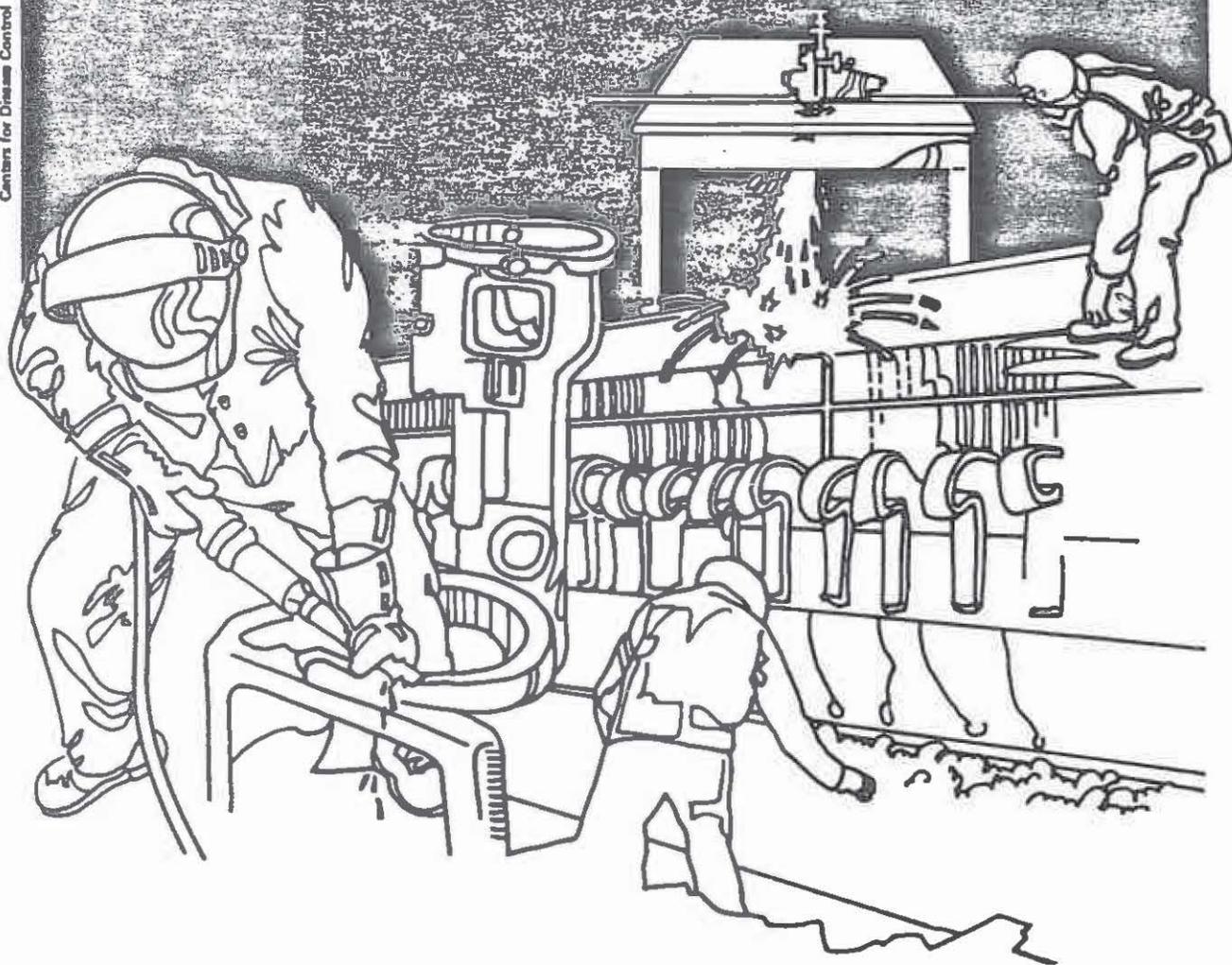


NIOOSH



Health Hazard Evaluation Report

HETA 80-187-1395
SAFT AMERICA, INCORPORATED
ST. PAUL, MINNESOTA

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.

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

HETA 80-187-1395
DECEMBER 1983
SAFT AMERICA, INCORPORATED
(formerly Gould, Incorporated)
ST. PAUL, MINNESOTA

NIOSH INVESTIGATORS:
James M. Boiano, IH
Jane A. Lipscomb, RN, MS
Gary M. Liss, MD, MS

I. SUMMARY

On July 2, 1980, the National Institute for Occupational Safety and Health (NIOSH) received a request to evaluate cadmium and nickel exposure and a number of health complaints among workers employed at Saft America, Incorporated (formerly Gould, Incorporated), St. Paul, Minnesota. The plant manufactures nickel-cadmium (NiCad®) cells and batteries. There were approximately 225 hourly workers employed at the plant at the time of the evaluation.

On August 12-13, 1980, NIOSH investigators conducted an initial walk-through evaluation. On November 17-19, 1981, 42 personal breathing zone air samples were collected to determine worker exposure to cadmium and nickel dusts. Time-weighted average cadmium exposures ranged between 3 and 284 $\mu\text{g}/\text{m}^3$. Levels in eighteen (43%) of the samples exceeded the NIOSH recommended standard of 40 $\mu\text{g}/\text{m}^3$. Levels in five (12%) of the samples exceeded the NIOSH ceiling limit and the OSHA standard of 200 $\mu\text{g}/\text{m}^3$. Time-weighted average nickel exposures ranged between 6 and 630 $\mu\text{g}/\text{m}^3$. Concentration in thirty-three (79%) of the samples exceeded the NIOSH recommended standard of 15 $\mu\text{g}/\text{m}^3$. None of the samples exceeded the OSHA standard of 1000 $\mu\text{g}/\text{m}^3$.

NIOSH obtained and analyzed biological monitoring data (parameters of cadmium absorption and of renal function) which the company had collected for 305 production employees between December 1980 and June 1982. Blood cadmium levels were 10.0 ng/ml or above in 82 (27%) of the workers. Blood cadmium levels were more frequently elevated among those who had worked in the terminal and/or coiling (high-exposure) areas [63 (32%) of 199] than among those who had worked in other (low-exposure) areas [19 (18%) of 106] ($p=0.01$). Among workers in the high-exposure areas, the prevalence of abnormal blood and urine cadmium concentrations was significantly related to duration of employment in these areas.

