratory collapse and death.[7] The association between inhaled cobalt metal and the development of lung fibrosis is supported in studies conducted in swine.[20]

The reported latency period from exposure to disease varies from a few to 20 years.[7] It is unclear whether this variable latency is related to individual susceptibility or different levels of exposure between studies.

A series of reports[21-23] describe lung function test results among 155 Swedish cemented carbide workers and 74 controls matched for sex, age and smoking history. Persons exposed to an average of 0.06 mg/m^3 airborne cobalt showed obstructive on pulmonary function changes over the week that did not improve over the weekend. Smokers were more affected than non-smokers.

Several investigations have suggested evidence of bronchitis among hard metal workers.[10-24] Asthma has been reported[10,11,25,26] as early as within one month after initial exposure.[6] The development of asthma seems to be a true sensitization to cobalt. The occurrence of allergic lung sensitization has heightened plausibility in view of the occurrence of documented cobalt allergic dermititis that has been reported among workers using cobalt containing materials.[26,27]

Sjogren et al[28] reported three non-smoking hard metal workers having symptoms and signs compatible with allergic alveolitis, the symptoms, signs and chest X-ray findings clearing with removal from work; and re-exposure leading to reoccurrence of symptoms and chest X-ray findings. All three had contact eczema and were sensitive to cobalt on skin patch testing. These workers were located in the section of the plant with the lowest measured air cobalt levels. They utilized a

cutting fluid when grinding tungsten carbide that caused a portion of the cobalt to dissolve and become ionized in the cutting fluid. Ionized cobalt, reportedly reacts with proteins[29], so it is biologically plausible that it could form a hapten, and possibly induce an allergic response.

There are a number of other physiological effects associated with cobalt. Cardiomyopathy (enlargement and dilation of the heart) has been associated with heavy consumption of beer made in the 1960's with cobaltous sulfate or cobaltous chloride as a foam stabilizer.[30-35] The signs and symptoms of the affected individuals (the majority who drank between 2 or more liters of beer per day) included abdominal pain, shortness of breath, lowered blood pressure, heart enlargement, pericardial effusion, tachycardia and electrocardiographic abnormalities. The amount of cobalt ingested daily by a 6 liter per day drinker was about 5-10 mg/day, much higher than that inhaled and absorbed by a worker breathing 10-15 m^3 at the current Federal OSHA limit of 0.1 mg/m^3.[7] Cobalt has been used to treat anemias because it has been shown to increase hemoglobin and hematocrit levels in humans.[36-44] Hypothyroidism and goiter have been associated with oral daily cobalt chloride doses of 2-10 mg/kg administered over a 2-4 month period in a small percentage of people.[7] Additional effects reported in humans but for which there is limited information available, include disturbed kidney function, hyperglycemia, mild to moderate changes in liver function tests and impaired sense of smell.[7]

The OSHA standard for cobalt is 0.1 mg/M^3. The American Conference of Governmental Industrial Hygienists has a proposed limit of 0.05 mg/M^3.

Cadmium

Cadmium is a toxic heavy metal which may enter the body either by ingestion (swallowing) or by inhalation (breathing) of cadmium-containing dust or oxide fume. Once absorbed into the body, cadmium accumulates in organs throughout the body, but major depositions occur in the liver and kidneys. Acute inhalation exposure to high levels of cadmium can cause pneumonia or pulmonary edema, as well as liver and kidney damage.[48] Chronic exposure may lead to emphysema of the lungs and kidney disease, or cancer of the prostrate.[49] There is also limited evidence that occupational cadmium exposure may be associated with lung cancer.[48,49]

NIOSH recommends that worker exposures to cadmium dust or fume be limited to not more than 200 ug/m^3 during a 15-minute ceiling period or to a threshold limit value (TLV) of not more than 40 ug/m^3, as a time-weighted average (TWA) over a 10-hour shift. The Occupational Safety and Health Administration (OSHA) standard for cadmium dust exposure is 200 ug/m^3, and for cadmium fume exposure 100 ug/m^3, using an 8-hour TWA for each.

Nickel

Airborne exposure to inorganic nickel compounds can cause erosion and perforation of the nasal septum and impairment of the sense of smell. Skin exposure can cause allergic contact dermatitis and those workers who become sensitive to nickel may also develop asthma.

