

Effects of Welding on Health, I



American Welding Society



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Effects of Welding on Health

A literature survey and evaluation to establish the state-of-the-art and to point the directions for future research 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
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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. Appendix A of this report reviews AWS sponsored studies and others in order to clarify the occupational exposures that are summarized in Chapter 1. Chapters 2 and 3 contain information related to the effects of exposure to byproducts of welding operations on humans and on laboratory animals. Chapter 4 covers studies of the mutagenicity of welding fume condensate.

Introduction

According to census figures (Ref. 1), there were an estimated 572,000 welders and cutters in 1970. Sosnin (Ref. 2) has estimated that today there are close to one million individuals whose occupation requires a substantial amount of welding; this latter estimate includes pipefitters and mechanics who view welding only as one of the tools of their trade. Therefore, the health and safety of this large occupational group is an important concern.

Welders are not a homogeneous group. They work under a variety of conditions: outdoors, indoors in open as well as confined spaces, underwater, and above ground on construction sites. They also utilize a large number of welding and cutting processes. However, many of these have in common the production of fumes, gases, radiation, and other potentially harmful agents. The review that follows attempts to assess the effects of these agents on the health of welders.

The discussion concentrates on health effects due directly to the welding process. It does not attempt to

treat safety problems, nor does it cover in any detail the accompanying hazards that, although often found in the welding environment, are not created directly by the welding process.

Occupational health depends upon the conditions and exposures received in the workers' environment. In Chapter 1 of this report, the physical and chemical agents to which welders may be exposed and the conditions of their generation are summarized; details are provided in Appendix A. In Chapter 2, the effects of this exposure on welders and others in the environment are reviewed. Since welders are exposed to a number of factors simultaneously, the chapter is subdivided by organ system, not by agent. Effects on the respiratory system, skin, eyes, and internal organs are considered. Chapter 3 presents the results of studies in experimental animals; the review concludes with a discussion of special studies of the mutagenicity of welding fumes.

Executive Summary

Most commonly used welding processes emit fumes, gases, electromagnetic radiation, and noise as byproducts of their operation. During welding, workers are potentially exposed to all of these agents. The fumes are chemically very complex, arising primarily from the filler metals and any electrode coatings or cores. The potential exposure varies with the process and welding conditions employed.

Numerous reports of the effects of welding on health have been published. Many cases of acute poisoning, due to excess exposure to one or more gases and fumes, have been documented. However, other than lung involvement, few chronic effects have been attributed to welding, and, in almost all the studies of chronic effects, welding of ferrous metals was involved. This does not necessarily mean that welding ferrous or any other metals presents no routine health risks. Frequently, published studies have not been appropriately designed to detect the effects of chronic exposure and have not adequately examined special groups (for example, welders of aluminum or stainless steel) with unique exposure factors. Conclusions resulting from this review are presented briefly in the following sections.

Acute Poisoning

Exposure to ozone generated during gas shielded arc welding, especially of aluminum, may produce irritation and inflammation of the respiratory tract, excess fluid and hemorrhage in the lungs, and sometimes headache, lethargy, and eye irritation. Severe cases could be fatal. Ozone is generated at a distance from the arc by the action of the emitted ultraviolet radiation ($<240\text{ nm}$) on atmospheric oxygen. Nitrogen oxides, which are produced in welding arcs and flames by thermal oxidation of atmospheric nitrogen, produce similar respiratory tract effects.

Metal fume fever occurs in welders who inhale zinc-containing fumes, although other fume components may also produce the symptoms of this condition; for example, a metallic taste, chills, thirst, fever, muscle aches, chest soreness, fatigue, gastrointestinal pain, headache, nausea, and vomiting. The symptoms usually subside within 1 to 3 days of exposure without residual effect.

Chronic Lung Conditions

Protracted inhalation of welding fume particles leads to their accumulation in the lungs, a condition seen as dense areas on chest x-ray films. The severity of changes in lung x-rays is proportional to the length of welding experience; however, the changes seen in such x-rays are not necessarily associated with reduced lung function or disease.

In general, welders of ferrous materials have been found to have an increased frequency of respiratory symptoms such as chronic bronchitis. No conclusive studies have been made to determine whether reduced lung function, emphysema, or other chronic respiratory conditions generally occur more frequently in these groups than in nonwelders.

Significant levels of ozone are produced in certain gas shielded welding operations. Lengthy exposure of experimental animals to this gas has induced the formation of fibrous tissue and caused emphysema-like and other effects in the lungs. No studies of the long-term effects of this gas on welding populations exposed to it have been undertaken.

Studies of the effects of welding fumes on experimental animals have indicated excess deposition of fibrous tissue in the lungs when a variety of metals were welded. However, in these experiments, fume concentrations were always very high, and experimental conditions were incompletely described; therefore, it is dif-

difficult to relate the importance of these findings to the welding experience.

Eye

Ultraviolet (>170 nm), visible, and infrared radiation are emitted by the welding arc. Welders not wearing eye protection and others in the vicinity of the arc are at risk to the effects of this radiation.

Ultraviolet radiation produces the condition known as "arc eye," an acute inflammation of the external structures of the eye; the symptoms disappear within 1 to 2 days. Infrared radiation penetrates the interior of the eye and can cause burns on the retina.

There is no evidence that cataracts occur more frequently in arc welders than in nonwelders; the results of studies designed to detect cataracts in welders have yet to be published.

Skin

Exposed skin is susceptible to the effects of ultraviolet radiation from the arc (for example, erythema) and of any fume components capable of skin sensitization or irritation. Chromium compounds, which occur in stainless steel welding fumes, are a frequent cause of dermatitis.

Cardiovascular Disease

Electrocardiograms and blood pressures have been measured in several studies. In most of these, electro-

cardiograms of welders did not differ significantly from control groups of nonwelders, and welders have been shown to have normal or slightly lower blood pressure than the control groups.

Carbon monoxide is generated from carbon dioxide in the gas shielded welding processes. It combines avidly with hemoglobin, reducing the oxygen-carrying capacity of the blood. Exposure to carbon monoxide may present an added health risk to those with heart disease. No studies of heart disease in welders performing carbon dioxide shielded welding have been documented.

Nervous System and Other Organs

Lead and manganese can occur in the fumes of certain welding operations; both are toxic to the nervous system. Except for those attributable to these two substances, no reliable reports of effects of welding exposure to the nervous system have been published.

There is no significant evidence that welders have a higher incidence of other chronic diseases.

Carcinogenicity

Based upon available information, there does not appear to be any evidence that exposure to welding fumes and gases induces lung or other forms of cancer. Further study is required of groups exposed to specific kinds of fumes. The welding arc emits ultraviolet radiation of wavelengths that produce skin tumors in animals, but no study of this effect in welders has been reported.

Technical Summary

The welding exposure is unique. There is no material from any other source directly comparable to the composition and structure of welding fumes. Exposure to byproduct gases accompanies fume exposure; therefore, ability to extrapolate from exposures received by workers in other industries to those in the welding environment is limited.

Welders are potentially exposed to a large number of chemical and physical agents. Several questions of the effects of welding on health are apparent:

1. Do welders have a higher incidence of chronic respiratory tract disease than nonwelders?
2. Do they have a higher incidence of respiratory tract cancer or other malignancies?
3. Does the ingestion and inhalation of welding fumes and gases result in any other systemic diseases?

Since many different materials and processes are used, these questions should be asked for specific subgroups of welders. In available studies, adequate data to make these distinctions often have not been reported, and any conclusions made apply to welding in general. However, it should be noted that most welding involves ferrous-based metals.

Chronic Respiratory Disease

Protracted inhalation of welding fume particles leads to their accumulation in the lungs. Deposits of these fume particles in the lungs have been noted as dense areas on chest x-ray films. The severity of the changes in the x-rays has been shown to be proportional to the duration of welding exposure (Refs. 3, 4, and 5). However, this evidence of siderosis and pneumoconiosis is

not necessarily associated with clinical evidence of lung disease.

Ten major epidemiological studies have contributed to the assessment of risk of chronic restrictive or obstructive lung disease or both in welders. All studies attempted to correct for the confounding effects of smoking. Most obtained previous occupational or medical histories or both (Refs. 3, and 6 through 15). However, in a number of studies (Refs. 3, 8, 9, 11 through 14, 16, and 17), the subjects were shipyard welders in whom concomitant exposure to asbestos is likely; in only one case (Ref. 8) was an attempt made to correct for this confounding effect. Although SMAW or FCAW or both were used by welders in all of the subject populations, descriptive details of the exposure varied between studies and were generally inadequate. All studies were published during 1964 or later except for one (Ref. 3) published in 1947; the latter may not be applicable to current conditions.

The most important criticism of all of these studies is that they were of the "point prevalence" or "cross-sectional" type. In this design, workers at a plant or plants at a particular point in time are studied. Serious bias may enter, because workers who are ill, have died, or have taken up other employment for health-related reasons are omitted. Point prevalence studies are likely to detect only subclinical disease.

Of nine studies that investigated respiratory symptoms, seven (Refs. 3, 7, 9, 11, and 15 through 17) reported an increased prevalence in welders compared to a reference group; in two (Refs. 6, 10, and 12 through 14), no significant difference was found. In only two (Refs. 9 and 16) could a pulmonary function deficit be

reasonably attributed to the welding experience; this deficit was stated to be subclinical in one study (Ref. 9) and was probably subclinical in the second. Thus, from these studies, it appears that exposure to the welding fumes or gases or both caused irritation to the respiratory tract, but not of a sufficient degree to produce significant pulmonary dysfunction (Ref. 18) among current workers. However, the strength of these conclusions is seriously weakened by the bias inherent in point prevalence surveys.

There have been studies in which laboratory animals inhaled welding fumes and gases, or fume concentrate was administered to them intratracheally. Soviet and other Eastern European investigators have observed evidence of pulmonary fibrosis in rats (Refs. 19 through 26) and guinea pigs (Ref. 27) that usually involved experimentation with covered electrodes. It is not possible to implicate a particular agent or agents or to evaluate the importance of these findings because of deficiencies in these experiments, such as failure to report one or more of the following: electrode composition, fume level, fume composition, concentration of gases, and exposure schedule of the animal populations; in addition, dose-response relationships were not investigated.

The pulmonary hazard potential varies with the process and metals being welded. In most situations, fume production is the major source of atmospheric contamination; however, in certain gas shielded welding operations, ozone can be produced in significant amounts. The highest rate of ozone production occurs during GMAW (argon) of aluminum and its alloys (Ref. 28). Significant quantities are also produced in other GMAW and GTAW operations. Because the ozone is not produced in the arc, but is generated over a distance by the action of arc-emitted UV radiation on atmospheric oxygen, ozone removal is difficult. Exposure to welding radiation-produced ozone represents an acute respiratory hazard. The effects of chronic exposure to low levels of ozone in humans are unknown, but, in experimental animals, emphysematous and fibrotic and other pathologic pulmonary changes have been observed (Ref. 29).

Cancer

Epidemiological studies of cancer risk have only incidentally included welders (Refs. 30 through 35). Examination of a mixed group of welders was a specific goal in only one study (Refs. 34 and 35). This was a prospective study of lung cancer in 14 different occupational groups. Other studies involving welders include: a survey by NIOSH (Ref. 30) to examine causes of death in Washington state males; a cohort mortality study in Dow Chemical Company employees including maintenance welders (Ref. 31); a study of lung cancer deaths and cases in Los Angeles County (Ref. 32); and a case-control study of lung cancer patients in 11 California hospitals (Ref. 33). In three of these studies, no correction was made for the confounding effect of smoking (Refs. 30 through 32).

The results of three of the studies (Refs. 30, 32, and 33) indicated that further study of lung cancer in welders was warranted; but, in two of these, neither the effects of smoking nor occupational exposure was examined, and the population of welders was very small in the third. In the most reliable study (Refs. 34 and 35), an increased risk of lung cancer was not observed. Thus, there is no substantial evidence to indicate that exposure to welding byproducts causes lung cancer; reports of well designed epidemiological studies are not yet available.

Significant evidence is not available from epidemiological studies to indicate whether welders are exposed to an increased cancer risk for organs other than lungs. No reliable study of the carcinogenicity of welding fumes and gases in experimental animals has yet been reported.