Kidney function evaluation found very few elevated blood urea nitrogen (BUN) or serum creatinine concentrations. However, urinary beta-2 microglobulin, which is reported to be increased in persons with kidney damage caused by cadmium, was elevated (greater than 370 ng/ml) in six workers, all of whom had worked in the high-exposure areas at some time. Urine cadmium concentrations were 10.0 $\mu\text{g}/\text{l}$ or above in 82 (27%) of the 305 workers. Urine cadmium was more frequently elevated among those who had worked in high-exposure areas [64 (32%) of 99] than among those who had not [18 (17%) of 106] ($p=0.004$).

Based on the results of this evaluation, NIOSH has determined that a health hazard from overexposure to cadmium and nickel dusts existed at Saft America, Incorporated, St. Paul, Minnesota. Recommendations to reduce environmental exposures to these metals are presented in Section VIII of the report.

KEYWORDS: SIC 3691 (Storage Batteries), nickel, cadmium, NiCad® batteries, blood cadmium, urinary cadmium, kidney function, BUN, beta-2-microglobulin, serum creatinine.

II. INTRODUCTION

On July 2, 1980, the National Institute for Occupational Safety and Health (NIOSH) received a request from an authorized representative of several employees, members of Local 110, International Brotherhood of Electrical Workers (IBEW), to evaluate workers exposed in the manufacture of nickel-cadmium batteries at Saft America, Incorporated (formerly Gould, Incorporated), St. Paul, Minnesota. NIOSH investigators conducted an initial survey August 12-13, 1980, which included a walkaround tour and discussions with management, labor representatives, and employees. NIOSH personnel conducted a followup industrial hygiene survey November 17-19, 1981 and collected air samples for cadmium and nickel. The company and union were informed of NIOSH's progress via two interim reports. Interim Report No. 1 (November 1980) summarized the activities and findings of the initial survey. Interim Report No. 2 (March 1982) presented the air sampling results and preliminary recommendations from the followup survey.

III. BACKGROUND

A. Plant History and Workforce

Saft America, Incorporated, Portable Battery Division, St. Paul, Minnesota, manufactures a variety of rechargeable nickel-cadmium (NiCad®) cells and batteries for both industrial and domestic use. The plant began operation in 1959 under the ownership of Gould, Incorporated, the previous owner. Saft purchased the plant in July 1982, and has retained the hourly workforce formerly employed by Gould.

At the time of the followup survey in November 1981 approximately 225 hourly workers were employed at the plant. Because of layoffs this was about 80 workers less than the workforce at the time of the initial survey. Approximately 80-85% of the November 1981 workforce was female; most were involved in cell/battery assembly operations.

B. Process Description

The manufacture of NiCad® batteries involves the following processes: preparation of positive (nickel) plaques, preparation of negative (cadmium) plaques, fabrication of positive and negative electrodes, assembly of positive and negative electrodes into cells, and assembly of cells into batteries.

Preparation of Positive Plaques - Nickel powder is added to a mixture of carboxymethylcellulose, antifoam agent, and water to form a slurry. Nickel-plated, perforated or "wiped", sheet metal is continuously fed through a container filled with the nickel slurry prior to entering a vertical furnace where sintering occurs under an inert atmosphere. The sintered material or plaques are wound in spools, transferred to a vacuum chamber, impregnated with nickel nitrate, then heated to convert the nickel nitrate to nickel hydroxide. The impregnation process may then be repeated until the desired weight gain of active material is achieved. The plaques are subjected to a charge-discharge cycle, and then washed, dried, and spooled.

Preparation of Negative Plaques - Unlike the positive plaque process, negative plaques are manufactured by a predominately wet, fully automated, single machine process. Cadmium hydroxide is electrochemically deposited onto a nickel-plated screen. When the desired thickness is achieved, the plaque is then washed, electrically charged/discharged, compressed, reoxidized, washed, dried, and spooled.

Fabrication of Positive and Negative Electrodes - Negative plaques (with the perforated base metal) and positive plaques are further processed in the terminal department. Two slitter machines, one for each type of plaque, are used to slit the plaque into many strips of similar width. The strips are spooled and then fed into terminal machines. These machines cut the positive or negative strips to the desired length and abrade a small portion of each strip down to the base metal. Metal tabs (terminals) are then mechanically fastened to the base metal.

Negative plaques with the "wiped" metal base are processed in a tab brushing machine. This machine removes the deposited nickel metal from the smooth sections of the plaque. The brushed plaques are then stamped by a punch press into electrodes.

Assembly of Positive and Negative Electrodes into Cells - Cells are initially assembled in the coiling department. Workers in this area operate coiling machines. They obtain a positive electrode and its negative electrode counterpart and place a polyethylene separator between them. This "sandwich" is then coiled and inserted into a cylindrical container.

The partially completed cells are further processed by workers along the cell assembly line. Assemblers in this area spot weld the terminals to the container, fill the container with electrolyte, insert insulators, place the cap, and then hermetically seal the cell in a crimp press.

All cells are tested for capacity in the circuit test room.

Assembly of Cells into Batteries - Approved cells are transferred to the product assembly area. Here, workers label and package cells or combine cells in varying combinations to make batteries. All batteries are inspected and tested prior to shipment.

C. Plant Industrial Hygiene and Medical Surveillance

The company began an environmental surveillance program for cadmium and nickel exposed workers in 1973. Since that time all air sampling and most of the analysis had been done by the company. Records of air sampling data have been maintained by the company since the beginning of the program.

In June 1980, the company began a medical surveillance program for cadmium exposed workers. At that time the company hired a consultant to develop a protocol for this program. In December 1980, the company began screening production workers for biological indicators of cadmium exposure. Between December 1980 and June 1982, 305 production workers participated in the medical screening. As part of the medical surveillance program workers received a physical examination by a local consulting physician.

In June 1982, NIOSH medical investigators contacted the company's medical department regarding the status of their medical screening program. At that time, NIOSH was informed that the company had completed the screening and that the data were going to be analyzed by the University of Minnesota. At that point NIOSH reviewed the protocol and requested the company's medical data upon completion of the analysis. By October 1982, the data analysis was still not underway, so the company provided NIOSH with the raw data.

