

# Effects of Welding on Health, II

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**American Welding Society**

# Effects of Welding on Health II

*An up-dated (January 1978-May 1979) literature survey and evaluation of the data recorded since the publication of the first report, to understand and improve the occupational health of welding personnel.*

Research performed at the Franklin Research Center under contract with the American Welding Society and supported by industry contributions.

Prepared for:  
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## Preface

This literature review has been prepared for the Safety and Health Committee of the American Welding Society to provide an assessment of current knowledge of the effects of welding on health, as well as to aid in the formulation of a research program in this area, as part of an ongoing program sponsored by the Committee. Previous work has included studies of fumes and gases, radiation, and noise generated during various forms of arc welding. Conclusions based on this review and recommendations for further research are presented in the introductory portions of the report. Chapter 1 summarizes the occupational exposures. Chapters 2 and 3 contain information related to the effects of exposure to byproducts of welding operations on humans and laboratory animals. Chapter 4 covers *in vitro* studies.

Referenced materials are available from the Franklin Institute.



# Introduction

The health of welders has always been a concern of the American Welding Society (AWS). Much of the earlier literature has been reviewed by The Franklin Research Center (FRC); findings were presented in October 1978 to the Safety and Health Committee of AWS in the report *Effects of Welding on Health* (Ref. 1).

Since then, much scientific study has been reported, and it was found necessary to update the earlier report. As the present work focuses mainly on health effects of welding, newly published methods of sampling and analyses of welding fume have not been emphasized.

Like workers in the metals industry, welders are generally exposed to a variety of occupational risks such as cuts, bruises, and sometimes more severe injuries due to the handling of massive metal objects. Furthermore, due to the nature of their work, welders are exposed to other occupational hazards such as heat, radiation, and the inhalation of fumes that might cause acute or chronic health effects. It is the purpose of this review to evaluate and present an update to include information on the effects of welding on health published from the start of 1978 through May 1979.

The reader is cautioned that the papers reviewed were examined only for manual consistency. No independent checks of the experiments were performed. This report must be read in conjunction with *Effects of Welding on Health* by Villaume et al.



## Executive Summary

Although welders are exposed to various potentially hazardous substances, overt clinical disease is an infrequent phenomenon among adult welders. This is due to continually improved industrial hygiene techniques, research findings, and the fact that repeated exposure to a given compound in small concentrations could stimulate various defense mechanisms in the human body to effectively deal with the "invader." Tolerance (that is, reduced sensitivity of the body to the toxic effects of chemicals) and the development of various immune responses are factors that contribute to the lack of overt toxicity syndromes in certain cases. On the other hand, life-style factors, such as smoking or drinking habits, use of drugs, and personal hygiene, could not only change the clinical picture of metal exposure, but also give rise to totally misleading findings. It follows, therefore, that we are faced with two situations that might lead to erroneous conclusions: first, subclinical cases that are considered normal on medical examinations; second, clinical cases that are inaccurately blamed on welding exposure. However, these two groups have in their biological systems a variable amount of "extrinsic substances." It is clear, therefore, that biological monitoring for the presence of various chemicals in biological fluids or tissues, or both, constitutes one accurate measure by which health personnel can estimate the level of exposure to a given toxic substance, particularly the complex mixture known to be involved in welding processes, especially when used in conjunction with careful environmental monitoring techniques.

Regular testing of the urine, blood, hair, or enzymatic patterns of welders could be used to alert the industrial hygienist to the possibility of overexposure to a given compound. There are two limitations to the use of biochemical testing:

(1) The only criterion in such analysis is the deviation from what would be expected to be "normal." Certain

biological tests, especially those that involve enzymatic systems, have a wide range of "normal" or control values.

(2) The toxicokinetics (that is, the absorption, distribution, and excretion) of different metals do not obey a fixed pattern of behavior; thus, while some metals are cumulative, others are not. With the development of modern technology, it would be very gratifying to develop sensitive and specific diagnostic tests to detect any ailment in welders long before any "irreversible" damage might occur.

Before recommending specific biochemical tests for determining the extent of welder exposure to fumes, it was found necessary to review the merits of the analysis of various heavy metals in biological fluids.

