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On Welding, Wheezing, and Whimsy

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introduction

The biological effects of exposure to welding fumes have generated much' interest and considerable discussion over the past three to four decades. A plethora of papers has been published. many of which have waxed hyperbolic on the real and imaginary hazards of this occupation,[1-4] while a lesser number have indicated that welding, provided appropriate precautions are taken. is relatively harmless.[5-7] A strident minority seem willing to accept the viewpoint that whenever a welder develops a respiratory disease. exposure to welding fumes is responsible.[1-4] Over the course of the last 20 yr. both pulmonary fibrosis and emphysema. not to mention decreased libido. dementia. and motor neurone disease,[8] all have been attributed to the inhalation of welding fumes. A critical review of the biological hazards of welding is long overdue.

A multiplicity of processes is involved during welding. and the character and effects of the effluents emitted are influenced by the type 01 welding being performed. by the welding rods used. by the industrial hygiene practices that are in operation (these include the use of respiratory protective devices such as masks). by the personal habits of the welder himself. and. possibly. by genetic factors.''' In most West European and Sorth American countries. monitoring of welding fumes is the usual practice and is frequently mandatory.

Over the years welding has been evolving from a process that initially was relatively simple. and in which bare iron electrodes were used almost exclusively. into a complex technology in which numerous different electrodes are used.''.''' These now consist of a central core and various outer coats that are comprised of a variety of different agents. The effluent gases from the process contain a variety 01 fumes and gases. with some of the latter posing. under certain conditions. significant respiratory hazards. including death. The emission and type of fumes vary according to the kind of welding taking place. with manual metal arc and metal inert gas welding having the highest emissions and tungsten inert gas and submerged arc welding giving off the lowest emissions.[9,11] The volume of the fumes and gases that are emitted during the process depends on the type of electrode used and the shielding gas. Also important are the metallic constituents of the electrodes, surface coating, and any other contaminants. The generation of fumes also is influenced by the voltage used. by the welder's or cutter's attention to industrial hygiene. and by the adequacy of ventilation in the workplace. McMillan,[9] in a recent paper. showed that 30 different electrodes were being used in the Royal Navy Dockyards. The electrodes that were used contained 19 different ferrous and nonferrous alloys. with ferrous alloys making up around 90%. The principal elements concerned were iron. manganese. cobalt. chromium, nickel. silver, cadmium, zinc, and aluminium. He mentioned four main types of base metal surface coating, including metallic, bitumastic. various paints. and lubricants. Table I lists most of the agents encountered in welding fumes along with their TLVs®.

The likelihood of welding fumes reaching the lung parenchyma depends on the aerodynamic diameter of the fumes. with the smallest being most likely to reach the alveoli. Manual metal arc welding generates larger particles which tend to agglomerate.'''' When the fume concentration is high. as in manual arc welding, it has been suggested that more deposition occurs in the small airways and parenchyma of the lung. In contrast. stainless steel welders who use tungsten inert gas welding are exposed to lower fume concentrations and probably less pulmonary deposition. The inhalation of fumes. however. also depends on other factors, including the concentration of fumes in the breathing zone of the welder. the breathing pattern of the welder. and the ventilation present at the workplace.

TABLE I
Threshold Limit Values of Certain Agents Found in Welding Fumes

Agent	TLVQ
Iron oxide	5 mg/m³
Calcium oxide	2 mg/m³
Manganese	1 mg/m³
Cadmium	0.05 mg/m³
Zinc oxide	5 mg/m³
Fluorides	2.5 mgim'
Magnesium oxide	10 mgim"
Nickel	1 mg/m³
Chromium (metal)	0.5 mgim"
Lead	0.15 mg/m³
Mercury	0.01 mg/m³
Vanadium peroxide	0.05 mg/m³
Copper	0.2 mg/m³
Beryllium	0.002 mg/m³
NO_2	3 ppm
NO	2 ppm
O_3	0.1 ppm
Phosgene	0.1 ppm

Deposition of Welding Fumes and Their Retention In the Lungs

During life, many millions of particles are deposited in the lungs. For the most part, such particles have no significant effect on lung function.[11] Only when exposure has been excessive and prolonged is there a deleterious effect. In this regard, it is essential to distinguish between a physiological and adaptive response to the inhalation of moderate numbers of such particles and an injury or disease that results from the continued inhalation of many such particles. An adaptive response implies that any panicles deposited in the lungs or airways are removed or rendered innocuous by the normal defense mechanisms of the body. In contrast, a disease indicates that the particles have induced a response. namely an injury, and the tissue has responded in a pathological rather than physiological manner.

Toxicity of Fumes and Gases Emitted during Welding

It is suggested that a number of criteria should be fulfilled prior to incriminating a particular substance as hazardous or toxic. In this connection it must be remembered that all substances, including water and oxygen. are toxic under certain circumstances and that the dose and the duration of exposure are of paramount importance.

If a particular agent is to be designated as toxic, the following criteria should be fulfilled.

(1) The disease or effects induced are specific and can be recognized and diagnosed with a reasonable degree of certainty.

(2) Although the clinical presentation of the specific disease induced by the toxin may vary to some extent. there are certain symptoms. physical signs. and laboratory tests that are common to the condition. A majority of these symptoms. signs, and laboratory tests needs to be present or positive prior to making a diagnosis.

(3) In the case of an environmentally induced disease, there needs to be an adequate history of exposure. and such exposure must be to a known and recognized hazard.

