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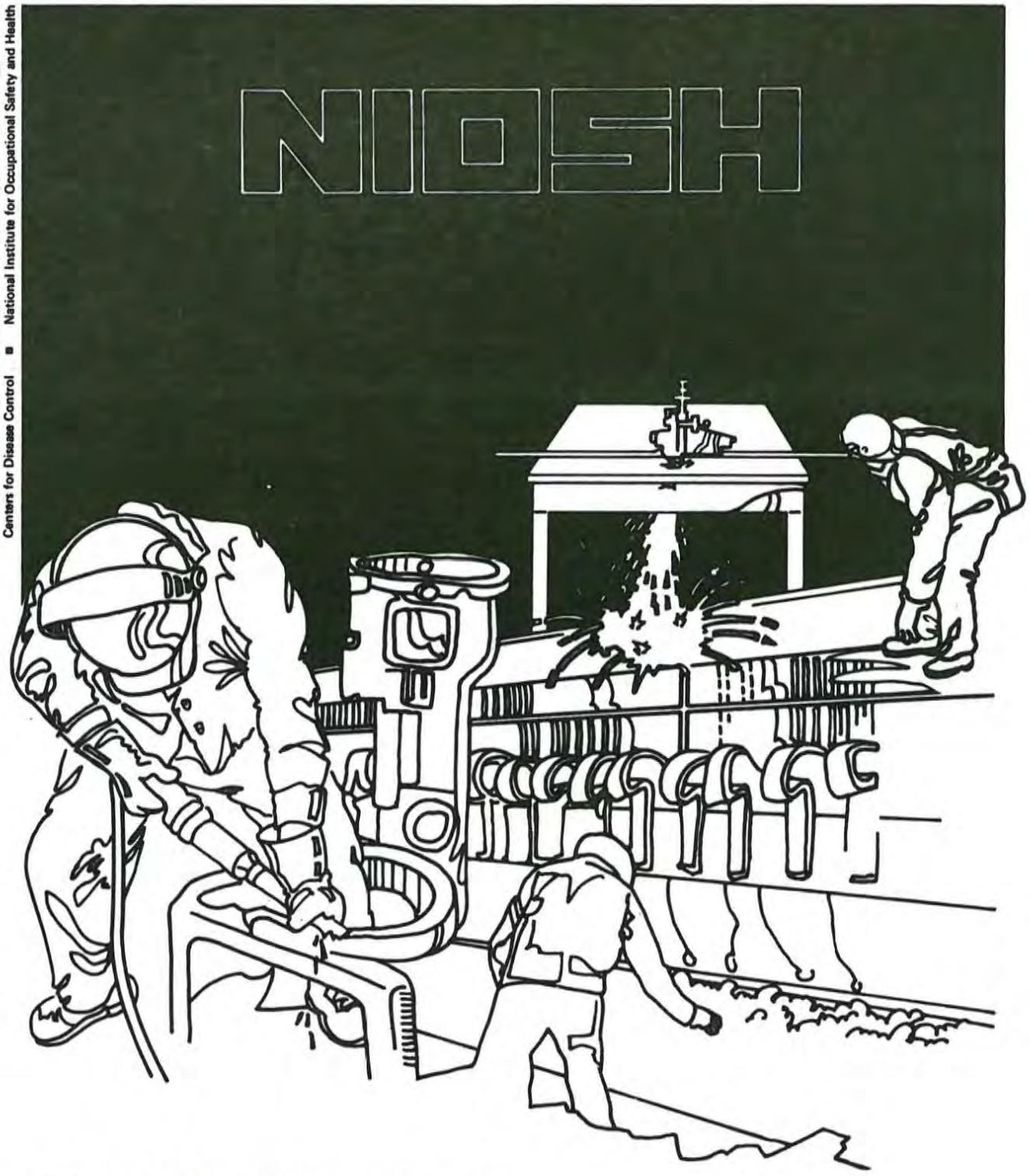

Health Hazard Evaluation Report

HETA 80-234-1196
MASTER METALS, INCORPORATED
CLEVELAND, 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.

HETA 80-234-1196
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MASTER METALS, INCORPORATED
CLEVELAND, OHIO

NIOSH INVESTIGATORS:
James M. Boiano, I.H.
P. Lynne Moody, M.D.

I. SUMMARY

In September 1980, the National Institute for Occupational Safety and Health (NIOSH) received a request to evaluate exposures to metals at Master Metals, Incorporated, a secondary lead smelter in Cleveland, Ohio. Health complaints consisting of digestive disturbances, constipation, and headaches were reported.

On September 25-26, 1980, and May 6-7, 1981, NIOSH investigators conducted an environmental and medical evaluation of the production workers . Further medical testing was conducted on October 20-22, 1981. Environmental measurements were made to determine worker exposure to lead, arsenic, arsine, stibine, and sulfur dioxide. The medical evaluation consisted of blood lead determinations, confidential questionnaires, blood pressure measurements, and blood and urine tests of kidney and erythropoietic function.

Air lead concentrations in 17 personal samples ranged from 159 to 4830 micrograms per cubic meter of air (ug/M^3), all exceeding the OSHA lead standard of 50 ug/M^3. General area air samples indicated that nonproduction areas, as well as production areas, were contaminated with lead. Airborne arsenic concentrations in four of eleven personal samples exceeded the NIOSH recommended criterion of 2.0 ug/M^3; one sample exceeded the OSHA standard of 10 ug/M^3. Personal and general area air samples for arsine, stibine, and sulfur dioxide were below the limits of detection.

NIOSH medical surveys consistently demonstrated: (1) high lead absorption, with 42 to 90% of the blood leads above 60 ug per deciliter in the three NIOSH surveys; (2) a high prevalence of low hemoglobins (25 to 42%) with grossly elevated free erythrocyte protoporphyrins, and (3) a high prevalence of kidney function abnormalities (39 to 54%).

On the basis of the environmental and medical data, NIOSH had determined that a serious health hazard of overexposure to airborne lead and arsenic existed at Master Metals, Incorporated, Cleveland, Ohio. A high prevalence of anemia and kidney function abnormalities suggests long-standing, severe lead toxicity among workers. Recommendations on engineering controls, work practices, and housekeeping to control these hazards are presented in Section VII of this report.

KEYWORDS: SIC 3341 (Secondary Smelting and Refining of Nonferous Metals), lead, blood lead, arsenic, arsine, stibine, sulfur dioxide, lead nephropathy, anemia, n-acetyl-beta-D-glucosaminidase.

II. INTRODUCTION

On September 2, 1980, the National Institute for Occupational Safety and Health (NIOSH) received a request for a health hazard evaluation from United Steelworkers of America - Local 735 to investigate health complaints of workers at Master Metals, Incorporated, in Cleveland, Ohio. Reported symptoms included digestive disturbances, constipation, and headaches. NIOSH conducted an environmental/medical evaluation at this facility on September 25-26, 1980, and May 6-7, 1981, and further medical evaluation on October 20-22, 1981, to determine worker exposure to lead, arsenic, and other contaminants during lead smelting.