### Analyses for Metallic Compounds

It is becoming increasingly clear that analyses of blood, urine, expired air, and other specimens of biological origin, though not always reliable, have value in conjunction with environmental monitoring to determine the degree of exposure to environmental substances. The main justification for this joint approach lies in the fact that direct analysis of environmental air, though providing an accurate assessment of the amount of potentially toxic substances in ambient air, fails to precisely reflect the degree to which these materials are actually absorbed by different biological systems. Since the degree of toxicity is directly related to the amount of toxic substance delivered to the target organ, it follows that detection of these substances in biospecimens potentially provides a better measurement of exposure and, hence, the degree of toxicity. It is, therefore, suggested that the joint determination of the amount of metals or gases, or both, and their metabolites in biospecimens from welders (such

as blood, urine, hair, nails, saliva), plus environmental monitoring, could be used to measure the extent of exposure, and better estimate the total risk.

## Urine Analysis for Metallic Compounds

Generally speaking, urine analysis constitutes a practical method for monitoring the extent of exposure. Its value in the diagnosis of exposure to certain metals is dubious. Urine analysis of workers exposed to various metals showed that significant excretion in excess has been observed for such elements as nickel (Refs. 2 and 3);

chromium (Refs. 4 and 5); cadmium (Refs. 6, 7, and 8); lead (Refs. 9 and 10); arsenic (Ref. 11); fluorides (Refs. 12, 13, and 14); and selenium (Ref. 15). Some observations in recent publications are summarized in Table 1.

## Blood Analysis for Metallic Compounds

In 1977, Ulfvarson and Wold (Ref. 16) estimated the concentration of 17 trace elements (lead, strontium, rubidium, beryllium, gallium, zinc, copper, cobalt, iron, manganese, chromium, calcium, potassium, sulfur, phosphorus, silicon, and magnesium) in whole blood samples

**Table 1**  
**Observations from recently published urine analysis studies**

| Metal  | Occupation | Observations  | Reference                      |
|--|------------|---|--------------------------------|
| Nickel   | Welders    | 1. Correlation between exposure and excretion was demonstrated for levels 1 to 5 mg/m <sup>3</sup> .                      | 3. Norseth and Gundersen 1978  |
|  |            | 2. No correlation was found in welders exposed to less than 1 mg/m <sup>3</sup> .   |                                |
| 3. No increase in urinary Ni excretion was found in welders exposed to 0.1 mg/m <sup>3</sup> . |            |   |                                |
|  |            | 1. Moderate increase in urinary Ni excretion was observed in arc welders.   | 185. Bernacki and Parsons 1978 |
|  |            | 2. Measurement of urinary Ni is more sensitive and practical than that in serum for determining the extent of exposure.   |                                |
| Chromium   | Welders    | 1. Urinary Cr level is a good indicator of short term exposure.   | 4. Tola et al. 1977            |
|  |            | 2. A high degree of correlation was found between inhaled Cr and its concentration in urine.                              | 5. Gylseth et al. 1977         |
| Lead   | Welders    | 1. Exposure to Pb for short times increased its level in urine; prolonged exposure resulted in a less prominent increase. | 9. Cramer et al. 1973          |
|  |            | 2. Urinary ALA levels were increased after exposure to lead.  |                                |

of 71 persons utilizing various welding methods. They could not find a correlation between the level of exposure and blood levels. However, other authors found that the serum level of various metals is substantially increased in workers exposed to metals such as zinc (Ref. 17), copper, chromium, and nickel (Refs. 18 and 19), lead (Ref. 20), and cadmium (Ref. 6). Various observations are shown in Table 2.

The "normal" amount of various metals in urine and blood as well as methods of their determination are mentioned elsewhere (Refs. 22 through 26).

### Perspiration Analysis for Nickel

Sunderman (Ref. 27), in a review of the toxicity of nickel, maintained that the concentration of nickel in perspiration was approximately 20 times greater than that in urine. If this is the case, determination of nickel in perspiration may constitute a useful method for the estimation of exposure.

### Biochemical Tests for Metallic Exposure

Biochemical tests show changes in the function of various organs due to excessive exposure to a given compound. Nonetheless, due to the wide range of "normal" values, slight fluctuations in these values due to early toxicity cannot be easily detected.

Recently, some specific and highly sensitive tests have been developed to detect dysfunction of the target organs in early stages of poisoning. Such tests are exemplified by the determination of serum ornithine carbamyl transferase (OCT) levels and greyscale ultrasonography as tests for the detection of early liver damage. It is well known that antimony, beryllium, cadmium, cobalt, copper, and molybdenum bring about liver damage in humans. The effects of metallic elements usually present in a welding atmosphere on serum protein composition in humans are summarized in Table 3. Tables 4 and 5 summarize findings in experimental animals (modified from DeBruin, Ref. 28).

