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RESEARCH PAPER

Arc welding of steels and pulmonary fibrosis

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Abstract This paper summarises the increasing epidemiological and experimental evidence of the causal link between exposure to high concentrations of welding fume exposure for prolonged periods of time and the subsequent development of pulmonary fibrosis in a relatively small number of people. It is not yet clear which components of welding fume or gases are the cause, but the most likely culprits are the soluble transition metals which may cause the formation of free radicals. The most likely work scenario leading to pulmonary fibrosis due to welding fume is of high fume exposure, without effective local extraction and respiratory protection, in confined spaces for long periods of time. Avoidance of high exposures for long periods of time is required to prevent this condition. For clarity, the term "pulmonary fibrosis due to prolonged exposure to welding fume at high concentration" is suggested when there is shown to be a causal link in an individual, and we recommend that the terms siderofibrosis and arc welder's lung are abandoned.

Keywords (IIW Thesaurus) Welders health · Fume · Arc welding · Pulmonary fibrosis

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1 Introduction

The purpose of this document is to provide an up-to-date review of the epidemiological and experimental evidence linking prolonged steel welding fume exposure at high concentration and the subsequent development of pulmonary fibrosis. A previous paper in *Welding in the World* [1] has reviewed the evidence for airway disease in welders, the general nature of welding fume and provided generic guidance on the control of fume [Box 1] and complements this paper.

Box 1. Recommendations from Commission VIII on control of fume and the prevention of airways disease in welders [1]

- Systems of work are employed that will minimise the fume and gas exposure of the welder and others in the workplace, and, if possible, welding in confined spaces is avoided if technically feasible;
- Welding processes and procedures that produce lower amounts of fume emissions are used wherever possible;
- Surface coatings are removed prior to welding;
- The welders keep their head out of the welding plume;
- Local exhaust ventilation is used when arc welding in order to keep the level of fume exposure as low as reasonably practicable and at least to national standards. Local exhaust ventilation reduces background levels of fume and protects others in the workplace. It is always desirable and may be needed even if an air-fed helmet is used to reduce levels to acceptable standards;
- Local exhaust ventilation is placed in close proximity to the workpiece when welding and grinding, including when undertaking TIG welding;
- Local exhaust ventilation is continued after the completion of the welding to clear the remaining welding fume and gas;
- The welder uses an improved helmet (a helmet with an apron to reduce fume and gases from getting under the helmet), rather than a standard helmet as a minimum but ideally uses a purified air-fed helmet;
- The welder uses a technique of counting to five at the end of the welding process before lifting an air-fed helmet to allow the fume levels to reduce before breathing ambient air containing welding fume. This technique is not appropriate for standard or improved helmets;

- If the welder needs to do any grinding in the area, local exhaust ventilation and a suitable dust mask are used;
- Welders are strongly advised not to smoke as this exposure acts with welding fume to cause more damage than would be the case with welding fume or smoking alone.

Once we have defined some terms commonly used to describe pulmonary fibrosis in welders, we will go through the historical background and describe the experimental, radiological and clinical evidence currently available. We will describe a typical case of pulmonary fibrosis due to welding fume and provide guidelines regarding appropriate clinical investigation and occupational management. Finally, we suggest key topics for further research and areas needing the development of international consensus.

Excluded from this review are the medical causes of pulmonary fibrosis and pulmonary conditions following exposure in welders to short-duration very high-dose exposures to welding fume and gases; aluminium; tungsten and cobalt; and asbestos and crystalline silica.

1.1 Definitions

Pneumoconiosis is a medical condition caused by dust in the lungs, for instance, as evidenced by X-ray, computerised tomography (CT) scan or histology, and may be for example due to exposure to silica, mixed dusts, metals [2–4], or organic matter. It is modulated by host factors such as genetics, the immune response and smoking [5].

Pneumoconiosis due to welding fume includes the following terms:

- *Siderosis* which is evidence of iron in the lung on a chest X-ray or histology without fibrosis;
- *Siderofibrosis* which is the development of fibrosis within the lungs as a result of exposure to iron as evidenced by histology;
- *Pulmonary fibrosis due to welding fume* where there is histological proof of fibrosis that is thought to be due to welding fumes or gases (but not necessarily due to iron).

