Respiratory Hazards of Welding

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Welding produces a variety of substances toxic to the respiratory tract. A thorough occupational history, based on an understanding of the various work processes, is essential to the prompt identification of potential welding-associated hazards. Several key questions can establish if the worker has been exposed to some of the more potent airborne emissions from welding. Although this trade is not associated with extraordinary morbidity and mortality in occupational health statistics, a wide variety of acute and chronic respiratory disorders are recognized. There is considerable overlap in the presentation of several of the acute disorders, frequently posing a diagnostic dilemma. The health hazards of welding are changing with the evolution of increasingly sophisticated technologies. Although exposure to welding fumes in general is associated with a 30% to 40% increased mortality from lung cancer, it remains controversial whether or not stainless steel welders are at heightened risk.

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Welding is a process of joining metals by melting and fusing. The trade of welding, as the term is loosely used, is actually a variety of tasks, involving the cutting of metal objects, grinding, brazing, and in some applications, soldering. It has been estimated that welders account for between 0.5% and 2.0% of the general workforce (1). Few occupations are as fundamental to industrial development as welding. Among the industrial trades, skill at welding is a highly prized ability, and skilled welders are seldom out of work.

This article will consider both the chronic and acute respiratory effects associated with welding. Emphasis is placed on the accurate diagnosis of welding-related lung disease. It is important to note, however, that welding is also associated with effects on other body systems (largely ocular and musculoskeletal injuries) and that numerous exposures to toxic compounds are possible, some with well-known extrapulmonary toxic effects (2).

As with any occupational disease, a thorough knowledge of work processes and potential toxic exposures is a prerequisite for the prompt identification of welding-related health problems. The following section will explain aspects of welding that may assist the clinician.

ARC WELDING

Although there are many different welding technologies (at least 220 variations by some counts), a relatively small number of welding techniques are common in industry. Moreover, there are basic principles of welding common to all technologies. This section will explain the essential elements of welding to assist the clinician in obtaining an occupational history and to provide the basis for the prompt identification of potential respiratory hazards. Important terms that are commonly encountered in welding or the medical literature on welding will be italicized as they are introduced.

To melt the constituents of a weld, an energy source is required. For most

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types of welding, heat is derived from an electrical *arc* formed from a current that bridges a gap from an *electrode* to the *base metal* to be welded. Hence the term *arc welding*, the essential features of which are displayed in Figure 1. In addition to generating heat, the arc produces a substantial amount of ultraviolet radiation.

The arc is used to melt not only the base metal but also an additional metal, referred to as a *filler metal*. The filler metal can either be added as a separate *wire* or *rod* or can be derived from the electrode itself. In the former instance, the electrode is said to be *nonconsumable* because it provides an energy source only and is not consumed in the formation of the weld. Nonconsumable electrodes are commonly made of tungsten or carbon. In contrast, a *consumable* electrode not only conducts the current needed to form the arc but also provides the filler metal needed to form the weld.

The site where molten filler and base metal combine is referred to as the *weld pool*. Contaminants in the weld pool can decrease the strength of the weld. The most important of these are gases commonly present in the surrounding air: hydrogen, oxygen, and nitrogen (3). Because these gases are much less soluble in solid than in liquid metal, they can result in an unacceptably porous, brittle weld with cooling. Oxidation of the metal in the nascent weld is an additional source of weakness. It is therefore necessary to protect the weld pool by using a *shielding atmosphere*, blanketing it by applying an inert gas such as argon. An alternative strategy is to use *flux*, applied either from a core within the electrode or as a separate paste. Flux contains various agents, which, in addition to producing shielding gases, can serve to stabilize the arc, clean the welding surface, enhance the mechanical integrity of the weld, and decrease oxidation.

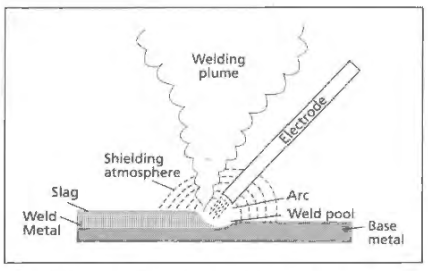

FIGURE 1. *Schematic illustration of the components of arc welding.*

Slag refers to nonmetallic material originating from either a coating over a consumable electrode or as a constituent of flux that protects and insulates the cooling weld. Once the weld has formed, it is usually necessary to remove the slag by cleaning the welded material. The completed weld, therefore, is a complex mixture consisting of a coalescence of base and filler metals that may also include constituents of flux and a slag coating.

The various types of arc welding are distinguished by the type of shielding used. The four most prevalent welding techniques are shielded metal arc welding (SMAW), gas metal arc welding (GMAW), flux cored arc welding (FCAW), and gas tungsten arc welding (GTAW). The choice of a particular technology depends on many factors, including the thickness and type of base metal to be welded, the size and strength of the weld desired, the speed or volume of welding, the position of the material to be welded (i.e., vertical or horizontal), and cost. A combination of these four processes satisfies the requirements of most industries.

Shielded Metal Arc Welding (SMAW)

SMAW, commonly known to welders as "stick welding," is a versatile process using short consumable electrodes consisting of a core of filler metal covered by materials to produce slag and shielding gases. SMAW is the traditional and, in North America at least, still the most common welding process. In Europe, this form of welding is referred to as manual metal arc welding (4). SMAW is inexpensive and readily performed by relatively unskilled workers.