The results of the September 1980 survey were presented in a November 1980 interim report. This report presented environmental and biological sample results and seven recommendations to reduce employees' exposure to inorganic lead. The results of the May 1981 survey were presented in a letter report issued in August 1981.

Prior to the first followup visit, the Occupational Safety and Health Administration (OSHA) conducted an environmental evaluation at the facility in April 1981. Based on their findings, a citation was issued for lead overexposure.

III. BACKGROUND

A. Plant History

Master Metals, Incorporated, is a secondary lead smelter. This facility reclaims lead from scrap batteries to produce pure (99.97% or greater purity) lead and antimonial lead alloys. The plant has been in operation since the early 1930's and since then has been involved in three ownership changes, the most recent in September 1979 when it was purchased from National Lead Industries.

Master Metals, Incorporated operates one blast furnace, one reverberatory furnace, and up to eight kettle furnaces. Major plant areas include: raw material receiving; battery breaking or decasing; smelting, refining, alloying, and casting (SRA&C); ingot storage; and baghouses. SRA&C operations are continuous, 7 days a week, 3 shifts per day. All other operations, including maintenance activities, are conducted during the first two shifts.

Minimum shift manpower requirements for the receiving, decasing, and SRA&C operations are one, four and six workers, respectively. These workers include two battery unloaders, one saw operator and one helper in the former operation, and one furnace feeder, one blast furnace operator, two casters, one baghouse operator and one forklift operator in the latter operations. (More workers were involved in these operations during the initial site visit in September 1980, but were laid off because of lead scrap shortages.)

B. Process Description

The scrap batteries are received by truck, manually unloaded, and then decased in a sawing operation. The lead scrap from the battery plates and terminals is retrieved and stockpiled outdoors. As needed, the scrap is transported by front-end loader, dumped into a weigh-hopper, and mechanically transferred into the top of the blast furnace. Coke, scrap iron, limestone, return slag and dross, and flue dust are also charged. During smelting, crude lead is continuously tapped from the blast furnace into one of two holding kettles. Depending on the demand, the crude lead is either alloyed with antimony, arsenic and/or tin or further refined to pure lead in a reverberatory furnace or kettle furnaces. Once the product specifications are met, the pure or alloyed lead is manually piped into water-cooled molds, drossed, cooled, then stacked on pallets for warehousing and subsequent shipment.

Flue dusts from the smelting and refining operations are collected in two baghouses. The metallurgical baghouse, a two-story brick building which contains nine bag cells, is used to collect flue dust emissions from the blast and reverberatory furnaces. The bags are manually rapped by the baghouse operator to remove filtered flue dust, which is then collected and recycled. The sanitary baghouse collects fugitive emissions from the kettles. The filtered dust is automatically removed from the bag filters. The dust is collected in steel bins and recycled.

C. Personal Protective Equipment and Hygiene

Respiratory protection is required in most areas of the smelter. Full-face, air-supplied respirators are used routinely by the decasers and baghouse operator. The remaining employees are required to wear high-efficiency filter, half-face respirators. The company has a respirator program which incompasses all aspects outlined in 29 CFR 1910.1025. The adequacy of the fit-testing aspect of the program was questionable since beards were prevalent among workers, especially during the initial survey. Other personal protective equipment worn in this smelter includes safety glasses, safety shoes, hard hats, and protective clothing (all company supplied). Face shields were worn by the blast furnace operator and casters.

Double change room and shower facilities are provided.

D. Housekeeping

The floor of the SRA&C building and surrounding smelter grounds were hosed down with water at least once a shift to reduce dust reintrainment. Raw material stockpiles were sprayed with water by fixed sprinklers.

IV. EVALUATION DESIGN AND METHODS

A. Environmental

The September 1980 initial survey was limited to SRA&C operations, where the highest lead concentrations were anticipated. Fourteen full-shift personal and general area airborne samples for lead were collected on 37-mm mixed cellulose ester membrane filters using personal sampling pumps calibrated at 1.5 liters per minute (Lpm). Analysis was performed according to NIOSH Analytical Method S-341.[1]

On the May 1980 follow-up survey, NIOSH investigators sampled for lead, arsenic, arsine, stibine, and sulfur dioxide. Lead monitoring focused on the maintenance men and forklift operators and their work areas since these jobs/areas were not sampled during the initial site visit. Sixteen personal and general area samples were collected and analyzed for lead by the methods previously described. No sampling was done on the battery unloaders or decasers since the battery breaking operation was idle due to repairs.

Full-shift, time-weighted average (TWA) samples for arsenic, arsine, and stibine were obtained from ten SRA&C workers using a sampling train consisting of a 13-mm mixed cellulose ester membrane filter (for particulate arsenic) followed by a 150-mg charcoal tube (for arsine and stibine). The sampling train was connected to a personal sampling pump calibrated at 0.2 Lpm. The filters were analyzed for arsenic according to NIOSH Method P&CAM No. 286.[2] The charcoal tubes were analyzed for arsine and stibine via NIOSH Method P&CAM No. S-229.[1]

In addition to the full-shift arsenic samples, fifteen-minute personal samples for arsenic were collected on the blast furnace operator and casters. Up to three short-term samples were collected on five workers during slag tapping and casting operations since the peak exposure potential appeared to be greatest during these tasks. Samples were collected on 37-mm mixed cellulose ester membrane filters using calibrated personal sampling pumps operating at 1.5 Lpm. NIOSH Analytical Method P&CAM No. 286 was used to analyze these filters.[2]

The short-term arsenic samples discussed above should not be confused with the traditional "ceiling" designation occasionally assigned to "fast-acting" chemicals which require 15-minute exposure determinations. The 15-minute sampling period is advised by NIOSH to avoid spurious sampling results produced by "background" concentrations of inorganic arsenic (in light of the extremely low recommended standard and documented occurrences of ambient arsenic levels as high as 1.4 ug/M^3).

Direct-reading, long-term detector tubes were used to evaluate SRA&C worker exposure to sulfur dioxide. Five personal samples and one general area air sample were obtained by drawing workroom air through the detector tube using a low-flow personal sampling pump calibrated at a flow rate of 0.010 Lpm. Samples were collected for approximately four hours, and the tubes were read immediately.

B. Medical

NIOSH conducted an initial site visit and two follow-up medical studies at Master Metals. During the initial visit in September 1980, NIOSH interviewed a systematic sample of 35 of the 93 then current hourly and supervisory personnel selected from seniority lists, and obtained a blood sample for a blood lead on 33 of the 35 workers interviewed. The questionnaire used for the interviews, elicited demographic information, symptoms associated with lead and arsenic toxicity, other health problems, and job information, (activities, duration, dustiness, respirator type). Blood lead assays were performed by a NIOSH contract laboratory, with split samples analyzed by NIOSH. In addition to collecting our own questionnaire and blood lead data, NIOSH investigators reviewed available company blood lead records for the preceding year on those workers in our survey sample. This review was conducted to obtain a longitudinal view of blood lead levels at the plant.