**Table 2**  
**Observations from recently published blood analysis studies**

| Metal     | Occupation                                   | Observations  | Reference                    |
|-----------|--|---|------------------------------|
| Chromium  | Autoworkers                                  | Cr blood levels were significantly high.  | 18. Clausen and Rastogi 1977 |
| Lead      | Autoworkers                                  | 1. Increased blood Pb levels was observed in 59 percent of workers.<br>2. Significant correlation between blood Pb levels and ALA-D activity was found.                         | 19. Clausen and Rastogi 1977 |
| Manganese | Workers in Mn alloy plant                    | Mn concentration in blood did not increase with the increase in exposure time.  | 21. Tsalev et al. 1977       |
| Nickel    | Autoworkers                                  | Ni blood levels were significantly high.  | 18. Clausen and Rastogi 1977 |
| Zinc      | Various occupations with high oxide exposure | 1. Serum Zn concentrations are high and could be used to diagnose occupational exposure.<br>2. Serum Zn level is high in patients with zinc fever, especially on the first day. | 17. Jaremin 1977             |



**Table 3**  
**Serum protein composition in human exposure**

| Compound                             | Exposure condition                         | Observations   | Interpretation  |
|--------------------------------------|--|--|---|
| Lead (inorg.)                        | Occupational workers, prolonged exposure   | General hypoalbuminemia with a corresponding rise of alpha and beta globulins in chronic plumbism; behavior of gamma globulin is not uniform; values either above or below normal were encountered. Slight protein shifts may occur in the absence of distinct signs of toxicity; changes are more pronounced with increasing severity of clinical signs and with length of contact. | Liver insufficiency, shown by functional tests; possible RES involvement. |
| Cadmium                              | Occup. exp.                                | Decline of albumin; rise of various globulins.   | Hemolytic action?   |
| Manganese                            | Occup. exp., chronic                       | Patients show a fall in albumin, and a rise in alpha, alpha <sub>2</sub> and gamma globulin; hyproproteinemia.   | Probable liver involvement.   |
| Beryllium                            | Chronic poison                             | Fall in A/G in chronic berylliosis; globulin rise; the course and stage of disease; hypergammaglobulinemia.  | Etiology not defined; fall in blood SH.                                   |
| Selenium                             | Se factory                                 | Decline of total protein content.  |   |
| Copper                               | Cu mining plants                           | Dysproteinemia, most marked in subjects with signs of poisoning.   | Hematological disorders (rise in blood Cu, Fe).                           |
| Molybdenum                           | Chronic exp. (CuMo plant)                  | Fall in A/G ratio; rise of globulins, particularly the alpha fraction.   | Liver dysfunction.  |
| Nickelcarbonyl, Ni (CO) <sub>4</sub> |  | Dysproteinemia; marked rise of alpha globulin.   | Lung edema; hematological changes.  |
| Carbon monoxide, CO                  | Occup. exp., coke furnace workers (a.s.o.) | Marked shift from albumin to globulin, notably beta globulin in heavy prolonged exposure (500 ppm and more).   | Etiology not well defined.  |

## Recommended Tests

### 1. Analysis of urine for:

(a) *Chromium*. Occupational exposure to chromium is reflected by an immediate increase in its concentration in urine. However, both blood and urinary content of chromium remain elevated for several years after the cessation of exposure.

(b) *Fluorides*. The concentration of fluorides in human urine is closely correlated to the amount that obtains access to the body either by the oral route or by inhalation. A characteristic feature of fluoride exposure is the short onset time for its appearance in urine. Urine analysis is, therefore, considered to be the best means of estimating fluoride exposure.

### 2. Analysis of blood for:

(a) *Copper*. Determination of copper in blood is a more reliable method for assessing the degree of exposure than is estimating its level in urine.

(b) *Lead*. Serum and urine levels of lead are reliable measures in the diagnosis of early lead exposure. Prolonged exposure to lead, however, produced lower urinary lead levels than immediate exposure (Ref. 8). Nonetheless, there is a high correlation between lead levels in ambient air and that in body fluids. This correlation permits the determination of the level of exposure and allows predictions of the total lead body burden. A more accurate diagnostic test for the severity of lead exposure is the estimation of the activity of ALAD (lead causes a decrease in ALAD level that is not related to smoking).