Gas Metal Arc Welding (GMAW)

GMAW is an increasingly common, semiautomatic welding process in which a solid consumable metal electrode is fed into the arc. A gaseous shield of either argon and/or carbon dioxide is provided from a separate source. The result is a slag-free, high-speed welding process requiring little operator training that is well suited to production line work. Because slag does not need to be removed from the weld, this type of welding is easier to automate and reduces labor costs.

In Europe, the terms *MIG* (metal inert gas) and *MAG* (metal active gas) welding reflect whether inert gas (argon) or active gas (argon-carbon dioxide) is used in shielding. In North America, the commonly used term "MIG welding" includes both "MIG" and "MAG" processes.

Fluxed Core Arc Welding (FCAW)

FCAW closely resembles GMAW in that a continuous consumable metal electrode is fed into the arc. However, in FCAW, the electrode is actually a cylinder of filler metal with a central core of flux. The flux may be the only source of shielding gas (*self-shielded FCAW*), or a dual system can be used, using supplemental gas, usually carbon dioxide, from an auxiliary source (*gas-shielded FCAW*) (3).

Gas Tungsten Arc Welding (GTAW)

GTAW, better known as *TIG welding* (tungsten inert gas) is a process in which a nonconsumable tungsten electrode provides the arc, and a separate hand-fed rod provides the filler material. A shielding gas such as helium or argon is used. GTAW requires a highly skilled operator and has a low rate of production, but it is well suited to thin plates, fine work, and difficult materials such as aluminum and magnesium. This technology is used for relatively small welds.

Related Technologies

Two techniques related to welding are *soldering* and *brazing*. These are both joining methods that differ from welding in that only the filler metal, but not the base metal, is melted. Soldering refers to the use of a filler metal with a melting point below 450°C, whereas in brazing, the filler metal melts above 450°C.

In addition to being used to join materials, welding procedures can be modified to cut metal. However, most of the arc welding techniques do not concentrate sufficient heat to cut reliably. The cutting system most widely used is called *oxyfuel gas cutting*. This technique involves two steps. Oxygen together with a fuel source (usually acetylene, referred to as an *acetylene torch*) is first used to heat the metal. A

high-velocity stream of oxygen then oxidizes and blows away the molten metal, producing the cut.

POTENTIAL RESPIRATORY HAZARDS

The respiratory hazards generated during welding are all produced either by the action of heat or by ultraviolet radiation from the arc. The combination of gases and fumes produced is referred to as the *welding plume*. Considering each of the components of welding separately, potential substrates for respiratory toxins include the following:

1. The base metal
2. The filler metal
3. Coatings on the base metal
4. Coatings on the filler metal or consumable electrode (which includes slag)
5. Flux
6. The surrounding atmosphere
7. Electromagnetic fields
8. Contaminants

The filler metal provides the major source of the fume, with the base metal only making a significant contribution if it is coated. Because the constituents of flux and electrode coatings are similar, six sources require separate consideration. After a survey of the hazards from each component is provided, the various respiratory disorders associated with welding will be discussed.

Base and Filler Metal Fumes

Welding activity frequently involves steel that comes in various alloys. Steel is simply an alloy of iron with a small (usually less than 0.40%) amount of carbon. *Stainless steel* (SS) is designed to resist corrosion (particularly oxidation) by the addition of at least 18% chromium. Nickel is also a constituent of SS, generally found in levels of 8% to 11%. Other types of steel (containing little or no chromium) are referred to as *mild steel* (MS), *low alloy steel*, or *plain carbon steel*. Welding of SS has been shown to produce airborne chromium of which a significant portion is present in the hexavalent state (5).

Cadmium may be encountered as a filler metal ("silver solder") used in brazing. Other metals encountered as base or filler that may produce adverse respiratory effects include copper, magnesium, and, rarely, cobalt (6). Under the conditions of welding, metal fumes are invariably present as oxides, as for example, magnesium oxide.

Base Metal Coatings

A coating of zinc is frequently applied to steel (known as *galvanization*), leading to the production of zinc oxide when welded. Paints are an important source of several noxious agents that are readily volatilized with welding or, more commonly, cutting. Lead is perhaps the best known such additive; however, mercury is also used as an antibacterial and antimildew agent. Rust-proofed metals may contain a coating of phosphate, a potential source of phosphine gas. Sheet metal may be plated with cadmium.

Flux and Electrode Coatings

Fluorides and silicates are sometimes constituents of flux or electrode coatings. Asbestos was also once widely added as a powder; however, this practice ceased in the 1950s (5). One particularly important constituent of flux, used as a core in soldering, is colophony, a pine pitch that is the causative agent of electronic worker's asthma.

Gases Produced from the Atmosphere

Several important compounds are produced by the action of the welding arc on surrounding atmospheric gases. Ultraviolet radiation produced by the arc converts oxygen into ozone. Greater amounts of ozone are produced when welding on aluminum as a base metal or when using shielding gases, especially argon. Thus, the combination of GTAW on aluminum is of particular concern with respect to ozone. Similarly, oxides of nitrogen can be produced from ambient nitrogen. Oxyacetylene cutting is known to produce some of the highest levels of these compounds.