(4) The agent responsible preferably can be identified in the body tissues or fluids. vir. in blood. urine. *etc.*

(5) The toxin of concern. when absorbed into the body, should produce the same effects in all susceptible subjects and preferably in experimental animals also.

(6) The latency period between exposure to the toxin and the development of physical effects should be reasonable. As a general rule. the longer the latency period. the more onerous and difficult the task of establishing a causal relationship between a chemical in the environment and the effect that is observed.

The potential hazards of welding may be classified as follows:

(i) Acute toxic effects, respiratory and systemic:
(ii) Chronic toxic effects;
(iii) Chronic respiratory effects: and
(iv) Carcinogenic effects.

Acute Effects

Irritation of the eyes and respiratory tract constitutes by t the vast preponderance of the acute effects of exposure welding fumes. Rarely, severe tracheitis. bronchitis. a pulmonary edema or pneumonia (leading to respiratc insufficiency and failure and, occasionally. to death) m occur. The detection of a cause and effect relationsh between exposure to welding fumes and symptoms of irri tion. such as excess watering of the eyes and a cough. usually relatively easy, but the incrimination of the partic lar irritant fume or gas is often difficult. Moreover. a sin agent seldom is responsible. In contrast. in those exposu followed by the development of severe respiratory insuf ciency or death. it is usually possible to identify and rec nize the offending agent. In this connection. cadmium. t oxides of nitrogen. ozone, and phosgene all have be incriminated. and. on occasion. may cause acute respi tory insufficiency and, rarely, death.

Specific Agents

Cadmium

Acute exposure to cadmium fumes may occur as a result various welding and cutting operations.[13] Such incide are rare but have been described well. Most have occurred confined, poorly ventilated spaces where dismantling ope tions have been taking place. At such places. oxyacetyle cutting or welding has been taking place and. subsequent it has been found that the bolts. nuts. or various other pa used in the metal frame of the structure had been cadmi plated. Those involved in the dismantling usually unaware that cadmium was present. and hence. the risk such work is unappreciated. Most of such hazards ar when cutting or welding involves metal containing a hi percentage of cadmium (5% to 15%). usually as an all Acute cadmium exposure and poisoning have been repor from California and from Canada.[14,15]

The acute effects of exposure to cadmium fumes increasing shortness of breath. upper respiratory tract irri tion. cough. and severe tracheitis and are often follow relatively quickly by the development of overt pulmon edema.[16] The latter is associated with frothy and. occasi ally, blood-stained sputum and may take 3 to 5 hr develop. The affected subject often complains of a charac istic metallic taste in his mouth. In those persons who ha been exposed to high concentrations. the subsequent dev opment of emphysema is common.

Oxides of Nitrogen

The oxides of nitrogen include four gases: nitrous ox (N_2O) which is used as an anaesthetic. nitric oxide (N which is rapidly oxidized to nitrogen dioxide, which exist: two forms NO_2 and N_2O_4. Both forms of nitrogen diox are relatively insoluble and exist as a darkish red-brown which, when inhaled in high concentrations. has a pung odor.

Exposure to moderate concentrations of nitrogen diox above the TLV. but of insufficient intensity to affect . lower respiratory tract. causes cough, irritation of the e

and nose. and little else.[16] In contrast, high concentrations are more likely to reach the small airways and alveoli and lead to the development of pulmonary edema and, occasionally. death.

Both arc welding and flame cutting lead to the generation of the oxides of nitrogen. The more serious life-threatening episodes occur when the welder is working in a confined space with poor ventilation.'''' Most fatal exposures have occurred in ships' hulls, tanks. or boxcars.

Since the oxides of nitrogen are relatively insoluble, exposure to low concentrations can be tolerated with few, if any, effects other than mild irritation. With massive exposures. the onset of symptoms is relatively rapid. usually within 10 to 15 min, and consists of paroxysmal coughing, excessive lacrimation. and irritation of the upper respiratory tract. Within 15 min. pulmonary edema may develop. When this occurs. the subject starts to cough up copious amounts of blood-stained sputum. becomes increasingly short of breath. and has obvious tachypnea and tachycardia. The rapid development of such symptoms usually is of poor prognostic significance and indicates a massive exposure. Should the exposures have been somewhat less massive but still severe. the cough and other symptoms may clear up for a few hours with the subject feeling appreciably better. but some 4 to 8 hr later. pulmonary edema ensues. When the latter is present. the affected subject is grossly tachypneic. cyanosed. and mid and late crackles are present all over both lungs.

During the first few hours. the chest radiograph may be normal unless the subject has symptoms of pulmonary edema, when fluffy exudates are seen throughout both lung fields. Thus. a clear chest x-ray directly after exposure does not mean the subject will not develop pulmonary edema later.

In many instances. the acute episode clears up completely. but 7 to 14 days later. the subject becomes acutely short of breath. develops fever. and shows extensive nodulation throughout both lungs on his radiograph. The latter is caused by bronchiolitis obliterans. The administration of steroids for 2 to 3 weeks shortly after exposure is believed by many to prevent the development of bronchiolitis obliterans; however. no prospective randomized trial has been carried out to confirm the efficacy. Even if the administration of steroids is delayed until the development of bronchiolitis. the majority of those affected show complete resolution.

The development of pulmonary fibrosis in welders has been attributed to exposure to nitrogen dioxide.[1,4] This hypothesis will be discussed later.

