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RESEARCH PAPER

Arc welding and airway disease

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Abstract Fume from arc welding has long been suspected to cause various lung diseases. This paper, developed by Commission VIII of the International Institute of Welding (Health Safety and Environment), summarises the epidemiological evidence for fume and airway disease including asthma, chronic obstructive airway disease and accelerated loss of lung function. Whilst the epidemiological evidence for the link between these conditions remains limited, a precautionary approach is recommended by Commission VIII and clear guidelines regarding control of fume are given.

Keywords Arc welding · Fume · Gases

1 Introduction

The purpose of this document is to provide background information to enable welders, welding engineers and safety and occupational health staff to understand the epidemiological research on the potential adverse effects of welding fume on the welder's airways. It does not contain information on animal or human toxicology as this is well described by the reviews of Antonini [1-3] but aims to provide detailed background information on the difficulties and pitfalls of undertaking and interpreting epidemiological research on this topic together with an overview of the findings of existing epidemiological research. A separate Commission VIII review of pulmonary fibrosis in welders is currently underway and will be published in the future.

Welding fume is the condensation product of the gas and vapour formed when metal is welded, mostly using a filler, and is a mixture of the metal(s) being welded, the shielding gas, the consumable filler and the products of the chemical reactions occurring as a result. Most is derived from the consumable. Components of welding fume are potentially toxic and include metal oxides (such as complex oxides of iron, aluminium, chromium, nickel, and manganese) and inorganic compounds (such as fluorides and noncrystalline silicates). In addition, gases (such as ozone, oxides of nitrogen and carbon monoxide) and degradation products of organic coatings (paints, plastics and oils) may be present with the particulate fume. The respiratory exposures of welders depend on the type of welding undertaken, the base metal, the constituents of the consumable, the frequency and duration of welding, whether the welding is undertaken in an open or closed environment and whether respiratory protection and or local exhaust ventilation is used.

Whilst most of the fume produced by mild or carbon-steel welding is a complex oxide (spinel) of iron (80–95 %) and manganese (1–15 %), a number of other metals and inorganic compounds may also be present. The gases produced during welding, carbon monoxide, ozone, nitrogen oxides, may reach very high concentrations in the vicinity of the welding plume but may also form and accumulate away from the welding area. The particles in welding fume are predominantly in the ultrafine size range with a close correlation between ultrafine and total particle concentrations. Standard welding helmets may [4] or may not [5] reduce the level of fume that the welder is exposed to but cannot be relied on as a method of controlling fume exposure.

Lung function can be measured in many different ways, and for the nonmedical person, the nomenclature can cause considerable confusion. Standard lung volumes are available

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for different populations, and the values are affected by ethnicity, gender, height, weight and disease. The most reproducible and commonly used lung function value in the medical literature is the volume of air that can be exhaled forcibly in 1 s after maximal inhalation and is known as the FEV_1. In the best hands, between-test-variability of FEV_1 is less than 40 ml. The effect of improving technique with repeated measurement is an increase in FEV_1 of 33±20 ml [6]. There is diurnal variation in lung function with peak values at noon. Overall, the FEV_1 rises from baseline by a mean of 80 ml between 9:00 a.m. and midday and then falls back to baseline levels by the evening. This variation is greater in younger people and smokers and less in older people and nonsmokers. Likewise, the total amount of air that can be forced out of the lung after maximal inspiration, the forced vital capacity (FVC), increases by 200 ml between morning and midday and then falls slowly during the day. Again, this variation is greater in smokers, but in contrast to FEV_1, it is greater in older people than younger ones. Variation in both FEV_1 and FVC is greater in those who have chest symptoms [7].

There are various factors that affect normal lung volumes. Firstly, lung volumes change with time: they increase with age until around the age of 25 at which point they peak and then fall naturally with age. The loss in FEV_1 at age 25 is around 25 ml/year, and at age 75, the normal loss has increased to 60 ml/year [6]. Lung volumes in persons under 25, therefore, will be increased with age and decreased by disease, and this can make interpretation difficult. After the age of 25, the tendency will be for both disease and age to cause a decline in lung volumes: assessing the effect of a potential respiratory toxin on lung function will, therefore, involve looking for an accelerated decline in lung function compared to others at the same age.