Many other combustion products such as carbon monoxide and carbon dioxide are present but only rarely in clinically significant amounts. However, under improper working conditions, particularly during welding in a confined space, asphyxiation becomes a very real hazard when atmospheres containing carbon monoxide are combined with large amounts of shielding gases.

Electromagnetic Fields

Interest in the human health effects of exposure to electromagnetic fields (EMFs) has been a topic of controversy for some years. Recently, the exposure of welders to EMFs has come under scrutiny (7,8). Kirschvink and colleagues (7) found that magnetite could naturally be located in human cells; therefore, changes in the intensity of the EMFs could be expected to induce abrupt currents in the exposed cells. These findings could indicate that such currents may be induced by alternating magnetic fields during welding. Studies are under way to evaluate exposure to EMFs during the application of different welding methods (8). At present, no conclusions can be drawn regarding the importance of this exposure in welders.

Contaminants

In addition to the integral components of welding, many substances may be present inadvertently in the welding area. Such contaminants commonly include various greases and solvents. One of the best known examples is phosgene gas ($COCl_2$) produced by the action of ultraviolet radiation from the arc on chlorinated hydrocarbons commonly used as degreasers (such as perchloroethylene and trichloroethylene). GTAW on aluminum can generate very high levels of phosgene.

TABLE 1. *Typical fume generation rates of common welding technologies (11).*

Technology	Fume Generation Rate (mg/min)
Shielded metal arc welding (SMAW)	300–800
Gas metal arc welding (GMAW)	200–500
Flux cored arc welding (FCAW)	900–1300
Gas tungsten arc welding (GTAW)	3–7
Oxyfuel gas cutting (of steel)	200–700

General Approach to Assessing Exposures in Welding

The main component of welding fumes is iron oxide (9). For arc welding, the majority of the particles are less than 1 μm and are thus readily respirable (10). Comparing the various types of welding technologies (Table 1) provides a crude indication of the relative "cleanliness" of each practice. However, it cannot be overstated that welding fumes represent a complex mixture of gases and that additional attributes influencing exposure such as size distribution and aerodynamic properties of fume particles have been shown to vary considerably with various welding methods (12).

Several other factors influence the amount of fumes to which a worker is exposed. These include variables such as the degree of confinement, the rate of progression of welding, and the adequacy of ventilation. Other less obvious factors include the work configuration. For example, the total fume concentration generated by SMAW has been shown to vary from about 1 to 28 mg/m^3 under nominally identical conditions, apparently as a result of differences in posture and welding position (11). Karlsen and coworkers (13,14; Karlsen JT, Torgrimsen T, Langård S. Exposure to solid aerosols during TIG and MIG/MAG on stainless steel, unpublished, 1996) have presented data on fume concentrations and concentrations of different elements during the use of different welding methods.

The helmets used by welders to protect themselves from the heat and ultraviolet radiation of the arc have been shown to provide a variable amount of respiratory protection. One study found that levels of iron oxide measured inside the helmet were 36% to 71% of the levels outside (16). A more recent comparison of fume concentrations inside welding helmets to those of the personal breathing zone revealed a mean ratio of only 0.9 (17). Studies have shown that filter masks provide a more appropriate exposure protection than air-stream helmets (18).

The previous two sections may be summarized by considering some relevant questions that the clinician who is faced with respiratory symptoms in a welder may wish to ask (Table 2). In addition to the history, several other methods exist to ascertain potential exposures. Material safety data sheets that document the constituents of the filler metal or consumable electrode should be available to the worker. The American Welding Society uses a classification number that is stamped on some filler metals and electrodes, which allows for identification through a published listing. When this number is unknown or unavailable, it is also possible to cross-reference through trade names (19,20).

ADVERSE RESPIRATORY EFFECTS

That welders may be subject to some untoward health effects peculiar to their occupation is beyond doubt, but the scope and severity of these effects have often been a source of uncertainty and, at times, of controversy (21). Although it is generally true that welders as a group do not seem to have an extraordinarily high burden of morbidity compared with other industrial workers or to the general population, there is evidence that hazards are changing with the introduction of new technologies, hence also disease risks.

Acute Respiratory Effects of Welding

The literature on the acute respiratory effects of metal fumes is not entirely clear. Two clinical entities are widely recognized: metal fume fever and toxic pneumonitis. However, an accumulation of case reports indicates that a spectrum of intermediate presentations exists. There is no consensus on terminology for either of these conditions, and the term "metal fume fever" is often used to describe what is clearly a toxic pneumonitis (22).