Because consistently high blood lead levels were found in all but a few workers sampled during the September site visit, and because occupational lead nephropathy in modern American industry is a subject of some controversy, NIOSH returned in May 1981 to screen all hourly workers still employed at the plant. Medical testing included several well-established parameters of kidney function (blood urea nitrogen or BUN, and serum creatinine) as well as two less frequently used measurements of tubular function: urine concentrating ability after a 12-hour water fast, and the measurement of a renal proximal tubular enzyme, n-acetyl-beta-D-glucosaminidase (NAG), in a spot urine sample. NIOSH tested urine for gross glucose and protein by the "dipstick" method and microscopically examined urinary sediment for the presence of cells or other evidence of glomerular or tubular disease. Blood lead measurements were repeated and evidence of hematopoietic effect was sought by measuring free erythrocyte protoporphyrin (FEP), hemoglobin, and hematocrit. In addition, a medical questionnaire was used to obtain information on various risk factors for kidney disease (hypertension, diabetes, kidney stones, prostatic hypertrophy, recurrent urinary tract infections, analgesic abuse, and heroin use). Blood lead, hemoglobin, and FEP analyses were performed by a NIOSH contract laboratory. The Clinical and Biochemical Support Section, Technical Services Branch, NIOSH, performed confirmatory blood lead analyses, and BUN and creatinine. The Department of Laboratory Medicine at the

University of Connecticut, Farmington, performed the analyses for NAG and creatinine on the urine samples. Microscopic urine sediment analysis and hematocrits were done at the study site by a member of the NIOSH medical staff.

The May medical follow-up study found a high prevalence of both abnormal BUN and serum creatinines (29% and 54%, respectively). Because of these findings, NIOSH returned for a second medical follow-up visit in October 1981 to attempt a more specific characterization of glomerular function and other associated effects of kidney disease. Workers were asked to collect all their urine during an 8-hour work shift. Blood samples were drawn for determination of creatinine, BUN, calcium, phosphorus, and uric acid, and a creatinine clearance was calculated for each worker. In addition, NIOSH investigators again measured blood lead, FEP, and hemoglobin, as well as serum iron, iron binding capacity, and binding capacity saturation, in an effort to clarify other features of hematopoietic status. All workers then at the plant were asked to participate. NIOSH also requested that the union contact any workers recently laid off who had participated in the last study and ask them to participate again. The effort to find a local group of age-, race-, and sex-matched workers was pursued at some length through the local and international unions, but a suitable comparison group could not be found. Instead, as a rough comparison, reference was made to a group of paint manufacturing workers recently evaluated by NIOSH on whom blood tests (especially BUN and creatinine) were performed by the same NIOSH contract laboratory within the same year.

V. EVALUATION CRITERIA

Environmental

To assess the concentrations of air contaminants found during this investigation, three primary sources of criteria were used: (1) NIOSH recommended standards, (2) American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLV's), and (3) Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PEL), U.S. Department of Labor (29 CFR 1910.1000). These criteria are generally established at levels intended to protect workers occupationally exposed during an 8- or 10-hour workday over a normal working lifetime. The environmental criteria of the substances evaluated in the study along with brief descriptions of their primary health effects are presented in Table I. For evaluation purposes, those which are the most stringent and thus afford the best health protection for the worker, will be applied.

For those substances which were found to be in excess of the limits expressed in the evaluation criteria the following discussion is presented.

A. Lead

1. General Toxicological Considerations

Inhalation of lead dust and fumes is the major route of lead exposure in industry. A secondary source of exposure may be from ingestion of lead dust contamination on food, cigarettes, or other objects. Once absorbed, lead is excreted from the body very slowly. The absorbed lead can damage the kidneys, peripheral and central nervous systems, and the blood-forming organs (bone marrow). These effects may be felt as weakness, tiredness, irritability, digestive disturbances, high blood pressure, kidney damage, mental deficiency, or slowed reaction times. Chronic lead exposure is associated with infertility and with fetal damage in pregnant women.

Blood lead (PbB) levels below 40 micrograms/100 grams (ug/100g) whole blood are considered to be acceptable levels (in adult males) which may result from daily environmental exposure in industrialized societies. However, fetal damage in pregnant women may occur at blood lead levels as low as 30 ug/100g. Lead levels between 40 to 60 ug/100g in lead exposed workers indicate excessive absorption of lead and may result in some adverse health effects. Levels of 60 to 100 ug/100g represent unacceptable elevations which may cause serious adverse health effects. Levels over 100 ug/100g are considered dangerous and often require hospitalization and medical treatment.

The OSHA standard for airborne lead is 50 ug/M^3 on an 8-hour time-weighted average daily exposure.[3] For secondary lead producers the currently acceptable air level is 100 ug/M^3 with final reduction to a level of 50 ug/M^3 by November 1983. Secondary lead producers must achieve the 100 ug/M^3 level through engineering and administrative controls, and must protect workers at the 50 ug/M^3 permissible exposure limit through any combination of controls, including the use of proper respirators. The standard also dictates that by November 1982 workers with blood lead levels greater than 50 ug/100g must be immediately removed from further lead exposure and in some circumstances workers with lead levels less than 50 ug/100g must also be removed. At present, medical removal of workers is necessary at blood lead levels of 60 ug/100g or greater. Removed workers have protection for wage, benefits, and seniority for up to 18 months until their blood levels adequately decline and they can return to lead exposure areas.

2. Lead Nephrotoxicity

Lead has long been recognized as a nephrotoxin, and exposure to high levels has resulted in overt renal disease. "Granular

contracted kidneys" were observed in lead industry workers in the late 19th and early 20th centuries; lead-poisoned children developed the acquired Fanconi syndrome (elevated glucose, amino acids, and phosphate in the urine), and a high proportion of Australian lead-poisoned children developed chronic renal failure in young adult life; and saturnine nephropathy has been described in chronic imbibers of lead-contaminated moonshine whiskey.[4] Although several investigators have described nephropathy in contemporary American lead workers,[5,6,7,8] and increased mortality from nephritis has been reported in a large cohort mortality study of lead smelter workers exposed between 1946 and 1970,[9] occupational lead nephropathy appears to have declined with the advent of improved industrial hygiene standards.

Despite improvements, significant occupational lead exposure still occurs. Although lead is among the best studied of all occupational toxins, many questions about its effects remain. It is assumed that most acute effects of lead nephrotoxicity are reversible.[10] However, the prevalence of lead nephropathy, the effect of chronic lead exposure at levels currently found in American industry, and the relationship between episodes of acute intoxication, chronic lead exposure, and chronic nephropathy, have not been elucidated.