IV. METHODS AND MATERIALS

A. Environmental

On November 17 and 18, 1981, NIOSH collected 42 personal breathing zone air samples during first shift production operations to assess workers' exposures to cadmium and nickel. Employees monitored included those from the sintering, vacuum impregnation, electrodeposition, terminal, coiling, cell assembly, circuit test, and battery assembly areas. NIOSH investigators focused the monitoring on workers in the terminal and coiling departments since company surveillance records indicated that cadmium exposure was highest in these areas. Half of the samples NIOSH collected were obtained from employees in these two departments.

All samples were collected on mixed cellulose ester membrane filters using personal sampling pumps calibrated at 1.5 liters per minute (Lpm). Sampling times averaged seven and one-half hours. The filters were analyzed for cadmium and nickel by atomic absorption spectrophotometry according to NIOSH Method P&CAM 173.¹ The limits of detection for cadmium and nickel were 2 and 3 ug/sample, respectively.

Company environmental surveillance records of documentable quality (1977-1982) were obtained and reviewed.

B. Medical

NIOSH obtained and analysed the company's biological monitoring data. The following information was collected on all production workers participating in the company screening: demographic information; employment history, including length of employment in areas with elevated cadmium exposure (i.e., coiling and terminal areas); and smoking history. Biological monitoring data included: blood cadmium, blood creatinine, blood urea nitrogen (BUN), urine cadmium, urine beta-2 microglobulin, urine creatinine, urine cadmium/creatinine ratio, urine total protein, and specific gravity. Spot urine samples rather than 24-hour collections were used for all urine analyses. Metpath Laboratory, Teterboro, New Jersey performed all laboratory analyses. Metpath's reference ranges for relevant test results performed in their labs are as follows:

blood cadmium	0.0 - 10.0 ng/ml
blood urea nitrogen (BUN)	6.0 - 25.0 mg/dl
serum creatinine	0.5 - 1.7 mg/dl
urine cadmium	0.0 - 10.0 ug/l
urine beta-2 microglobulin	4.0 - 370 ng/ml

Test results were reported as actual values in most cases, with the exception of 39 blood cadmium levels, which were performed between June and December of 1981. During this period, Metpath reported blood cadmium levels of less than 10 ng/ml as "less than 10 ng/ml" rather than as specific results. Therefore, when comparing mean blood cadmium values between high-exposure and other production workers, we eliminated these 39 values. When making dichotomous comparisons (normal/abnormal), we considered values of less than 10 ng/ml "normal" values.

Because the company screened only production workers, there was no group of unexposed workers to serve as a comparison group. Therefore, we grouped workers into the following two exposure categories: low-exposure - consisting of those who had never worked in one of the designated elevated exposure areas; and high-exposure - consisting of those who had worked in the terminal and/or coiling areas.

All biological test values were log-transformed for statistical analysis. A Student t-test was used to compare mean assay values between high and low exposed workers. A Wilcoxon rank sum test was used to compare means of results which did not follow a normal (i.e., Gaussian) distribution. A chi-square test was used to compare prevalence of abnormal results between the two exposure groups. Spearman's correlation coefficient was used to determine correlations between blood cadmium, urine cadmium, and beta-2 microglobulin. A logistic regression model, obtained from the Statistical Analyses System (SAS) program package¹, was used to describe simultaneously the effect on blood and urine cadmium of exposure (i.e., years in high-exposure areas) and of other factors, such as age, sex, smoking history, and length of employment, which might be confounding or modifying the effect of exposure.

V. EVALUATION CRITERIA

A. Environmental 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 (TLV's), and 3) the U.S. Department of Labor (OSHA) occupational health standards. Often, the NIOSH recommendations and ACGIH TLV's are lower than the corresponding OSHA standards. Both NIOSH recommendations and ACGIH TLV's 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 standards, 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 only 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.

B. Cadmium

Cadmium is a toxic heavy metal used in the manufacture of batteries, pigments, and jewelry, and as a neutron absorber in nuclear reactors. Cadmium may enter the human body either by ingestion (swallowing) or by inhalation (breathing) of cadmium metal or oxide.

Absorption and Measurement

Approximately 6 to 10 percent of ingested cadmium and 15 to 30 percent of inhaled cadmium is absorbed into the body.³ Cadmium is transported from the site of absorption by the red blood cells and plasma. It is deposited in organs throughout the body, but major depositions occur in the liver and kidneys. Under normal conditions, the kidneys accumulate the greatest concentrations of cadmium. Cadmium is excreted from the body very slowly, and thus accumulates in the cortex of the kidneys.

The blood cadmium concentration is the best biological indicator of recent cadmium exposure and absorption.^{4,5} The normal blood cadmium level is below 1.0 microgram (ug) per deciliter [10 nanograms per milliliter (ng/ml)] of whole blood.⁶ Chronic cadmium exposure can be assessed by measuring the cadmium content of the kidneys by the technique of neutron activation analysis.⁷ The urine cadmium concentration, although used widely as an index of exposure, is primarily an indicator of cadmium-induced kidney damage; the urine cadmium concentration does not ordinarily begin to increase until after injury has occurred to the kidneys.⁸

Acute Toxicity

Acute inhalation exposure to cadmium can cause pneumonia or pulmonary edema,⁹ as well as liver and kidney damage.¹⁰ Ingestion of toxic quantities can produce nausea, vomiting, and diarrhea. Exposure to an airborne concentration of cadmium of 40 mg/m³ is considered immediately dangerous to life.

Chronic Toxicity

Occupational exposure to cadmium is more commonly chronic than acute. Chronic occupational exposure to cadmium can produce several toxic effects, of which the most important are emphysema of the lungs and chronic kidney disease.³ Also, occupational cadmium exposure has been associated with cancer of the prostate gland, and there is limited evidence that occupational cadmium exposure may be associated with lung cancer.¹¹⁻¹⁴

Apart from malignancy, kidney disease is the toxic effect of chronic cadmium exposure which is of principal concern.³ Although much remains to be learned about the development of kidney disease in persons exposed to cadmium, the process appears to be a gradual one.⁸ Also, the process is dose-related; persons with greatest lifetime absorption of cadmium are at greatest risk of kidney disease (nephropathy). The initial signs of cadmium nephropathy are subtle. Affected workers will usually have no symptoms in the early stages, and their kidney function test results may still be within the broad range of normal, although their test results will tend over time to move toward the end of the normal range.