TABLE 2. *Key questions in the occupational history for welders.*

	Possible Toxins/Comments
What type of welding were you performing?	
Gas tungsten arc welding	Ozone
Soldering	Numerous respiratory sensitizers
Oxyacetylene cutting	Oxides of nitrogen
What base/filler metal were you welding?	
Aluminum	Ozone
Stainless steel	Chromium, nickel
Sheet metal	Cadmium
Was there any coating on the base metal?	
Paint	Mercury
Rust proofing	Phosphine
Galvanized	Zinc
Were you working with sheet metal or silver solder?	Cadmium
Were you working with flux/coated electrodes?	Silicates, fluoride
Was the base metal clean or dirty?	
Degreasers	Phosgene
What type of ventilation and personal protection were you using?	
Confined space	Asphyxiation
In what position were you welding?	Higher exposure in downhand position

Metal Fume Fever. Metal fume fever is a self-limited but very uncomfortable condition closely resembling influenza most frequently associated with exposure to fumes of zinc oxide. The condition is very common among welders and goes by many names, including "Monday morning syndrome," "foundry fever," "the smothers," "spelter shakes," "welder's ague," and "brass chills." It typically begins 4 to 6 hours after exposure with fever, chills, headache, nausea, shortness of breath, myalgias, and a sweet metallic taste in the mouth. The illness runs its course over 1 or 2 days without specific treatment (23,24). One peculiar feature of metal fume fever is the phenomenon of tolerance or tachyphylaxis in which workers are asymptomatic with repeated exposure. This tolerance is lost after holidays or weekends, hence the term "Monday morning syndrome."

One thousand ninety-two cases of metal fume fever were reported in 1994 to poison control centers in the United States (25). Blanc and Boushey (26), extrapolating from the "Doctor's First Report" system in California, derive a figure of 1500 to 2500 cases annually in the United States. However, because of their familiarity with this disease and its benign course, many welders do not seek medical attention for this condition, leading to substantial underreporting.

Metal fume fever is a poorly understood entity. It is clinically indistinguishable from several other disorders that result from the inhalation of a diverse array of agents including organic dust toxic syndrome caused by various bioaerosols and polymer fume fever caused by the pyrolysis products of tetrafluoroethylene resins (Teflon; du Pont, Wilmington, DE). All of these conditions also share the phenomenon of tolerance (27). Recently, the term "inhalation fever" has been advocated to encompass these conditions (27).

Several pathophysiologic models have been proposed for metal fume fever, the most plausible of which is based on cytokines (28). In this model, the inhaled zinc oxide causes pulmonary macrophages to synthesize and release cytokines. The key cytokine in this process may be tumor necrosis factor-alpha (TNF-α) (28). The released cytokine(s) would then initiate a cascade of mediators leading to the host of systemic flu-like symptoms. TNF, interleukin-6, and interleukin-8 have all been found in elevated levels in the supernatant of lavage fluid obtained from subjects exposed to elevated levels of zinc oxide (28). As Blanc and Boushey (26) point out, the lung plays a key role in initiating metal fume fever and does not merely serve as the route of exposure because the symptoms of metal fume fever are not elicited by the ingestion or intravenous administration of toxic levels of zinc.

Although there is no dispute that exposure to zinc oxide fumes (almost always in the context of welding or cutting galvanized metal) causes metal fume fever, many authors also refer to a long list of other metals as putative causes, variously implicating copper, magnesium, manganese, selenium, silver, aluminum, nickel, cadmium, mercury, chromium, iron, tin, and antimony (23,24,29). A careful review of the literature supports only zinc, and much less readily, copper and magnesium as proven causes of metal fume fever (28,30). Mixed exposures in welders render any conclusions about a causative role for single agents on the basis of a case report tentative. There is no doubt that zinc oxide causes the vast majority of cases of metal fume fever among welders. Other possible causes should not be entertained unless zinc has been convincingly excluded. Copper and magnesium can probably cause metal fume fever but do so more rarely. This impression is supported by the paucity of case reports implicating these metals despite widespread use in industry.

Another common misconception concerning metal fume fever highlighted by Blanc and Boushey (26) is that it can only be caused by freshly formed fumes of zinc oxide. Metal fume fever has been reproduced in individuals exposed to commercially available finely ground zinc dust, and two cases have been reported in workers using a rotary wire buffer on galvanized metal tanks (30,31). Zinc oxide must be present as ultrafine (less than 1 μm) particles to be effectively delivered to the alveoli. Sufficiently small particles are far more readily generated by the high temperatures of welding than by most physical means. Thus the overwhelming preponderance of cases of metal fume fever in response to exposure to zinc oxide fumes simply reflects the physical constraints governing the route of exposure.

Attempts to measure serum and whole blood zinc in cases of metal fume fever have been made with inconsistent, often uninterpretable, results (32). Moreover, very little is known about the toxicokinetics of zinc after inhalational exposure.

Pneumonitis. In contrast, certain metal fumes, the prototype being cadmium, can cause diffuse and profound alveolar damage, resulting in an acute toxic pneumonitis (33,34). The clinical picture consists of an insidious course of noncardiogenic pulmonary edema and acute respiratory distress syndrome (ARDS). This condition may be life-threatening, and supportive treatment is often required. However, the onset of ARDS can be delayed by up to 48 hours from the time of exposure. During this prodromal period, the signs and symptoms of toxic metal pneumonitis are very similar to those of metal fume fever. For this reason, some clinicians advocate the inclusion of cadmium pneumonitis in the differential diagnosis of any welder presenting with the clinical picture of metal fume fever (34).