Ozone

Ozone is produced by the action of ultraviolet light on nolecular oxygen and exists as a layer in the stratosphere. ncreased concentrations of ozone may be present in and round the welding arc. Thus. metal arcs and gas tungsten rcs are particularly prone to produce ozone.[18,19] Both of hese types of arcs frequently are used to weld aluminium. Concentrations at and about the TLV and somewhat above t lead to irritation of the nose and eyes and are said to cause headache and general irritability. The typical Los Angeles smog and its irritant effects on the respiratory tract are in the main a consequence of ozone and other oxidants. As the concentration increases. tightness of the chest develops and headache becomes worse. Visual disturbances are reported also.[20] A dull persistent chest pain may result from exposure to high concentrations. Animals. when exposed to extremely high doses. may develop pulmonary edema. For the most part. exposure to ozone can be minimized so that no symptoms develop with the application of ventilation and relatively simple industrial hygiene. Rarely, personal protection is needed.

Phosgene

The decomposition of chlorinated hydrocarbons such as trichlorethylene and perchlorethylene may lead to the formation of phosgene.'''' Metal inert gas electrodes are particularly prone to lead to the formation of high concentrations of phosgene. Both of these agents are contained in degreasants which often are used in the same part of the shop where welding is taking place.

Phosgene is an almost odorless and colorless gas which liquefies at 8°C. Its inhalation causes pulmonary constriction by paralyzing the sympathetic system and leads to the massive transudation of fluid into the lungs. The initial symptoms are those of cough and. after 2 to 8 hr. pulmonary edema may develop.[22]

Exposure during welding is exceedingly uncommon. but it must be remembered that degreasing .solutions should be kept well away from all welding processes.

Metal Fume Fever

This condition was formerly known as brass founders' ague or Monday morning fever.[16] It usually develops in welders and oxyacetylene cutters several hours after the subject has left his work. The symptoms are those of a flu-like illness with fever, chills. and a cough. The subject is often prostrated for several hours. but usually within 12 to 18 hr. he starts to feel better, and the symptoms have cleared up completely within 48 to 72 hr. In general. the disease is self limiting and pulmonary complications are rare. Many workers recognize the symptoms and tend to ignore them. Moreover. with repeated exposures. attenuation of the symptoms takes place.''''

The disease often is attributed to an allergic response, but there is no evidence in favor of this hypothesis. Indeed. the condition occurs often on the evening of the first working day. It may recur on each Monday for several weeks but. as time goes by, become less severe. Were it an allergic response. there would be a period before the subject became sensitized. The evidence would suggest that something in the welding fumes attracts polymorphar leucocytes to the lungs, and the fever. chills, and cough result from the liberation of pyrogens. There is abundant evidence to suggest that the disease is induced by the inhalation of superheated. ultramicroscopic particles of various metals. but in particular. zinc. copper, and magnesium are the commonest causes.[16] Nickel. cobalt. and selenium also may be responsible. No

specific treatment is known. although many welders believe that drinking large quantities of milk helps.

Chronic Toxic Effects

Most of the chronic toxic effects arise from the constituents of the covered electrodes or from repeated exposures to fumes originating from material that is being welded.[24,25] Lead and manganese often are present in the electrodes. and small concentrations may be present in welding fumes.[7,20] For the most part. lead levels arising from welding are well below the TLV. Manganese concentrations are more likely to rise transiently above the permitted level.[20]

Manganese

Prolonged exposure to manganese may lead to manganese poisoning. Affected subjects may develop Parkinsonism, and the symptoms include languor and somnolence during the day time along with muscle pain. an unsteady gait, weakness, and involuntary movement.[16] To the author's knowledge, no subjects from either North America or Western Europe have been described with manganese poisoning resulting from welding, although there have been sporadic reports from Eastern Europe.[7,20]

Lead

Lead is an occasional constituent of the electrode and has not been reported in concentrations above the TLV in the welding environment for many years. Along the way, occasional outbreaks of lead poisoning occurred in welders and oxyacetylene cutters during shipbuilding and scrapping.[26]

Chromium

There is little doubt that significant exposure to chromium occurs. especially during stainless steel welding. In contrast. mild steel welding is not associated with exposure to high concentrations of chromium.

The effects of exposure to chromium have been studied by Tola et al.[27] These investigators studied six welders throughout the workweek and made determinations of the total fume and chromium in the air. along with measurements of the total chromium content of the urine. Urinary concentrations of chromium were a good indicator of short-term exposure to chromium.

A number of subjects with occupational asthma have been described among stainless steel welders. It has been suggested that chromium is the responsible agent.[7,20]

Nickel

Nickel also is produced during stainless steel welding, and many of the same risks associated with chromium are associated with nickel. Nickel is a recognized cause of asthma and perhaps may play a role in the development of occupational asthma that has been uncommonly reported in stainless steel welders.[28,29]

Other Trace Elements

Traces of copper. cobalt. iron. and manganese all occur in welding fumes. Ulfvarson and Wold[30] determined the blood concentration of these elements in 81 stainless steel and aluminium welders and in 68 nonwclders. The data, in the main. suggested that excessive concentrations of these constituents were not found in the blood and also pointed out that the concentrations noted could not necessarily be attributed to welding fumes.

Chronic Respiratory Effects

A number of known chronic pulmonary effects or diseases are associated with welding. These include siderosis (Arc Welders' Lung), bronchitis. emphysema. asbestosis and other asbestos-induced pulmonary conditions. and silicosis.