Eye

Another major concern is effects to the eyes. Injury may be caused by ultraviolet radiation or flying particles, and irritation may be produced by certain fumes and gases (Ref. 36), particularly ozone (Ref. 37). In one Swedish shipyard in 1976, 4,000 welders had 11,000 eye accidents (Ref. 38). In 1977, 7,000 eye injuries in 3,000 welders were reported in another Swedish shipyard, 30 percent of which were due to UV radiation (Ref. 39). Ross (Ref. 40) reported 459 non-lost-time injuries among 400 heavy engineering welders in 1971; 43 percent involved the eye, one third of which were classified as arc eye.

A potential hazard is welding arc radiation. Ultraviolet, visible, and infrared radiation are produced by the welding arc. Welders wearing no eye protection are at risk when the arc is accidentally struck. Assistants and supervisors not adequately protected are also at risk.

Arc eye (or keratoconjunctivitis) is an acute, self-limited irritation and inflammation of the superficial structures of the eye (Refs. 41 and 42), resulting from cumulative unprotected exposure to the UV radiation from the arc (Ref. 43). Cascini (Ref. 44) noted that in 20 cases exposure to the arc lasted 0.33 to 2 hours. The symptoms disappear 22 to 52 hours after the exposure, and no permanent injury appears to remain (Refs. 42 and 45).

Although the eye is not penetrated by the ultraviolet portion of the spectrum, inner structures are affected by wavelengths of 400 to 1400 nm. Radiation in this region of the spectrum can cause lenticular cataracts and chorioretinal burns (Refs. 41 and 46).

Historically, a high incidence of cataracts has been observed in glass blowers and foundrymen after many years of unprotected exposure to radiation emitted by molten glass and metal, respectively (Ref. 41). Massive doses of IR radiation were required to produce lenticular damage in rabbits (Jacobsen et al., 1963, as cited in Ref. 46). This contrasts with the infrequent exposure in modern welders, which, except in cases of gross negligence, is of short duration.

In 1967, Edbrooke and Edwards (Ref. 46) reviewed available evidence and concluded that welders did not develop cataracts more frequently than the general population; unfortunately, no well documented epidemiological study in welders has been published. Although quite rare, cases of retinal damage after unprotected exposure to arc radiation have been reported (Ref. 47).

The distant and near vision of welders does not appear to be significantly different from that of nonwelders, based upon examinations of heavy engineering (Ref. 148) and shipbuilding welders (Refs. 3 and 48) and control groups. However, as in other studies of the eye, these are cross-sectional rather than cohort studies.

Other Organs and Systems

Cardiovascular System

No unusual EKG findings were observed among a mixed group of 402 welders who had at least five years of welding experience and had not been exposed to other pulmonary occupational hazards (Ref. 49). Likewise, EKG abnormalities in 35 shipbuilding welders did not occur more frequently than in a control group (Ref. 3). However, Italian investigators claim that the ratio of the P wave duration to the length of the PR segment was significantly higher in 58 steel arc welders than in controls (Refs. 50 and 51). The EKG should be monitored in future studies.

The blood pressure measurements of welders do not differ from those of nonwelders (Refs. 48 and 52) nor do they have lower blood pressure (Refs. 3, 53, and 54). The latter is possibly due to the fact that welders may be more physically fit than persons in sedentary jobs.

The production of carbon monoxide from carbon dioxide used in gas shielded welding operations and by the oxyacetylene flame is noteworthy. Carbon monoxide avidly combines with hemoglobin to reduce the oxygen-carrying capacity of the blood. Exposure to 100 ppm for 4 hours shortened the time of onset of exercise pain in patients with ischemic heart disease and altered their EKG (Ref. 55). It has been shown to aggravate peripheral arterial disease (Ref. 56) and lowered the threshold for ventricular fibrillation in dogs and monkeys with experimentally-induced heart disease (Ref. 57).

Blood

In 1976, Ross and Hewitt (Ref. 52) found no significant differences in hemoglobin levels between 350 heavy engineering welders and a group of controls. In 1947, Dreessen (Ref. 3) noted no significant differences in hemoglobin levels between 3,000 shipbuilding welders (including both sexes and blacks and whites) and non-

welders. Likewise, in 1964, Marchand et al. (Ref. 49) reported normal hemograms in 402 welders with varied welding experiences, and Schuler et al. (Ref. 58) found no anemia in 23 welders from Santiago, Chile. Thus, under the conditions of the above studies, welding does not appear to affect hemoglobin levels.

Although leukocytosis is reported to be a common finding in metal fume fever (Ref. 59), Chmielewski et al. (Ref. 60) reported normal white blood cell counts in 20 welders before and after they worked on galvanized metal in confined spaces. Also, the leukocyte counts were not different from those of controls among heavy engineering welders (Ross and Hewitt, Ref. 52). Data on other blood parameters are unavailable.

Nervous System

Potential exposure to manganese occurs whenever this metal is used in electrode coatings and cores or in electrode wire. Potential lead exposure occurs during welding and cutting of any metal coated with lead-based paint. Both are poisonous to the nervous system. Information on effects to the nervous system is unavailable, except for those effects attributable to manganese or lead.

Gastrointestinal Tract

Symptoms of metal fume fever include diarrhea, nausea, and abdominal pain (Ref. 60). Chromate, which may be generated in stainless steel welding fumes, is an irritant to mucosal tissue (Ref. 61). Although Dreessen et al. (Ref. 3) noted no increase in gastrointestinal complaints in shipyard welders compared to controls, Rozera (Ref. 62), in a study of 620 Italian metallurgical and metal machining welders, found increased morbidity from ulcerative and other digestive system diseases in them compared to other workers in the industry. Significant corroborating data are lacking.

Other Organs

Except for a few cases of cadmium poisoning, information on effects to the kidneys is unavailable. No reliable reports of adverse effects on liver function were located. One report on a small group of 25 foundry welders revealed no clinical evidence of endocrine dysfunction or significant differences in plasma cortisol, urinary corticosteroids, plasma and urine epinephrine or norepinephrine, compared to 10 controls (Ref. 63).

Welders may be exposed to fluoride dust and vapors from certain FCAW and GMAW operations and SAW fluxes. Chronic fluorosis is a syndrome characterized by increased density of bones and ligaments due to fluoride deposition. However, no data are available that identify a relationship between exposure to fluoride-containing fumes and disorders of bones or ligaments or both (Ref. 64).

Recommendations

Available data are not adequate to reach many conclusions regarding health effects, if any, of the welding experience. Recommendations to fill in the most important information gaps are listed as items 1 through 4 below. In addition, potential health considerations should become an integral part of the development of new welding products; items 5 and 6 are suggested to aid in studying health hazard potential during the development stage.

1. *Prospective epidemiological study of chronic lung disease.* All of the studies reported to date are of the “cross-sectional” type and are subject to bias from lost workers (Ref. 65). The study should start with a cohort of welders, all employed at the start of the study in the same or similar welding environments; a control group of the same size should be included. Previous occupational exposures, smoking habits, and medical histories should be obtained. The groups should then be followed for a defined period with periodic observation, using symptom questionnaires, chest x-rays, and simple pulmonary function tests. All illnesses and hospitalizations should be carefully documented. All medical examinations and analyses should be performed without knowledge of occupation. Careful environmental monitoring should be pursued so that specific cause-and-effect relationships, if any, can be elucidated (Ref. 18). Increased information can be gained by combining the subject group with an historical cohort, starting 3 to 5 years in the past.

This study would also provide an excellent opportunity to monitor EKG, clinical chemistry, and blood metal levels. The size of the welder and reference groups to be chosen, as well as the duration of the study, depend upon the particular welding environment being examined.

Two milieus should be examined. First, a study of SMA or FCA welding of ferrous alloys is warranted. Such welding is commercially important, and, therefore, large numbers of individuals are potentially exposed. Results of earlier cross-sectional studies indicated that a large population and a long study period (5 years or more) may be required to pick up any effects when good industrial hygiene practices are observed (Ref. 18).

The second group recommended for prospective monitoring are welders using gas metal arc processes where ozone generation is significant. If the atmosphere in this type of welding environment represents a significant health risk, it may be detectable with a smaller sample.

2. *Epidemiologic studies of carcinogenic potential.* No reliable studies are available that assess the carcinogenic potential of any particular welding situation. Investigation of carcinogenicity will require selection of a cohort of welders and nonwelding controls, in existence far enough in the past that any cancer would have had enough time to develop. A reconstruction of past industrial hygiene measures at the plant under investigation and retrospective collection of relevant personal and occupational information on the subjects (or next of kin) should be undertaken. The incidence and mortality patterns in the exposed and control groups must then be ascertained. Death and morbidity data on other chronic diseases will also be concomitantly obtained.

An increased risk of respiratory tract cancer has been associated with exposure to certain chromates (Ref. 66) and nickel compounds (Ref. 67). Therefore, welders of stainless steel and other chromium and nickel alloys should be studied. The National Cancer Institute is

currently sponsoring a study of welders of stainless steel (Ref. 68).

Shielded metal arc or flux cored arc welding of ferrous materials or both account for a major portion of today's welding operations. Therefore, effects of exposure to this milieu in the absence of chromium and nickel (for example, in mild steel welding) should be considered if stainless steel welding is found to be associated with an increased carcinogenic risk.

Results from an epidemiologic study of this design can provide information only on the carcinogenic risk under industrial hygiene conditions and processes that existed 15 to 30 years ago. Therefore, any cancer epidemiology data should be supplemented with information on current exposure conditions (see item 6).

3. *Prospective study of eye injuries.* Although neither heat cataracts nor retinal injury have been reported to be a problem in welders, published supporting data are poor. Cohorts of current welders should be chosen, and they should undergo periodic eye examination for a length of time sufficient to detect cataract development. If possible, workers who seek other employment should also be followed, since attrition may be due to eye problems. This study might be undertaken independently or as part of item 1.

4. *Design of employee records forms.* Model forms should be developed to facilitate collecting and centralizing all job description, environmental monitoring, personnel, and other information necessary to monitor the health of welder populations. These should be made available to all firms employing large numbers of welders.

5. *Screening of fume for fibrogenic potential.* Fume from SMAW and FCAW is a complex mixture composed of unoxidized and oxidized metal from the electrode and material originating from the coating and core, respectively. Although significant evidence of restrictive lung disease was not uncovered in the limited cross-sectional studies discussed above, experiments in animals indicate possible fibrogenic potential.

Silicates constitute a significant portion of many coatings and cores, releasing silica or silicates or both into the fumes when these electrodes are burned. Another source of silica is in the oxidation of alloying elemental silicon. Crystalline forms of silica, which are known to produce pulmonary fibrosis (Ref. 69), have not been found in welding fumes (Refs. 70 and 71). However, even amorphous silica (Refs. 72 through 75, and 305), such as that formed by evaporation from liquid silica (Ref. 72), or amorphous hydrated silica

fibrils obtained by acid treatment of chrysotile (Ref. 73) may have fibrogenic potential. Therefore, consideration should be given to fibrogenic potential when developing new electrodes.

Ideally, electrodes should be screened for the fibrogenic potential of their fumes, using a rapid and inexpensive assay. An *in vitro* assay meeting these requirements is the alveolar macrophage test (Refs. 73 and 74). This assay is in the developmental stage and should be first examined for its utility in screening fume condensate.

If the test is validated, electrodes with standard or "typical" coating and core compositions should be developed for such a program, as it is important to be able to correlate biological effects with coating or core compositions. Results on "typical" electrodes can then be made available to aid manufacturers in coating and core formulation.

Based on results of the screening assay, highly suspect fume condensates can be further tested using appropriate experiment animal models. Experimental designs using intraperitoneal (Ref. 73) or intratracheal administration are relatively economical. Chronic inhalation experiments, which more closely mimic the type of exposure potentially received by welders, can be very expensive.

6. *Mutagenicity and in vitro carcinogenicity screening of fume condensate.* A number of tests are available to screen for mutagenic and carcinogenic potential (Ref. 76). The most well known of these is the Ames test of mutagenicity in *Salmonella*. This test has gained some acceptance because of the good correlation between results in it and *in vivo* carcinogenicity (Ref. 77). However, most results are on organic compounds. Therefore, any test proposed must be validated for solid inorganic compounds.

In the Ames test, samples of stainless steel welding fumes have been found to be mutagenic, whereas fumes from mild steel welding have not (Ref. 78). Based on these results, a study on stainless steel fume in rats has been initiated (Ref. 78). Therefore, process changes that minimize mutagenic potential should be examined for welding stainless steel. After each change, fumes should be collected and bioassayed. Other chromium- and nickel-containing fumes should be examined.