Table 3 summarizes the causes of pneumonitis in welding. All of the agents listed are known to cause toxic pneumonitis at high-level exposures. However, only cadmium, phosgene, ozone, and nitrogen dioxide have been implicated through case reports in welders. Beryllium, in addition to a direct toxic effect on the lung, can also cause a granulomatous pneumonitis with relatively lower-level chronic exposure. Only the latter disease has been observed in a welder (38). Phosphine gas may be produced from the heating of rust-proofed metal coatings; manganese is a common component of various alloys; and mercury is used as an antifouling agent in paint. Although the potential for exposure to these toxic compounds exists, there have been no case reports describing pneumonitis in welders caused by any of these three agents. In general, case reports of pneumonitis from welding occur sporadically in the literature and should be viewed as very rare occurrences in this setting. The agent most commonly implicated is cadmium, which should represent the greatest concern.

Differentiating metal fume fever from toxic pneumonitis. Many authors em-

TABLE 3. *Causes of pneumonitis in welding and related technologies.*

Level of Evidence	Agent	Reference	Comment
Case report of toxic pneumonitis in welders	Cadmium	Blejer and colleagues, 1966 (ref. 33)	Acute, fatal pulmonary edema after brazing with silver solder; patient initially attributed symptoms to "welder's fever"
	Phosgene	Sjögren and colleagues, 1991 (ref. 35)	Acute pulmonary edema after gas tungsten arc welding of steel washed with trichloroethylene
	Ozone	Kleinfeld and colleagues, 1957 (ref. 36)	Acute pulmonary edema after gas metal arc welding
	Nitrogen dioxide	Norwood and colleagues, 1966 (ref. 37)	Acute pulmonary edema after oxyacetylene torch cutting of metal
	Beryllium	Monie and Roberts, 1991 (ref. 38)	Suspected chronic granulomatous pneumonitis from welding using a consumable electrode composed of a copper alloy with 2% to 3% beryllium; no case reports of acute toxic pneumonitis from welding
Reports of toxic pneumonitis in other settings; potential for exposure in welding	Phosphine	Waldron, 1994 (ref. 39)	Produced from rust-proof metal coatings containing phosphate
	Manganese	Lloyd Davies and Harding, 1949 (ref. 40)	Constituent of numerous alloys; shown to cause acute pulmonary edema in animals; studies in humans have shown increased prevalence of pneumonia; exposures in welders have resulted in chronic neurologic effects without pulmonary involvement
	Mercury	Doig and Challen, 1964 (ref. 41)	Used as an antifouling agent in some paints; brief mention is made of "metal fume fever" followed by "systemic mercury poisoning" in a man brazing a cylinder coated on the inside with a mercury amalgam

phasize the radiographic differences between metal fume fever and toxic pneumonitis, stating that the chest radiograph is typically normal in metal fume fever (26,34). Nonetheless, an accumulation of case reports indicates that such radiographic changes do occur in a significant minority of cases (32,42–46). Although the timing of chest radiographs is not always stated in these reports, the general pattern is one of transient, diffuse, nodular infiltrates occurring sometime between 4 to 18 hours after exposure.

Functional changes have also been observed in metal fume fever. Hypoxemia (43,46), obstructive changes (44,47), and restrictive changes (46,48) have all been noted. Reductions in the diffusing capacity of carbon monoxide may be the most sensitive functional change in metal fume fever (49). The timing of these changes is similar to that of the radiographic abnormalities, occurring between 2 to 24 hours after exposure.

Bronchoalveolar lavage (BAL) has been performed in subjects with metal fume fever with consistent findings of a marked polymorphonuclear leukocytosis at 20 (49) and 24 hours (46) after exposure. These results are very similar to those observed in cases of toxic pneumonitis.

The case reports and investigations are summarized in Table 4, which compares and contrasts metal fume fever and toxic pneumonitis (adapted from PD Blanc, personal communication, 1996). The available observations suggest that a continuum of clinical presentations exists between metal fume fever and toxic metal pneumonitis, with considerable overlap between the two. Key factors determining the extent of pulmonary involvement include the inherent toxicity of the metal involved and the level of exposure. Low-level inhalational exposure to more toxic metals such as mercury and cadmium can cause a self-limited, flu-like illness indistinguishable from metal fume fever (50). High-level exposure to a less toxic metal such as zinc in one case has led to hypoxemia, requiring supplemental oxygen and a marked micronodular infiltrate that resolved only after 3 days (43).

The clinician faced with acute respiratory symptoms in a welder should ascertain what the most likely exposures are based on a knowledge of the

work practice. If, for example, the welder was working with galvanized steel, metal fume fever is a more likely diagnosis. Conversely, if the history reveals that the worker was brazing with silver solder or cutting sheet metal, close observation is required for at least 72 hours after exposure. Depending on the circumstances and clinical findings, hospital admission may be warranted.

There is clearly a need for further prospective investigations of metal fume fever to clarify the nature and timing of radiographic and functional changes and to identify cofactors that may explain atypical presentations. There are limited data on direct measurements of cadmium in urine and blood in cases of cadmium pneumonitis. For urinary cadmium, levels are well above the normal range and peak several days after exposure (51). However, most of these studies have been performed on samples several days after exposure, when metal fume fever should no longer be a diagnostic consideration.