Siderosis

This condition was first described by Zenker.[31] but the two subjects he described also had tuberculosis. Neither of Zenker's patients was a welder. and it was not until 1936 that Doig and McLaughlin[32] described welders' siderosis. They noted that welders and oxyacetylene cutters developed a condition that radiographically resembled silicosis. Small. rounded opacities were present throughout the lung fields. and the radiographic appearances could not be distinguished with certainty from those of classical. simple silicosis. With the passage of time. it has become evident that in general the opacities seen in welders' siderosis tend to be more opaque and more circumscribed than those seen in silicosis. Moreover, over a period of several years. some subjects show radiographic clearing with regression of both the number and size of the opacities.[33]

Complicated siderosis as characterized by the presence of one or more large shadows has been reported. but in these subjects there always has been concomitant exposure to an additional fibrogenic agent other than iron oxide.[34,35] In two of the reports, a mass was resected. and it was only afterwards that it became apparent that both subjects had a history of sand-blasting. As such it was the presence of silico-tuberculosis that was responsible for the development of the conglomerate masses noted.[34,35]

Studies of lung function in welders with siderosis have shown little. if any, respiratory impairment.[36-43] Although it has been suggested that there may be minimal increases in the stiffness of the lungs. such changes have not been associated with significant symptoms.[44] Thus, of the 16 subjects studied by Stanescu et al.,[44] 7 had slight dyspnea and 3 had minimal sputum production. These symptoms more appropriately should be attributed to bronchitis and small airways disease than to parenchymal lung disease. Moreover. specific lung compliance and the elastic recoil at total lung capacity were not affected.

Other studies have reported to show changes in small airways function. but once again these changes are better attributed to bronchitis than to any lung stiffness associated with the deposition of iron in the parenchyma.[45] Measurements of the diffusing capacity in welders' siderosis likewise have been normal.[46]

Several investigations of the pathological changes that occur in welders' siderosis have been published. and there is

general agreement that the inhalation of ferric oxide, the main constituent of welding fumes, does not lead to fibrosis.[36,47-49] Although a. number of case reports have described subjects with lung fibrosis and attributed the fibrosis to the welding fumes. most are unconvincing. Other and far more likely explanations for the fibrosis. including exposure to other fibrogenic agents, were available. Moreover. epidemiological confirmation has been lacking.[9]

Microscopically, welders'siderosis is characterized by the deposition of iron particles in the lung parenchyma. While some of these particles can be seen to be lying free in the alveoli. most have been ingested by alveolar macrophages. The alveolar septa are not thickened and there is no alveolitis.[36] Iron particles present in the alveoli stain a deep blue with Prussian blue stain. In this connection. there have been occasional reports in which the granular pneumocytes in fibrosing alveolitis or. as it is sometimes known. chronic interstitial fibrosis of the lungs have shown intense blue coloring of the cytoplasm with appearances rather similar to those seen in hemosiderosis and in welders' siderosis.[50] One such case recently occurred in a welder. and it was alleged that the presence of iron had lead to the development of the fibrosis."[1] It was evident. however. that the subject had idiopathic pulmonary fibrosis. The little iron that was present in his lungs was not ferric oxide but hemosiderin.

Bronchitis

A number 01 well controlled epidemiological studies in welders have been carried out in which the prevalence of respiratory symptoms and pulmonary impairment has been quantified.[38,40-42,52-54] Some have demonstrated an increased prevalence of bronchitis.[38,41,43,52,54] The latter. however. has not been associated with disabling airways obstruction. and indeed. in most studies it has not been possible to show an increased prevalence of chronic airflow limitation except in smokers.[38,40-42,52,54]

A number 01confounding tactors exist that contribute to the increased prevalence of respiratory symptoms in those exposed to welding fumes. These include the observation that welders tend to smoke more than the general population[53,55] and that welding often is associated with exposure to other significant hazards. such as asbestos and silica. with the latter being particularly prevalent in those employed in

The bronchitis that develops in welders is a nonspecific response to irritant fumes originating from the welding process. The irritants may be gaseous or particulate. The bronchitis that aifects welders may be regarded as a form of industrial bronchitis and has the same effects on lung function and the same pathological features that are found in other workers who develop bronchitis from other industrial exposure, *e.g.*, cement workers. gold and coal miners, foundry workers, *etc.*[56] While the bronchitis that appears in welders sometimes may be associated with a minimal reduction of ventilatory capacity and minor obstruction to flow located mainly in the large airways, it is never disabling nor is it associated with the development of emphysema.[44-45,54] Moreover, cessation of exposure to the irritants usually leads to a decrease or. indeed, complete resolution of. the symptoms of bronchitis.

Emphysema

The only accepted occupational cause of emphysema in welders is cadmium. Much doubt exists as to whether low exposures to cadmium fumes lead to the development of emphysema, but there is some recent evidence to this effect.[16] Other studies have suggested that there is a fibrotic response.[57] In general, however. exposures to cadmium fumes in welding are well below the TLV. and it is only when the process involves welding or cutting alloys with a significant percentage of cadmium that a hazard exists.

Asbestosis and Other Asbestos-Induced Pulmonary Conditions

Although it was not until the 1960s that it was realized that welders were exposed to hazardous concentrations of asbestos,[58,59] it is now abundantly clear that those who were employed in shipyards (and occasionally elsewhere) in the 50s and 60s may have had sufficient exposure to asbestos to cause asbestosis. mesothelioma, and lung cancer.[9,25,60] It was frequently the custom for welding. pipefitting, and lagging to be carried out at the same time in a confined, poorly ventilated area of the ship. While pipefitters wore respiratory protection. welders seldom did because it was believed that intermittent exposures were not harmful. Such coincident exposures have a bearing on many of the so-called "symptomatic cases of welders' siderosis." Reference to this will be made later.