Other *in vitro* assays that measure mutagenic and carcinogenic potential (such as sister chromatid exchange (SCE) or *in vitro* transformation) should also be considered (Ref. 76). Positive screening results in any individual assay or battery of assays should be verified in appropriate animal studies.

Chapter 1

The Exposure

Details of the physical environment created by the welding process are presented in Appendix A. They are summarized here to provide the reader with the necessary background to evaluate information presented in Chapters 2, 3, and 4.

Welders are potentially exposed to the particulates, gases, radiation, and noise created as byproducts of most of the welding processes currently in use. The hazard potential of this exposure will depend primarily upon the composition and quantity of the fumes and gases generated and the spectrum and intensity of radiation emitted, which in turn depends upon the welding process employed and the metal being welded. It also depends upon the length of time spent in this type of environment and the effectiveness of control measures, such as ventilation and personal protective equipment.

Over 80 different welding processes in commercial use have been identified by the American Welding Society (Ref. 79). The health literature concerns exposure to some of the most widely used processes, including shielded metal arc welding (SMAW), also called covered electrode welding; flux cored arc welding (FCAW); gas metal arc welding (GMAW); gas tungsten arc welding (GTAW); and oxygas welding. All of these produce fumes, gases, radiation, and noise to varying degrees.

Fumes

In arc welding, fumes arise principally from the filler metal of consumable electrodes and any covering or core materials they may contain. The composition of the filler is usually similar to that of the metal being welded. Vaporization, then incomplete reaction with atmospheric oxygen, followed by rapid condensation are the main steps in their formation. The base metal weld pool is much cooler than the electrode tip, so the base metal is a significant contributor to total fumes only when it contains a volatile protective coating or volatile alloying elements. The total fume generation rate in any welding process is affected by the welding current, arc voltage, electrode polarity, electrode diameter, speed of welding, and welding practices.

In the shielded metal arc process, the consumable electrode is coated with materials that decompose to provide a protective gas shielding to the molten weld metal and fluxing. Therefore, in addition to the electrode, metal fume components arise from coating materials, which often include cellulose, calcium and magnesium carbonates, fluorspar (CaF_2), rutile (TiO_2), silicate minerals (feldspar, mica, asbestos), clay, sodium and potassium silicates, iron, silicon, and manganese. Fume composition varies widely depending upon the

coating compositions, which may be divided into four classes: acidic, rutile, neutral, and basic (containing CaF_2). Tables A3 and A4 in Appendix A present the major components formed when steel is welded with electrodes having each of these types of coating.

This process is widely used for welding carbon and low alloy steels in structural applications. Table A5 lists the concentration of selected elements in fumes from using electrodes for steels and nickel alloys.

Because of the added burden of the coating materials, a relatively high fume generation rate is produced with this process. The rate increases with increasing current.

In the flux cored arc process, a continuous filler wire electrode with a core containing fluxing, deoxidizing, and slagging agents is used. Some electrodes contain additional materials that decompose to provide a shielding gas, while the gas is externally supplied in others. Many of the core materials are the same as those used in the coatings of shielded metal arc welding electrodes. Table A9 presents the percentages of several elements in the fumes from several common flux cored arc electrodes. Due to the added contribution from core material and the generally high level of arc current used, this process generates fumes at a high rate.

In gas metal arc welding, fumes originate from the continuous bare wire electrode (with minor contribution from base metal) only. No core or coating is present. Thus, the composition of the fumes should be much more easily predicted. Shielding is provided by an externally supplied inert gas (usually argon), argon with a small quantity of oxygen or carbon dioxide, carbon dioxide, or helium. The composition of fumes produced when welding is done with various currently available electrodes is presented in Table A12. When carbon dioxide is used as a shielding gas, a higher rate of fume generation occurs than when argon or argon-5 percent oxygen (Refs. 80 and 81) is used. The rate also increases with increasing arc current.

The gas tungsten arc process is adapted to welding a wide range of materials: carbon, low and high alloy steels, aluminum and its alloys, magnesium and its alloys, copper and copper-nickel alloys, brasses, silver, and others. Gas tungsten arc welding is similar to gas metal arc welding except that a nonconsumable (tungsten) electrode is used. An external filler wire (not in the electric circuit) may or may not be used. When filler is used, it is relatively cool compared to the arc temperature, yielding generally a low rate of fume generation. When no filler is used (and fusion is produced by melting together the two base metals) the rate is insignificant (Ref. 81). The plasma arc welding process is similar to the gas tungsten arc process, and for this reason the fume generation rates are generally similar.

The submerged arc process is used to weld relatively thick plates at high metal deposition rates, commonly plain carbon and low alloy steels (Ref. 82). In use the arc is not visible; it is submerged under a flux. The end of the electrode and the molten weld metal are surrounded and shielded by an envelope of molten flux,

upon which rests a layer of unmelted flux. Since the arc and the weld metal are protected by flux, the fume generation rate is minimal when compared to those of shielded metal, gas metal, and flux cored arc welding. The major source of fumes is fluorides in the flux (Ref. 82).

Significant levels of fumes may be generated by the oxygas process in welding galvanized steel or an alloy containing a volatile metal (Ref. 82). Otherwise, the rate of fume production is relatively low, since the temperatures produced by the oxygas flame are much lower than arc temperatures (Ref. 82).

Table A16 and Figure A1 in Appendix A emphasize the relative fume producing capabilities of various welding processes.

In addition to fume level, duration of exposure to these fumes, location of welding, use of personal protective equipment, and engineering controls, the effects of welding fumes on the health of welders will also depend upon the composition and particle size distribution of the fumes. Some fume constituents may pose more of a potential hazard than others, depending upon their inherent toxicity. Of special concern are silica or silicates or both, fluorides, copper, chromium, nickel, and manganese.

The principal source of silica or silicates or both is the coating of shielded metal arc electrodes and the core of flux cored arc electrodes, present in the form of silica, ferrosilicate, kaolin, feldspar, mica, talc, and water-glass (sodium silicate). Some low alloy steels and aluminum alloys also contain elemental silicon. The crystalline forms of this compound are responsible for producing silicosis. However, attempts to locate crystalline phases of silica in welding fumes have been unsuccessful (Refs. 70 and 71).

The major source of fluorides in welding fumes is also the covering on shielded metal arc electrodes and the core of flux cored arc electrodes. Basic (low hydrogen) covered electrodes and self-shielded flux cored electrodes contain large amounts of fluorspar (calcium fluoride). The flux used in submerged arc welding is another source. The fluoride compounds present in the fumes from basic covered electrodes have been found to constitute 5 to 30 percent (as fluorine) of the total fumes. These are present mainly as calcium, sodium, and potassium fluorides.

The highest concentrations of copper are found when copper and its alloys are welded. Another minor source is from copper-coated gas metal arc electrodes.

Chromium arises in the fumes when stainless and high alloy steels are welded. The hexavalent, trivalent, and zero valent oxidation states of this element are present in the fumes. Some compounds of hexavalent chromium have been identified as carcinogens and mutagens (Refs. 66 and 83). Stern found that the total concentration of chromium in the fumes from welding mild steel (unalloyed) was less than 0.05 percent in the shielded metal arc process and 0.005 percent in the gas metal arc process. Shielded metal arc welding of stainless

steel (15 to 25 percent Cr) with basic or rutile type covered electrodes yielded a fume content of 2.4 to 6.4 percent chromium, while it ranged from 9.8 to 13.8 percent chromium with the gas metal arc process. Of the chromium in the stainless steel welding fumes, Stern (Ref. 78) found 5 to 33 percent to be in the zero or trivalent state (all of which was insoluble in water), and 67 to 95 percent to be in the hexavalent oxidation state (of which 0 to 13 percent was insoluble in water), when the shielded metal arc process was used. In gas metal arc welding of stainless steel using argon or an argon-oxygen mixture, 98 to 99.86 percent of the chromium in the fume was zero or trivalent (all of which was water insoluble), and 0.14 to 2 percent was hexavalent (of which 60 to 90 percent was insoluble in water) (Ref. 78). The relative amount of hexavalent chromium in the fumes from the gas metal arc process depended upon the amount of oxygen in the shielding gas (Ref. 78). In contrast, Virtamo and Tuomola (Ref. 84) found that in using basic covered electrodes to weld stainless steel (18 percent Cr), 1.8 to 3.1 percent of the fumes was chromium, of which 0.57 to 2.2 percent was hexavalent. Fumes from a rutile type covered electrode were 3.6 percent chromium, of which 2.5 percent was hexavalent. The gas metal arc process produced fumes of 0.40 to 0.96 percent chromium, 0.2 to 0.32 percent being hexavalent (Ref. 84).

Nickel is produced in fumes produced in welding stainless steel (typically 8 to 15 percent Ni) and nickel alloys.

Manganese is used in the coating of some shielded metal arc electrodes and in the core of flux cored arc electrodes. In a recent study of Pattee et al. (Ref. 70), manganese accounted for 0.3 to 8.8 percent and 1.0 to 13.5 percent of the total fumes produced by these two processes, respectively. Also, special steels with a high manganese content are another source of manganese oxides in welding fumes (Ref. 80).

Although fumes generally arise from the electrodes and the base metals, other sources may contribute a considerable fraction to the fumes in special cases. Zinc from galvanized steel and pigments and decomposition products from welding on painted surfaces are of special importance.

The particle size distribution is an important factor in determining the hazard potential of welding fumes, since it is an indication of the depth to which particles may penetrate into the respiratory system and the percentage of particles that will be retained therein. Particles in the range of 1 to 7 μm in diameter represent the most serious hazard due to penetration into the alveolar region (Ref. 85). Welding fume particles are essentially all less than 1.0 μm in diameter (Refs. 70, 86, and 87).

Hedenstedt et al. (Ref. 88) found that about 90 percent of the fume particles generated during the welding of stainless steel were less than 1 μm in diameter. However, the particles produced by the shielded metal arc process were relatively larger than those formed during gas metal arc welding. Stern (Ref. 78) found that the

mass median diameter produced in shielded metal arc welding of stainless steel was approximately 0.3 to 0.5 μm , whereas with the gas metal arc process it was approximately 0.25 μm . By extrapolating from a least squares plot, Heile and Hill (Ref. 81) estimated the mean particle diameters to be 0.03 μm and 0.12 μm from welding steel by the gas metal arc and flux cored arc processes, respectively. During gas metal arc welding with carbon dioxide, the mean particle size was greater than when argon was used. Although most welding fume particles are less than 1 μm in diameter when formed, results of one study (Ref. 85) indicate that they may agglomerate and increase in size with time.

Gases

Ozone, nitrogen oxides, carbon monoxide, and carbon dioxide are the principal gases produced during arc and oxygas welding. Phosgene, hydrogen chloride, and diacetyl chloride are produced incidentally from the photochemical (welding radiation) oxidation of chlorinated hydrocarbon solvents that may be present in the atmosphere.

Ozone is produced from atmospheric oxygen in a photochemical reaction by radiation of a wavelength shorter than 210 nm emitted from the welding arc. At wavelengths shorter than 175 nm, the process is so effective that virtually none of this radiation penetrates further than a few centimeters beyond the arc; the effectiveness decreases with increasing wavelength (Ref. 89). At wavelengths in the range of 220 to 290 nm, ozone absorbs ultraviolet energy and decomposes back to diatomic oxygen (Ref. 28).

The rate of formation of ozone depends upon the wavelengths and the intensity of ultraviolet radiation generated in the arc, which in turn is affected by the material being welded, the type of electrode being used, shielding gas (if it is used), and the welding variables (such as voltage, current, and arc length) (Ref. 90).

Gas shielded arc welding processes present a much greater problem from ozone production than the shielded metal arc (Refs. 28, 91, and 92) or flux cored arc (Ref. 28) processes. During the welding of aluminum and aluminum-magnesium alloys, Vorontsova (Ref. 91) found no detectable amounts of ozone when the shielded metal arc process was used; when argon was used as the shielding gas, the gas metal arc process produced ozone levels approximately four times higher than did gas tungsten arc welding. Sampling 6 inches from various arcs, Lunau (Ref. 28) found low levels (0.12 to 0.24 ppm) when mild steel was welded with either the shielded metal arc or flux cored arc process. Higher levels (0.27 to 2.1 ppm) were found during gas tungsten arc welding of mild steel. The highest (2.3 to 14.5 ppm) were attained during argon shielded metal arc welding of aluminum or aluminum-based alloys (Ref. 28). One possible explanation for the higher production of ozone during gas metal arc welding, relative to the shielded metal arc or flux cored arc processes, is the higher level

of fumes produced by the latter, which tends to block the emission of ultraviolet radiation.