Lead has its clearest nephrotoxic effects on the brush border cells of the proximal convoluted tubule, producing mitochondrial and cellular swelling and nuclear "inclusion bodies". Chronic tubulo-interstitial disease with tubular atrophy, interstitial fibrosis, and contraction of the renal cortex with glomerular obliteration characterizes the chronic interstitial nephritis which has been associated with lead exposure, but which is pathologically indistinguishable from interstitial nephritis caused by other agents.[10] In terms of tubular function, acute tubular damage may produce leakage into the urine of tubular intracellular enzymes, small proteins, and other substances normally handled by the proximal tubules. Progressive loss of tubular function with chronic interstitial disease may lead to impaired handling of uric acid and loss of urinary concentrating ability. Loss of glomerular function is probably a secondary phenomenon associated with extensive interstitial fibrosis. To date, neither a clear dose-effect relationship nor the precise pathophysiological mechanisms relating acute proximal tubular damage to chronic interstitial nephritis have been established for lead nephropathy.

Conventional laboratory markers of kidney dysfunction used in occupational field studies have most often been confined to crude measurements of glomerular function, utilizing blood urea nitrogen (BUN) and serum creatinine. Both parameters have

limitations. BUN may be elevated with dehydration, or with digestion of blood products in the gastrointestinal tract, as may occur after bleeding in the stomach. Creatinine is generally regarded as a more specific measure of glomerular function, but neither creatinine nor BUN become elevated as a result of renal insufficiency until 50 to 75% of functioning nephrons are lost. Creatinine clearance calculated on a complete 24-hour urine collection is regarded as a fairly sensitive index of glomerular function, but the collection of timed single shift urines during the workday is of uncertain reliability.

Because loss of glomerular function usually represents a more advanced stage of renal disease than tubular dysfunction, markers of tubular function are regarded as desirable tools for evaluating early or acute lead nephrotoxicity. Elevated glucose, phosphorus, and amino acids have been demonstrated in the urine of acutely lead-intoxicated children.[4] Although measurement of beta-2 microglobulin in the urine of long-term cadmium workers has revealed consistent elevations, and has also been elevated in mercury vapor- and nickel-exposed workers, this was not the case in a study of lead workers with evidence of moderately high lead absorption (blood lead levels not exceeding 62 ug/dl).[11,12]

Measurement in the urine of enzymes normally confined to renal proximal tubular cells has been regarded with increasing interest in recent years, since proximal tubular damage is either the earliest lesion in many forms of renal disease or may be an ongoing and recurrent lesion in many processes. N-acetyl-beta-D-glucosaminidase (NAG) is a proximal tubular enzyme which has been measured in both normal populations and in pediatric patients with active renal disease of various etiologies, including both primary tubular and glomerular disease. When corrected for creatinine excretion in the urine, the NAG/creatinine ratio is remarkably constant during a 24-hour period and can be measured on a random urine specimen.[13,14] The ratio appears to be stable throughout life between the extremes of early childhood and old age. Because NAG is confined to the proximal tubular cells, because elevations of urinary NAG have been seen in workers with hypernickeluria,[12] and because lead is known to damage the proximal tubules, NIOSH investigators postulated that the NAG/creatinine ratio in urine might be a potentially useful marker of early or acute lead damage to the kidney.

B. Arsenic

As with lead, inhalation of arsenicals is the most common route of arsenic exposure in the occupational setting. Long-term or chronic

arsenical poisoning due to ingestion is rare. However, it can be a concomitant of inhaled inorganic arsenic from swallowed sputum and from food or smoking materials contaminated from dust in the air, on surfaces, or on hands. Chronic industrial arsenic intoxication manifests itself in three different sundromes: (1) complaints of weakness, loss of appetite, occasional nausea and vomiting, sense of heaviness in the stomach with some diarrhea; (2) complaints of conjunctivitis and inflammation of the mucous membranes of the nose, larynx, and respiratory passage. Perforation of the nasal septum is common, and is probably the most typical lesion of the upper respiratory tract in occupational exposure to arsenical dust. Skin lesions are also common; and (3) complaints of symptoms of peripheral neuritis, initially of the hands and feet, which is essentially sensory. In more severe cases motor paralysis occurs.

Numerous epidemiologic studies have shown that chronic exposure to arsenic compounds can cause lung cancer and lymphomas as well as other forms of cancer.[15]

NIOSH has recommended that airborne concentrations of inorganic arsenic be controlled to prevent exposures in excess of 2.0 ug/M^3 as determined by a 15-minute sampling period.[15] This standard was designed to minimize the possibility of developing lymphatic and respiratory cancer. The OSHA standard for inorganic arsenic is 10 ug/M^3 as averaged over an 8-hour work-shift.[3]. The ACGIH TLV for arsenic and its soluble compounds is 200 ug/M^3.[16]

VI. RESULTS AND DISCUSSION

A. Environmental

1. Initial Site Visit: September 1980

Environmental sampling in September 1980 revealed lead exposures in excess of the 50 ug/M^3 PEL for all SRA&C activities per se (i.e., furnace feeder, furnace operators, and casters); air levels ranged from 262 to 780 ug/M^3 (Table II). A possible contributory factor for the excessive lead exposures was the absence of ventilation controls for the lead tapping and casting operations and the lack of flush connections between the kettle cover and ducting during the refining process. The highest lead exposures observed were for the baghouse operators. Two full-shift samples obtained from these workers revealed lead levels of 4830 and 3830 ug/M^3, which are considerably above the OSHA PEL of 50 ug/M^3. Observations of the job revealed that the highest lead exposure resulted during the rapping operation where the worker enters a baghouse chamber and manually raps the bag filters to dislodge filtered flue dust. These workers wore full-facepiece, airline-supplied respirators during this operation.

With the exception of the baghouse operators, all other workers wore half-face respirators with high-efficiency filters. Of the workers monitored during the initial survey, about half had beards. This condition severely reduces the efficiency of the respirators since facial hair interferes with proper seal.[17] The presence of facial hair was not as common during the followup visit.

Two general area air samples for airborne lead, obtained from the SRA&C building between the receiving kettles and at the blast furnace, showed concentrations of 140 and 250 ug/M^3, respectively. One area sample taken in the employees lunchroom indicated an airborne lead concentration of 52 ug/M^3. This finding offers the distinct possibility that workers are consuming food and beverages or smoking/chewing materials that are contaminated with lead.

2. May 1981

The May 1981 follow-up survey sampling results are presented in Tables III and IV.

A total of 6 personal samples for lead were obtained, 2 from mechanics and 4 from forklift operators (Table III). The mechanics had lead exposures of 294 and 178 ug/M^3 (average, 236 ug/M^3), while the forklift operators' lead exposures ranged from 143-325 ug/M^3 (average, 209 ug/M^3). Two area air samples for lead were taken in each of five areas in the plant, including both production and nonproduction areas (Table III).

Each area except for the lunchroom and the control room had an average air lead level over 100 ug/M^3. These results show that airborne lead is widespread throughout the plant and, more importantly, that workers are unsuspectingly being exposed in areas where one would presume that air lead levels would be low, i.e., lunchroom, office (reception and first aid areas), and control room. Most of the contamination in these areas probably resulted from workers tracking in lead-laden "mud" from the yard. After it dried, ordinary activities could make this material airborne and a source of lead exposure to workers.