### 3. Analysis of hair for:

(a) *Lead*.

(b) *Cadmium*. It seems that there is no evidence of a quantitative correlation between the urinary cadmium level and either the degree or the duration of exposure. The amount of cadmium in urine increases significantly during continuous exposure and persists for several years

**Table 4**  
**Effect of elemental compounds on serum enzymes**

| Compound(s)              | Conditions of treatment (species)  | Enzyme(s) tested                             | Effect |
|--------------------------|--|--|--------|
| Lead (Pb) salts          | Multiple treatment, rabbit   | GOT, GPT                                     | Inc.   |
|                          | Acute treatment  | CPK, hexokinase                              | Inc.   |
|                          | Chronic pois. sheep  | GPT  | Inc.   |
|                          | Repeated i.p. injection, rat   | LDH  | Inc.   |
|                          | Oral dosage, guinea pig  | LDH, GOT-1 & 2, G1-DH                        | Inc.   |
|                          | Acute i.v. treatment   | Alk. Ph.                                     | Dec.   |
|                          | Chronic adm., sheep, rat   | Pseudo Ch.E                                  | Dec.   |
| Beryllium (Be) salts     | Chronic dosage   | Als. Ph.                                     | Inc.   |
|                          | Chronic treatment, rabbit  | Transaminase, MDH, ICDH, rat                 | Inc.   |
|                          | Lethal poisoning intra-tracheal injection                                    | Alk. Ph.                                     | Inc.   |
| Manganese (Mn) compounds | Repeated i.v. dosage   | Transaminase LDH                             | Inc.   |
|                          |  | Adenosine deaminase                          | Inc.   |
|                          |  | Pseudo Ch.E                                  | Dec.   |
| Cobalt (Co) salts        | Heart damaging doses, rat  | Transaminase, ALD, CPK, LDH                  | Inc.   |
| Cadmium (Cd) salts       | Multiple high dosage   | Transaminase                                 | Inc.   |
|                          | Repeated doses<br>Chronic pois.<br>Subacute treatment<br>Chronic, s.c. doses | Pseudo Ch.E                                  | Inc.   |
|                          |  | GOT, GPT, LDH                                | Inc.   |
|                          |  | GOT, G1-DH                                   | Inc.   |
|                          |  | Alk. Ph.                                     | Inc.   |
| Amylase                  | Inc.   |  |        |
| Copper (Cu) salts        | Repeated adm., cattle  | Transaminase LDH                             | Inc.   |
| Zinc (Zn) salts          | Various treatments   | LDH  | Dec.   |
| Molybdenum (Mo) salts    | Acute intake   | Pseudo Ch.E                                  | Dec.   |
|                          |  | Alk. Ph.                                     | Dec.   |
| Nickel (Ni) chloride     | Chronic, i.v. intoxication, rabbit   | GOT, GPT, LDH, ALD                           | Inc.   |
| Selenium (Se) salts      | Varying doses  | Pseudo Ch.E                                  | Dec.   |
| Inorganic fluoride (F)   | Fluorotic dosage, cattle, chicken, rabbit                                    | Alk. Ph.                                     | Inc.   |
| Carbon monoxide (CO)     |  | ALD, CKP, GOT, LDH, MDH & SHD<br>Pseudo Ch.E | Inc.   |

**Table 5**  
**Serum lipoprotein changes in animal poisoning**

| Compound           | Animal species | Treatment, condition   | Result   |
|--------------------|----------------|--|--|
| Lead acetate       | Rabbit<br>Rat  | Long term i.e. administration<br>Intraperitoneal application | Increase of beta lipo-<br>protein, with associated<br>hypercholesterolemia |
| Zinc salt          | Rabbit         | Acute doses  | Shift from alpha to beta<br>fraction; rise of beta/<br>alpha ratio         |
| Cadmium<br>salt    |                | Chronic doses  | Increase of beta/alpha<br>ratio  |
| Carbon<br>monoxide | Rabbit         | Chronic exposure (2500 h,<br>100 ppm)                        | Rise in beta/alpha<br>quotient   |
| Silica             | Guinea<br>pig  | Intraperitoneal  | Rise in beta lipopro-<br>tein; hyperlipemia                                |
| Quartz<br>powder   | Dog,<br>rabbit | Intrabronchially, intra-<br>venously                         | Rise in beta lipopro-<br>tein fraction                                     |

after the cessation of exposure. Therefore, little or no information can be obtained from blood or urinary levels of cadmium; however, this can be used as a rough estimate of the amount of cadmium stored in the body. Hair samples are, therefore, recommended to estimate the exposure.