Because the kidney has an enormous reserve capacity, results of the usual renal function tests--blood urea nitrogen (BUN), serum creatinine, and serum uric acid--will not become frankly abnormal until one-third to one-half of kidney function has been lost.¹⁵ For that reason, more sensitive screening tests of renal function have been sought. These include measurement of serum concentrations of 1,25-dihydroxy vitamin D (which may be decreased),¹⁶ and measurement of urine concentrations of cadmium and of the protein, beta-2 microglobulin (both of which are reported to increase in persons with kidney damage caused by cadmium).¹⁷ Also, aminoaciduria, renal glycosuria, or hyperphosphaturia may develop.

When any of these test results are abnormal in a person exposed to cadmium, or even when two or more test results are in the high normal range, there exists a possibility of kidney damage. In that circumstance, more complete evaluation of the individual worker's kidney function is required.

NIOSH currently recommends that workers exposure to cadmium dust not exceed an air concentration of 40 ug/M³ as a time-weighted average (TWA) for up to a 10-hour workday, or to a ceiling concentration greater than 200 ug/M³ for any 15 minute period.¹⁸ NIOSH also has established an action level for cadmium of one half the recommended exposure limit (20 ug/M³) which, if worker exposures exceed, the company should institute appropriate engineering and/or administrative controls. The Occupational Safety and Health Administration (OSHA) Standard for cadmium dust exposure is 200 ug/M³, as an 8-hour TWA.¹⁹

C. Nickel

Nickel is a toxic metal used in the manufacture of various iron alloys, batteries, and as a catalyst in the hydrogenation of fats and oils. Routes of absorption in the occupational setting are similar to that of cadmium. Nickel has been associated with both noncarcinogenic and carcinogenic adverse effects.^{20,21} Among the former, contact dermatitis has been well-documented. Nickel is a common cause of allergic contact dermatitis (ACD). This is seen more frequently among women than among men and is noted for its tendency to remain for years.²² Fisher describes this element as the third most frequent cause of ACD. Perspiration, pressure, and friction may affect the severity of nickel dermatitis in sensitized individuals.²² Most authors have found no significant relation between nickel dermatitis and atopic dermatitis. Nickel has been reported as a rare cause of occupational asthma.^{23,24} Nickel carbonyl is a severe respiratory irritant and produces a syndrome characterized by headache, nausea, vomiting, and perhaps unconsciousness, followed after one to two days by cough, dyspnea, pulmonary edema and even death.²⁵ Several epidemiologic studies of respiratory cancer in nickel refinery workers in Wales, Canada and Norway²⁰ have confirmed the carcinogenicity of nickel. Other organs that can be affected are the nasal sinuses and larynx. There is a suggestion that those starting work in nickel refineries after 1930 may have only a small elevated risk for lung cancer. In the studies in Wales, the average latency period from entry until death was about 27 years for lung cancer and 22 years for nasal cancer, but the latency was shorter in the Canadian studies. The actual carcinogen may be an insoluble nickel compound such as nickel subsulfide.

NIOSH recommends that worker exposure to nickel dust not exceed 15 $\mu\text{g}/\text{m}^3$, as a 10-hour TWA.²⁰ The current OSHA standard for nickel metal is 1000 $\mu\text{g}/\text{m}^3$, as an 8-hour TWA.²⁰

VI. RESULTS AND DISCUSSION

A. Environmental

The air sampling results for cadmium and nickel are summarized by job classification in Table I. Individual exposure data are presented in Appendix A.

Time-weighted average (TWA) exposure concentrations for cadmium ranged between 3 and 284 (micrograms per cubic meter of air) ug/m^3 . Eighteen (45%) of the samples exceeded the NIOSH recommended standard of $40 \text{ ug}/\text{m}^3$. Five (12%) of the samples exceeded the NIOSH recommended ceiling concentration of $200 \text{ ug}/\text{m}^3$ and the OSHA 8-hour TWA standard of $200 \text{ ug}/\text{m}^3$.

Airborne cadmium exposures were highest in the terminal department. Samples obtained from eleven employees including 7 terminal machine operators, 3 slitter machine operators, and 1 salvage operator, ranged from 61 to $284 \text{ ug}/\text{m}^3$ (mean: $163 \text{ ug}/\text{m}^3$). As expected, those workers who processed negative material generally had much higher cadmium than nickel exposures. Although each of the machines in this department was equipped with local exhaust ventilation, the effectiveness of the ventilation was suspect since a considerable amount of dust was observed in most of the downdraft hoods servicing these machines. This indicates that the cadmium and/or nickel particulates are not being effectively captured and removed from these machines, consequently contributing to overall exposures to the workers in this area.

The coiling machine operators appeared to be the second highest cadmium exposed group in the plant. Seven of 10 operators monitored had cadmium exposures in excess of the NIOSH recommended standard of $40 \text{ ug}/\text{m}^3$. Levels ranged from 26 to $192 \text{ ug}/\text{m}^3$ (mean: $61 \text{ ug}/\text{m}^3$).

Unlike the terminal department employees who normally handle either positive or negative material during the workshift, the coilers handle an equal number of positive and negative electrodes while operating the coiling machines. This balance is reflected in the similarity between their cadmium and nickel exposures.

Cadmium exposures for workers monitored in the remaining areas of the plant were below the NIOSH recommended standard of $40 \text{ ug}/\text{m}^3$. However, three samples obtained from the nickel recovery operator and the employee who operated both the tab brusher and the punch press exceeded the NIOSH action level of $20 \text{ ug}/\text{m}^3$.

Review of the company's environmental surveillance records from 1977 to 1982 revealed the following information regarding cadmium exposures. In 1977-78 the company only monitored workers in the terminal department. Cadmium exposures ranged from 80 to $880 \text{ ug}/\text{M}^3$, with an average of $290 \text{ ug}/\text{M}^3$. These levels were somewhat higher than those measured during this survey. The reason for this could have been due to the fact that none of the machines

in the terminal department were, at that time, equipped with local exhaust ventilation. (The ventilation system was completely installed in 1979). In 1979 the company began monitoring workers in the ceiling department with inclusion of remaining production areas by 1982. Cadmium exposure levels for this period were similar to those levels measured during this evaluation.