The formation of ozone during submerged arc welding should be negligible, since there is no visible evidence of an arc. During oxygas welding, the flame is not hot enough to emit radiation of sufficient energy to generate ozone.

Changes in shielding gases and metals produce their effect on ozone generation rate by causing arc spectral changes. Those that increase the intensity of radiation at wavelengths shorter than 210 nm will increase the ozone generation rate. It appears that the highest levels of ozone are generated by the use of argon shielding in gas metal arc welding of aluminum (Refs. 28 and 89). Frant (Ref. 89) claimed that ozone was generated at 15 to 20 times higher levels when argon was substituted for helium in the welding of aluminum or copper. Similarly, less ozone is generated when carbon dioxide, rather than argon, is used (Ref. 89).

Alloying elements can play an important role in the ultraviolet light spectrum and, consequently, in ozone production. Lunau found that in welding aluminum the addition of 5 percent magnesium to the aluminum partially suppresses ozone generation, whereas the addition of 5 percent silicon enhances it.

Oxides of nitrogen are formed by the direct oxidation of atmospheric nitrogen at the high temperatures produced by the arc or flame (Refs. 93, 94, and 95). Table A21 in Appendix A presents the levels of nitrogen dioxide found during various types of welding operations; in only one case did the concentration in or around the welder's helmet appreciably exceed 2 ppm.

Carbon dioxide and carbon monoxide are formed by the decomposition of organic compounds in electrode coatings and cores, from inorganic carbonates in coatings, from carbon in weld metal, and in the oxyacetylene flame. Carbon monoxide is generated by the decomposition of carbon dioxide used in gas shielded arc welding processes.

Radiation

Electromagnetic radiation from the ultraviolet, visible,

and infrared portions of the spectrum is emitted by most arc welding processes. The arc generates line spectra characteristic of the materials involved, superimposed upon a continuum of radiation (Ref. 39).

Virtually no radiation of a wavelength shorter than around 175 nm should strike the welder; as previously stated, absorption of this by oxygen is so effective that the intensity should be reduced essentially to zero after it passes through a few centimeters of air (Refs. 28 and 89). Atmospheric oxygen will also absorb the 175 to 210 nm wavelength radiation.

The intensity of at least the radiation in the ultraviolet portion of the spectrum is also attenuated by the fumes produced. During shielded metal arc welding, UV irradiances at 4.8 m from the electrode were found to be 12 to 100 times higher when a high rate (not specified) blower was used to remove fumes than when only natural ventilation was available (Ref. 96). UV irradiance increases with increasing current, and Lyon et al. (Ref. 97) demonstrated that the increase in actinic ultraviolet (200 to 315 nm) irradiance was roughly proportional to the square of the current for gas tungsten arc, gas metal arc (CO₂), and flux cored arc (CO₂) welding of mild steel. Reflection from highly polished surfaces also increases exposure to radiation.

Noise

All of the commonly used welding processes produce noise. Levels for various processes measured with a type 1 meter by Rodman et al. (Ref. 98) are presented in Table A23. In addition to noise produced by the welding equipment itself, welders are often exposed to noise from other operations in their workplace.

Other Factors

Heat may present a problem when welding is done in confined spaces or when metal that has been preheated to improve welding characteristics is used. In addition to any direct effects, heat and exertional stress may also increase a welder's susceptibility to the effects of other harmful agents.

Chapter 2

Effects of Welding on Health

A major problem in evaluating many of the human toxicity studies has been the lack of information provided by the author(s) as to the nature of exposure: namely, the welding fume and gas concentrations in welder's breathing zones, the type of process and protection being used, length of time spent working in confined spaces, and other pertinent data. Another consideration is the way in which a particular group of welders was chosen for evaluation. A random sample is essential. If only healthy workers are examined, for example, the results would be different than if the study included welders presently on sick leave or unemployed. Because of the influence of cigarette smoking, alcohol use, drug use, diet, previous occupational exposure, and physical constitution, among other factors, upon the health of a population, the effects of welding exposures cannot be isolated and studied without considering these variables. For these reasons, there are not very many conclusive reports on the health of welders in the available literature. It is in this light that reports on the effects of welding on health are presented.

Respiratory Tract

The discussion is divided into sections dealing with acute and chronic lung conditions, although there is some overlap. Metal fume fever is considered in a sepa-

rate chapter, even though lung damage is presumed to play a role in this condition.

Acute Diseases Due Directly to Occupational Exposure

In examining acute pulmonary diseases in welders, it is always necessary to evaluate the adequacy of any respiratory protective equipment and ventilation in use. Acute pulmonary diseases in welders working in inadequately ventilated confined areas and exposed to toxic concentrations of mixtures of various welding fumes and gases are described in case reports dating from 1929 to the present. No specific intoxicant could be identified in some reports (Refs. 99, 100, and 101). The development of varying degrees of pulmonary edema, with the possibility of heart failure, acute pneumonia, bronchitis, or combinations thereof, is often observed, regardless of the toxic substance(s) involved.

The patient may become cyanotic, developing severe breathing difficulty and chest pain, prior to becoming unconscious. Death can occur in minutes or hours. If the patient recovers, an acute pneumonia is likely to persist for up to two weeks.

Inhalation of sufficient concentrations of nitrogen oxides (Ref. 41), ozone (Ref. 29), phosgene (Ref. 102), or fumes containing cadmium (Ref. 103) can cause acute pulmonary edema, acute pneumonia, and acute bronchitis. The more important hazardous agents are discussed individually.

Ozone

In certain welding situations, exposure to ozone is a serious potential health hazard. Since ozone is generated by the action of ultraviolet radiation on molecular oxygen, it can be present in substantial concentrations several feet from the arc. It is a special hazard in GMA and GTA welding. Argon shielded welding produces more ozone than helium shielded arcs (Ref. 104). Appendix A further discusses the formation of ozone.

Ozone is an irritant gas. At concentrations above 0.1 ppm, drying of the mucous membranes of the mouth, nose, and throat occur. At approximately 1 ppm, headache and general irritation are noted. Visual disturbances have resulted after exposure to ozone levels of 0.2 to 0.5 ppm for 3 hours. Respiratory tract irritation, pulmonary congestion, and edema may result from exposure to 1.5 to 2 ppm inhaled for periods of 1 hour or more. Deterioration of defensive mechanisms of the lung has been reported (Ref. 29). Exposure to 1 ppm of ozone for 10 minutes can precipitate asthma attacks. Respiratory irritation during mild exercise may occur with exposure to 0.5 ppm for 30 to 60 minutes. Both effects are reversible (Ref. 29).

Kleinfeld and Giel, 1956 (Ref. 105), studied severe acute ozone intoxication in welders. Pulmonary edema and hemorrhage, possibly fatal, or temporary pulmonary insufficiency in less severe exposures are characteristic. Secondary effects include irritation of the mucous membranes, headache, and lethargy. Low concentrations produce general depression or sleep in certain individuals.

Three features of ozone intoxication are noteworthy. The first is the delayed onset and severity of the condition. The second is the fact that symptoms such as marked dyspnea, chest pain, or cough are much worse than the clinical signs, which indicate only minimal pulmonary involvement. The third is that residual symptoms persist for months after exposure, even though the physical signs and chest x-ray films indicate the lungs have cleared (Ref. 105).

Moles and Collins, 1957 (Ref. 106), studied less severe effects of ozone exposure in GMA welders over a 3 month period. Ozone levels in the workroom air exceeded 0.1 ppm in all determinations. Eye and respiratory tract irritation complaints were voiced when the inert gas used was a mixture of 98 percent argon and 2 percent oxygen, but not when pure argon, helium, or carbon dioxide was used. Increased current density and gas flow rate increased the severity of these complaints. Other workers at various distances and directions from the arc also simultaneously experienced eye and respiratory tract complaints due to ozone formation by the ultraviolet radiation. Symptoms disappeared after termination of welding with the Ar-O₂ mixture, leaving no evidence of residual effects or disability. Use of respiratory protective equipment and ventilation (if any) was not specified.

A case of acute pulmonary edema in a 47-year-old welder developed 4.5 to 5 hours following his welding in a 5,000 gallon aluminum tank with inadequate

ventilation and no respirator. The metal had been preheated and was then welded by the argon tungsten arc process. Recovery was rapid; lung function values returned to normal within 11 days of the incident. Kurta, 1976 (Ref. 107), noted that the toxic agents inhaled were most likely a mixture of ozone and nitrogen oxides, although no measurements of these gases were reported.

Kleinfeld, 1970 (Ref. 6), reported the case of a 51-year-old welder who developed ozone pneumonitis after 240 minutes of exposure to gas shielded arc welding (GMAW) in a poorly ventilated area with an ozone level of 1.8 mg/m³ (0.9 ppm). NO₂ levels were stated to be minimal. The lungs cleared in 6 days.

Three other cases of ozone intoxication in male welders were reported by Kleinfeld et al., 1957 (Ref. 108). These men worked in a 200 x 90 x 10 ft room with the welding area in one corner. No supplementary ventilation was provided. One GMA and two GTA welding machines were used in this area by 8 workmen on two shifts. The work was done on nickel. Atmospheric ozone levels reached 9.2 ppm, but levels of nickel carbonyl and nitrogen oxides were stated to be negligible.

The first case, in a 51-year-old, was marked by a sudden severe headache, substernal pressure, and dyspnea, all of which occurred during GMA welding. Pulmonary edema developed. A chest x-ray film showed diffuse peribronchial infiltrates similar to acute pneumonia. After remaining in critical condition for two days due to persistent lung congestion, the welder slowly recovered over the course of two weeks, when chest x-ray films showed the lungs had cleared. Nine months later, he still experienced unusual fatigue and exertional dyspnea. The man had been a "conventional" arc welder for 10 years, but had switched to operating the GMA welding machine two weeks before the episode of pulmonary edema.

Another case occurred in a welder operating a GMA welding machine for three days; previously, he had 14 years of experience in arc welding. He noted burning of the eyes and throat for 3 days, a cough, a sensation of choking, and dyspnea. A chest x-ray showed small scattered infiltrations in both lung fields. He was hospitalized and treated for bronchopneumonia. Nine days later he was discharged with cleared chest x-ray films. For the next 9 months, he still experienced fatigue and frequent head colds, although clinical examinations revealed no abnormalities (Ref. 108).

Nitrogen Oxides

Overexposure to nitrogen oxides can cause pulmonary edema, acute pneumonia, and bronchitis and can lead to chronic lung diseases including emphysema and bronchiolitis fibrosa obliterans (Ref. 109). Nitric oxide (NO), nitrogen dioxide (NO₂), dinitrogen trioxide (N₂O₃), dinitrogen tetroxide (N₂O₄), and dinitrogen pentoxide (N₂O₅) generally occur together. N₂O₅ is unstable above 0° C but may occur in the presence of ozone. When inhaled, certain nitrogen oxides may react with water to yield nitrite and nitrate ions. Nitrosation by N₂O₄ (and possibly NO), changes in acidity with the formation

of nitric acid, oxidations by N_2O_3 and N_2O_4 , nitrite ion effects, and the reaction of NO with hemoglobin may be responsible for the toxicity (Ref. 110).

Exposure to nitrogen oxides can lead to violent dry coughing; high concentrations cause laryngospasm. Pulmonary edema and methemoglobinemia lead to cyanosis, with a possibility of death in a few minutes. Recovery usually follows hospitalization of the subject. The pulmonary edema may lead to a chemical pneumonitis, which then can induce a chronic inflammatory response leading to fibrotic changes. A predisposition to pneumonia during recovery has been suggested (Ref. 110).

Cadmium

Cadmium fume exposure is rare (Ref. 111). Hazard occurs whenever cadmium alloys are welded in an enclosed space (Ref. 112). Cadmium-containing or cadmium-plated metals, including some stainless steels, will evolve the oxide fumes when heated during welding processes. Cadmium fume overexposure represents an extreme health hazard and medical emergency (Ref. 113).