Whilst siderosis is common, amounting to a significant percentage of the workforce in many studies, particularly in older workers, the development of symptomatic pulmonary fibrosis due to welding fume is very rare, in the order of 200 cases reported in the world medical literature over the last 75 years.

In the past, the term "arc welder's lung" was used in a non-specific way and has been used to describe welders with pulmonary fibrosis but could also cover non-fibrotic responses of the lung to welding fume.

2 Historical background

Following the introduction of welding technology, it became apparent that some arc welders who weld steels developed abnormal chest X-rays due to the iron content of the fumes [6]. Debate followed about whether iron from welding fume might result in pulmonary fibrosis [7–9], and by the early 1960s, the conclusion in the UK [10] and by some in the USA [11] was that this was unlikely to be the case. Subsequent mortality studies have not shown an increased rate of death from either pneumoconiosis or pulmonary fibrosis in welders [12–17].

Nevertheless, there has been long standing concern in Germany [18, 19], Japan [20, 21], Eastern Europe [22, 23] and by some in the USA [24–27] that some compounds in the fumes and gases might cause pulmonary fibrosis with high cumulative exposures.

Recently, the German government has prescribed pulmonary fibrosis due to prolonged exposure to welding fume at high concentration as an industrial disease [28], and this action prompted us to review the evidence and write this review.

3 The evidence that welding fume can cause pulmonary fibrosis

3.1 Experimental evidence

Many animal studies of welding fume exposure have been undertaken using both direct instillation of welding fume into the lungs of rodents [29–34], inhalation studies using fresh welding fume [29, 35–41] and in vitro studies of rodent macrophages [42, 43].

Once deposited within the lungs, the insoluble fraction, the "spinels" comprising complex iron-manganese oxides, will remain until removed by phagocytosis or expectoration, but the soluble fraction will dissolve in the macrophages and alveolar tissues and will steadily be carried out of the lungs in the blood and lymphatics. Soluble metals in welding fume include chromium and nickel in stainless steel and manganese that is not bound into spinels in mild steel. It is thought that the most reactive part of the fume is the soluble fraction [42], which may cause free-radical production within the tissues of the alveolus. Manual metal arc (MMA) welding gives rise to a greater amount of soluble fume than metal inert gas or metal active gas welding (MIG/MAG) [33]. Whilst fume contains silicates, these are amorphous and not crystalline and therefore do not pose a risk of fibrosis.

Following sacrifice of experimental animals exposed to welding fume, it is possible to show the sequence of events

in the alveoli and bronchioles [44, 45]. These changes are similar to those found in humans with pulmonary fibrosis due to welding fume and are more marked with exposure to stainless steel than mild steel welding fume [31, 33, 37, 41, 46] and more marked in MMA than MIG/MAG [33]. Initially, welding fume deposits in the alveoli and bronchioles and is phagocytosed by macrophages and after a few days by polymorphonuclear cells. Such cells may move up into the bronchi to be expectorated once they have reached the top of the mucociliary escalator. Macrophages may accumulate in the alveoli as a result of persistent levels of welding fume exposure. As the macrophages become overwhelmed by fume, more macrophages are recruited to ingest the fume, some of which pass into the tissue of the alveoli and the interstitial tissue and some of which die in the alveolar air space. Some of the welding fume and macrophages pass into the lymphatic system to be removed from the alveolar interstitial tissue. Deposits of macrophages and welding fume particles increase around the bronchioles and arterioles. With time, fibrosis may develop within these deposits, which may distort the adjacent bronchioles, either leading to narrowing of the small airways or a traction dilatation. The lung demonstrates patchy areas of fibrosis. Areas of emphysema may develop in the peribronchial areas partly as a result of traction on the alveoli from adjacent areas of fibrosis. The ability of the lung to recover and the risk of development of pulmonary fibrosis depend on the cumulative exposure to fume, that is, the product of dose and duration [45, 47].

3.2 Radiological evidence

Several studies have described the radiological investigation of welders' chests including chest X-ray and CT. The chances of developing radiological evidence of siderosis gradually increase with duration of exposure, lower exposure showing a delayed onset of the condition [48, 49].