Time-weighted average nickel exposure concentrations ranged between 6 and 630 $\mu\text{g}/\text{m}^3$ for all workers monitored during the survey. Thirty-three (79%) of the 42 samples exceeded the NIOSH recommended standard of 15 $\mu\text{g}/\text{m}^3$. Nickel exposures above the NIOSH criterion were present in all production areas monitored except the circuit test and battery assembly departments. None of the samples exceeded the OSHA nickel standard of 1000 $\mu\text{g}/\text{m}^3$.

The highest nickel exposure levels were obtained on workers in the sintering, terminal, coiling, and punch press areas. Workers in these areas routinely handle dry sintered nickel sheets or strips during the course of their workday. Three workers in the sintering area, a slurry mixer and two furnacemen, were monitored. The slurry mixer, who is the only employee in the plant who handles dry nickel powder, had a TWA nickel exposure of 630 $\mu\text{g}/\text{m}^3$ while processing 20 10-lb. batches of mix. NIOSH personnel observed that during the manual addition of the powder to the solution in the mixer bowls, some of the powder was expelled from the bowl onto the counter and floor. This condition contributed to the relatively high airborne nickel dust levels in this area. The sinter furnace operators had nickel exposures of 243 and 431 $\mu\text{g}/\text{m}^3$.

The eleven terminal department employees monitored were exposed to nickel particulate at concentrations ranging from 19 to 174 $\mu\text{g}/\text{m}^3$. Those workers processing positive electrodes, as expected, generally had much higher nickel than cadmium exposures. Nickel exposures for the ten coiling machine operators monitored ranged between 24 and 105 $\mu\text{g}/\text{m}^3$, with a mean of 50 $\mu\text{g}/\text{m}^3$. One worker who operated a positive electrode punch press for about two hours was exposed to a nickel concentration of 952 $\mu\text{g}/\text{m}^3$. When combined with an exposure of 64 $\mu\text{g}/\text{m}^3$ while operating the tab brusher machine for the balance of the workday, the calculated TWA nickel exposure was 325 $\mu\text{g}/\text{m}^3$. Independent exposure monitoring during these two operations revealed that the punch press operation is capable of generating substantial amounts of nickel particulate.

The only monitored departments having airborne concentrations below the NIOSH 15 ug/m³ criterion were circuit test and battery assembly. In both areas the cells are sealed, i.e., there is no physical contact with the negative or positive electrodes. Although there is some contact with the electrodes in the cell assembly operation, exposures above 15 ug/m³ probably resulted from the proximity of this operation to the coiling and punch press areas.

B. Medical

Between December 1980 and June 1982, 305 (98%) of 311 production workers participated in the company's medical screening program. As of October 1982, 46 of these 311 workers had been terminated, and 114 had been layed off (still maintained active employee status). Eighty-seven percent of the participants were females, 88% were white. The mean age at the time of testing was 40 years (median = 41 yrs, range: 20-65 yrs). The mean length of employment was 8.9 years (median = 9 yrs, range: 1-23 yrs). Two hundred twenty-three (73%) workers said that they were current or ex-smokers.

There were 199 workers in the high-exposure group and 106 workers in the low-exposure comparison group. Workers in the low-exposure group were slightly older (42 yrs vs. 40 yrs), had a smaller percentage of females (73% vs. 95%), and had a slightly higher mean length of employment (9.1 yrs vs. 8.8 yrs) than the high-exposure group.

Eighty-two (27%) of all workers had blood cadmium levels greater than 10 ng/ml. Sixty-three (32%) of the 199 workers in the high-exposure workers had blood cadmium levels above 10 ug/dl in contrast to 19 (18%) of the 106 low-exposure workers (p=0.01) (Table II). The geometric mean blood cadmium level of workers in the high-exposure group, 9.3 ng/dl, was significantly higher than that of the other production workers, 6.4 ng/dl (p=0.0001). The highest blood cadmium level was 69 ng/dl (terminal department inspector). Among workers in the high-exposure areas, the proportion of workers with elevated blood cadmium increased as duration of employment in high-exposure areas increased (p=0.0001) (Table III).

Kidney Function Evaluation

BUN values in two of 305 workers tested at Saft/Gould were above the lab reference value of 25 mg/dl. Neither of these worked in high exposure areas, although their total length of employment was 21.3 years and 10.3 years. One blood creatinine value was above

the reference range of 1.7 mg/dl. This worker had a history of 14 years in the high exposure areas. Mean blood creatinine was 1.2 mg/dl in both the low and high-exposure groups. Mean BUN in the high-exposure group was 15 mg/dl vs. 16 mg/dl in the low-exposure group ($p < 0.05$). Although this difference was statistically significant, a difference of 1.0 mg/dl creatinine is not biologically significant.

Urine beta-2 microglobulin concentrations were elevated above the upper reference limit of 370 ng/ml in 6 workers, including two with very high values: 1,924 and 10,700 ng/ml. All 6 workers had worked as inspectors in the terminal department (high-exposure group) for cumulative times ranging from 0.5 to 12.4 years (median 5.7 yr) and their length of employment at the company ranged from 12.3 years to 22.2 years, all longer than the median of 9 years for all employees.

Urine cadmium concentration is used widely as an index of cadmium exposure. Urine concentrations above 10 ug/l may indicate kidney damage in cadmium exposed workers. Eighty-two (27%) of the workers had urine cadmium concentrations of 10 ug/l or greater. Sixty-four (32%) of 199 high-exposed workers had elevated urine cadmium in contrast with 18 (17%) of 106 other production workers ($p = 0.004$) (Table IV). The geometric mean urine cadmium concentration in the high exposure group was 8.5 ug/l, compared to 5.4 ug/l in other production workers ($p = 0.0001$). As with blood cadmium, the proportion of workers having elevated urine cadmium generally increased as duration of employment in high-exposure areas increased ($p = 0.0005$) (Table V). There was a weak positive correlation between urine cadmium and both blood cadmium and urine beta-2 microglobulin levels, $r = 0.27$ ($p = 0.0001$) and $r = 0.19$ ($p = 0.0007$), respectively.