Sampling results for arsenic, arsine, and stibine appear in Table IV. Personal sampling results for arsenic indicate that four of eleven SRA&C workers, including 2 blast furnace operators and 2 casters, were potentially exposed to arsenic at concentrations in excess of the NIOSH recommended criterion of 2.0 ug/M^3. One employee, a caster, was potentially exposed to arsenic at a concentration in excess of the OSHA PEL of 10 ug/M^3. His daily TWA arsenic level was 93 ug/M^3, about 9

times above the OSHA standard. Casters' full-shift TWA exposures ranged from 0.69 to 93 ug/M^3 (average, 20 ug/M^3). Six 15-minute samples obtained from 2 casters ranged from nondetectable to 1.3 ug/M^3. Two full-shift samples from the blast furnace operator revealed arsenic concentrations of 0.81 and 0.98 ug/M^3. Eight 15-minute samples taken from three blast furnace operators ranged from nondetectable to 2.72 ug/M^3. Three full-shift samples, two taken from the furnace feeder and one from a laborer, ranged between 0.23 and 0.67 ug/M^3. General area air samples positioned at the blast furnace launder and near the refining kettles revealed arsenic levels of 1.91 and 0.95 ug/M^3, respectively.

Arsine and stibine concentrations in all ten personal and both general area air samples were below the analytical limits of detection (0.02 ug/sample for both analytes).

None of the five personal or one general area air samples revealed any detectable sulfur dioxide.

B. Medical

1. Initial Site Visit: September 1980

All 35 of 93 workers chosen systematically from seniority lists agreed to be interviewed, and 33 consented to have blood drawn. Demographic characteristics of the workers sampled included the following: all 35 were male, and 83% were black. NIOSH found that blood lead levels were high throughout the plant, virtually irrespective of work area; all but two were greater than or equal to (>) 40 ug/dl (Table V). The mean blood lead level among the 33 workers sampled was 68 ug/dl. Twenty-four percent of the sample had a blood lead level >70 ug/dl, the blood lead level then- legal for secondary lead smelter workers under the OSHA standard. Eighteen of the 33 workers for whom blood lead levels were measured had been employed at the plant for more than one year. Company blood lead records for 15 of the 18 were available, and a comparison of mean blood lead levels from July 1979 and July 1980 showed that blood lead levels had been high for at least the past year (Table VI). Although extensive blood lead records were not available, union representatives indicated that blood levels among workers had been high at the plant for many years.

No relationship between blood lead levels and specific symptoms was noted. However, the blood lead levels of those workers reporting one or more symptoms compatible with lead toxicity was higher than the mean blood lead level of those reporting no symptoms compatible with lead toxicity (Table VII), and the absence of statistical significance was slight ($0.05<p<0.10$).

The 18 workers employed more than 1 year had a significantly higher mean blood lead level (PbB = 65.9±12.6 ug/dl) than the 15 workers employed less than or equal to (≤) 1 year (PbB = 47.3±13.6 ug/dl), ($p<0.01$ by Student's t test, Table VII).

2. May 1981

By May, only 35 hourly workers were still at the plant after extensive lay-offs. Thirty-three of them were available for study. While all 33 agreed to provide urine samples, 28 agreed to provide blood as well. Study participants had been employed for a mean duration of 11.6±11.2 years, and had a mean age of 39.8 years. Twenty six of the 33 (78%) were black.

None of the study participants gave a prior history of kidney disease, heroin use, or analgesic abuse. Seven workers said they had been told on at least one occasion by a physician that their blood pressure was elevated, although only one took antihypertensive medication. One had a history of prostatic hypertrophy treated by transurethral prostatectomy and one was an insulin-dependent diabetic. One gave a history of gout. Nine workers reported symptoms consistent with lead colic. Seven workers gave a history of chelation. Blood pressure measurements were obtained on 32 workers, and 12 were elevated (systolic pressure ≥140 mmHG, and/or diastolic ≤90 mmHG), 6 of these in the longest employed and oldest group.

Because blood lead had been related to duration of employment in the first survey, and because renal effects of lead may be cumulative and dose-related, duration of employment was used as a crude indicator of increasing exposure and, presumably, increased body burden of lead. Workers were grouped by duration of employment into 4 groups (<1 year, 1 to 4 years, 5 to 9 years, and >10 years) for evaluation of questionnaire and laboratory data (Table VIII).

The NIOSH laboratories defined a serum creatinine >1.3 mg/dl as elevated, as was a BUN >20 mg/dl. An abnormal urinalysis was defined as more than 2 red cells and/or 2 white cells, clumps of epithelial cells, or the presence of glucose or more than a trace amount of protein. Normal values for the NAG/creatinine ratio were determined by the laboratory performing the test on a group of presumably healthy laboratory volunteers. Normal values for hemoglobin and hematocrit were designated as 14.0 to 18.0 gm%, and 40 to 55% respectively.

NIOSH found that mean hemoglobin concentration declined with increasing duration of employment, and the proportion of low hematocrits increased with increasing duration of employment (Table VIII). FEP levels were high in all employment groups,

participants, they had a mean age of 39 years, and 78% were black.

Again, a remarkably high prevalence of low hemoglobins was noted: 13 of 31 (47%) had a hemoglobin below 14.0 gm%. There were no significant differences in the mean serum iron levels, iron binding capacity, or percent saturation between the groups of workers with and without low hemoglobin (Table IX).

The laboratory that performed the tests defined an abnormally elevated serum creatinine as >1.5 mg/dl, and an elevated BUN as >20 mg/dl. Eight persons had an elevated BUN and/or creatinine by these criteria (7 had an elevated BUN, and 4 an elevated creatinine). Calculated creatinine clearances, adjusting for body surface area, indicate that 6 persons had a reduced creatinine clearance (less than 80 ml/minute). Two of the six persons with a reduced creatinine clearance also had an abnormal creatinine. Of the 7 workers with an elevated BUN, 3 had no other abnormality of kidney function. A total of 12 of 31 workers (39%) had at least one abnormal parameter of kidney function.

Again, workers were grouped by duration of employment (Table X). The same pattern of increasing proportions of renal abnormality with increasing age and duration of employment is apparent in Table IX as was seen in the May study (Table VIII). Workers were then grouped into dichotomous categories of having none or at least one abnormal parameter of renal glomerular function (elevated BUN, creatinine, and/or reduced creatinine clearance (Table XI). Those workers with evidence of abnormal kidney function were significantly older and had been employed longer than workers with normal kidney function. As in the May study, there was no significant relationship between abnormal glomerular function and reporting a known risk factor other than lead exposure, for renal disease. Only one worker, who gave a history of gout, had an elevated serum uric acid. All employees had normal blood phosphorus levels, and one employee had a marginally elevated serum calcium. The characteristics of workers with abnormal glomerular function are summarized and compared with the characteristics of those with apparently normal glomerular function in Table XI.

Creatinine clearance did correlate significantly with urine volume ($p=0.03$), and urine specific gravity also correlated significantly with urine volume ($p=0.0001$). These findings suggest that inadequate collection may have accounted for some of the diminished creatinine clearances, and that as a group, these workers have retained their urine-concentrating capacity.