Silicosis

Welders are often coincidentally exposed to silica. In the past. work practices involved exposure to silica and the hazard attached to such exposures was seldom appreciated and usually unrecognized. Should a subject be exposed to both free silica and ferric oxide. a condition known as silicosiderosis develops."[1] Conglomeration in this condition is not uncommon. and the impairment and disability similar to that observed in classical silicosis occurs. Amorphous silica is the usual form of silica found in welding fumes: however. this is nonfibrogenic.

Carcinogenesis and Welding

Welders may be exposed to a number of carcinogenic materials while at work. Of particular concern in the past has been asbestos. and the increased incidence of lung cancer observed in welders. for the most part. can be attributed to coincident asbestos exposure. Although it has been suggested that chromium and nickel, both of which are found in the fumes generated during stainless steel welding. are a cause of lung cancer, no increased incidence of lung cancer in welders over that of other shipyard workers has been demonstrated which cannot be explained by exposure to asbestos or by the increased smoking habits of welders.[55,62] While a number of chromosome studies in welders have been carried out showing that numerous aberrations occur. their significance is dubious in view of the fact that it is difficult to demonstrate an increased incidence of cancer in man.[63,64]

Epidemiological Studies

Over the past several years, a number of studies have shed some light on the chronic effects of welding, especially in regard to the prevalence and effects of bronchitis. Hunnicutt and colleagues,[38] in a group of welders from the Newport News shipyard, found that the prevalence of symptoms such as cough and sputum were significantly higher in welders than in nonwelders. There also was an increased prevalence of airways obstruction, but only smoking welders were affected. Similar findings resulted from a study of Boston shipyard welders.[41] In this study it was recognized that many welders had significant exposure to asbestos. Peters and his colleagues[41] concluded that no detectable ventilatory defect was present in welders who did not smoke. Anne Fogh and her colleagues[40] observed similar findings in 156 Danish welders. They concluded that there was no significant difference between welders and controls in the occurrence of chronic bronchitis and ventilatory function after controlling for smoking. Similarly, Sjogren and Ulfvarson,[52] although able to show an increased prevalence of bronchitis, could detect no effect on lung function in a group of 269 welders. of whom 64 were aluminium welders: 56. stainless steel welders: and 149. road track welders. Antti-Poika et al.[42] found similar findings in a group of Finnish arc welders.

In a series of well carried out and detailed studies. McMillan[53,54,65,66] investigated the health of welders employed in the Royal Navy dockyards in Britain. In a well controlled retrospective study, McMillan analyzed the morbidity and incidence of respiratory disease over a 5-yr period.[65] Five relatively comparable groups who also worked in the shipyard were included as a reference population. These included boiler makers, shipwrights. electrical fitters. painters. and joiners. He concluded that there was no evidence of a significant excess of chronic respiratory disease in the welders. Moreover. in 16 subjects who were discharged from the dockyards because of respiratory disease. only 2 were welders. and in these there was nothing to suggest that the disease responsible for their stopping work was related to their job. He did notice, however. that welders had somewhat more upper respiratory illnesses than other groups but that the absence caused by these illnesses was of the same duration.

McMillan and Heath[66] measured lung function in 25 welders (with a reference population of 25 electrical fitters) at the beginning and end of a shift and related any changes in ventilatory capacity to the gas and fumes present in the subject's breathing zone. No significant differences were found in lung function changes over the day. Later McMillan[53] reported on the relationship between absence attributed to respiratory disease. exposure to welding, and smoking habits in three groups of workers in the Devonport Naval Dockyards. He studied welders, shipwrights, and boiler makers, all of whom were intermittently exposed to welding fumes. McMillan noted that a higher proportion of welders were smokers. Those welders who were also smokers had more time off work than did the nonsmokers. Nonsmoking welders. however, had a lower absence and disease incidence than did nonsmoking shipwrights and boiler makers. H concluded that if welders do suffer more respiratory diseas and, thus, more respiratory symptoms than controls. i would be expected that there would be proportionately mor exsmokers among welders than controls, when in reality th converse was present.

McMillan also has published a general review[67] of th health of welders in naval dockyards and has concluded tha there was no evidence of a causal relationship betwee welding and respiratory diseases or other ill health. with th exception of injuries, among welders in Her Majesty's dock yards. He felt there may be a small minority of welders wh are unusually susceptible to the effects of fumes and gase and that such persons have obstructive airways disease suc as asthma or emphysema. Neither the asthma nor the emphy sema. however, were related to welding exposure.

Because it has beensuggested that most studies of welder have been carried out in welders with relatively short expc sures to welding fumes (less than 15 yr). McMillan an Pethybridge[54] decided to examine I35 welders aged 45 an over who also had prolonged exposures. The average dura tion of welding was 33.1 yr. Those exposed had detaile. clinical. radiological. and pulmonary function examination and were compared with a comparable control group age 45 and over. McMillan and Pethybridge concluded tha prolonged exposure to welding fumes did not cause signit cant clinical abnormality nor any serious impairment of lun function. They expressed the opinion that minimal airway obstruction may result from exposure to welding iume Rather similar findings have been reported by Hayden an colleagues[43] in welders employed in three engineering facto ries in the British Midlands. These investigators conclude however, that welders had no increased risk of chron obstructive lung disease.[67] Similar conclusions have bet reached by others elsewhere.[40-42]

Symptomatic Lung Disease in Welders

Over the last 30 to 40 yr. a number of welders have bet described in whom symptomatic pulmonary disease w present. There has been a tendency to associate the sym toms and the disease present in the welders with their occ pation. This has occurred despite the fact that the nature the disease and the type of impairment. whether restricti or obstructive. have differed. In addition the absence consistent pathophysiological effects and of supporti epidemiological evidence to confirm the association cas doubt in the validity of the association of welding to t disease described.