A number of case histories of cadmium overexposure in welders have been reported (Refs. 114 through 117). Intoxication may develop immediately or have a delayed onset. The clinical picture may at first resemble metal fume fever (Ref. 118), but is much more severe; it includes mild throat irritation and a foul taste in the mouth initially, followed in one to two hours by acute upper respiratory tract symptoms of cough, dyspnea, and pain, along with headache and chills. This progresses to severe pulmonary edema and possibly bronchopneumonia. Acute gastroenteritis may also occur. In cases of metal fume fever, in contrast, the symptoms usually subside in 12 hours or less (Ref. 114).

According to Christensen and Olson, 1957 (Ref. 115), the chest x-ray picture of acute cadmium intoxication resembles bronchopneumonia, but these findings are out of proportion to the clinical presentation. Whether or not permanent lung or kidney injuries result in nonfatal cases remains to be investigated, according to Beton et al., 1966 (Ref. 114). Christensen and Olson emphasized that cadmium fumes are not exceedingly irritating and do not have a pronounced odor. They estimated 2500 mg/m³ of cadmium fumes in air is lethal to man (Ref. 115).

General Acute Respiratory Diseases in Welders

A higher incidence of pneumonia or other acute respiratory symptoms in welders would be a possible indication of harmful effects of welding fumes and gases. In reviewing the London Decennial report for 1951, Doig and Challen found that deaths from all causes in welders were slightly higher than expected, 1196 compared to 1092 expected, yielding a standard mortality ratio of 110 (observed-to-expected mortality; if observed and expected are the same, the value would be 100). A substantial part of the excess was due to pneumonia; there were 70 deaths from pneumonia in welders against 31

expected. This increased pneumonia risk was not age-related, but seemed to be constant throughout the welders' working lives. It was unclear whether the acute pneumonias were caused by infectious microorganisms or by occupational overexposures to toxic substances in the welding environment (Ref. 94).

In other welders, Collen, in 1947 (Ref. 119), came to an opposite conclusion regarding pneumococcal pneumonia rates. No significant difference between 12,100 shipyard welders and all other shipyard workers exclusive of welders was found for either morbidity or mortality frequency due to pneumococcal pneumonia. The case fatality rates were 5.2 percent for welders and 5.8 percent for all other workers. The pneumonia in the welders, who were treated at the Permanente Foundation Hospital, were no different in severity, incidence of complications, or treatment days required than for all shipyard workers (Ref. 119).

In a cross-sectional study undertaken in Finland by Antti-Poika et al., 1977 (Ref. 7), 157 arc welders were compared to 108 male controls having similar smoking habits and socioeconomic status. Welders were exposed to fumes from basic electrodes used for mild unpainted steel for at least 3 years, at least 3 hours per day in shops; 25 welders had worked in enclosed spaces. Ventilation or respiratory protection in use was not specified. Previous exposures to dust in other occupations occurred in 59 welders (38 percent) and 17 controls (16 percent). Questionnaires on acute respiratory disease experience revealed the prevalences shown in Table 1. Only colds, hoarseness, and sore throats were significantly more prevalent in the welders than in the controls. The authors were able to conclude that serious acute disorders of the respiratory tract were no more common in male welders than in matched controls.

Siderosis and Mixed Dust Pneumoconiosis

Protracted inhalation of welding fume particles leads to accumulation in the lungs of otherwise healthy workers (Ref. 120). Deposits of the welding particles and dusts in the lungs can be noted on chest x-ray films as areas that are denser than the normal appearing lung fields. These densities may be small nodules, termed pinhead or micronodular opacities, or linear markings. In some chest x-ray films of welders, a combination of the two, that is, a reticulonodular pattern, is seen. Some authors refer to this as "snowstorm" lungs. This is not necessarily a clinical lung disease, although chest x-ray findings sometimes can be mistaken for asbestosis (Ref. 58). A number of cases in which biopsy or autopsy have been performed have been reported (Refs. 58 and 121 through 129).

The prevalence of chest x-ray evidence of mixed-dust pneumoconiosis in welders varies from none at all (Ref. 8) to percentages as high as 71 percent of welders examined (Ref. 130). The details of many epidemiologic investigations are presented below and are summarized in Table 7 at the end of this section. In several of the

Table 1
Prevalence of acute respiratory diseases
in welders in Finland

Condition	Welders			Controls			P
	Number questioned	Positive response number	%	Number questioned	Positive response number	%	
More than 2 colds per year	115	27	23	82	9	11	<0.05
Frequent attacks of nasal catarrh without cold	157	22	14	98	10	9	N.S.
Frequent attacks of hoarseness without cold	157	33	21	81	5	6	<0.01
Frequent attacks of sore throat without cold	157	68	43	81	22	27	<0.05
Pneumonia	156	14	9	106	8	8	N.S.
Acute bronchitis	157	21	13	106	10	9	N.S.

N.S. = Not statistically significant

Antti-Poika et al., 1977 (Ref. 7).

more recent studies, no abnormal roentgenological changes were observed, possibly indicating improved conditions. In some studies, neither the type of welding process nor the adequacy of industrial hygiene measures (if any were provided) were presented in detail by the authors.

In groups in which exposure to welding fumes has resulted in abnormal lung film changes, the frequency and severity of those changes have been shown to be related to the duration of occupational exposure. Dreessen et al., 1947 (Ref. 3), studied arc welders in steel ship construction at 7 shipyards in the United States. From a random sample that included over 3,000 welders (about 15 percent of the total welding workforce), 4563 x-ray films were evaluated. More men than women had exaggerated lung field markings: 36.1 per-

cent and 17.3 percent, respectively. Male welders and nonwelders were compared on the basis of age groups to determine the presence of abnormal chest x-ray markings. The results are shown in Table 2. Age was proportional to the prevalence of exaggerated lung field markings in both welders and nonwelders working in the shipyards.

Dreessen et al. also evaluated the association between the length of occupational exposure to welding and the occurrence of exaggerated lung field markings. They divided the sample into those men with and those without previous industrial exposures, obtaining the results shown in Table 3.

Table 2
Prevalence of abnormal chest x-ray films in
U.S. shipyard workers by age

Age (yr)	Male welders, %	Male nonwelders, %
15-24	2.6	1.4
25-34	8.4	5.3
35-44	9.9	6.3
>45	9.9	11.5

Dreessen et al., 1947 (Ref. 3).

Table 3
Prevalence of abnormal chest x-ray films in
U.S. shipyard welders by duration of exposure

Duration of welding exposure	Male welders with no previous industrial exposure, %	Male welders with previous industrial exposure, %
<10 months	5.3	5.9
10-19	5.4	7.6
20-29	6.2	10.6
>30	11.0	12.3

Dreessen et al., 1947 (Ref. 3).

Table 3 shows that the duration of exposure was associated with an increased prevalence of exaggerated lung field markings, and it is possible that prior exposure to other dusty industrial environments could have influenced the abnormal chest x-ray films as well. Further analysis of the chest films suggested welders' siderosis in 1.9 percent of the welders examined, but none in the nonwelders. Among white males, the prevalence was 3.4 percent; for nonwhite males it was 1.3 percent; and for white females the prevalence was zero.

A study by Britton and Walsh, 1940 (Ref. 131), reviewed chest x-ray films of 286 welders with 5 years or more (average 9 years) of exposure to arc or oxygas welding. Their ages ranged from 22 to 63 years. Most welders worked in well ventilated areas. There were 121 arc, 49 oxygas, and 66 "mixed" welders, all working in 14 plants involved in machinery manufacture. Mottled or stippled lung fields were revealed on chest x-ray films of 24 (8.4 percent) of the welders. Increased hilar or linear lung markings were found in 47 (16.4 percent). In 186 welders (65.0 percent), the chest x-rays were considered normal.

Garnuszewski and Dobrzynski, 1964 (Ref. 4), reported a chest x-ray survey of 307 shipyard welders. Abnormalities appeared in chest films of 192 (60 percent) of the welders and were interpreted as pulmonary siderosis coexisting with silicosis. In repeat chest films taken 1-1/2 years later, 31.7 percent of the cases showed a rapidly progressive course. The prevalence of this abnormality was directly proportional to the duration of welding exposure. All workers with more than 10 years'

experience were affected. Inadequate ventilation in ship compartments and dockyard shops was probably one factor influencing the x-ray abnormality.

Dobrzynski, 1973 (Ref. 5), reviewed chest x-ray films of 1027 manual arc welders during the period from 1961 to 1972, with respect to type of exposure (see Table 4). The duration of exposure to welding was associated with the frequency of appearance of abnormal chest films. Only 1 percent of group I welders (hull welders in confined space) with abnormal chest films had worked for less than 3 years, while over 60 percent of the cases occurred in welders with over 13 years of exposure. The chest x-ray picture was characterized by linear and micronodular shadows, which the authors classified as pneumoconiosis due to exposure to iron, silica, and other dusts in the hull welders' environment. Dust levels were not reported.

Of 210 arc welders from various industries in Santiago, Chile, 32 had clear-cut pulmonary siderosis, according to Schuler et al., 1962 (Ref. 58). The authors claimed that the welders were exposed solely to iron oxide fumes, without external silica contamination. However, electrodes were covered with carbonates and oxides or a fine mesh of asbestos. Ages of the welders ranged from 30 to 60; the mean was 45 years. The majority had been welding for at least 15 years, with a range of 5 to 25 years. Normal lung function test results and the absence of respiratory disease symptoms were noted in 84 percent of the welders, including those with the abnormal chest films.

Kierst et al., 1964 (Ref. 132), reported chest x-ray

Table 4
Prevalences of abnormal chest x-ray films in
manual arc welders according to working conditions

Group	No.	Working conditions	Abnormal chest x-ray	Mean age (years)		Mean exposure (years)	
				Welders with abnormal chest x-rays	Welders with normal chest x-rays	Welders with abnormal chest x-rays	Welders with normal chest x-rays
I	650	Hull welders in confined space	77 (12%)	38.6 ± 7	32.9 ± 9	14.0 ± 5	9.2 ± 6
II	46	Container welders outside ship-building industry	6 (13%)	41.5 ± 6	40.3 ± 8	13.4 ± 5	11.5 ± 9
III	156	Shipyard welders in open air	5 (3%)	35.5 ± 5	34.6 ± 8	11.0 ± 2	10.2 ± 5
IV	175	Welders in other industries	4 (2%)	37.7 ± 9	38.2 ± 9	11.7 ± 7	12.2 ± 6

Dobrzynski, 1973 (Ref. 5).

findings for a group of 171 welders employed in the Gdynia Shipyards in Poland, who were outpatients registered at an occupational disease medical facility. Exposure to fumes from rutile covered electrodes used in SMA welding was noted. No levels of fumes or other information were provided. The patients and hull fitters were evaluated for pulmonary disease and lung function in groupings based upon chest x-ray findings. The results are presented in Table 5. The ages of the welders ranged from 18 to 62 years; only 12 were over age 45. The length of welder employment was 1 to 32 years. Chest x-ray films were normal in 30 (19.3 percent, 2 to 16 years exposure). In 63 (36.9 percent, 2 to 32 years exposure), there were localized shadows considered to be on the borderline of normal. There were 64 welders (37.4 percent, 2 to 21 years exposure) with scattered nodular shadows, and 11 welders (6.4 percent, 6 to 16 years exposure) with "snowstorm" type shadows (diffuse micronodular pattern). Both the nodular and micronodular chest films were classified as mixed siderosis and silicosis. Among a control group of 43 hull fitters with 1 to 15 years of occupational exposure in the shipyards and aged 18 to 45 years, borderline chest x-ray findings were noted in 18 (41 percent); the rest had normal chest films. For both welders and controls, the percentage of abnormal chest films was proportional to the duration of occupational exposure.

The relatively mild clinical course of the disease was emphasized. The siderosilicosis caused little pulmonary fibrosis or interference with pulmonary circulation, even after long exposures to welding fumes, in contrast to true silicosis, which would have caused obvious disability.

Spacilova and Koval, 1975 (Ref. 129), compared chest films of arc welders working in confined areas to those of arc welders working in well-ventilated conditions in industries in Prague. The mean age, the mean duration of occupational exposure, and the number of welders for both groups are listed in Table 6. In 18 (78 percent) Group A workers and 12 (96 percent) in Group B, chest films revealed abnormalities. In 26 of the 30 films, alterations were slight; but, in 4 chest films of welders in Group A with 25 or more years of occupational exposure to welding, nodular opacities indicative of fibrosis were noted. The substance(s) causing the opacities was not identified. Eight of those from both groups had chronic bronchitis and reduced pulmonary function. Pulmonary function was normal in the remainder.