In the univariate analysis presented above, blood and urine cadmium was associated with duration of employment in a high exposure area among workers. Because cigarette smoking, age, and sex are known to cause an increase in the body burden of cadmium⁵, a regression model was used to evaluate the association between these variables, a history of employment at Saft and in high exposure areas, and blood and urine cadmium levels. Results of this analysis demonstrate that when controlling for the effects of sex, age, and a history of ever smoking cigarettes, blood and urine cadmium levels are significantly associated with years of work in a high exposure area, and neither age or smoking status contributed significantly (Tables VI, VII). Blood cadmium but not urine cadmium was associated with total years of employment at Saft/Gould (Table VI). Sex was also associated with blood and urine cadmium levels, with females having lower levels.

VII. CONCLUSIONS

The data collected during this investigation indicate that workers at Saft/Gould were overexposed to cadmium and nickel in the past and at the time of the survey. Results of the medical screening provide definitive evidence that workers (especially those in the terminal and coiling areas) have had increased cadmium absorption. This was related to duration of exposure in "elevated area" and is compatible with the elevated environmental cadmium concentrations measured. Based on the company's biological monitoring data, a number of workers appear to have early signs of cadmium-induced kidney disease. Although very few abnormal BUN and serum creatinine values were observed, these are insensitive tests of kidney dysfunction.

VIII. RECOMMENDATIONS

1. The presence of dust accumulation in the downdraft exhaust hoods of the slitter and terminal machines indicate that the systems' design velocities are inadequate for the removal of the collected particulates. In light of this observation, the design specifications for the systems should be reviewed to determine if their design duct velocities are at least 3500 feet per minute. If the design specifications specify a minimum duct velocity of 3500 fpm, a system performance evaluation should be conducted to determine the actual operating duct velocities. System adjustments should be made accordingly.
2. Based on the high nickel exposure of the positive punch press operator (952 ug/m^3), and the relatively high nickel (up to 105 ug/m^3) and cadmium (up to 192 ug/m^3) exposures of the coiler machine operators, these machines should be equipped with local exhaust ventilation.
3. In departments where exposures to cadmium exceed the NIOSH action level of 20 ug/m^3 , and exposures to nickel exceed the 15 ug/m^3 criterion, workers should be provided with appropriate respirators. Respirators certified for use in atmospheres containing nickel and/or cadmium dusts should be considered only an interim control measure until effective engineering controls are instituted.
4. Based on the observation that nickel powder is not effectively contained when the powder is added to the mixer, NIOSH recommends that the existing ventilation be further evaluated with respect to reducing airborne nickel levels.

5. The practice of bringing food, beverages and smoking materials in the production area should be discontinued.
6. A housekeeping program should be emphasized whereby equipment, work tables and floors are vacuumed at the end of each day.
7. We recommend medical follow-up by a specialist in kidney function (nephrologist) for all workers with a persistently elevated BUN, or serum creatinine, or with excessive excretion in urine of beta-2 microglobulin.
8. The company should continue their medical monitoring program. In addition to comparing individual workers test results with laboratory reference ranges, all workers' results should be compared with their previous test results and any substantial worsening over time should be investigated by a nephrologist.
9. Workers who are found to have blood and urine cadmium concentrations in excess of the laboratory's range of normal values should be followed closely with periodic retesting. Workers found to have evidence of kidney damage should be moved to an area of the plant where they will not be exposed to cadmium.

IX. REFERENCES

1. National Institute for Occupational Safety and Health. NIOSH manual of analytical methods. Vol 1, 2nd ed. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1977. (DHEW (NIOSH) publication no. 77-157-A).
2. Statistical Analyses System (SAS) User's Guide. SAS Institute Inc. Copy N.C. 1981 edition.
3. Webb M: Cadmium. Br Med Bull 1975;31:246-50.
4. Lauwerys R, Roels H, Regniers M, et al. Significance of cadmium concentration in blood and in urine in workers exposed to cadmium. Environ Res 1979;20:375-91.
5. Kjellstrom T. Exposure and accumulation of cadmium in populations from Japan, the United States, and Sweden. Environ Health Perspect 1979;28:169-97.
6. Bernard A, Buchet JP, Roels H, et al. Renal excretion of proteins and enzymes in workers exposed to cadmium. Eur. J. Clin. Invest. 1979;9:11.

7. Ellis KJ, Vartsky D, Zanzi I, et al. Cadmium: in vivo measurement in smokers and nonsmokers. *Science* 1979;205:323-25.
8. Friberg L, Piscator M, Nordberg GF, et al. Cadmium in the environment. Cleveland: CRC Press, 1974.
9. Baker EL, Peterson WA, Holtz J, Coleman C, Landrigan PJ. Subacute cadmium intoxication in jewelry workers: an evaluation of diagnostic procedures. *Arch Environ Health* 1979;34:173-77.
10. Scott R, Paterson PJ, Mills EA, et al. Clinical and biochemical abnormalities in coppersmiths exposed to cadmium. *Lancet* 1976;2:396-98.
11. Lemen RA, Lee JS, Wagoner JK, et al. Cancer mortality among cadmium production workers. *Ann NY Acad Sci* 1976;271:273-79.
12. Piscator M. Role of cadmium in carcinogenesis with special reference to cancer of the prostate. *Environ Health Perspect* 1981;40:107-20.
13. Thun M, Schnorr JM, Smith AB et al. Mortality Among a Cohort of U.S. Cadmium Production Workers - An Update. National Institute for Occupational Safety and Health. (document in review)
14. International Agency for Research on Cancer. Monograph on the evaluation of carcinogenic risk of chemicals to Non-Cadmium and Cadmium Compounds, 1976; 11:39-74
15. Page LB, Culver PJ. A syllabus of laboratory examinations in clinical diagnosis. Cambridge: Harvard University Press, 1962.
16. Rosen JF, Chesney R, Hamstra A, DeLuca H, Mahaffey K. Reduction in 1,25-dihydroxyvitamin D in children with increased lead absorption. *N Engl J Med* 1980;302:1128-31.
17. Kazantzis G. Renal tubular dysfunction and abnormalities of calcium metabolism in cadmium workers. *Environ Health Perspect* 1979;28:155-59.
18. National Institute for Occupational Safety and Health. Criteria for a recommended standard: occupational exposure to cadmium. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1976. (DHEW publication no. (NIOSH) 76-192).