In some instances. analyses of lung tissue have demo strated numerous elements to be present in the lung pare chyma in excess concentrations. and it has been assum that their presence has led to the fibrosis and the oth pathological responses observed.[2,3] Their mere presence the site of the histological changes has been assumed to sufficient to associate the pathological changes observ with the agent that has been deposited in the lung. Th despite the fact that other regions of the lung in which

pathological changes have been present often have equal amounts of the foreign agent present.

Charr,[67-69] in a series of repprts extending over a number of years. described a number of welders with respiratory insufficiency. In no instance was an adequate smoking history given. and clearly, no consideration was given to the possible role cigarette smoke had in inducing the pulmonary diseases. In some instances those affected showed the presence of emphysema and, in other instances, fibrosis. It is interesting to note that the majority of the men Charr described worked in the Philadelphia naval shipyard as welders.'"') The first subject in a series of subjects described by Charr[67] had obvious emphysema. and the reproduction. in his paper. of the chest radiograph makes this apparent. The so-called fibrosis that existed was simply some accentuated markings in the lower zones. No smoking history was given. The second case was a man who had been a welder in a shipyard and had been noted to be cyanosed and have clubbed digits. The chest x ray showed exaggeration of the lung markings in the lower half of each lung field. Clearly, the presumptive diagnosis should have been asbestosis. The subject of the third report had worked as a welder for only 6 yr, mostly with steel. He was noted to be short of breath on exertion. In this instance. the description suggests this subject had increased markings and emphysema. Cases 4 and 5 were poorly described. and neither a smoking history nor pulmonary function data were given. Case 6 had worked in confined spaces where he was likely to be exposed to asbestos. He had cyanosis and his sputum was noted to be positive for tubercle bacilli. He was treated with antimicrobial therapy. as was Case 7. Clearly, this series of cases was poorly investigated. had inadequate smoking and occupational histories, and had no definitive evidence as to the type of respiratory impairment, if any, available.

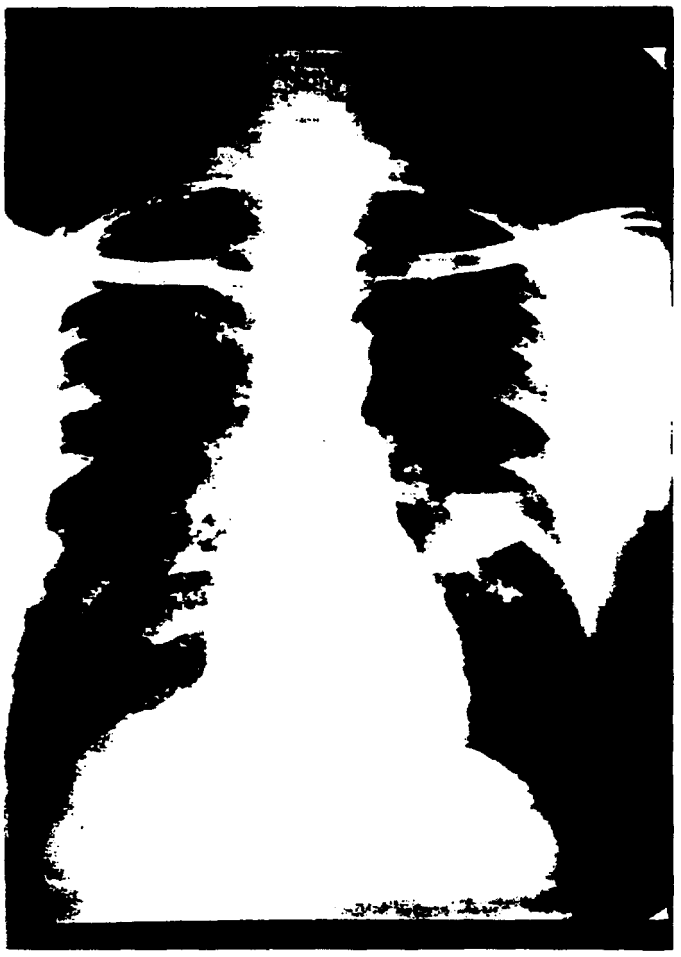

Figure 1—Chest radiograph of a welder with a mass in the superior segment of the left lower lobe. The welder in this instance also had sandblasted in a shipyard and had worked in the proximity of sandblasters for a considerable time. The lesion was resected as a carcinoma but proved to be conglomerate silicosiderosis.

A further series of three cases is described by Charr.[69] Here again. Case 1 is described as having pulmonary function tests showing respiratory insufficiency consistent with emphysema and fibrosis. Since the type of pulmonary impairment in these two conditions is totally dissimilar. it is difficult knowing how such tests would indicate both fibrosis and emphysema as being present. Case 2 likewise had been employed in the Philadelphia shipyard. and again it was noted that his fingers were clubbed and that he had numerous wheezes throughout the chest. Again no smoking history was given. and yet again basal reticulonodular shadows were described in the lower half of the field. suggesting pulmonary fibrosis. A biopsy showed extensive fibrosis. and indeed. one photomicrograph in Charr's manuscript[69] suggests a remnant of an asbestos body is present. Similarly, Case 3 again showed clubbing of the fingers. polycythemia, and hypoxemia. There was a diffuse increase in linear markings throughout both lungs but mostly affecting the lower two-thirds of the lungs. In this instance. lung function tests had been done. and there was a generalized reduction of lung volumes. This patient had not undergone lung biopsy. and again. because he had been employed in the Philadelphia naval shipyard. a diagnosis of asbestosis must be considered most likely.