Gillon and Marchand, 1963 (Ref. 133), found 27 cases (5.1 percent) of reticular or nodular densities in both lung fields, particularly in the 7th and 8th intercostal spaces, without apical involvement or diaphragmatic or cardiac abnormalities, after examining chest x-ray films of 500 arc welders. All welders had more than 5 years of occupational exposure to SMA or GTA welding. Those with silica exposure or histories of lung disease were excluded from the study. Lung function tests (not specified) remained normal in the welders with abnormal chest films.

There appears to be no reliable correlation between the prevalence of pneumoconiosis and frequency of appearance of lung function deficit (Refs. 7, 8, and 134 through 136). Studies that have investigated both are presented in the following section.

Table 5
Clinical, functional, and radiological respiratory system abnormalities
in Polish shipyard welders

Chest x-ray picture	Number (%) of welders	Number (%) of controls	Clinical and functional findings in welders
Group I: no chest x-ray abnormality	30 (19%)	25 (59%)	None
Group II: slight localized shadowing	63 (37%)	18 (41%)	None
Group III: linear shadows and small nodular opacities in peripheral lung fields	64 (37.4%)	-	Chronic bronchitis, pulmonary emphysema, and effort dyspnea in 6 welders; decreased vital capacity in 19 welders
Group IV: "snowstorm appearance," diffuse micronodular opacities	11 (6.4%)	-	Chronic bronchitis and effort dyspnea in 11 welders; beginnings of cor pulmonale in 5 welders; decreased vital capacity in 5 welders

Kierst et al., 1964 (Ref. 132).

Table 6
X-ray abnormalities and working conditions
of welders in Prague

	No.	Abnormal x-ray	Age		Exposure (yrs)		Working conditions
			Mean age	Range	Mean duration	Range	
Group A	23	18	42	30-56	18	10-32	13% of yearly worktime in confined spaces; dust levels of 49- 145mg/m ³
Group B	14	12	46	31-64	16	3-34	Open-air welding; no dust; basic elec- trodes used

Spacilova and Koval, 1975 (Ref. 129).

Table 7
Prevalences of welders with chest x-ray pictures
characteristic of siderosis or mixed-dust pneumoconiosis

Ref.	Year	Workplace	Prevalence, %	Duration of work (years)
7	1977	Nonshipyard	0	36 (mean)
8	1973	Shipyard	0	-
5	1973	Ship's hull, confined space; container weld- ing (nonship- building); shipyard, open air; other industry	12 13 3 2	9.8 (mean) 11.7 10.2 12.2
10	1969	Manufacturing	32	-
9	1964	Shipbuilding	34	10-35
4	1964	Shipyard	60	-
133	1963		5.1	-
58	1962	Various	15	5-25
3	1947	Shipyard	Men 36.1 Women 17.3	-
130	1944	Manufacturing	71	>6
131	1940	Manufacturing	35	>5

Chronic Respiratory Conditions

The chest x-ray findings, presented in the previous section, do indicate evidence of exposure of the lungs to welding fumes. A number of cases (Refs. 134 through 138) of chronic dysfunctional respiratory conditions, including pulmonary fibrosis, emphysema, and chronic bronchitis, have been reported in welders; but the association between these conditions and welding fumes and gases must be elucidated through epidemiologic investigation. An understanding of these diseases and of the pulmonary function tests used in their determination is necessary to an understanding of the discussion that follows, and they are, therefore, discussed in detail in Appendix C.

The welders' risk of developing chronic lung or respiratory tract disease depends upon many interrelated factors: welding related factors such as the availability, adequacy, and use of ventilation, respirators, and related respiratory protective equipment; fume levels and composition; and concentration of gases in the welding environment, which will differ in the confines of small containers, enclosed and open-air welding in shipyards, small shops, and large, well-ventilated industrial workplaces. Other risk factors not related to welding include aging, lack of physical fitness, cigarette smoking, urban air pollution, hereditary predisposition, and prevalence of certain lung diseases in populations living in a given geographical location.

Epidemiologic studies may contain certain biases that can distort their conclusions. Sources of bias include: the selection of study population (especially, use of the cross-sectional survey, which does not consider absent, ill, dead, or otherwise missing welders); the design of interviews and questionnaires; and the selection of a control group for any comparisons that need to be made.

A NIOSH survey of death records of males over age 20 in Washington state during the years of 1950-1971 revealed a statistically significant excess of deaths in the 1376 welders and flame cutters from all diseases of the respiratory system, including cancer, chronic bronchitis, and pulmonary emphysema ($p < 0.05$) compared to all male deaths in Washington during these years (Ref. 30).

Ten cross-sectional epidemiological studies have been reported that can contribute to the assessment of chronic lung effects in welders. These are presented in detail below. The studies are summarized in Table 25 at the end of this section.

Hunnicut et al., 1964 (Ref. 9), compared the pulmonary functions of arc welders, under age 60 with 10 years or more of experience, to those of nonwelders, all employed at a shipbuilding plant with approximately 20,000 employees in 1962. After excluding those with significant chest and lung lesions, asthma, a past history of exposure to known irritating dusts or fumes, or with cardiovascular disease, 100 welders and 100 controls were randomly chosen. No data on welding processes or materials, ventilation, or fume and gas levels were presented. Reports of respiratory symptoms were tabulated according to smoking habits for both groups, as shown in Table 8.

Of the welders, 34 percent had x-ray evidence of pulmonary siderosis, having worked as welders an average of 18.3 years; the remainder had worked as welders for an average of 18.9 years. No other x-ray abnormalities were noted.

The prevalence of respiratory symptoms in welders who smoked cigarettes was twice that of nonsmoking welders. The controls reported respiratory symptoms less frequently than welders, regardless of smoking habits.

Pulmonary function tests revealed that 46 percent of the welders and 23 percent of the controls had impaired timed spirometers: they were unable to expel 70 percent of the maximal expiratory volume in one second (FEV_{1.0}). The welders also had significantly lower ($p = 0.01$) maximal expiratory flow rates and maximal midexpiratory flow volumes than did the controls. The respiratory impairment of the workers was indicative of obstructive lung disease in 37 percent of welders and in 10 percent of the controls, while restrictive lung disease was diagnosed, on the basis of pulmonary function testing, in 3 percent of welders and 11 percent of controls. Six percent of the welders and 2 percent of the controls had combined obstructive and restrictive lung disease.

When lung function was evaluated in terms of cigarette

Table 8
Prevalence of chronic respiratory symptoms
in shipyard welders

Symptom	Welders (N=100)	Controls (N=100)	Smoking welders (N=71)	Nonsmoking welders (N=29)	Smoking controls (N=59)	Nonsmoking controls (N=41)
Cough	25	11	21 (30%)	4 (14%)	10 (17%)	1 (2%)
Expectoration	18	8	14 (20%)	4 (14%)	7 (12%)	1 (2%)
Wheezing	13	2	12 (17%)	1 (3%)	2 (3%)	0 (0%)
Dyspnea	12	2	9 (13%)	3 (10%)	2 (3%)	0 (0%)

Hunnicut et al., 1964 (Ref. 9).

Table 9
Abnormal lung function prevalences in shipyard welders

		Obstructive lung dysfunction		Restrictive lung dysfunction		Combined lung dysfunction		Total	
	N	N	%	N	%	N	%	N	%
Welders:									
Smokers	71	29	41	2	3	5	7	36	51
Nonsmokers	29	8	28	1	3	1	3	10	34
Controls:									
Smokers	59	6	10	8	14	1	2	15	25
Nonsmokers	41	4	10	3	7	1	2	8	20

Hunnicut et al., 1964 (Ref. 9).

smoking habits, the data shown in Table 9 were reported.

Welders who smoked had a twofold increase in prevalence of abnormal pulmonary function tests when compared to controls who smoked. Smoking and welding were stated to have a "cumulative effect" on lung dysfunction. The dysfunction, however, was considered to be subclinical, since the welders experienced no apparent respiratory difficulties.

Table 10 summarizes data on welding, smoking, pulmonary function, and symptoms of respiratory disease for the shipbuilding welders and controls studied. Hunnicutt et al. concluded that symptoms of lung disease (cough, expectoration, dyspnea, and wheezing) followed the pulmonary function results among the several groups, being present twice as often among welders who smoked as among welders who did not smoke cigarettes.

Kleinfeld et al., 1969 (Ref. 10), and Kleinfeld, 1970 (Ref. 6), compared respiratory symptoms, pulmonary function, and chest x-ray abnormalities in welders with age- and residence-matched controls with no occupational exposure to dust. The 25 welders were also divided into those with less than or more than 20 years of welding exposure (12 and 13 welders, respectively). The results are presented in Table 11.

The welders were from a plant manufacturing sheet metal products from stainless steel and, presumably, mild steel. Iron oxide fumes inside face shields ranged from 0.65 to 47 mg/m³. Ozone concentrations were found to be negligible; 2.7 mg/m³ of fluoride fumes were found in an air sample collected outside the face shield of an FCA welder. No other information about welding processes, materials, or type of ventilation was provided.

There were no significant differences in pulmonary function test results either between welders and controls, or between welders exposed for less than or greater than 20 years. Although 32 percent of welders had chest x-ray evidence of pulmonary siderosis, which was more frequent in the welders with over 20 years of exposure,

there were no significant differences in the prevalence of cough, dyspnea, or wheezing between welders and controls. The number of cigarette smokers (at least one pack per day for 5 years or more) was similar in welders and controls. However, the small sample size should be noted.

Male welders were not at a greater risk of developing serious respiratory diseases than other males with similar smoking habits and socioeconomic status, according to Antti-Poika et al., 1977 (Ref. 7). The study included 157 currently employed arc welders who were exposed to total fume levels up to 9 mg/m³ measured outside the helmet, using basic covered electrodes to weld, mainly, mild unpainted steels.

The welders had at least 3 years' experience and welded at least 3 hours per day in shops; 25 welders had worked in confined spaces. Fifty-nine welders (38 percent) and 17 controls (16 percent) had previous exposures to dusty occupations other than welding. Various criteria by which the welders and controls were compared are presented in Table 12.

No x-ray evidence of pneumoconiosis or any other chest findings that could be related to welding were observed.

Pulmonary function in all welders currently employed was similar to those of the controls. However, the forced vital capacity (FVC) and one-second forced expiratory volume (FEV_{1.0}) became significantly lower with increasing age and duration of exposure to welding fumes and gases. It was remarked that no differences were noted in pulmonary function due to smoking habits or type of welding processes and materials to which welders were exposed (for example, stainless steel welding or metals coated with anticorrosion primers).

Although there was a significantly greater prevalence of simple chronic bronchitis in welders than in controls, persistent cough and breathlessness were more frequent complaints among the controls than among the welders. There were no differences in rates of simple chronic bronchitis due to age, smoking habits, duration of weld-

Table 10
Chronic respiratory symptoms with and without lung dysfunction by
smoking habit and occupation

Symptom	Welders								Unexposed controls							
	Smokers (N=71)				Nonsmokers (N=29)				Smokers (N=59)				Nonsmokers (N=41)			
	Normal lung function		Lung dys-function		Normal lung function		Lung dys-function		Normal lung function		Lung dys-function		Normal lung function		Lung dys-function	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
Cough	6	9	15	21	3	10	1	3	8	14	2	3	0	0	1	2
Expectoration	4	6	10	14	3	1	1	3	5	8	2	3	0	0	1	2
Dyspnea	4	6	3	7	2	7	1	3	1	2	1	2	0	0	0	0
Wheezing	2	3	10	14	1	3	0	0	1	2	1	2	0	0	0	0

Hunnicut et al., 1964 (Ref. 9).

ing exposure, or welding processes and materials used.

The mean values of pulmonary function tests were similar for all welders with or without chronic bronchitis (Ref. 7).