Epidemiologic studies have shown that nickel refinery workers have an increased risk of lung, nasal, and kidney cancer. The nickel compounds most commonly found during refining are nickel sulfate, nickel sulfide, and nickel oxide, but prudence dictates that all nickel compounds should be considered carcinogenic until further studies are conducted. NIOSH recommends a TWA exposure limit of 15 ug/M^3. The OSHA permissible exposure limit is 1 mg/M^3 for an 8-hour TWA exposure.[50]

Silver

Exposure to silver dust can cause a local or generalized impregnation of mucous membranes, skin and eyes. This condition called agrygria appears as grey-blue pigmentation. Inhalation of silver dust may cause such pigmentation of the respiratory tract and may cause a mild bronchitis.[51]

The OSHA standard for silver metal and soluble compounds is a TWA of 0.01 mg/m^3. The American Conference of Governmental Industrial Hygienists recommends a TWA of 0.1 mg/m^3 for silver metal, and 0.01 mg/m^3 for soluble metal compounds.

VI. RESULTS AND DISCUSSION

A. Environmental

Results of the personal and area air samples collected for particulates and metal analyses are presented in Table 1. Results indicate personal exposures to cobalt ranging from 26 to 63 ug/M^3. One of the two personal samples exceeded the ACGIH TLV of 50 ug/M^3. Area concentrations were lower, ranging from 2.8 ug/M^3 to 14 ug/M^3. Tungsten concentrations were 365 ug/M^3 and 925 ug/M^3 for the personal samples and ranged from below the limit of quantitation (LOQ) up to 261 ug/M^3 on the area samples (TLV - 1000 ug/M^3). Nickel concentrations were 3.9 ug/M^3 and 9.3 ug/M^3 for the personal samples (PEL - 15 ug/M^3), while the area concentration ranged from below the LOQ to 1.8 ug/M^3. Cadmium and silver concentrations were well below their evaluation criteria.

Analyses of the three bulk liquid cutting fluid samples showed cobalt concentrations of none detected, 441 ug/10 ml of liquid and 3290 ug/10 ml liquid. Variability in cobalt concentration is probably related to the age of cutting fluid and the type of work conducted.

Analyses of settled dust samples for cobalt showed concentrations ranging from 0.2% up to 2%. (Table II).

All the local exhaust ventilation provided to the shop was handled by a single, recirculating system. As a result, a sample for cobalt and nickel determination was collected on the exhaust side of the ventilation. This was done to determine if metals were being entrained in the local exhaust system and being recirculated back into the work area. The results indicated a cobalt concentration of 30 ug/M^3 and a nickel concentration of 7 ug/M^3 being discharged back into the work area. Analyses of the fiberglass filter in the ventilation system also indicated a cobalt content of approximately 0.3%.

The settled dust samples, together with the levels of contamination present in the recirculated exhaust air, indicate high cobalt contamination through out the facility.

B. Medical

Three workers participated in the medical survey. Two were grinders with 15 and 85 months employment at the company. The other employee was a truck driver who spent approximately three hours per day in the shop, but did no grinding. They did not routinely use respirators, and their work clothes were laundered at home. Two workers reported frequent sore throats; one worker reported frequent rash and eye irritation. All three participants reported coughing 4-6 times a day, for four or more days per week; they also reported occasional episodes of wheezing. One worker reported shortness of breath after walking on level ground for a few hundred yards. All three participants smoked cigarettes.

There were no significant changes over the workshift in pulmonary functon tests when pre- and post-shift FEV_1, FVC, and FEF_{25-75} were compared. The FEV_1/FVC ratio in two workers was mildly reduced. One of them had a cough and respiratory infection at the time of testing.

Both the deionized water field and laboratory blanks showed evidence of cobalt contamination. One bottle of deionized water was used for both laboratory and field blanks, and it is possible

that the bottle was contaminated in the field. Despite the problems with the field and laboratory blanks, the cobalt concentrations in urine samples obtained at home, both pre- and post-shift, were in good agreement with those in the corresponding samples collected in the workplace restroom (Table III). The worker with highest air cobalt level also had the highest pre- and post-shift urine cobalt concentrations, which were appreciably higher than those of the office workers (Table IV). The other two workers, however, had urine cobalt concentrations that were mostly within the corresponding pre- or post-shift range found in the office workers.

VII. Conclusions

One of the workers who had a personal breathing zone air sample for cobalt had a concentration that exceeded the evaluation criterion.