Additional welders with definite pulmonary impairment or so-called "symptomatic welders' siderosis" have been described. Freide and Rachow'"' described a 40-year-old welder who was admitted to hospital in obvious heart failure. No smoking history was given. but it was noted that he had worked on construction. The physical examination indicated that he had an apical systolic murmur and gross cardiomegaly with obvious peripheral edema. The lung function tests showed restrictive impairment with low lung volumes. and yet the authors state that the ventilatory pattern was that of marked obstruction. In doing so they do not give the forced expiratory volume in 1 sec (FEV_1) value or provide any evidence of airways obstruction. Following treatment of his heart failure. his vital capacity improved greatly and went up from 1.5 L to 2.77 L. This improvement could not have taken place if the subject had pulmonary fibrosis induced by welding since fibrosis does not suddenly disappear with the administration of diuretics. The chest radiograph was reported as showing diffuse pulmonary fibrosis and a severe degree of emphysema. and again it must be noted that these two conditions have totally divergent effects on lung function. Much was made of the presence of

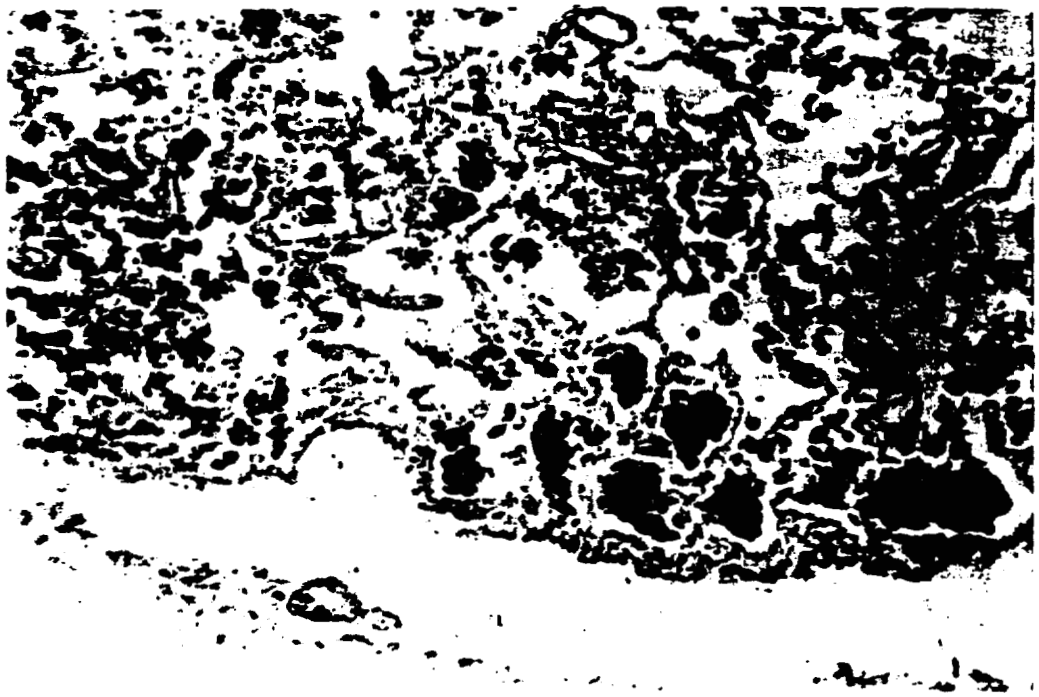

Figure 2—Lung biopsy of a welder. The black-staining material represents deposits of iron in intra-alveolar macrophages. They have been stained with Prussian blue It can be seen that there is no thickening of thealveolo-capillary membrane. There is no fibrosis. The iron content of the subject's lung was 25 times the normal value.

large quantities of iron in this subject's lungs. but it seems certain that the iron was hemosiderin present in the macrophages. and the latter would more aptly have been termed "heart failure cells." In their discussion. the authors'"' talk of nonspecific fibrosis leading to emphysema and to other secondary features of pulmonary hypertension. Such hypotheses are no longer acceptable. and clearly the authors described a subject who happened to be a welder and also developed unrelated heart failure.

A further case report was described by Meyer. Kratzinger. and Miller.[35] This subject was noted to have an enlarging mass lesion in the right upper lobe. Although he was a welder and he had been in a ship's company for 24 yr. it is recorded that he also had welded in close proximity to sand-blasting. For this subject. the history makes it clear that the subject had never smoked. Subsequently, the mass in the right upper lobe, cavitated and was resected. Pathologically. the mass showed conglomerate fibrosis or massive fibrosis and exudative granulomata. The histology was reported as compatible with tuberculosis. The concentration of silica present in this man was stated to be 30% of the total "iron" content. This phrase in itself is somewhat confusing, but it is quite clear that the subject in question had stiff lungs and obvious silico-tuberculosis. To invoke any other diagnosis would seem to be flying in the face of reason.

Similarly, Mann and Lecutier[72] described another welder who had a lobectomy. and pathologically, tuberculosis was noted to be present. The lung volumes were slightly reduced, but here again silica was noted to be present in the lung and again the incrimination of welding as the cause of this man': problem was based on extremely tenuous evidence.