Welding Fumes and the Respiratory Tract. There have been numerous studies examining respiratory symptoms and pulmonary function among groups of welders, often with contradictory results (1,21). Many have been difficult to interpret because of differences in design, exposure assessment, and attention paid to out-migration from the occupation (1). It is thought that many of the least healthy welders leave this physically demanding occupation. This out-migration may bias cross-sectional surveys, making it more difficult to identify an excess of respiratory symptoms (1).

Studies of shipyard workers in the United States and the United Kingdom have suggested a marked excess of respiratory symptoms such as cough with sputum production, wheezing, and shortness of breath (1). These findings have been associated with age, duration of employment, smoking, past history of respiratory disease, and a history of welding aluminum (52,53). A recent prospective cohort study found statistically significant increased symptoms of work-related cough, sputum production, wheeze, and chest tightness (54). All of these symptoms improved when the subjects were away from work.

Studies of pulmonary function suggest that some welders may experience reductions in airflow, vital capacity, and diffusing capacity, the latter two apparently more likely among welders who smoke (1,52,53,55–57). The degree to which these changes progress after they appear is in some doubt (55), but an effect of welding at least on the flow in small airways seems to be independent of smoking or asbestos exposure (56). There does seem to be an extensive interaction between welding-related exposures and smoking, however, potentiating whatever effect is produced (56). There is also preliminary evidence that, as proposed by McMillan (58), there may be a subgroup of welders at particular risk because of atopy and associated airways reactivity and that these workers are more likely to migrate out of the occupation and be lost to follow-up (59).

The particulate phase of welding fumes, similar to other dusts, may induce mild respiratory irritation and bronchitis. Whether specific agents produced during welding may lead to occupational asthma is less clear, in view of the multiple exposures involved. The evidence implicating nickel is most compelling (60,61). One report suggested an etiologic role for chromium based on the case of a welder with contact dermatitis to chromium salts who experienced airflow obstruction after welding galvanized steel (62). However, other authorities state that chromium metal, in contrast to chromium salts, is nonallergenic and should not be considered a cause of occupational asthma (61). A similar late bronchospastic reaction has been attributed to zinc (62,63). There is mounting evidence that these metals cause occupational asthma through an IgE-dependent mechanism (60,61).

Other potent respiratory sensitizers have been documented, but exposure to these agents is restricted to specialized types of soldering. Agents incriminated as causes of asthma, through non–IgE-dependent mechanisms, in solderers include colophony, aminoethyl ethanolamine, potassium aluminum tetrafluoride, aluminum chloride, and zinc chloride (64). Isocyanates have also been shown to be produced during soldering of polyurethane-coated wires (65). In addition, clinicians should be vigilant for the possibility of reactive airways dysfunction syndrome, which has been described in welders (66).

Although various agents encountered in welding and related technolo-

TABLE 4. *Comparison of features of metal fume fever and toxic metal pneumonitis**

	Metal Fume Fever Prototype: Zinc Oxide	Toxic Metal Pneumonitis Prototype: Cadmium
Onset	Dose related; 4–8 hours postexposure	Dose related; 4–8 hours postexposure
Fever	Sine qua non	Common
Chest radiograph	Normal or fleeting infiltrates	Noncardiogenic pulmonary edema
Arterial blood gases	Normal or minimal transient changes	Marked hypoxemia; widened alveolar-arterial gradient
Complete blood cell count	Polymorphonuclear leukocytosis	Polymorphonuclear leukocytosis
Pulmonary function testing	Normal or transient changes	Frank, restrictive defect
Bronchoalveolar lavage	Polymorphonuclear leukocytosis	Polymorphonuclear leukocytosis
Prognosis	Self-limiting	Life-threatening
Sequelae	?None	Postacute respiratory distress syndrome

* Adapted from PD Blanc, personal communication, 1996.

gies have been implicated as causes of asthma, this disorder is generally an infrequent occurrence in this setting. A recent cross-sectional study estimated that welding fume exposure caused asthma in 1% of the welders ascertained (67). In one of the few prospective cohort studies to address this issue, one new case of asthma was diagnosed in a group of 51 welders followed up for 3 years (54).

It has long been observed that workers in a variety of trades exposed to metal fumes, particularly those of manganese, have increased rates of infectious pneumonia (40). A recent study has confirmed this finding in welders (68). Interestingly, the observed excess mortality from lobar pneumonia in this study was not observed in men beyond the age of retirement, implying a reversible effect.

Chronic Respiratory Effects of Welding

Arc Welders' Pneumoconiosis. In 1936, Doig and MacLaughlin (69) reported on 16 young, healthy British men who had worked only as arc welders. Six of them had radiographic changes reminiscent of miliary tuberculosis or silicosis, and three others had slight interstitial "stippling" on which different radiologists disagreed. In this first article, Doig and MacLaughlin described the condition, declared that it appeared reasonably benign, and correctly attributed the chest radiograph appearance to iron oxide inhalation (69). There was little added to this report for the next dozen years until Doig's second report on this specific entity, in 1948, which pointed out that "arc welders' siderosis," as it was called, was unique among the pneumoconioses in that it may be completely reversible when exposure ceases (70). A distinctly abnormal chest radiograph showing an extensive micronodular infiltrate can gradually become less dense and ultimately may clear completely.