This study could not determine whether the chromic respiratory symptoms reported by the participants were due to occupational cobalt exposure, smoking, or some other cause.

VIII. RECOMMENDATIONS

1. General housekeeping practices in the grinding shop need to be improved.

2. The present local exhaust ventilation system should be modified so that it discharges air outside the work air rather than recirculating contaminated air. The discharge of the local exhaust ventilation air into the work area appears to be a major contributor to the cobalt and nickel concentrations. NIOSH recommends that air containing toxic contaminants not be recirculated. If air is recirculated, the fiberglass filter used in this plant is not the appropriate filter type for use in an industrial environment.

3. Workers exposed to cobalt should have pre-placement and periodic medical evaluations, which should routinely include pulmonary function tests performed using standardized procedures.[1] Chest X-rays need not be taken more often than every 2-5 years unless there is some other medical reason.[52]

4. Although the health significance of the urine cobalt concentrations measured in the Eccles employees is unknown, prudence would dictate that excess cobalt absorption be kept to a minimum because cobalt has been associated with a number of health effects.

IX. REFERENCES

1. "ATS Statement - Snowbird Workshop on Standardization of Spirometry. American Thoracic Society", Am Rev Respir Dis 119:833-838, 1979.

2. Knudson RJ et al. Maximal Expiratory Flow-Volume Curve", AM Rev Respir Dis 113:587-600, 1976.

3. Slavin W, Carnick G, Manning D and Truszkowska E: "Recent Experiences with the Stabilized Temperature Platform Furnace and Zeeman Background Correction", Atomic Spectroscopy 4 (3):69-86, 1983.

4. Annino JS, Clinical Chemistry Principals and Procedures, 3rd Ed., Little Brown and Company, Boston, 1964.

5. Schumacher-Wittkopf E and Angerer J., Praxisgerechte Methode Zur Kobalt Bestimmung in Harn. Int Arch Occup Envir Health, 49:77, 1981.

6. Coates OE and Watson J, Diffuse Interstitial Disease in Tungsten Carbide Workers, Annals of Internal Medicine, 75:709-716, 1971.

7. Criteria for Controlling Occupational Exposure to Cobalt. HEW Publication No. (NIOSH) 82-107. Cincinnati, Ohio, USDHEW, PHS, CDC, NIOSH, 1982.

8. Schroeder, HA, Nason AP, Tipton IH: "Essential Trace Metals in Man - Cobalt", J Chronic Dis, 20:869-90, 1967.

9. Scherrer M, Parambadathumalail A, Burki H, Senn A, Zurcher R: (Three Cases of "Hard Metal" Pneumoconiosis). J Suisse Med, 100:2251-55, 1970 (Ger).

10. Reber E, Burckhardt P: Hard Metal Pneumoconiosis in Switzerland, Respiration, 27:120-42, 1970 (Ger).

11. Bech AO: Hard Metal Disease and Tool Room Grinding, J Soc Occup Med, 24:11-16, 1974.

12. Husten K: Hard-metal Fibrosis of the Lungs, Arch Gewerbepathol Gerwerbehyg, 15:721-92, 1959 (Ger).

13. Einbrodg HJ, Kuhne W: (Lung dust and morphological picture of a hard metal lung) in Reploh H, Klosterkotter W (eds.): Fortschritte der Staublungenforschung. Monograph on the Fourth International Dust-Lung Meeting of April 3-5, 1962, in Munster, Westfalen. Dinslaken, Niederrheinische Druckeri, 1963, pp. 217-26 (Ger).

14. Collet A, Liot F, Gallouedec C, Roussel G, Martin J, Reuet C, Brouet G: Electron Microscopy Study of Various Cellular Aspects of Pulmonary Fibroadenomatoses with Diffusion Disorders - Discussion on the Etiological Role of Cobalt and Tungsten Carbide, Rev Tuberc (Paris) 27:357-81, 1963 (Fre).

15. Rochemaure J, Ancla M, Trinquet G, Meyer A: A case of pulmonary bifrosis - Possible role of tungsten dust, J Fr Med Chir Thjorac, 26:305-12, 1972 (Fre).

16. Joseph M: Hard metal pneumoconiosis. Australas Radiol, 12:92-95, 1968.

17. Bartl F, Lichtenstein ME: Tungsten carbide pulmonary fibrosis - A case report. Am Ind Hyg Assoc J, 37:668-70, 1976.

18. Teyssier L, Guerin L, Frey N, Lesobre R: Pulmonary fibrosis observed in the hard metal industry. Arch Mal Prof, 36:53-56, 1975 (Fre).