Elsewhere. Patel and colleagues[73] describe a case of wha they term "Arc Welders' Lung." Their subject had sever restrictive impairment but only minimal obstruction. Onc again his occupational history recorded that he had worke in a naval dockyard for 18 yr. The phvsical examinatio again suggested interstitial fibrosis. compatible with asbes tosis. Although this subject had obvious pulmonary fibrosis which the authors attributed to the parenchymal depositio: of iron, a biopsy of his lungs did not stain with Prussian blue indicating that there was little. if any, excess iron present i the lungs.

The series of symptomatic cases referred to above. alon with other sporadic cases that appear in the literature. ar unconvincing in that first. the type of pulmonary impair ment described has differed from case to case. and seconc many of the subjects have had other exposures to harmfi agents (including silica) and, thereby epidemiological cor firmation has been entirely lacking. Despite this. som workers have suggested that the reason most epidemiolog cal studies have failed to demonstrate subjects with fibros is a consequence of the fact that the men employed in welc ing have been employed in the industry for only a limite time. Yet. when one reviews the ages of the symptomat cases described above, the mean exposure time is relative limited. While two of Charr's[67-69] cases had been expose for 30 and 40 yr, respectively. and one for 24 yr. all the othe had been exposed for less than 20. and in one instance. t

welder had worked for as little as 6 yr. Freide and Rachow's[71] subject had welded for only 11 yr; that of Patel et al.,[73] 18 yr; and that of Myer et al.,[35] 24 yr. The early subjects described by Charr[67] had a mean duration of welding of 13 yr. Most of them had normal lung function. one dying of an aneurysm while another had emphysema, but Once again no smoking history was available. Thus, there is little evidence to support the contention that the only reason that epidemiological confirmation is lacking is that the welders studied have had inadequate or brief exposures to welding fumes. In those case reports in which welding has been suggested as the cause of the impairment. the time spent in welding has been less than 15 yr and, in several instances. less than 10.

Elsewhere, there have been a number of review articles suggesting that welding fumes. under certain conditions, may be fibrogenic. Thus. Stern and colleagues'" reviewed 3600 pathology cases in the Liebow Pulmonary Pathology collection at the University of California's School of Medicine in San Diego, and of the total, 29 subjects were found who were welders and were noted to have pulmonary fibrosis also. Of the 29.8 had a history of either exposure to talc or asbestos. Thus, the fibrosis well may have resulted from exposure to these agents. A further 10 subjects had no available occupational history. That leaves 11 subjects with fibrosis in whom idiopathic pulmonary fibrosis had not been excluded. After a long and exhausting review that included a detailed description of the agents contained in the lungs, the authors concluded that the fumes from manual arc welding of stainless steel were fibrogenic. They then went on to suggest that nitrogen dioxide was the fibrogenic agent responsible for the development of the fibrosis in the small group of welders. an argument that is less than convincing and is. moreover. epidemiologically indefensible.

The late Dr. Liebow. an international authority on pulmonary fibrosis. had lung sections referred to him from all 'over the world. It is quite clear there was a predilection for referral of tissues from subjects who had occupational exposure to fumes and particles. By the process of *post* hoc. *ergo propter* hoc. it was concluded that the fibrosis noted in them was a result of occupational exposure. Such an attitude assumes that idiopathic pulmonary fibrosis or fibrosing alveolitis does not occur in welders and other persons with occupational exposure.

Similarly. in a long and tendentious review of the toxicity of the higher oxides of nitrogen. Guidotti puts forward the argument that low concentrations of nitrogen dioxide induce pulmonary fibrosis."' In reality. there is little or no evidence to suggest that this occurs in man, and indeed. exposure to the higher oxides of nitrogen leads initially to the development of pulmonary edema and. subsequently. to bronchiolitis obliterans. Moreover. there is substantial variation between species in susceptibility to nitrogen dioxide and the pathological changes induced by this gas. To argue from some unconvincing evidence in hamsters that the same reaction occurs in humans is completely unjustified. In animals. for the most part. NO_2 induces a subacute bronchiolitis and. subsequently. centrilobular emphysema.

Similarly, Abraham and Hertzburg,[3] in a study of 93 subjects with idiopathic pulmonary fibrosis. concluded that because 75% of them had a history of exposure to dust or fumes. the latter played a role in the development of the fibrosis. Here again these subjects were derived from Liebow's collection and represent an exceedingly biased group. Before any conclusions can be drawn from such a group of subjects, it is obviously necessary to carry out a careful case control study. As such, this has been sadly lacking.

Pertinent in this regard are the findings of McMillan,[9] who found 11 of 328 with lung fibrosis. All were related to asbestos exposure. Moreover, in a carefully controlled case control study, neither the diffusing capacity nor the total lung capacity were significantly different in welders as compared to control subjects.[54]

Thus. the evidence to suggest that welding fumes induce pulmonary fibrosis is exceedingly tenuous in the absence of a clear-cut history of exposure to recognized fibrogenic substances. such as asbestos. silica. or cadmium. There is little or noevidence to support the contention that nitrogen dioxide. chromium. or any other metal emitted in welding fumes will induce pulmonary fibrosis. although the introduction of new technology may lead to new and unrecognized hazards.

Summary

The various exposures. hazards. and diseases that may be accounted by welders are described. The hazards of welding may be subdivided conveniently into acute toxic effects. chronic toxic effects, chronic respiratory effects, and carcinogenetic effects. Each is considered in turn and their effects on morbidity and mortality are discussed. The evidence suggests that welding is not a particularly hazardous occupation provided care is taken to limit exposure to the toxic effects of any fumes that are generated. Nonetheless. the technical aspects of welding are constantly undergoing change. and continual vigilance is necessary lest a new process introduces a formerly unrecognized hazard.

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