(c) *Nickel*.

4. Fecal analysis for manganese.

The amount of manganese excreted in urine represents

but a small fraction of the total amount in the body. It follows that the value of the determination of manganese in urine as an index of the exposure level is questionable. The blood level in workers exposed to manganese could be as high as four times the normal value. Determination of blood levels of manganese is of greater significance than that of urine. Manganese exposure is claimed to be accurately determined by estimating its level in the feces (Ref. 29). This is dependent on the fact that manganese is preferentially excreted via bile.

## Technical Summary

According to a recent population survey in the U.S. (unpublished report, Bureau of Labor Statistics 1979), there are an estimated 713,000 welders and flame cutters who are actively employed. Welders are exposed, in varying degrees, to occupational hazards such as cuts, bruises, and more severe injuries involved in the handling of metal objects. Furthermore, because of the nature of their work, welders are exposed to heat, radiation, noise, and welding fumes that may cause acute or chronic health effects.

It is the objective of this document to update an earlier report titled *Effects of Welding on Health*, to evaluate the present state of knowledge of the effects of welding on health, and to provide recommendations to the American Welding Society for future research. To accomplish these three objectives, scientific studies published between the start of 1978 and May 1979 were evaluated. The information was evaluated and arranged in four chapters: The Exposure, Effect of Welding on Human Health, Toxicologic Investigations in Animals, and In Vitro Studies.

### The Exposure

In this chapter, studies performed on welding fumes, gases, radiation, and noise are summarized.

### Fumes

The objective of engineers and industrial hygienists is to reduce the concentration of fumes in the welder's environment. This goal can be achieved by: (a) use of welding methods that produce the least volume of fumes, and (b) adjustment of the rate at which fumes are removed, i.e., ventilation requirements. The use of inert gas welding was found to reduce the diffusion of metal vapor and, hence, fume generation, as shown by Kobayashi et al. (Refs. 30 and 31). From an industrial hygiene

viewpoint, the publications of Stern (Refs. 32 through 35) are of interest. In a series of articles, Stern suggested a classification of welding electrodes according to what he called the "electrode profile." Each type of electrode, according to Stern, should carry a label showing its productivity in kg/hr as compared to the air flow (in m<sup>3</sup>/sec) required to bring the generated fumes to the recommended TLV's. A formula for calculating the ventilation requirements was developed by Magnusson (Ref. 36).

### Gases

The sources of gases that are apt to be produced during welding are discussed. Gases cited are: nitrogen oxides, ozone, carbon monoxide, and, under certain unique conditions, phosgene or phosphine.

### Radiation

Welders are exposed to three types of radiation: ultraviolet, visible, and infrared. Hinrichs (Ref. 37) maintained that, as in the case of fumes, the extent of radiation differs from one welding process to the other. Highest emissions were found in gas metal arc welding and the lowest in plasma arc welding.

Visible light produced during welding was found to be predominantly in the short wave (420 to 430 nm) spectrum. Zaborski (Ref. 38) attributed these emissions to atomic and molecular radiations from vapors and gases in the arc. Ozone is generated by ultraviolet emission at some distance from the arc. Exposure to ultraviolet light could cause conjunctivitis, or "welder's flash." Shade shield recommendations by NIOSH (ANSI Standard Z87.1-78) should be followed. It is important to recognize that certain chemicals, such as the furocoumarins and psoralens, present in various foods and drugs have the capacity to increase photosensitization to UV radiations. Ray burns of the skin should be interpreted not only in terms of light intensity but also in terms of possible photosensitization.

The most dangerous effect of infrared radiation is the development of cataracts, which was found to be caused by wave lengths below 1400 nm.

## Noise

Excessive exposure to noise could be a substantial health hazard to welders. Noise is associated with other shop operations as well as with welding.

## Effects of Welding on Human Health

In this chapter, the effects of welding fumes on human physiological systems are discussed. The data about the possible carcinogenicity of welding fumes, human fatalities, and recent epidemiological studies are summarized and evaluated.

## Background

It has been shown by Tierney (Ref. 39) that health hazards associated with welding fumes constitute only a small fraction of the total occupational risk of welding. Hazards from mechanical or accidental events that, fortunately, could be easily eliminated constitute the major bulk of risk. The scope of this report is limited to the effects of welding fumes on health.