19. Siegesmund KA, Funahashi A, Pintar K: Identification of metals in lung from a patient with interstitial pneumonia. Arch Environ Health, 28:345-49, 1974.

20. "Cobalt Metal Inhalation Studies on Miniature Swine", Am Industrial Hyg Assoc J, 36:17-25, 1975.

21. Alexandersson R: Studies on Effects of Exposure to Cobalt: II. Reactions of the Respiratory Organs of Various Exposure Levels in the Hardmetal Industry. Arbete Och Halsa, 2:1-34, 1979 (Swe).

22. Alexandersson R: Studies on Effects of Exposure to Cobalt: VI. Exposure, Uptake, and Pulmonary Effects on Cobalt in the Hardmetal Industry. Arbete Och Halsa, 10:1-24, 1979 (Swe).

23. Alexandersson R, and Hedenstierna G: Studies on Effects of Exposure to Cobalt: III. Ventilation Capacity, Distribution of Inhaled Gas, and Closing of Respiratory Passages During Ongoing Work and After Periods of Non-Exposure. Arbete Och Halsa, 7:1-25, 1979 (Swe).

24. Jobs H and Ballhausen C: The Medical and Technical Viewpoints of Metal Ceramics as a Source of Dust. Vertrauensorzt KranKenKasse, 8:142-48, 1940 (Gre).

25. Bruckner HC: Extrinsic Asthma in a Tungsten Carbide Worker. Ann Arbor Reports, 9:518-9, 1967.

26. Pirila V: Sensitization to Cobalt in Pottery Workers. Acta Derm Venereol, 33:193-198, 1953.

27. Rystedt I and Fischer T: Relationship Between Nickel and Cobalt Sensitization in Hard Metal Workers. Contact Dermatitis, 9:195-200.

28. Sjorgren I, Hillerdal G, Anderson A and Zelterstrom O: Hard Metals Lung Disease: Importance of Cobalt in Coolants. Thorax, 35:653-659, 1980.

29. Hellsten E, Blomberg M, Henrikson-Enflo A, Sundbom M and Vokal H: "Kobalt (Copbalt)" Stockholm: University of Stockholm, Institute of Physics , 1976, Report 76.

30. Alexander CS: Cobalt-beer cardiomyopathy - A clinical and pathologic study of twenty- eight cases. Am J Med, 53:395-417, 1972.

31. Morin YL, Foley AR, Martineau G, Roussel J: Quebec beer-drinkers' cardiomyopathy - Forty-eight cases. Can Med Assoc J, 97:881-83, 1967.

32. The mystery of the Quebec beer drinkers' cardiomyopathy. Can Med Assoc J, 97:930-31, 1967 (editorial).

33. Sullivan JF, Egan JD, George RP: A distinctive myocardiopathy occurring in Omaha, Nebraska - Clinical aspects. Ann NY Acad Sci, 156:526-43, 1969.

34. Kesteloot H, Roelandt J, Willems J, Claes JH, Joossens JV: An inquiry into the role of cobalt in the heart disease of chronic beer drinkers. Circulation, 37:854-64, 1968.

35. Kerr A Jr: Myocardiopathy, alcohol, and pericardial effusion. Arch Intern Med, 119:617-19, 1967.

36. Berk L, Burchenal JH, Castle WB: Erythropoietic effect of cobalt in patients with or without anemia. N Engl J Med, 240:754-61, 1949.

37. Kato K: Iron-cobalt treatment of physiologic and nuitritional anemia in infants. J Pediat, 11:385-96, 1937.

38. Coles BL: The use of cobalt in some common anemias of childhood. Arch Dis Child, 30:121-26, 1955.

39. Robinson JC, James GW III, Kark RM: The effect of oral therapy with cobaltous chloride on the blood of patients suffering with chronic suppurative infection. N Engl J Med, 240:749-53, 1949.

40. Lindblad G, Wegelius R: Effect of cobalt on the reticulocyte counts of young premature infants. Ann Paediat Fenn, 3:103-08, 1957.

41. Rohn RJ, Bond WH: Observations on some hematological effects of cobalt-iron mixtures. J Lancet, 73:317-24, 1953.

42. Wolf J, Levy IJ: Treatment of sickle-cell anemia with cobalt chloride. AMA Arch Intern Med, 93:387-96, 1954.

43. Coles BL, James U: Use of cobalt and iron in the treatment and prevention of anemia of prematurity. J Lancet, 75:79-82, 1955.