Dreessen et al., 1947 (Ref. 3), found an excess of upper respiratory tract abnormalities (such as nasal congestion, sinusitis, and pharyngitis) in their study of

over 3,000 welders and 1000 nonwelders employed in seven United States shipyards in steel ship construction. The excess of upper respiratory symptoms was especially noteworthy in male welders who smoked. In females, the difference between smokers and nonsmokers who welded was less pronounced than in the males. In a breakdown of data according to race and occupation,

Table 11
Chronic respiratory system clinical and functional variables in sheet metal welders

Variable	All welders	Controls	P	Welders with less than 20 years exposure	Welders with 20 years or more exposure	P
Number	25	20		12	13	N.S.
	Mean (range)	Mean (range)		Mean	Mean	
Age	48.8 (25-70)	46.7 (25-64)	N.S.	54.0	54.7	N.S.
Duration exposure (yrs.)	18.7 (3-32)	---	---	---	---	---
	N (%)	N (%)		N (%)	N (%)	
Smokers (1 pack per day for 5 yrs.)	14 (56%)	11 (55%)	N.S.	6 (50%)	8 (61%)	N.S.
Chronic cough	2 (8%)	2 (10%)	N.S.	---	---	---
Dyspnea	0 (0%)	0 (0%)	N.S.	---	---	---
Wheezing	3 (12.0%)	0 (0%)	N.S.	---	---	---
Chest x-ray, siderosis	8 (32.0%)	0 (0%)	<0.01	2 (16.7%)	6 (46.2%)	N.S.
	Mean $\pm$ s.e.	Mean $\pm$ s.e.		Mean $\pm$ s.e.	Mean $\pm$ s.e.	
FEV (% predicted)	87.0 $\pm$ 2.8	91.6 $\pm$ 3.4	N.S.	89.3 $\pm$ 2.7	84.8 $\pm$ 5.6	N.S.
FEV _{1.0} (% VC)	75.1 $\pm$ 2.1	72.9 $\pm$ 2.7	N.S.	77.3 $\pm$ 3.3	73.2 $\pm$ 2.9	N.S.
RV (% predicted)	86.4 $\pm$ 3.6	90.1 $\pm$ 5.7	N.S.	---	---	---
TLC (% predicted)	83.1 $\pm$ 1.9	88.0 $\pm$ 2.4	N.S.	---	---	---
RV/TLC (% predicted)	100.8 $\pm$ 8.4	99.6 $\pm$ 5.8	N.S.	---	---	---

N.S. = Not statistically significant.

Kleinfeld et al., 1969 (Ref. 10).

Table 12
Prevalences of chronic respiratory clinical and functional abnormalities in welders in Finland

Variable	Welders, % (N=157)	Controls, % (N=108)	P
3-4 yrs. exposure to welding	10	---	---
5-14 yrs. exposure to welding	51	---	---
15 or more yrs. exposure	39	---	---
Persistent cough	9	10	N.S.
Dyspnea	29	33	N.S.
Simple chronic bronchitis	24	14	<0.01
Mucopurulent chronic bronchitis	5	5	N.S.
	Mean $\pm$ S.D.	Mean $\pm$ S.D.	
FVC (liters)	4.95 $\pm$ 0.8	4.83 $\pm$ 0.8	N.S.
FEV _{1.0} (liters)	3.96 $\pm$ 0.7	3.94 $\pm$ 0.8	N.S.
100 x FEV/FVC	80.0 $\pm$ 6.8	81.4 $\pm$ 7.5	N.S.

N.S. = Not statistically significant

Antti-Poika et al., 1977 (Ref. 7).

Table 13 presents the percentages of workers with positive findings.

No permanent disability was reported in the welders, although the prevalence of a so-called upper respiratory symptom complex was considered by the authors to be statistically and clinically significant. The nasal congestion and pharyngitis were stated to have been caused by exposure to welding fumes and gases.

The prevalences of cough, chronic bronchitis, exertional dyspnea, wheezing, and respiratory system abnormalities in welders employed at the Galati shipyard in Romania were studied by Barhad et al., 1975 (Ref. 11). From a total of 700 welders, 153 males were chosen: all with at least 5 years of welding experience; no evidence

of major chronic chest wall, heart, or pulmonary diseases; and no previous exposures to substances considered to be respiratory hazards. The arc welders worked in large shops and confined spaces. Total dust in the welders' breathing zones was 6 to 36 mg/m³, but levels as high as 151 mg/m³ were found in confined spaces; nitrogen oxides ranged from 1.1 to 1.7 mg/m³, carbon monoxide from 6 to 17 mg/m³; and manganese fumes ranged between 0.6 and 3.3 mg/m³, depending upon process and materials. Shielded metal arc welding with basic covered electrodes, flux cored arc welding, and oxygas welding were utilized. A group of 100 controls was chosen from among maintenance workers and turners in areas free of welding fumes and gases. They

Table 13
Percent prevalences of chronic respiratory abnormalities in U.S. shipyard welders

	Welders				Nonwelders			
	Total	White males	Non-white males	White females	Total	White males	Non-white males	White females
Upper respiratory tract abnormality	40.5	47.7	37.9	30.3	30.9	36.4	32.6	22.4
Nasal congestion	18.6	22.2	18.2	13.5	14.2	17.2	15.9	9.3
Pharyngeal irritation	24.4	29.9	31.5	15.4	16.6	21.1	19.6	9.3
Upper respiratory tract symptoms	31.8	36.1	27.3	26.5	21.3	24.3	17.4	17.3

Dreessen et al., 1947 (Ref. 3).

Table 14
Prevalences of chronic respiratory signs and symptoms in
Romanian shipyard welders

Criteria	Welders (N=173)	Controls (N=100)	P
Cough	38 (22%)	14 (14%)	N.S.
Chronic bronchitis	34 (17.7%)	13 (13%)	N.S.
Dyspnea (during level walking)	34 (19.7%)	5 (5%)	<0.001
Wheezing, asthma attacks, or both	27 (15.6%)	7 (7%)	<0.05
Positive physical findings	24 (13.9%)	12 (12%)	N.S.

N.S. = Not statistically significant

Barhad et al., 1975 (Ref. 11).

suffered no chronic pulmonary diseases and were matched by age and smoking habits with the welders.

For the welders, the mean age was 34 years. There were 81 smokers (47 percent), 59 nonsmokers (34 percent), and 33 exsmokers (19 percent). They were given a questionnaire to determine the frequency of various respiratory system complaints, which were reported as in Table 14. There was a significantly higher prevalence of dyspnea and wheezing among the welders than among the controls. When the groups were separated by smoking habits, the results revealed most striking differences between exsmoking welders and controls, as shown in Table 15. In this group, the occurrence of symptoms might have been an inducement to stop smoking.

Chronic bronchitis occurred 1.5 times more frequently in welders than in controls. When smoking habits and age were considered, the analysis shown in Table 16 was provided.

The difference in prevalence of chronic bronchitis in the over 40 age group in the welders and the controls was close to the 5 percent significance level (borderline significance).

An evaluation of lung function, on the basis of smok-

ing, was accomplished for 125 welders. Results are given in Table 17.

Of the 125 measurements, 25 (20 percent) showed ventilatory impairment, defined as an FEV_{1.0} below 81 percent of predicted, or an FEV_{1.0}/VC ratio below the age-specific limits of normal, or both. The influence of smoking on lung function was regarded as minimal in the welders. Twenty-one of the 25 were of the restrictive type and 4 were considered obstructive impairments.

Respiratory disease symptoms and lung function in 156 welders were studied by Fogh et al., 1969 (Refs. 12 through 14). The welders, who ranged in age from 30 to 60 years, all had more than 5 years of welding experience in shipyards or as engine-, boiler- or tankmakers, and were exposed most often to fumes from the welding of mild steels. A control group consisted of 152 randomly selected employees of the same plants as the 156 welders, but who worked in areas where welding was not normally performed.

The prevalence of chronic bronchitis was similar in the welders and the controls. The data are presented in Table 18.

Smoking habits were identical for the welders and

Table 15
Prevalence of dyspnea during level walking in
Romanian shipyard welders

	Welders	Controls
Smokers	24%	6%
Ex-smokers	33%	6%
Nonsmokers	7%	3%

Barhad et al., 1975 (Ref. 11).

Table 16
Percent prevalence of chronic bronchitis
in Romanian shipyard welders

	Welders	Controls
Nonsmokers	12	3
Smokers	26	21
Over age 40	21.5	9.8

Barhad et al., 1975 (Ref. 11).

Table 17
Evaluation of lung function in Romanian
shipyard welders

	Nonsmokers	Ex-smokers	Smokers
Number	40	24	61
% of predicted forced vital capacity (FVC) (mean)	87.2%	85.4%	87.7%
% of predicted one second forced expiratory volume (FEV _{1.0}) (mean)	91.1%	90.8%	88.9%
100 x FEV _{1.0} /FVC (mean)	80.1%	80.5%	78.2%

Barhad et al., 1975 (Ref. 11).

the controls. Mean values for one second forced expiratory volume (FEV_{1.0}) were similar for both groups: 3.7 ± 0.68 liters for welders and 3.7 ± 0.77 liters for controls. Both means were 102 percent of predicted FEV_{1.0} levels. However, the welders showed increasing FEV_{1.0} impairment with increasing use of tobacco. The difference between welders and controls with identical smoking habits was not statistically significant. There were significant differences in FEV_{1.0} between nonsmoking welders and light smoking welders ($p < 0.05$) and between nonsmoking welders and heavy smoking welders ($p < 0.01$). These differences were not noted when nonsmoking controls were compared to light or heavy smoking controls.

Table 18
Prevalence of chronic bronchitis symptoms
in welders

Symptoms	Welders	Controls
Absent	104 (67%)	111 (73%)
Present	52 (33%)	41 (27%)
Total	156 (100%)	152 (100%)

Fogh et al., 1969 (Ref. 12).

Thirteen welders (8 percent) had obstructive pulmonary impairment while the impairment was questionable in 8. For the controls, 17 (11 percent) had impaired lung function, of which 12 were of the obstructive type, 5 were of the restrictive type, and 6 were questionable. There was no statistically significant difference between the welders and the controls in this regard.

Cough and sputum were proportionally related to tobacco use. There were no significant differences between smoking welders and smoking controls in relation to these symptoms. However, there were significant differences between nonsmoking and smoking welders ($p < 0.001$) and between nonsmoking and smoking controls ($p < 0.05$) in relation to the presence of cough and sputum. No increases in cough and sputum were noted for nonsmokers and exsmokers in relation to aging. Among both the welders and the controls who did not smoke, forced expiratory volumes were significantly higher than in the 2 corresponding groups of smokers.

In welders, but not in controls, respiratory symptoms increased with age: 25 percent of the welders under age 50 had symptoms, while 55 percent of the welders over 50 years old had symptoms. This difference was highly significant ($p < 0.001$). However, the groups studied were small, making the results difficult to evaluate, according to the authors (Ref. 12).

Peters et al., 1973 (Ref. 11), studied respiratory disease in welder and control populations. Sixty-one welders were selected from a shipyard where repair work was done. Exposures to fumes from coated and uncoated metals, low and high alloy steels, aluminum alloys, and bronze, from the use of SMAW, GTAW, and GMAW techniques were encountered in an intermittent "job shop" manner, in the shop and in confined (poorly ventilated) spaces on shipboard. A group of 61 pipecoverers was matched to the welders by age and duration of employment in the shipyard. Sixty-three pipefitters were selected as an unexposed control for the welders and the pipecoverers.

No siderosis was detected on x-ray films, and no other x-ray abnormalities that could be related to welding exposure were noted.

In all 3 groups, smoking habits were similar, as were

ages and heights. (Height is an important consideration in evaluating lung function studies.) Ten percent of welders and 9 percent of pipefitters had chronic cough. Three each of the welders and the pipefitters experienced shortness of breath. The results of pulmonary function tests were similar in the welders and the pipefitters: tests included forced vital capacity, one second forced expiratory volume, peak expiratory flow rate and volume, and total lung capacity and residual volume, among others.

In 36 smoking welders, the peak expiratory flow rate was significantly higher ($p < 0.05$) than in 25 current nonsmoking welders. The nonsmokers were further divided into those who never smoked ($N=10$) and ex-smokers ($N=15$). The 10 who never smoked were approximately 10 years younger than the exsmokers. No ventilatory defects were noted in those who never smoked. Forced vital capacity and one second forced expiratory volume were considerably below expected values in the exsmokers, being similar to the results for the smokers.

Peters et al. noted that welders, pipecoverers, and pipefitters all had similar exposures to asbestos in the shipyard, which complicates comparisons of these 3 groups in terms of respiratory disease criteria. Furthermore, both the pipefitters and the pipecoverers could be exposed to welding fumes. Exposure to ozone and nitrogen dioxide, known pulmonary irritants in welding fumes, would be expected to contribute to airway obstruction, and pulmonary function tests would be expected to reveal a pattern of increased total lung capacity and residual volume. On the other hand, exposure to asbestos and other fibrogenic dusts would be expected to cause a decrease in total lung capacity and residual volume, characteristic of restrictive pulmonary disease. An increase in residual volume was noted in welders; it was proportional to the duration of exposure. This could have been a result of ozone and nitrogen oxide exposure during welding, or a result of smoking, or aging, or both. Either result tends to increase residual lung volume. The total lung capacity of the welders was in the normal range. In the asbestos-exposed pipecoverers, a decrease in total lung capacity was noted, which was partly consistent with expectations. Residual volume did not decrease.