## Toxicity to Various Organs

### Respiratory System

Factors that govern the toxicity of gases and particulates in welding fumes and various defense mechanisms that are available to the body to expel these undesirable substances were reviewed. The well known inhibition of the body's defense system by tobacco smoke and the synergistic effects of smoking acting in concert with other materials can lead to lung dysfunctions of varied types.

Reported cases of pneumoconiosis from particulates such as iron, aluminum, copper, and beryllium were discussed. The introduction of scanning electron microscopy enabled Guidotti and DeNee (Ref. 40) to study metal particles that were not otherwise visible. Using the same technique, Eskilsson et al. (Ref. 41) found that the most biologically active fumes contained particles rich in chromium often associated with potassium.

Whether or not aluminum and iron particles induce interstitial fibrosis in the lungs remains an unresolved question. Thus, while Patel et al. (Ref. 42) claimed that iron oxide is fibrogenic, Stettler et al. (Ref. 43) maintained that it is not.

Agents that produce pulmonary irritation, such as nitrogen oxides, ozone, and cadmium vapor, were discussed. Only one case of acute pulmonary edema was attributed to nitrogen oxides and ozone. The case involved the use of oxyacetylene and tungsten arc welding in a confined space. The patient was treated with ampicillin and

prednisone, and complete recovery ensued in eleven days. Cadmium fumes, on the other hand, induced pulmonary edema that ended fatally in one case (Ref. 186).

Lung function tests were used to study the effect of welding fumes on the lung. Since lung function tests are insensitive measures of lung dysfunction, test results may appear normal in the presence of other manifestations or disease. Despite this problem, the lung function tests are especially useful in the early detection of the pneumoconiosis.

### Eye and Vision

The effects of fumes and radiation on welder's eyes were discussed. It appears that ultraviolet radiation is the major source of complaints. Ross (Ref. 44) examined 926 welders over a six-year period. He could find no long-term effects on either near or far vision. He reported only one case of cataracts that was unrelated to infrared exposure.

### Ears and Hearing

An average hearing loss of 34 dB in the speech frequency range was reported (Ref. 44). Adequate protection against noise is necessary.

### Skin

Burns from hot metal and ultraviolet radiation are quite common among welders. In view of the material's meltability, nylon clothing does not provide adequate skin protection.

### Gastrointestinal Tract

Welders using low hydrogen electrodes complained of loss of appetite and stomach ache (Ref. 45).

### Cardiovascular System

Although dynamic arterial hypertension was reported by Gola and Galazka (Ref. 46), a detailed study is needed to confirm the effect of fumes on the cardiovascular system.

### Blood

Nickel, cobalt, chromium, iron, and molybdenum induced a hemolytic effect on human erythrocytes *in vitro* (Ref. 47). Only one case of granulocytopenia was reported in a welder who was employed for 20 years (Ref. 46). However, since several drugs and chemicals are able to induce such an effect, the association of granulocytopenia to welding fumes is questionable. Medicaments that might have been used by that welder (a 39-year old) were not discussed.

### Central Nervous System

Manganese, a component of welding fumes, seems to cause certain visual motor disturbances and reduction in short-term memory acuity. Although cases of definite neurological manifestations were reported in workers in a battery factory (not welders), no studies indicating that this is a problem in welders have been found recently. Past reports show that chronic manganism is seen only rarely in welders.

### Liver

Exposure to various metals and their oxides in welding fumes resulted in certain biochemical changes in the liver

that are discussed below under the heading Biochemical Changes.

#### Urinary System

Cases of urinary system involvement in welders were not published in 1978-79. However, exposure to cadmium and lead fumes produced damage to the proximal tubules of the kidney.

#### Allergic Reactions (Metal Fume Fever)

Generally, more febrile episodes were encountered in welders than in nonwelders. This is due to exposure to welding fumes, especially those that are rich in zinc oxide. Removal of the patients from the polluted environment and supportive measures constitute the main line of treatment.

#### Biochemical Changes

The most consistent biochemical change observed was the decrease in the activity of  $\delta$ -aminolevulinic acid dehydratase due to the exposure to fumes rich in lead. This is a reliable test because the result is not affected by smoking. Although serum and urine levels of lead were also increased, lead determination in these fluids is a reliable measure in the diagnosis of early lead exposure. Prolonged exposure produced lower urinary lead levels than immediate exposure.