44. Duckham JM, Lee HA: The treatment of refractory anemia of chronic renal failure with cobalt chloride. Q J Med, 45:277-94, 1976.

45. Shabaan AA, Marks V, Lancaster MC, Dufeu GN: Fibrosarcomas induced by cobalt chloride (CoCl2) in rats. Lab Anim, 11:43-46, 1977.

46. Heath JC, Daniel MR, Dingle JT: The Carcinogenic and Metabolic Effects of Cobalt and Other Metals. Annu Rep Br Enip Cancer Comp, 39:334-40, 1961.

47. Heath JC and Daniel MR: The Production of Malignant Tumours by Cobalt in the Rat - Intrathoracic Tumours. Br J Cancer, 15:473-78, 1962.

48. Scott R, Paterson PJ, Mills EA, et all: Clinical and biochemical abnormalities in coppersmiths exposed to cadmium. Lancet, 2:396-98, 1976.

49. Webb M: Cadmium. Br Med Bull, 31:246-50, 1975.

50. National Institute for Occupational Safety and Health. Criteria for a recommended standard: Occupational Exposure to Inorganic Nickel. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1977. (DHEW Publication No. (NIOSH) 77-164).

51. Proctor NH and Hughes JP: Chemical Hazards in the Workplace. J.B. Lippincott Co., Philadelphia, 1978.

52. American Thoracic Society. Surveillance for respiratory hazards in the occupational setting. Am. Rev. Respir. Dis. 1982;122:952-6.

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1. Eccles Saw and Tool Company, Cincinnati, Ohio
2. OSHA, Region V

For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.

Table I

Airborne Metal Concentrations
Eccles Saw and Tool Company
Cincinnati, Ohio
HETA 85-415
September 19, 1985

Job/Location	Sample Type	Sampling Time	Cobalt	Tungsten	(ug/M^3) Nickel	Cadmium	Silver
Grinder	PB2	0736-1612	26.0	365.4	3.9	(0.8)	2.0
Grinder	PB2	0738-1610	63.0	924.8	9.3	1.1	0.9
Knife Dept.	A	0756-1545	2.8	(45.6)	(1.1)	N.D.	N.D.
Cutter Grinder (North Wall)	A	0809-1551	11.5	261.5	1.4	N.D.	N.D.
Cutter Grinder (East Wall)	A	0812-1553	14.3	250.4	1.8	(0.3)	N.D.
Circular Saw Room	A	0818-1550	8.9		1.5	1.1	(0.2)

Table II

Cobalt Content In Settled Dust
Eccles Saw and Tool Company
Cincinnati, Ohio
HETA 85-415
September 19, 1985

Sample Location	Sample Type	Cobalt Content(%)
Top of circuit breaker box by air compressor, NW corner of circular saw room	Dust	2.0
Top of box by steel band saw	Dust	0.3
Top of light above cutter/grinder on east wall	Dust	1.4
Ledge beside grinder, south east corner circular saw room	Dust	0.2
Ledge of furnace	Dust	0.5

Table III

Biological and Environmental Cobalt Levels
Eccles Saw and Tool Company
Cincinnati, Ohio
HETA 85-415
September 19, 1985

Air Cobalt Concentration (mg/m^3)	Urine Cobalt Concentration (ug/gram of creatinine)			
	At Home		At Work	
	Pre-Shift	Post-Shift	Pre-Shift	Post-Shift
0.063	15.6	21.5	19.1	23.8
0.026	5.7	12.9	-	15.1
N.D.	9.5	7.2	4.9	8.0

Table IV

Pre and Post-Shift Urinary Cobalt Levels
Office Workers
Cincinnati, Ohio
HETA 85-415
September 19, 1985

	Cobalt Concentration (ug/g creatinine)	
	Pre-Shift	Post-Shift
Males		
	4.1	12.9
	*	3.9
	2.5	3.1
	1.8	13.2
	2.1	*
Females		
	3.3	2.6
	*	2.6
	*	3.6

* = Not determinable. The limit of detection for cobalt was 3.4 ug/l. If no cobalt was detected in the sample, no creatinine correction was possible. An entry of "*" may represent a urine cobalt concentration higher or lower than other, numerical values in the table.

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