Studies of CT scans of welders recruited from the workplace show changes of poorly defined centrilobular micronodules and branching linear structures [50, 51]. These changes reflect iron deposition in the airways but do not reflect changes within the tissue of the lung (unlike silicosis and coal worker's pneumoconiosis). Welders who have been referred for CT as a result of positive chest X-rays taken during health surveillance programmes show similar changes with the addition of occasional ground-glass opacities [52], occasional mild fibrotic changes and emphysema [53]. In the radiology literature, symptomatic welders who have been referred for clinical assessment, however, show the addition of the following: emphysema in smokers, "ground-glass opacities"; features of usual interstitial pneumonia such as "honeycombing"; and conglomeration and bronchiectasis [54, 55]. There is no histology reported in these papers.

3.3 Histological evidence

In total, there are over 200 cases of histologically proven pulmonary fibrosis thought to be due to welding of steels in the world medical literature over the last 75 years.

Individual case reports can be classified into three groups: those where pulmonary fibrosis was related to welding fume exposure [56–74], those where it was related to an occupational exposure other than welding fume and those where there is most probably a clinical reason for the pulmonary fibrosis. Finally, in some pathology samples, no or minimal fibrosis was found [8, 75–77]. There are additional occasional reports of localised collections of welding fume within the lung tissue and the development of conglomeration/progressive massive fibrosis [78–81].

One of the difficulties with these historical case reports is that the classification of pulmonary fibrosis has changed significantly over the years and therefore it is difficult to know, without thorough multidisciplinary retrospective review of the cases using modern criteria and methods, whether they all represent pulmonary fibrosis due to welding fumes or whether some of them are in fact pulmonary fibrosis in a person who happens to be a welder but where there is no causal connection between the occupation and condition.

Several case series of welders with histologically proven pulmonary fibrosis have been published [8, 19, 22, 26, 27, 76, 77, 82–94], the most detailed of which is a series of 15 welders by Buerke et al. [95]. Buerke et al. came to the same conclusion as others that the cases were related to high exposure to welding fume for a prolonged period of time. Cumulative exposure estimates in the study by Buerke et al. ranged from 115 to 4350 mg years m^{-3} with a median exposure estimate of 221 mg years m^{-3}. All were MMA welders, but some also undertook MIG, MAG or tungsten inert gas (TIG) welding. The majority undertook confined space welding. Only five were exposed to chromium and seven to aluminium. Five were non-smokers.

In contrast to the radiological papers, honeycombing is not mentioned in any of the case reports or series with histology. Many papers include comments about associated emphysema in addition to the fibrosis, either clinical [26], radiological [57, 74, 80] or pathological [56, 58, 60, 73], but emphysema may be more related to the effects of cigarette smoke than welding exposure [96].

Some papers describe iron overload as a result of the high cumulative exposure to iron fumes [53, 65, 67, 80, 94], and there are several case reports of cor pulmonale (right heart failure) [27, 57, 63, 95].

3.3.1 Pathological grading of pulmonary fibrosis due to welding fume

A proposed pathology grading has been suggested by Müller and Verhoff [91]:

- Mild pulmonary fibrosis due to welding fume shows alveolar and interstitial macrophages but has no fibrosis in the dust deposits.
- Moderate pulmonary fibrosis due to welding fume demonstrates an increased number of macrophages with significant fibrosis in the dust deposits.
- Severe pulmonary fibrosis due to welding fume shows heavy fume deposits, chronic fibrosis with focal accentuation and a close association between fibrosis and deposits.

Buerke et al. [93] indicated that welders with severe disease had a cumulative exposure level in the order of 200 mg years m^{-3} and welders with moderate disease of 115 mg years m^{-3}.

4 Pulmonary fibrosis in welders

Pulmonary fibrosis in welders is not necessarily due to exposure to welding fume or gases as welders are also subject to clinical conditions unrelated to workplace exposures. The most common clinical condition is idiopathic pulmonary fibrosis (IPF).

Epidemiological research has shown that IPF and usual interstitial pneumonia (UIP), the most common histological pattern seen in IPF, are significantly more common in persons exposed to metal dust but are not more common in welders than controls [97–102]. Furthermore, the case series and case reports of welders with pulmonary fibrosis in the literature where there is histology contain no definite cases and only one case suggestive of IPF [24]. The two case reports of IPF in a welder both had only a radiological diagnosis and no histology [54, 103]. Therefore, a welder who has a diagnosis of IPF/UIP can be reassured that it was not the welding fume that caused the condition.