For the 1978-1979 period, no published research on the effect of welding fume on the musculoskeletal, reproductive and endocrine systems, or on teeth and the oral cavity was found.

## Carcinogenicity of the Fumes

The reported induction of cancer due to the occupation of welding will be mentioned briefly. To guard against hasty conclusions, each case will be followed by a short comment.

#### Skin Cancer

The case reported by Raynor et al. (Ref. 48) involved a 59-year old welder who suffered a traumatic injury of the left thumbnail in which a "black welding flux" was introduced under the nail. An epithelioid cell malignant tumor was observed 13 years later in the dorsum of the distal phalanx of the involved thumb. Neither a detailed history of the patient's occupation before and after the accident nor the chemical composition of the "black flux" were given.

Examination of this case throws a great deal of uncertainty on the causative agent. In the absence of a chemical analysis of the "black flux," it would be very difficult to judge whether or not it was the agent that caused the cancer. It is well known that certain substances such as arsenic, cutting oil, and tars are capable of inducing skin cancer. Exposure of the traumatized tissues to any of the cancer-causing agents, either during work or at home, could have caused the development of cancer. An analysis of the previous occupational exposure of this patient and his activities during the 13 years would be necessary to reach a firm conclusion.

#### Brain Tumors

In this case, the patient was a 52-year old welder who had spent 22 years as an electric welder; prior to that, he had been a coal miner. Before admission to the hospital he spent two weeks welding zinc coated pipes. He then developed symptoms typical of metal fume fever. Analysis of serum-metal levels showed a fivefold elevation in zinc. The autopsy showed bronchial problems as well as "black lung" indications, tumors in the parietal lobe, and in the left hemisphere (Ref. 17).

Zinc is normally present in the brain, and it is unlikely that a two-week exposure to zinc fumes could have been the sole cause of the brain tumors. It is technically justifiable to assume that excessive zinc absorption might have contributed to the unveiling of latent clinical symptoms of preexisting brain tumors of unknown etiology.

#### Cancer in the Lung and Nasal Sinuses

Reported cases of lung and nasal sinus cancer were due to exposure to nickel in a nickel refinery (Ref. 49), but none have been reported in welders. Although nickel is present in welding fumes, there is no way of extrapolating the above results to welders because of the unknown interactions of nickel, iron, and other metals, gases, etc., that occur during welding. The evidence appears to show that typical nickel nasal erosions do not occur in welders.

#### Cancer of the Bladder

Perhaps the epidemiological studies performed by Milham (Ref. 50) offer the best framework for a study of cancer risk in welders. In his study, Milham analyzed the mortality patterns associated with exposure to metals in Washington State in the period 1950 to 1971. He concluded that welders and flame cutters develop more lung cancer and lung related diseases than expected. Welders also showed a high, though statistically nonsignificant, proportionate mortality ratio from cancer of the urinary bladder. Although this study is indicative, it is not definitive, and complete occupational exposure history of these people should be studied before reaching firm conclusions. Continuous irritation as from bladder stones could theoretically initiate tumorigenesis. Exposure to known cancer-producing substances, such as benzidine or betanaphthylamine, should also be taken into account.

## Toxicokinesis of the Fume

The fate of fluorides in welders' bodies was studied by Pantucek (Ref. 51). He found a close correlation between the concentrations of fluorides in air and that in urine of welders.

## Epidemiologic Studies

Epidemiologic studies conducted by Buncher et al. in 1977 (Ref. 52) revealed that skin injuries and the occurrence of cough in the morning in welders are the most significant findings. The epidemiologic study of Milham (Ref. 50) was mentioned earlier.

## Human Fatalities

Four fatalities were attributed to welding (Refs. 17, 44, 48, and 53). The duration of exposure ranged from 5 to 22 years and the causes of death were lung related.

## Toxicologic Investigations in Animals

Only four papers appeared in 1978-1979 that discuss the effect of welding fumes on experimental animals. The bulk of the literature shows that only single metallic components were used for the study. This is unfortunate, since results from such experiments are in no way applicable to the welding fumes. Six reasons were offered

at the start of this summary to explain why such experiments do not add to the understanding of the fume toxicity. However, these experiments were included in an attempt to find possible correlations between pathological lesions from metals and their level in biological fluids.