Although the pattern described above is considered to be the "classical" presentation of arc welders' siderosis, there have been extensive changes in technology since that time that have changed arc welders' pneumoconiosis from a relatively pure siderosis to a much more complicated disorder. The earliest workers in the cases described in the 1930s worked with bare rods and used very little respiratory protection. Subsequently, silicate coatings on rods became almost universal. Further refinements added additional sources of exposure to silicate dusts, nonmetallic particles, and asbestos, which occurred in both protective equipment and, up to the 1950s, rod coatings (AT Doig, personal communication, 1981). In the 1970s, it became apparent that the picture of arc welders' pneumoconiosis as an accumulation of ferric oxide (Fe_2O_3) in the lung was no longer adequate to explain increasingly reported observations of respiratory impairment among welders (71,72).

The initial findings of a benign, reversible course for arc welders' pneumoconiosis are explained by the observation that the cause of the radiographically visible shadows differs from other pneumoconioses. In the more common pneumoconioses caused by fibrogenic dusts such as silica and asbestos, the visible opacity is a summation shadow of the dust itself and the fibrosis surrounding the dust in the plane of projection. The opacities in arc welders' pneumoconiosis as described by Doig and McLaughlin (69,70) are projected primarily by the dust itself with little or no fibrosis (73,74). The response to deposition of iron oxide is a comparatively benign process. What fibrosis occurs is minor compared with the exuberant response to silica or asbestos. The iron itself, however, is exceedingly dense to radiographic penetration and projects a shadow that is easily visible. When macrophage activity reduces and disperses the concentration of iron particles in the lung, the opacities appear to diffuse (75,76).

Necropsy material from patients with arc welders' pneumoconiosis is seldom obtained because the condition is rarely life-threatening. In the earliest cases in which lung tissue had been recovered from arc welders, extensive deposition of iron was observed, but little or no fibrosis was visible (73). In the rare cases in which lung tissue has been obtained from modern arc welders with chest radiograph changes, there has been evidence of fibrosis and markers of other exposures such as silica (75,76). The silicate material in welding rods burns off as amorphous silica, not crystalline silica, but crystalline silica is commonly present in various welding-related work such as sandblasting and mold casting. Thus, arc welders' pneumoconiosis and fibrosis associated with welding generally seem to be in transition as a result of changing technology, passing from the "classical" picture of a predominantly dust-related disorder of little functional consequence to a mixed disorder with potentially greater clinical implications (2).

Very little work has been done on the role of high-resolution computed tomography (HRCT) in pneumoconiosis caused by welding. In one recent study, HRCT was performed on 21 arc welders with chest radiographs suggestive of pneumoconiosis (77). In 15 of the subjects, ill-defined micronodules concentrated in the centrilobular regions were observed. Emphysema was seen in seven cases, all of whom were smokers. It is known that HRCT is more sensitive than chest radiography in detecting parenchymal abnormalities in other pneumoconioses, but whether or not this applies to siderosis has not been investigated.

Lung Cancer. A number of studies have indicated the presence of excessive lung cancer risk in relation to fume exposure during welding (78–87). Some of the data were clearly confounded by asbestos exposure and cigarette smoking (88–90). In 1985, Peto (79) reviewed the data available at that time and indicated that the studies generally showed an excess of mortality from lung cancer among welders of 30% to 40% (standardized mortality ratio [SMR] = 130 to 140). Peto offered three alternative explanations.

First, the observed excess risk is not due to welding activity per se but resulted from confounding because of exposure to asbestos and smoking. Although welding itself does not involve the use of asbestos, it has been used extensively in the personal protective equipment supplied to welders and is to be found in the industries from which welders have been studied, such as shipbuilding (80). Furthermore, several studies have shown that smoking rates among welders are higher than those of the general population (81–83). In support of this argument, a case-control study of 391 men with lung cancer found no appreciably increased risk of lung cancer in welders when asbestos and smoking were taken into account (84). However, in another case-control study in Norway, among 176

incident, consecutive lung cancer cases in men, for whom a detailed work history was taken, 28 case subjects had been welders for more than 3 years (odds ratio = 1.9) (85).

A second explanation offered by Peto was that welding fumes do lead to a substantial increase in lung cancer but only after a prolonged latency period of 20 to 30 years after initial exposure. This theory would account for the findings of a cohort study on lung cancer mortality among 3247 welders from Washington State performed by Beaumont and Weiss (86,87). They found 50 lung cancer deaths versus 37.95 expected (SMR = 132) based on U.S. census data as a reference. After allowing for 20 years of latency, 39 deaths by lung cancer were found versus 22.4 expected (SMR = 174). This otherwise well-performed study lacked detailed exposure histories to assess the possible effect of confounders, and an internal case-control comparison was not conducted.

Thirdly, as proposed by Stern, MS welding may pose little or no excess risk but serves to dilute a much heightened risk from SS welding (90). SS welders are exposed to Cr^{VI} at levels that have been shown to be carcinogenic in other industries (5). In contrast, the levels of nickel to which SS welders are exposed are considered to be low relative to other industries in which nickel was believed to have increased the risk of lung cancer (91).

Three studies have recently been performed in Norway among shipyard workers, two among mainly MS welders (92,93) and one among Norwegian boiler-welders who had welded both MS and SS (94). In the first of these studies, an excess of lung cancer was found, with 7 observed cases versus 3.2 cases expected. In this study, bladder cancer was also in excess, with 22 observed cases versus 15.2 cases expected (92). However, when accounting for duration of exposure as a welder, there seemed to be no clear "dose-response" relation between duration of exposure and the occurrence of cancer; the highest excess occurred in the group with an intermediate exposure time.