There is no question that the employees at Master Metals are exposed to unacceptably high levels of atmospheric lead. Their blood lead levels and FEP levels are high, and a large proportion of the group (25 to 42%) have low hemoglobin levels. Rising FEP levels with increasing duration of employment, along with increasing prevalence of low hemoglobin in longer-employed groups, and similar iron studies in workers with both low and "normal" hemoglobin levels, point to lead-associated suppression of erythropoiesis.

Interpretation of the data on renal function is somewhat more ambiguous, for a number of reasons. The absence of a local, matched comparison group limits the extent to which the high prevalence of abnormal renal function parameters can be definitely associated with work in the lead industry. Reporting of other factors may be incomplete. Such elements as dehydration or blood products in the gastrointestinal tract (which may elevate the BUN), laboratory error, or inadequate urine sample collection (which may yield an apparently diminished creatinine clearance), may be responsible for some abnormalities.

These qualifications notwithstanding, there is an apparent dose-effect relationship when dose is equated with years of employment (which is in turn equated with body burden), a finding already shown in work by Lilis et al.[7] Because age is a known confounder for elevations of BUN and creatinine, and since age is also related to increasing duration of employment, the absence of a local comparison group diminishes the strength of the conclusion that can be made, although Lilis was able to control for age in her study. The relationship between a history of chelation and an abnormal BUN and/or creatinine observed in the second survey is of interest, although no distinction can be made between the nephrotoxic effects of elevated blood lead levels (because of which the workers were presumably chelated) and the immense burden of lead presented to the kidney as a result of chelation. The significant difference in FEP between workers with normal and abnormal renal function, and the higher percentage of low hemoglobin levels among workers with abnormal renal function, tend to support the notion that these workers have a greater body burden of lead and commensurately more profound biological effect, and that the renal abnormalities are both real and may be related to lead exposure.

In addition to examining the findings within this group, NIOSH investigators compared them to the results of a recent NIOSH medical survey of paint manufacturing workers at a plant in Kansas where moderate exposures to lead and cadmium were found.[17] Of the 96 workers sampled in that survey, 9 (9%) had a blood lead of 40 or greater, and none of the workers had elevations of blood or urine cadmium. All workers in both the second and third surveys at Master Metals had blood leads above 40. The tests of renal

function on the paint manufacturing workers were performed by the same laboratory that performed the tests of renal function in the October 1981 survey in this study. In the group of 96 paint workers, 7 (8%) had an abnormal creatinine, and 11 had an abnormal BUN. In our final survey, 4 of 31 (13%) had an abnormal creatinine, and 7 (23%) an elevated BUN. The worker groups are not perfectly comparable: there are more blacks in the Master Metals population than in the Kansas paint plant, and the mean age of the paint plant workers was somewhat higher (about 48 years) than that of Master Metals group (39) years. Nonetheless, they are both industrial populations whose blood tests were performed by the same laboratory within the same year. In addition, because the paint workers were exposed to moderately elevated levels of lead and to lower levels of cadmium, the comparison of the lead smelter workers with the paint workers should serve to underestimate the difference in renal abnormalities which might exist between lead smelter workers and an unexposed population. Despite this postulated diminution of the effects attributable to lead, the Master Metals group has about twice the prevalence of renal function abnormalities when compared with the Kansas paint plant workers.

Unfortunately the study did not show the NAG/creatinine ratio to be a useful screening test for early lead nephropathy. NIOSH investigators did not find consistent elevations above the normal in any group. This survey suggests that the NAG/creatinine ratio is (1) insensitive as a marker of otherwise subclinical tubular injury, (2) that tubular injury is not occurring among individuals on whom sufficient data was gathered to interpret that injury, or (3) that tubular injury due to lead exposure is simply not occurring in these workers. The study group was quite small, however, and there was not a well-matched comparison group. Further work with the enzyme in other lead-exposed populations may be warranted before the test is abandoned. Attempted measurement of distal tubular function - urine specific gravity - was similarly unsuccessful, not because of limitations in the test itself, but because of inadequate compliance with the necessary water deprivation.

Although this study did not discover a useful and reliable screening tool for lead effect on the proximal tubules, it did demonstrate once again that lead-associated renal disease may still be occurring in the American secondary lead smelting industry, and that it appears to be related to both intensity and duration of exposure. Because this particular plant is grossly out of compliance in its air levels within the plant and in the blood lead levels of hourly employees, NIOSH cannot extrapolate from this study and speculate on whether adherence to currently recommended exposure standards would indeed result in the disappearance of occupational lead nephropathy.

VII. RECOMMENDATIONS

1. There is an overwhelming need to reduce lead exposures at the Master Metals facility below the OSHA standard of 50 ug/M^3. Lead-associated disease is entirely preventable.

2. Arsenic exposures should be reduced below the NIOSH recommended criterion of 2 ug/M^3 to minimize the cancer risk of exposed workers.

3. In regards to engineering controls, NIOSH recently issued two publications, References 19 and 20, which present up-to-date information on control technology to reduce or eliminate worker exposures to contaminants in secondary lead smelters. Most of the recommendations presented below were derived from control methodology presented in these publications.

   a. The lead tapping launders and the pouring/casting operations should be ventilated to reduce exposures to the SRA&C workers.

   b. The blast furnace slag tapping hood should be made to fit together better to reduce gaps where sparks and fumes may escape.

   c. Automatic mechanical bag shakers should be installed in the metallurigical baghouse. Their installation would eliminate workers from entering the baghouse cells where lead exposures were significant. Both baghouses should be equipped with automatic, enclosed flue dust recycling systems to prevent workers from handling the flue dust.

   d. Hinged metal plates should be installed at the access ports of the kettle furnace hoods so that they can be shut during metal refining to prevent the escape of contaminants into the work environment.

   e. The ventilation system for the sanitary and metallurgical baghouses should be periodically cleaned and inspected to ensure design airflows are maintained and worker exposures are minimized.

   f. Those workers overexposed to arsenic (i.e., blast furnace operator and casters) should be provided with air-supplied respirators.

   g. A filtered air equipped cab should be installed in the front end loader. However, for this control to be effective the operator must not wear lead-contaminated clothing or shoes into the cab.

4. A smelter-wide cleanup should be undertaken. Lead dust and mud should be removed from yard surfaces, building surfaces, equipment, etc. Settled particulate which has been accumulated on building structural members should be removed using vacuuming techniques.

5. Dry sweeping and shoveling should be discontinued. Shoveling of wet materials and vacuuming of dry materials are preferable.

6. Smelter employees should not enter the lunchroom, office, or other "clean" areas with lead-contaminated clothing or equipment unless surface lead dust has been removed. Alternatively, smelter employees could be provided with overalls which can be worn over their work clothing and removed before entering these areas. A shoe/boot cleaning station should also be provided at the entrance to these areas to prevent tracking in of contaminants.