## In Vitro Studies

Stern (Refs. 33 and 34) and Maxild et al. (Ref. 54), using Salmonella to test for the mutagenicity of welding fumes, found that those fumes that evolved were in some instances mutagenic and in others not mutagenic. See the initial report, *Effects of Welding on Health*, p. 55, for a complete discussion of Maxild's results. Stern's work and Maxild's are essentially similar.

## Recommendations

Reports of human exposure published in the last two years show that a large amount of information on the health effects of welding has been gathered. The information is incomplete, some is questionable, and areas remain where more study and information are needed in order to develop methods for protecting welders. The following recommendations are submitted for consideration.

### Epidemiologic Studies

Epidemiologic studies have provided a great deal of knowledge about the toxicity of various substances such as lead, arsenic, and asbestos in the workplace. Since epidemiologic data are derived from observations on human populations, such studies are far more significant than animal studies. There are numerous variables that, if left unaccounted for in the analysis of any association between a given substance and the resulting health effect, may lead to either a false positive or a misestimation of the magnitude of the risk. For the occupation of welding, these variables include not only the welding fumes, which vary dramatically in their composition from minute to minute, but also exposure to other agents in the occupational setting, the life-style, and habits such as cigarette smoking, street drug usage, and dietary patterns. It is recommended that future epidemiologic studies carefully consider the following points:

(1) Although it is known that the most prevalent (obvious) health effects in the welding population are those that involve the eye and lung, any study must take into account other possible effects, positive or negative, on various physiological systems of welders. This would include considerations of such diseases as dermatoses, cataracts, coronary heart diseases, dental involvement, hearing loss, and cancer. For example, an unprotected

welder could suffer a loss of hearing with a subsequent social handicap and a reduction in his quality of life, although precautions may have been taken to protect other organs such as eyes and lungs.

(2) A query to NIOSH's National Occupational Hazard Survey Data Base yielded a list of about 1,000 agents to which welders and flame cutters (code 680) were exposed during the survey. Analyses of epidemiologic data should take into account other agents (that are known to be carcinogenic in man such as asbestos) to which welders were exposed in addition to the welding exposure. Cancer could be caused by these agents and erroneously attributed to welding.

(3) Obviously, smoking and other life-style factors of the welders studied must be taken into consideration.

(4) Reliable records should be kept of the type of metal(s) that is being welded, the method used (including the flux or shielding gas), the welding rods or electrodes (including coating or contaminants), as well as other agents such as degreasing solvents. The composition of fume should be analyzed, and industrial hygiene measurements of the workplace atmosphere should be performed periodically and recorded. Ideally, a detailed history similar to the 24-hour overall history used in nutritional studies should be applied to an assessment of past occupational exposure.

(5) Although the concentration of pollutants in air can be accurately measured, this may not reflect the actual dosage received or the body burden. Periodic testing of blood, urine, hair, and feces for levels of metals such as chromium, nickel, cadmium, etc., should be performed; results of these tests should be considered during analysis of the epidemiological findings. A health effect could be associated with the presence of metals in the body, when enough data has been collected to show what normal variations could be expected and which were excursions that indicate a start of potential damage.



## Experimental Studies

Metals such as arsenic, cadmium, cobalt, copper, lead, manganese, molybdenum, nickel, and zinc are teratogenic in experimental animals, but no mention in the literature of the teratogenic potential of welding fumes that contain these metals was found. To take appropriate measures for protecting female workers who are exposed to the fumes during childbearing age, it will be necessary to study any potential teratogenic effect of welding fumes in experimental animals.

Reproductive effects have not been sought in human epidemiological studies or in experiments with animal models. Such a study might be initiated to learn if fertility or other changes are evident among welders.

Interferon (a substance that plays an important role in cellular defense mechanisms such as phagocytosis) production in humans is known to be depressed by certain dust particles. Interferon is one of the defense mechanisms of the lung that may be breached while inhaling welding fumes, with a possible higher occurrence of lung diseases.

It therefore would be of great interest, at least from an academic viewpoint, to study the interrelationship between the presence of interferon and welding fumes. This study may lead to a better understanding of the role of interferon in protecting the lungs from possible damage and in clearing the lungs of both metal and organic particles.

## Safety Measures

Work practices in the U.S. have gone far to ensure the protection of welders from the hazards of their work. Good industrial hygiene practices, the continued improvement of personal protective devices, the health and safety standards, and the studies established and performed by the American Welding Society and by other technical societies in the U.K., Sweden, and Japan have played a large part in reducing the risk due to welding in the industrial environment.