19. Occupational Safety and Health Administration. OSHA safety and health standards. 29 CFR 1910.1000. Occupational Safety and Health Administration, revised 1981.
20. 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).
21. National Academy of Sciences. Committee on the medical and biological effects of environmental pollutants. Nickel. Washington, D.C., 1975.
22. Fisher AA. Contact dermatitis. 2nd ed. Philadelphia: Lea & Febiger, 1973.
23. McConnell LH, Fink JN, Schlueter DP, Schmidt MG. Asthma caused by nickel sensitivity. Ann Intern Med 1973; 78:888-890.
24. Malo J-L, Cartier A, Doepner M, Nieboer E, Evans S, Dolovich J. Occupational asthma caused by nickel sulfate. J Allergy Clin Immunol 1982; 69:55-59.
25. Proctor NH, Hughes JP. Chemical hazards of the workplace. Philadelphia: J.B. Lippencott Company, 1978.

X. AUTHORSHIP AND ACKNOWLEDGEMENTS

Evaluation Conducted and
Report Prepared by:

James M. Boiano
Industrial Hygienist
Industrial Hygiene Section

Jane A. Lipscomb, R.N., M.S.
Epidemiologist
Medical Section

Gary M. Liss, M.D., M.S.
Medical Officer
Medical Section

Evaluation Assistance: Steven H. Ahrenholz, M.S., C.I.H.
Industrial Hygienist
Industrial Hygiene Section

Roger Luckmann, M.D.
St. Paul - Ramsey Medical Center
St. Paul, Minnesota

Statistical Evaluation: William T. Stringer
Statistician
Support Services Branch

Originating Office: Hazard Evaluations and Technical
Assistance Branch
Division of Surveillance, Hazard
Evaluations, and Field Studies

Report Typed By: Jacqueline Grass
Clerk/Typist
Industrial Hygiene Section

XI. 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. Confidential Requestors
2. Saft America, Incorporated, St. Paul, Minnesota
3. IBEW - Local 110, St. Paul, Minnesota
4. NIOSH, Region V
5. 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

Summary of Cadmium and Nickel Exposures by Job Classification

Saft America, Incorporated, Portable Battery Division
 St. Paul, Minnesota
 HETA 80-187

November 18-19, 1981

Job Classification	Number of Workers Sampled	Airborne Concentrations (ug/m ³)			
		Cadmium		Nickel	
		Range	Mean	Range	Mean
Nickel slurry mixer	1	-	12	-	630
Sinter furnace operator	2	14-17	15	243-431	337
Vacuum chamber operator	2	10-21	15	29-61	45
Wet brusher	1	-	11	-	57
Ni recovery operator	2	-	22	9-24	17
E.D. machine operator	2	15-20	17	6-30	18
Tab brusher/punch press operator	1	-	31	-	325
Terminal machine operator	7	69-284	160	19-174	70
Slitter machine operator	3	61-270	199	27-108	57
Salvage operator	1	-	219	-	22
Coiling machine operator	10	26-192	61	24-105	50
Line spot welder	2	8-12	10	12-15	13
Cell assembler	2	-	11	15-20	17
Crimp press operator	2	4-11	7	7-14	11
Cell rack tender	2	4-5	4	6-7	6
Battery assembler operator	2	3-6	4	9-14	12
NIOSH Recommended Standard:		40 (200 ceiling)		15	
OSHA Standard:		200		1000	

TABLE II

Rates of Elevated and Normal Blood Cadmium Levels
Among 305 Production WorkersSaft America, Incorporated
St. Paul, Minnesota
HETA 80-187

October 1982

Exposure Group	> 10 ng/dl	< 10 ng/dl	Total
<u>high</u>	63 (32%)	136 (68%)	199
<u>low</u>	19 (18%)	87 (82%)	106
	82	223	305

Chi-square test = 6.64, df=1, p=0.01

Rate Ratio = 1.8

TABLE III

Rates of Elevated and Normal Blood Cadmium Concentrations
by Duration of Employment in High Exposure Area
199 Production Workers

Saft America, Incorporated
St. Paul, Minnesota
HETA 80-187

October 1982

Years In High Exposure Area	> 10 ng/dl	< 10 ng/dl	Total
0.1 -0.9	3 (6%)	44 (94%)	47
1.0 - 2.9	24 (33%)	49 (67%)	73
3.0 - 4.9	11 (37%)	19 (63%)	30
5.0 - 9.9	15 (44%)	19 (56%)	34
10.0 +	10 (67%)	5 (33%)	15
	63	136	199

Chi-square=25.21, df=4, p=0.0001

TABLE IV

Rates of Elevated and Normal Urine Cadmium Concentration
Among 305 Production Worker

Saft America, Incorporated
St. Paul, Minnesota
HETA 80-187

October 1982

Exposure Group	> 10 ug/l	< 10 ug/l	Total
<u>high</u>	64 (32%)	135 (68%)	199
<u>low</u>	18 (17%)	88 (83%)	106
	82	223	305

Chi-square=8.1, df=1, p=0.0044

Rate Ratio = 1.9

TABLE V

Rates of Elevated and Normal Urine Cadmium Concentrations
by Duration of Employment in High Exposure Area
199 Production Workers

Saft America, Incorporated
St. Paul, Minnesota
HETA 80-187

October 1982

Years in High Exposure Area	> 10 ug/l	< 10 ug/l	Total
0.1 - 0.9	7 (15%)	40 (85%)	47
1.0 - 2.9	18 (25%)	55 (75%)	73
3.0 - 4.9	15 (50%)	15 (63%)	30
5.0 - 9.9	18 (53%)	16 (47%)	34
10.0 +	6 (35%)	9 (40%)	15
	63	136	199