In contrast, desquamative interstitial pneumonia (DIP) and respiratory bronchiolitis lie on a continuum and share many features with the histology described in individual case reports and case series of welders [81, 83, 85]. These conditions, particularly DIP, have been linked with particle exposure including welding in some studies [104–106]. Given the changes in classification of the clinical conditions, it is now appropriate to review the link with welding fume for welders with pulmonary fibrosis to ascertain whether fibrosis due to welding fume is a separate condition to this continuum or whether in fact these are the same condition [96].

Finally, a few cases of pulmonary fibrosis in welders in the literature are related to exposure to crystalline silica in addition to welding fume [24, 59, 76, 77, 89] or asbestos [82, 85, 89, 107, 108]. Without formal assessment of these compounds within the areas of fibrosis, it is not possible to make the diagnosis and it is easy to miss the diagnosis if there is not a separate mineralogical analysis.

5 Assessment of the welder with pulmonary fibrosis

The assessment of a welder who is suspected of having pulmonary fibrosis should be undertaken by a multidisciplinary team experienced in both interstitial lung disorders and pneumoconiosis. The assessment should be undertaken to national guidelines and will normally include clinical history; examination; spirometry; carbon monoxide transfer factor; transferrin, an exposure assessment by an occupational hygienist or occupational physician and CT of the chest.

Lung biopsy is a highly invasive intervention and should not be undertaken to confirm the diagnosis of pulmonary fibrosis due to welding fume unless there are sound clinical reasons for undertaking the procedure and the risks are significantly outweighed by the benefits. Ideally, an open lung biopsy rather than a transbronchial biopsy is required, but the decision should be dictated by the clinical situation and access to appropriate expert advice. If a lung biopsy is clinically indicated, histological analysis should be supplemented by mineralogical assessment of lung tissue to assess welding fume, iron, silica and asbestos.

The welder will need clinical follow-up, as the diagnosis may need to be revised in the light of changes in the health of the welder with time.

Occupational management will depend on many factors but may require the removal of the welder from exposure, or at the very least, scrupulous use of effective local extraction together with a powered air-purifying respirator to limit the exposure to welding fume to less than 1 mg m^{-3}. A formal assessment of the other members of the workforce should automatically be undertaken by the occupational health/occupational hygiene team and a full review of the control of fume within the workplace and action taken to reduce the exposure levels to at least national standards by management and the occupational hygienist.

5.1 A typical case of pulmonary fibrosis due to steel welding fume

The typical clinical history for a case of pulmonary fibrosis due to welding fume will be as follows. The welder may complain of dyspnoea (shortness of breath) and less frequently a cough or may be a symptom-free welder with an abnormal chest X-ray. They may or may not be a smoker. Exposure

assessment would be expected to find that the welder was exposed to high levels of fume, for a significant duration, often in confined spaces without using effective extraction or respiratory protection. On examination, the clinician will very rarely find evidence of clubbing but may find crackles at the bases. Spirometry may be abnormal and may demonstrate obstructive, restrictive or mixed changes. The residual volume is likely to be raised. Transfer factor may be significantly reduced. Chest X-ray findings do not correlate with clinical findings but may occasionally demonstrate conglomeration or progressive massive fibrosis or evidence of pneumoconiosis related to iron exposure. High-resolution CT scans may show ground-glass opacities in symptomatic welders, but such changes are not usually found in asymptomatic welders. Histology of the lung, should it be undertaken, shows alveolar and interstitial macrophages and pulmonary fibrosis, as described above. Areas of patchy pulmonary fibrosis are strongly correlated with welding fume deposition on mineralogical analysis of the histological specimen, but there is no evidence of the presence of crystalline silica or asbestos. Once the welder is withdrawn from exposure, the condition does not, as a rule, progress in simple fibrosis due to welding fume.

6 Further research

Several areas of further work are required on this topic:

6.1 How do we best define and diagnose pulmonary fibrosis from welding fume and how do we distinguish it from other occupational and clinical causes?

There is a need for further international discussion regarding the existence of pulmonary fibrosis due to welding fume as a diagnosis with the development of consensus on the appropriate case definitions and distinguishing features of clinical and occupational causes of pulmonary fibrosis in relation to welding fume. The logical starting point would be the current German model [28]. Given the rarity of the condition and the difficulties in making the diagnosis, it may be that international reference histopathology laboratories dealing in pulmonary fibrosis could be established that could lead in both histological and mineralogical assessment and research in this area.