7. All equipment taken to the maintenance building should be free of lead to preclude exposures during equipment repair.

8. Facial hair should be prohibited where respiratory protection is required. Respirators cannot provide sufficient protection if facial hair interferes with proper seal.

9. The company should continue to wet building floors, raw material stockpiles, and heavily trafficked areas in the yard to aid in dust suppression.

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IX. AUTHORSHIP AND ACKNOWLEDGEMENTS

Report Prepared by:

James M. Boiano
Industrial Hygienist
Industrial Hygiene Section

P. Lynne Moody, M.D.
Medical Officer
Medical Section

Environmental Evaluation Assistance:

Cheryl Lucas and
Richard Hartle
Industrial Hygienists
Industrial Hygiene Section

Medical Evaluation Assistance:

Marian Coleman
Support Services Branch

Michael Bader, M.D.
Cambridge City Hospital

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Originating Office:

Hazard Evaluations and Technical Assistance Branch
Division of Surveillance, Hazard Evaluations, and Field Studies
Cincinnati, Ohio

Report Typed By:

Debra A. McDonald
Clerk-Typist
Industrial Hygiene Section

X. DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this report are currently available upon request from NIOSH, Division of Standards Development and Technology Transfer, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through the National Technical Information Service (NTIS), 5285 Port Royal, Springfield, Virginia 22161. Information regarding its availability through NTIS can be obtained from NIOSH Publications Office at the Cincinnati address. Copies of this report have been sent to:

1. Master Metals, Incorporated
2. Authorized Representatives of Employees, Local 735, United Steelworkers of America
3. NIOSH, Region V
4. 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. A copy of the summary page will be sent to all employees who participated in the NIOSH medical surveys.

TABLE I

Evaluation Criteria

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

Substance	Evaluation Criteria* NIOSH	ACGIH	OSHA	Primary Health Effects
Lead	50	150	50	Prolonged absorption of lead or its inorganic compounds results in gastrointestinal disturbances, anemia, neuromuscular dysfunction, and kidney damage. (See text for additional discussion)
Arsenic	2	200	10	Arsenic compounds are irritants of the skin, mucuous membranes and eyes. Arsenical dermatoses and epidermal carcinoma are reported risks of exposure to arsenic compounds, as are other forms of cancer. (See text for additional information)
Arsine	2	200	200	Arsine is a hemolytic agent of extreme toxicity and with poor warning properties. Characteristic features of acute arsine poisoning are abdominal pain, dark red urine, and jaundice.
Stibine	---	500	500	Stibine is a hemolytic agent in animals; it is expected that the same effect would occur in humans.
Sulfur Dioxide	1.3	5.2	13	Severe irritant of the eyes, mucous membranes, and skin, caused by the rapidity with which it forms sulfurous acid on contact with moist surfaces.

* The NIOSH criterion for arsenic is a time-weighted average (TWA) based on a 15-minute sampling period. The OSHA standards and ACGIH threshold limit values (TLV's) are TWA's based on an 8-hour workday. Values are in micrograms per cubic meter of air (ug/M^3), except for sulfur dioxide which is given in milligrams per cubic meter (mg/M^3).

TABLE II

Results from Personal and General Area Sampling for Lead

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

September 25-26, 1980

Date	Sample Location	Sample Description	Sampling Duration (min)	Sample Volume (m^3)	Airborne Concentration (ug/M^3)
9/25/80	SRA&C Building	Blast Furnace Operator	406	0.61	262
9/26/80	SRA&C Building	Blast Furnace Operator	402	0.60	367
9/25/80	SRA&C Building	Caster	399	0.60	316
9/25/80	SRA&C Building	Caster	396	0.59	593
9/25/80	SRA&C Building	Caster	395	0.59	406
9/26/80	SRA&C Building	Caster	-	-	780**
9/25/80	Front End Loader	Furnace Feeder	396	0.59	406
9/25/80	SRA&C Building	Reverb Operator	387	0.58	362
9/26/80	SRA&C Building	Reverb Operator	406	0.61	311
9/25/80	Baghouse/Plantwide	Baghouse Operator	386	0.58	4830
9/26/80	Baghouse/Plantwide	Baghouse Operator	399	0.60	3830
9/25/80	SRA&C Building	Between Receiving Kettles*	380	0.57	140
9/25/80	SRA&C Building	Blast Furnace*	372	0.56	250
9/25/80	Welfare Building	Lunchroom*	373	0.56	52

Laboratory Limit of Detection (ug/sample):	3
Evaluation Criterion:	50

* Area sample
** Conservative estimate due to pump failure

TABLE III

Environmental Concentrations of Lead

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

May 6-7, 1981

Date	Sample Location	Sample Description	Sampling Duration (min)	Sample Volume (m^3)	Airborne Concentration (ug/M^3)
5/6/81	Maintenance Building	Mechanic	430	0.645	294
5/7/81	Maintenance Building	Mechanic	411	0.616	178
5/6/81	Shipping and Receiving (S&R) Building/SRA&C Building	Forklift Operator	395	0.592	159
5/6/81	S&R Building/SRA&C Building	Forklift Operator	430	0.645	325
5/7/81	S&R Building	Forklift Operator	436	0.654	183
5/7/81	SRA&C Building	Forklift Operator	340	0.510	143
5/6/81	Maintenance Building	Shop Area	440	0.660	303
5/7/81	Maintenance Building	Shop Area	477	0.716	26
5/6/81	Office Building	Reception Area	455	0.682	176
5/7/81	Office Building	First Aid	458	0.687	28
5/6/81	S&R Building	East Wall	430	0.642	109
5/7/81	S&R Building	East Wall	479	0.718	125
5/6/81	Welfare Building	Lunchroom	415	0.623	21
5/7/81	Welfare Building	Lunchroom	487	0.730	40
5/6/81	SRA&C Building	Control Room	400	0.600	75
5/7/81	SRA&C Building	Control Room	459	0.688	78

Laboratory Limit of Detection (ug/sample):	3
Evaluation Criterion:	50

TABLE IV

Environmental Concentrations of Arsenic, Arsine, and Stibine
Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234
May 6-7, 1981