In order to better determine whether shipyard exposure to welding fumes and asbestos affected pulmonary function, the same authors compared their original 61 welders, 61 pipecoverers, and 63 pipefitters with a group of 94 pipefitters (controls) from another shipyard involved in new ship construction. These men had minimal or no exposure to welding fumes or asbestos. This time, statistically significant differences were noted between the original group and the new controls for forced vital capacity ($p < 0.005$), one second forced expiratory volume ($p < 0.005$), and peak expiratory flow rate ($p < 0.05$). These three pulmonary function test values were lowest in the welders.

A comparison of the same three pulmonary function

tests between the pipefitters and welders on the one hand and policemen who were matched by age and height on the other hand revealed considerably lower values in the welders on all three tests. The values for the policemen were not measured by the authors, but taken from recent literature. This was used as further evidence of the deleterious effect of shipyard welding exposure on the respiratory system. It should be noted that the degree of disability of the welders was subclinical, except for the presence of cough in 10 percent and shortness of breath in 3 percent, as described earlier (Ref. 8).

Ross, 1978 (Ref. 15), reported results of medical examinations of a group of 926 male manual metal arc welders in heavy engineering and shipbuilding occupations (no information on welding process, materials, or fume levels presented) and a group of 755 controls in other trades, excluding boilermakers, carried out over a 6-year period.

Past medical history revealed, both for welders who smoked pipes and cigarettes and for nonsmoking welders, a significant excess ($p < 0.05$) of pneumonia, bronchitis, and respiratory illnesses in older welders. No significant difference existed between the nonsmoking controls and welders in the amount of respiratory illness on the whole, or for pneumonia and bronchitis, with one exception: welders aged 50 to 59 had significantly more respiratory illness, pneumonia, and bronchitis than did the controls of that age group.

Productive morning cough was the only symptom that was significantly more prevalent in older smoking welders than in older nonsmoking welders (50 to 59 years old). Other symptoms, such as sore throat, catarrh, and dry cough, were equally prevalent in all welders regardless of age.

About 45 percent of the welders had been exposed to asbestos. No signs or symptoms of asbestosis were found, however.

Mean breathing test results for smokers and nonsmokers in each 10-year age group (20 to 60) indicated no significant differences between welders and controls for forced vital capacity, forced expiratory volume, or peak expiratory flow rate. Smoking welders in the 40 to 49 year age group had significantly greater ventilation restriction than nonsmokers of the same age group. Airway obstruction was noted in 24.4 percent of smoking welders and 20.3 percent of nonsmoking welders, on the whole.

Ross concluded that long term welding exposure caused a high incidence of respiratory illness in 50 to 59 year old welders who smoked. However, because many of the current welding processes are of relatively recent introduction, Ross felt it is probably too early to evaluate their adverse health effects, if any.

Wilhelmsen et al., 1977 (Ref. 16), investigated welders in the Gotaverken shipyards in Sweden, where most welding is performed indoors in an assembly hall or in small ship compartments. HT steel, containing 0.2 percent carbon, 0.1 to 0.5 percent silicon, 0.5 to 1.5 percent manganese, 0.5 percent phosphorus, and 0.05 percent

Table 19
Prevalence of chronic respiratory symptoms
in Swedish shipyard welders

	Welders, %		Clerks, %		50-yr.-old men, %	
	Non-smokers (N=75)	Smokers (N=44)	Non-smokers (N=53)	Smokers (N=37)	Non-smokers (N=157)	Smokers (N=182)
Morning cough for 3 months a year	19	19	0	15	6	36
Phlegm day or night for 3 months a year	16	30	6	17	1	12
Wheezing apart from colds	19	30	4	21	6	13
Walking uphill dyspnea	37	36	10	38	21	25

Wilhelmsen et al., 1977 (Ref. 16).

sulfur, 0.25 percent chromium, 0.30 percent nickel, 0.10 percent molybdenum, and 0.35 percent copper, with a 95 percent zinc and chromium primer containing polyvinylbutural, phenol-resins, and iron oxide red was used. Electrodes were the basic covered type, with high potassium and calcium content. The fume concentration was stated to be high, yet was simultaneously claimed never to exceed 1 mg/m³.

The mean age of the group of welders was 43.6 ± 9 years; the mean duration of exposure was 17.3 ± 8 years. For a control group of clerks, the mean age was 37.4 ± 5 years. A second unexposed control group, consisting of 339 50 year old men, was randomly selected from the local population.

The prevalence of cough, phlegm, wheezing, and effort dyspnea was ascertained in these three groups, according to smoking habits, giving the data in Table 19. Smoking welders complained of more symptoms than nonsmoking nonwelders. The difference was less marked when smoking welders and controls were compared.

The 119 welders were asked if welding, especially when high levels of smoke are present, caused cough, phlegm, wheezing, or dyspnea. The responses were as follows:

	Yes	No	Nonre- sponsive
Does welding in presence of much smoke cause cough?	73 (60%)	40 (39%)	6 (5%)
Does welding in presence of much smoke cause phlegm?	63 (52%)	50 (43%)	6 (5%)
Does welding in presence of much smoke cause wheezing?	36 (30%)	73 (12%)	10 (8%)
Does welding in presence of much smoke cause dyspnea?	48 (10%)	61 (82%)	10 (8%)

Table 20
Prevalence of chronic respiratory symptoms in
welders in Sweden

Symptom	Male welders (N=134)		Males in other occupations (N=7,321)	
	N	%	N	%
Chest pain walking uphill	22	16.9	1084	14.8
Intermittent claudication	17	13.5	930	12.7
Dyspnea walking uphill	34	26.8	1596	21.8
Sputum in morning	37	28.7	1706	23.3

Wilhelmsen et al., 1977 (Ref. 16).

Fifty percent or more of the welders thought there was an association between all symptoms and welding, with the exception of dyspnea.

Based on a cross-sectional examination of 7455 males in all occupations in Goteborg, representing a random population sample, Wilhelmsen claimed that welders experienced more respiratory symptoms than men in other occupations, but did not provide the results of statistical tests to support this claim (Table 20). The 134 welders in this sample worked in various industries and shipyards, under varying conditions (not specified). Over-reporting of symptoms is one bias that Wilhelmsen et al. ascribed to, influencing their data.

Pulmonary function tests were compared between the welders and clerks who were matched by age, height, and smoking status. There were no significant differences between the welders and the controls with respect to FEV_{1.0} (one second forced expiratory volume), VC (vital capacity), or flow-volume curves with flow measurements at different points in the vital capacity. However, there were significant differences in closing volume, closing capacity, and total lung capacity, as measured by N₂-dilution, between nonsmoking welders and matched nonsmoking clerks. The importance of these changes was not discussed.

Lung function was not found to deteriorate significantly among 23 nonsmoking welders who were tested on Monday before work (after a 2-day rest from welding), again on Monday after work, Thursday before, and Thursday after work (Ref. 16).

In a report by Ulrich et al., 1974 (Ref. 17), a clinical study was carried out in 80 shipyard welders,

58 of whom worked in semiconfined spaces (fume levels up to 340 mg/m³ in the breathing zone) and 22 in big halls (fume levels up to 192 mg/m³ in the breathing zone). There were 80 age-matched controls selected from the shipyard who were not exposed to welding fumes. The prevalence of respiratory symptoms and positive physical findings in these populations was tabulated as shown in Table 21.

Among the nonsmoking welders and controls, cough, expectoration, dyspnea, and nasal catarrh were evaluated as shown in Table 22.

Subjective breathing difficulties and irritation of nose and throat were more frequent in welders than in controls, although those working in confined spaces showed only slightly greater frequencies than those welding in assembly halls. Nonsmoking welders had a greater frequency of cough, expectoration, dyspnea, and nasal catarrh than did nonsmoking controls. Smoking welders had a higher frequency of dyspnea than did smoking controls. Furthermore, the effects of welding and smoking on dyspnea were additive, but the effects on cough and expectoration were not.

Results of lung function tests measuring vital capacity (VC) and one second forced expiratory volume (FEV_{1.0}) were similar for all welders and controls. However, significant decreases in FEV_{1.0} were noted both for welders in enclosed spaces in comparison to controls (p = 0.02), and for welders in confined areas in comparison to assembly hall welders (p = 0.05). Chronic bronchitis was considered to be the cause of the lowered FEV_{1.0} in the affected welders. Chest x-ray pneumoconiosis, present in 50 percent of the welders, influenced

Table 21
Prevalence of chronic respiratory tract signs and symptoms in shipyard welders

Symptom	Control (N=80)	Total welders (N=80)	Welders working in enclosed spaces (N=58)
Cough	29 (36%)	31 (38%)	24 (41%)
Expectoration	29 (35%)	30 (38%)	24 (41%)
Dyspnea	12 (15%)	25 (31%)	18 (31%)
Nasal catarrh	10 (13%)	13 (16%)	9 (15%)
Physical finding			
Lung wheezes	27 (34%)	29 (36%)	22 (38%)
Conjunctivitis	25 (31%)	52 (65%)	41 (71%)
Rhinitis pharyngitis	22 (28%)	52 (65%)	41 (71%)
Hypertension	5 (6.2%)	0	0

Ulrich et al., 1974 (Ref. 17).

Table 22
Prevalence of chronic respiratory symptoms in nonsmoking welders by exposure

Symptom	Control (N=25)	Total welders (N=32)	Welders not in confined spaces (N=12)	Welders in confined spaces (N=20)
Cough	3 (12%)	14 (44%)	5 (42%)	9 (45%)
Expectoration	3 (12%)	13 (41%)	5 (42%)	8 (40%)
Dyspnea	1 (4%)	6 (19%)	2 (17%)	4 (20%)
Nasal catarrh	2 (8%)	2 (6%)	1 (8%)	1 (5%)

Ulrich et al., 1973 (Ref. 17).

neither lung function nor respiratory symptoms (Ref. 17).

Two other studies are included for completeness. Pulmonary diseases in Chilean welders and nonwelders in the iron industry were analyzed by Benavides C., 1976 (Ref. 139). He studied the clinical findings of 69 arc welders and oxygas cutters and 69 age-, height- and weight-matched controls with similar clinical histories. The welders mainly used basic and acidic covered electrodes to weld mild steel, bronze, and other copper alloys. Work in semiconfined areas was performed by 93 percent of the arc welders. There were 28 smokers (at least 1 cigarette per day for 1 year), and 41 non-smokers and ex-smokers. An initial analysis of the welders and the controls gave the information in Table 23. No x-ray evidence of siderosis or pneumoconiosis was found in any of the welders.

Sixty-two welders were questioned about chronic

bronchitis symptoms: that is, dry or productive cough daily for at least three months per year for the previous 2 years. Chronic bronchitis occurred significantly more frequently in welders ($p < 0.05$), but neither age nor years of exposure could be correlated with frequency of chronic bronchitis.

Stanescu et al., 1967 (Ref. 140), carried out pulmonary function tests comparing 16 welders with x-ray evidence of siderosis to a group of 13 healthy unexposed men. These welders (mean age 40 ± 10 years) were randomly chosen from 70 welders employed at a metallurgical plant and were further selected on the basis of having over 7 years of arc welding experience. No data on welding process, materials, or fume levels were provided. The control group (mean age 36 ± 8 years) consisted of men not occupationally exposed to dust. The two groups were matched in height, age, and smoking habits.

Table 23
Respiratory function and prevalence of chronic respiratory disease in Chilean welders

Variable	Welders	Controls	P
Forced 1 sec. expiratory volume (FEV _{1.0})	3.3 liters (102.5% of predicted volume)	3.6 liters (107.6% of predicted volume)	
Forced vital capacity (FVC)	4.4 liters (109.4% of predicted volume)	4.7 liters (111.9% of predicted volume)	
100 x FEV _{1.0} /FVC	74.9	75.5	N.S.
Chronic bronchitis	22 (35%)	7.7%	<0.05

N.S. = Not statistically significant

Benavides C., 1976 (Ref. 139).