In the second study, there was a clear excess of lung cancer among 632 MS welders, with 9 cases observed versus 3.6 cases expected (93). There were 6 cases versus 1.6 cases expected in a subgroup of 255 heavily exposed welders. Because there were four cases of mesotheliomas in the whole shipyard population (although none had occurred among the welders), and smoking was not fully controlled for, exposure to asbestos as well as smoking could have confounded these results.

In the last of this series of studies, among 2957 boiler welders, there were 50 observed cases of lung cancer versus 37.5 cases expected; however, 3 cases of mesothelioma were also observed versus 1.1 cases expected (94). Smoking could not be controlled for in this study. In this study population, there were also an excess of leukemias, with 11 observed cases and 6.2 cases expected.

Few studies have been performed on "pure" SS welders. Sjögren (95) identified a group of 234 welders who had welded only on SS for 5 years or more between 1950 and 1965. The smoking habits of this study group were quite similar to the general male population, which was used as a reference. Three cases of lung cancer were found versus 0.8 expected. By including in the study group only subjects who had welded for at least 5 years, dilution of the results with persons at relatively low risk was avoided. Welders were selected who presumably had been exposed to very high air levels of SS welding fumes over a long period. When the same cohort was followed up until 1984, five cases of lung cancer were found versus 2.01 cases expected (96). In comparison to other studies, SS welding exposures were precisely characterized, and latencies for possible exposure-related cancer were accounted for in the design of this study. However, the number of cases was small, three and five cases, respectively, in these two follow-ups (95,96).

Becker and associates (97) performed a cohort study among 1224 SS welders who had used different welding methods. The only subgroup of welders with excess lung cancer was the group who performed SMAW and who had 20 to 29 years since first exposure, with 5 observed cases versus 2.1 cases expected, but in the whole SMAW subgroup, there were 5 cases versus 5.1 cases expected. The major inherent weakness of this study was the short time of observation.

One of the most recent studies was performed by Danielsen and colleagues (94). There was a subgroup of 606 SS boiler welders among whom there were 6 cases of lung cancer versus 5.8 cases expected and 1 case of mesothelioma versus 0.2 expected. However, when accounting for a presumed latency period of 15 years, the excess was reduced to a deficiency of 2 cases observed versus 3.4 cases expected, indicating that there was no excess. Hence, the results of this study do not lend support to the view that SS welding results in a higher risk of lung cancer than MS welding.

A recent meta-analysis of five studies on SS welders and lung cancer was performed by Sjögren and coworkers (98). Adjusting for smoking and asbestos exposure, they found a pooled SMR of 194 (95% confidence interval of 128–292) and concluded that there was a relation between SS welding and lung cancer and that SS welding should be separated from MS welding in the consideration of carcinogenic risk. However, others have argued that the risk of lung cancer in SS welders is no higher than those of MS welders (99,100).

The most significant recent contribution in this area was a study performed by the International Agency for Research on Cancer (IARC), which included both MS as well as SS welders, 11,092 in all (101). Welders from nine European countries participated, recruited mainly during the 1960s and the 1970s. There were 116 lung cancer deaths versus 86.8 expected. There were also 15 deaths of bladder cancer observed versus 7.9 expected and 12 versus 8.6 deaths of cancer of the kidneys, both cancer sites generally considered to be related to smoking. However, there were only 42 deaths caused by nonmalignant disease of the respiratory tract versus 61 expected, which might indicate that tobacco smoking did not play a major role as a cause of death in this study group.

There was a tendency for the SMR for lung cancer to increase with time since first employment both for MS welders, ever SS welders, and predominantly SS welders. The increase with time was most marked for predominantly SS welders than for the other two subcohorts. The authors concluded that there was an excess of mortality from lung cancer in welders but that it bore no relation to either SS welding in particular or duration of employment. Largely on the basis of this study, the

IARC concluded that welding was "possibly carcinogenic to humans (group 2B)" (5).

Thus, despite numerous studies, considerable controversy persists regarding the risk of lung cancer in welders (100,102). Smoking and asbestos exposure have been confounders in several studies and the extent to which these exposures account for the observed excess mortality from lung cancer in welders is a subject of ongoing debate. In most countries, SS welding is a more demanding job than MS welding, hence requiring greater skill. This could contribute to a lower prevalence and intensity of smoking in SS welders, making that subgroup less vulnerable to confounding. Such a difference in smoking habits could possibly mask a more hazardous workplace exposure situation. Further large case-control studies with detailed exposure histories and long-term follow-up are needed to clarify these issues (102).

CONCLUSION

Welders are exposed to a wide variety of potential respiratory hazards. It is therefore somewhat surprising that this trade is not usually associated with extraordinary respiratory morbidity. Nevertheless, as illustrated by the example of siderosis, there is evidence to suggest that, with the development of increasingly sophisticated welding technologies, the spectrum of hazards widens. In the future, clinicians should be aware of the possibility of new hazards as this ubiquitous industrial trade continues to evolve.

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