Date	Sample Location	Sample Description	Sampling Duration (min)	Sample Volume (m^3)	Airborne Concentration (ug/M^3)		
					Total Arsenic	Arsine	Stibine
5/6/81	SRA&C Building/Plantwide	Laborer	445	0.089	0.56	ND	ND
5/6/81	SRA&C Building	Caster	360	0.072	0.69	ND	ND
			15	0.023	0.87	--	--
			15	0.023	ND	--	--
			15	0.023	1.30	--	--
5/6/81	SRA&C Building	Caster	350	0.072	0.69	ND	ND
			15	0.023	ND	--	--
			15	0.023	ND	--	--
			15	0.023	ND	--	--
5/6/81	SRA&C Building	Caster	435	0.087	1.26	ND	ND
5/7/81	SRA&C Building	Caster	379	0.076	93	ND	ND
5/7/81	SRA&C Building	Caster	395	0.079	4.68	ND	ND
5/6/81	SRA&C Building	Blast Furnace Operator	430	0.086	0.81	ND	ND
			15	0.023	1.30	--	--
			15	0.023	ND	--	--
			15	0.023	ND	--	--
5/6/81	SRA&C Building	Blast Furnace Operator	15	0.023	2.17	--	--
			15	0.023	ND	--	--
			15	0.023	0.87	--	--
5/7/81	SRA&C Building	Blast Furnace Operator	459	0.092	0.98	ND	ND
			15	0.023	2.27	--	--
			15	0.023	ND	--	--
5/6/81	Front End Loader	Furnace Feeder	435	0.087	0.23	ND	ND
5/7/81	Front End Loader	Furnace Feeder	374	0.075	0.67	ND	ND
5/7/81	Blast Furnace	Above Lead Launder*	340	0.068	1.91	ND	ND
5/7/81	Casting	Near Refining Kettle*	335	0.067	0.75	ND	ND
Laboratory Limits of Detection (ug/sample):					0.02	0.02	0.02
Evaluation Criteria:					2	2	500

* Area Sample; ND = not detected; -- = not sample

TABLE V

Blood Lead Levels in Thirty-Three Employees by Job/Location

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

September 26, 1980

Battery Breaking	SRA&C	Baghouse	Shipping & Receiving	Health & Safety		Maintenance
				Welfare	Power Sweeper	
26*	22*	47	56	45	53	34*
48	40*	53	63	54	57	70
55	42*	86	70	82		92
64*	50		70			
76	54					
	54					
	57					
	47					
	60					
	62					
	67					
	69					
	70*					
Mean:						
54	54	62	65	60	55	65

Values in ug/100g
* Employees employed for 3 months or less.

TABLE VI

Comparison of Mean Blood Leads (PbB) on Fifteen Workers*

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

September 1980

	July 1979 (company records)	July 1980 (company records)	September 1980 (NIOSH survey)
PbB (ug/dl)	65 ± 10	57 ± 14	68 ± 12

* Of the 18 workers in our sample of 33 (from whom NIOSH obtained blood lead levels) who had worked more than 1 year, past company blood lead records were available on 15.

TABLE VII

Mean Blood Lead Levels in Workers Grouped by
Frequency of Symptom Reporting and Employment Duration

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

September 1980

	Symptoms of Lead Toxicity		Employment Duration	
	No Symptoms (13 workers)	>1 Symptom (20 workers)	<1 Year (15 workers)	>1 Year (18 workers)
PbB (ug/dl)	52 ± 15	61 ± 16	47 ± 14	66 ± 13
	($p<0.1$, Student's t test)		($p<0.01$, Student's t test)	

TABLE VIII

Comparison of Characteristics in Thirty-Three Workers Grouped by Duration of Employment

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

May 1981

Duration of Employment (Years)	Age (Mean)	PbB+ (ug/dl)	FEP+ (ug/dl)	Mean Hemoglobin+ (gm%)	Hematocrit+ (No./percent below 40)	BUN (mg/dl) (No./% above 20)	Creatinine (mg/dl) (No./% above 1.3)	Urinalysis Number/percent abnormal			Creatinine** (U/gm) Mean Value	Elevated Blood Pressure (Systolic>140 and/or >90 diastolic)
								Protein	Glucose	Cells*		
<1 year (2 workers)	23.5	62	154.5 ±99.7	15.0±1.2	1 of 2	0	1 of 2	0	0	0	3.6±1.0	1 of 1
1-4 years (12 workers)	27.8 ±5.5	70.4 ±9.7	298.3 ±72.2	15.2±1.1	4 of 10 (40%)	2 of 10 (20%)	4 of 10 (40%)	2 of 10	0	3 of 10	3.1±1.8	2 of 12
5-9 years (6 workers)	36.8 ±11.9	69.5 ±13.	411.8 ±281.3	14.9±1.0	4 of 6 (67%)	0	3 of 6 (50%)	1 of 6	1 of 6	2 of 6	3.6±1.0	3 of 6
>10 years (13 workers)	53.9 ±8.0	72.4 ±9.8	409.8 ±183.5	14.1±1.3	10 of 10 (100%)	6 of 10 (60%)	7 of 10 (70%)	0 of 10	0	5 of 10	2.9±1.9	6 of 13

+ (28 of 33 workers consented to give blood samples; PbB = blood lead; FEP = free erythroryte protoporphyrin; BUN = blood urea nitrogen)
* Abnormal cells = greater than 2 white cells, greater than 2 red cells, clumps of epithelial cells, and/or red or white cell casts
** (N-acetyl-B-glucosaminidase/creatinine) NAG/creatinine ratio on 38 normal lab personnel was 3.2 U/gm (S.D. = ±1.3, median 3.3, range 0.4 - 5.6)

TABLE IX

Hemoglobin Levels and Iron Studies in Workers

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

October 1981

Hemoglobin Levels	Mean Total Iron (normal = 40-170 ug/dl)	Mean Total Iron Binding Capacity (normal = 250-420 ug/dl)	Mean Percent Saturation (normal = 20-55%)
Low Hemoglobin (<14.0 gm%) (13 workers) range of Hgb values = 11.1-13.8 gm%	78±22	336±40	24±8
Normal hemoglobin (14.0-18.0 gm%) (18 workers) range of Hbb values = 14.0-16.8 gm%	81±28	362±53	22±7

TABLE X

Age and Kidney Function Characteristics of Thirty-One Workers
Grouped by Duration of Employment

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

October 1981

Duration of Employment	Mean Age (Years)	BUN (Number/percent above 20 mg/dl)	Creatinine (Number/percent above 1.5 mg/dl)	Creatinine Clearance (Number/percent less than 80 ml/min)
<1 year (No workers)	-	-	-	-
1-4 years (10 workers)	25±3	1 of 10 (10%)	0	0
5-9 years (8 workers)	36±9	1 of 8 (12.5%)	0	1 of 8 (12.5%)
>10 years (13 workers)	51±9	5 of 13 (38%)	4 of 13 (31%)	5 of 13 (38%)

TABLE XI

Characteristics of Workers With Normal and Abnormal* Renal Function

Master Metals, Incorporated
Cleveland, Ohio
HETA 80-234

October 1981

Renal Function Status	Age	Race	Duration of Employment (Years)	PbB (ug/dl)	FEP (ug/dl)	Hemoglobin <14.0 gm%	Risk Factors For Renal Disease**	Serum Uric Acid >8.5 mg/dl
Abnormal* (12 workers)	45±11	86% black	15.8±9.1	66±22	250±209	7 of 12 (58%)	1 of 12 (8%)	0
Normal (19 workers)	31±18	74% black	7.4±7.4	63±20	219±148	6 of 19 (32%)	4 of 19 (21%)	1

* Abnormal renal function = BUN >20 mg/dl, creatinine >1.5 mg/dl, and/or creatinine clearance <80 ml/min
** Other than elevated blood lead, e.g., hypertension, kidney stones, diabetes, prostatism

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