Chi-square=19.83, df=4, p=0.0005

TABLE VI

Regression Model of Blood Cadmium,
Exposure, and Demographic Variables

Saft America, Incorporated
St. Paul, Minnesota
HETA 80-187

October 1982

Variable	P Value
Years Employed	0.0001
Years in high exposure areas	0.0001
(Years in high exposure areas) ²	0.0025
Sex	0.0244
Age	0.1844
Never Smoked	0.9943

TABLE VII

Regression Model of Urine Cadmium,
Exposure, and Demographic Variables

Saft America, Incorporated
St. Paul, Minnesota
HETA 80-187

October 1982

Variable	P Value
Years in high exposure areas	0.0001
(Years in high exposure areas) ²	0.0002
Sex	0.0055
Years Employed	0.0976
Age	0.8264
Never Smoked	0.3122

APPENDIX A

Summary of Nickel and Cadmium Exposures by Job Classification

Soft America, Incorporated, Portable Battery Division
St. Paul, Minnesota

November 18-19, 1981
HETA 80-187

Date	Sample Location	Job Classification	Sampling Duration (min.)	Air Concentration (ug/m ³)		Remarks
				Cadmium	Nickel	
11-18	Mix room and furnace area	Nickel slurry mixer	497	12	630	Prepared 20 batches
11-18	Furnace area	Sinter furnace operator	465	17	243	Positive material
11-19	Furnace area	Sinter furnace operator	464	14	431	Positive material
11-18	Impregnation area	Vacuum chamber operator	478	21	61	Positive material
11-19	Impregnation area	Vacuum chamber operator	475	10	29	Positive material
11-19	Impregnation area	Wet brusher	242	11	57	Positive material
11-18	Impregnation area	Nickel recovery operator	450	22	9	
11-19	Impregnation area	Nickel recovery operator	462	22	24	
11-18	Electrodeposition machine A	E.D. machine operator	450	15	6	Negative material
11-19	Electrodeposition machine C	E.D. machine operator	472	20	30	Negative material
11-19	Impregnation and punch press areas	Tab brusher/punch press operator	429*	31*	325*	Positive electrodes
11-18	Terminal department	Terminal machine #4 operator	469	142	26	Negative cell type .250
11-18	Terminal department	Terminal machine #1 operator	421	284	19	Negative cell type 2.2
11-18	Terminal department	Terminal machine #8 operator	426	83	119	Positive cell type 1.2
11-18	Terminal department	Terminal machine #2 operator	450	267	61	Negative cell type 2.0
11-19	Terminal department	Terminal machine #2 operator	471	156	66	Positive cell type 2.2
11-19	Terminal department	Terminal machine #7 operator	444	125	27	Negative cell type 1.2
11-19	Terminal department	Terminal machine #8 operator	421	69	174	Positive cell type 1.2

NIOSH Recommended Standard (up to a 10-hr TWA):
OSHA Standard (8-hr TWA):

40 (200 ceiling) 15
200 1000

* Two filter samples were collected from this employee to assess the extent of nickel and cadmium exposure while operating each machine. The employee operated the tab brusher for 303 minutes and was exposed to 64 ug/m³ nickel and 22 ug/m³ cadmium. When operating the positive punch press for 126 minutes the nickel and cadmium exposure was 952 and 53 ug/m³, respectively. The cumulative TWA exposure for the workshift was calculated from the following formula: $E = (C_1T_1 + C_2T_2) / \text{total sampling time}$, where E is the equivalent TWA exposure for the workshift, C is the concentration in ug/m³ during a period of time T, and T is the duration of exposure in minutes at concentration C.

APPENDIX A (Continued)

Summary of Nickel and Cadmium Exposures by Job Classification

Saft America, Incorporated, Portable Battery Division
St. Paul, Minnesota

November 18-19, 1981

HETA 80-187

Date	Sample Location	Job Classification	Sampling Duration (min.)	8-hr. TWA ($\mu\text{g}/\text{m}^3$)		Remarks
				Cadmium	Nickel	
11-18	Terminal department	Slitter operator	446	270	27	Negative plaque
11-19	Terminal department	Slitter operator	453	265	35	Negative plaque
11-19	Terminal department	Slitter operator	438	61	108	Positive plaque
11-19	Terminal department	Salvage operator	427	219	22	Negative electrodes
11-18	Coiling department	Coiler operator machine #27	445	34	25	Cell size .100
11-18	Coiling department	Coiler operator machine #23	438	43	29	Cell size .150
11-18	Coiling department	Coiler operator machine #19	435	44	51	Cell size 2.0
11-18	Coiling department	Coiler operator machine #21	434	84	61	Cell size 2.0
11-19	Coiling department	Coiler operator machine #27	494	26	24	Cell size .100
11-19	Coiling department	Coiler operator machine #'s 28, 17	495	66	105	Cell size .225 and .750
11-19	Coiling department	Coiler operator machine #22	491	26	26	Cell size 2.0
11-19	Coiling department	Coiler operator machine #23	485	51	40	Cell size .150
11-19	Coiling department	Coiler operator machine #19	486	41	55	Cell size 2.0
11-19	Coiling department	Coiler operator machine #16	486	192	84	Cell size 4.0
11-18	Cell assembly, line 2	Spot welder	433	12	15	Electrodes in cell case
11-18	Cell assembly, line 3	Spot welder	480	8	12	Electrodes in cell case
11-19	Cell assembly, line 2	Assembler	430	11	20	Electrodes in cell case
11-18	Cell assembly, line 4	Assembler	474	11	15	Electrodes in cell case
11-19	Cell assembly, line 2	Crimp press operator	430	11	14	Electrodes in cell case
11-18	Cell assembly, line 5	Crimp press operator	472	4	7	Electrodes in cell case
11-18	Cycle test room	Cell rack tender	420	5	6	Cell case sealed
11-19	Cycle test room	Cell rack tender	464	4	7	Cell case sealed
11-18	Battery assembly area	Product assembler	417	6	14	Cell case sealed
11-19	Battery assembly area	Product assembler	432	3	9	Cell case sealed

NIOSH Recommended Standard (up to a 10-hr TWA):
OSHA Standard (8-hr TWA):

40 (200 ceiling) 15
200 1000

DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
ROBERT A. TAFT LABORATORIES
4676 COLUMBIA PARKWAY, CINCINNATI, OHIO 45226

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE, \$300

Third Class Mail



POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396