6.2 What is the aetiology of pulmonary fibrosis in welders?

There are many potential agents that could cause pulmonary fibrosis in welders including components of the fume (complex spinels of iron manganese oxides, soluble hexavalent chromium, titanium or manganese compounds) and gases (oxides of nitrogen, ozone) [49]. Other than hexavalent chromium, it is far from clear what the most likely cause of pulmonary fibrosis in welders might be, but hexavalent chromium does not explain all of the cases. It is not likely to be the iron-manganese oxide spinel which may simply be a marker of exposure. It is most likely to be a soluble element of the fume. It has been suggested that one component of the gases, particularly oxides of nitrogen, may play a part; however, this would not explain the close relationship with particles in the lungs and surrounding fibrosis and exposure to nitrogen dioxide results in a different clinical presentation of disease and pathology, namely bronchiolitis obliterans. Only one case in the literature is of this histological pattern [68]. Further research on the material science of the fume and gases itself rather than concentrating on purely the elemental composition of fume is likely to be rewarding. Such research needs to consider the potential synergistic effects of both the fumes and the gases produced in welding and the changes in the fume oxidation state and surface charge with time.

6.3 What is the interaction of welding fume and host factors such as smoking in the generation of pulmonary fibrosis?

With regards to host factors, firstly there is likely to be a synergistic effect of smoking and welding fume in the genesis of pulmonary fibrosis. Smoking is a significant risk factor for pulmonary fibrosis and is a very common habit among welders. Secondly, if emphysema is present in addition to pulmonary fibrosis, is this related to smoking or welding fume, or is there an interaction between the two? It is thought unlikely that genetics will turn out to be a major component, but with the current rapid progress in our understanding of the interaction between genes and occupational exposures in the genesis of occupational disease, it is wise to keep an open mind about this.

6.4 Does pulmonary fibrosis due to welding fume increase the subsequent risk of infection?

Many of the case reports and case series make mention of tuberculosis (TB) [22, 24, 26, 48, 50, 52, 56, 66, 76, 77, 82, 109, 110] although early commentators thought that there was no link between pulmonary fibrosis due to welding fume and the subsequent development of TB [24]. Given that other forms of pulmonary fibrosis due to occupational exposures such as silicosis are associated with TB and that there is an adverse effect of welding fume on macrophage function, it would not be a surprise if pulmonary fibrosis due to welding fume increased the risk of reactivation of latent tuberculosis. Whether this might be related to pulmonary fibrosis or macrophage dysfunction is worth investigating.

It is well recognised that pneumonia is more common in welders [111], half of the cases being from pneumococcal disease which can also present as meningitis [112]. The risk

of pneumonia falls to background levels on cessation of welding fume exposure [113] and therefore cannot be related to underlying pulmonary fibrosis.

6.5 Does pulmonary fibrosis in welders account for the unexplained increased risk of lung cancer?

There is an epidemiological link between welding and lung cancer that remains unexplained [114–116]. Pulmonary fibrosis (including respiratory bronchiolitis), also increases the risk of lung cancer [117, 118] although the link may not be causal. There are very few cases of lung cancer described in the current literature in cases of pulmonary fibrosis due to welding fume [76, 77, 85, 95], but this may be due to lack of follow-up. This warrants further investigation, firstly by following up welders with known pulmonary fibrosis, secondly by formal assessment of lung cancer specimens in welders looking for evidence of fibrosis such as respiratory bronchiolitis in the surrounding non-cancerous tissue and thirdly factoring in lung fibrosis and cumulative exposure estimates to the epidemiological studies on lung cancer and welders.

6.6 Nomenclature

Finally, it is suggested that the term siderofibrosis is abandoned for the sake of consensus and to avoid confusion, as it remains contentious as to whether iron plays any part in the development of fibrosis. Likewise, arc welder's lung is a non-specific term and should no longer be used. We prefer the descriptive term "pulmonary fibrosis due to prolonged exposure to welding fume at high concentration".

7 Conclusion

Arc welding of steels is associated with occasional cases of pulmonary fibrosis when the welder is exposed for a significant period of time and at high concentrations. We would like to reinforce the advice already provided to the welding industry on careful control of welding fume to minimise exposure at the workplace, particularly in confined spaces, and strongly reinforce the advice already provided by Commission VIII that welders should not smoke.

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