Secondly, the subject needs to fully understand how to perform lung function testing and the person's technique needs to be carefully mastered in order for there to be appropriate interpretation. Even then, the measured values tend to improve with experience and training.

Thirdly, smoking tobacco adversely affects lung function. In smokers, the overall loss of FEV_1 is increased from 70 to 80 ml/year [8] but often, there is no detectable difference in FEV_1 between smokers and nonsmokers until the age of 40 [9].

Finally, the effect of a reduction in airway calibre on air flow is given by $\text{Flow}=1/r^4$. Therefore, a small reduction in the diameter of the airway has a large effect on the ability to move air up and down that airway into the gas-exchanging part of the lung, the alveoli. Consequently, a relatively small constriction of the airway due to disease may have a significant effect on symptoms and lung function values.

1.1 Lung disease

It is important for the nonmedical reader to understand that clinical medicine is not an exact science and that clinicians are subject to variations in their labelling of conditions—diagnosis. Indeed, diagnostic labels in lung disease may be applied by different researchers in different ways and this is particularly true of occupational lung disease. For instance, a history of wheeze may be labelled as asthma, bronchitis or reactive airway disease by different researchers and the existence of welder's siderofibrosis as a clinical entity is accepted in some countries but refuted in others.

1.2 Study design and interpretation

There are many research papers on welding fume and lung function. The published studies have a variety of methodological problems which make generalisation and interpretation difficult as a result of problems with exposure assessment, measurement and interpretation of lung function assessment and challenges of undertaking epidemiological studies.

1.3 Exposure assessment

Assessment of the level of exposure is a key part of epidemiological research but is a concern in most research in this area. In particular:

- Most papers are written without the support of a multidisciplinary team to inform the nonmedical aspects of the research.
- Many papers do not make clear what is meant by the term welder nor do they provide details of exposure to welding fume and gases.
- Additionally, some papers include other professions who work with the welders.
- The selection of controls is a problem in many studies with controls being used that are also exposed to fume or other respiratory toxins or are substantially different in basic characteristics from the welders.

1.4 Measurement and interpretation of lung function

- Lung function measurement is not an exact science, and it is often difficult to distinguish changes in lung function from confounding factors such as normal physiological variability.
- There is an assumption that welders have the same normal range of lung function as the controls or general population at the start, but this may not be the case.
- There is a significant training effect with regard to use of a spirometer, and therefore, it is probably best to exclude the first group of results when establishing a baseline. This may account for some studies demonstrating an improvement in lung function in welders with exposure.

- Spirometry provides the researcher with many different values, but not all are as easy to interpret as the FVC and FEV_1.
- It is possible [10-14] that there is a cross shift change in lung function, and therefore, in any research study of long-term effects on lung function, formal assessment of lung function should be undertaken either prior to work or after inhalation of a beta 2 agonist to reverse any changes due to acute exposure.

1.5 Challenges of undertaking epidemiological studies

- Many studies have been undertaken in turbulent economic times resulting in loss to follow-up, in particular in welders, which makes interpretation of data difficult.
- A difficult problem is whether welders or controls with asthma are excluded from the study or included and if so, whether they are treated as a separate group.
- Young welders will have increasing lung volumes up to the age of 25, and therefore, this needs to be taken into account when undertaking research in apprentices.
- There is evidence that apprentice welders who develop problems with chest symptoms leave the training programmes, with the result that the people left at work are the symptom-free, self-selected, healthy survivors [15-17]. This “healthy worker effect” is a problem for all research in this area and needs to be taken into account by following up people who have left the workplace.
- Finally, particularly in developing countries, infection with tuberculosis can be a confounding factor and needs to be controlled for where this might be a significant problem.

2 Effect of welding on lung function and lung disease

2.1 Asthma and welding

Several publications report cases of asthma as a result of welding. The apprentice welder studies by El Zein [15] and Beach [17] indicate that early exit from a career as a welder may occur as a result of welding-induced asthma in a small number of apprentices, (in the order of 1–3 %). Additional case reports, for instance from the SHIELD reporting system [18], indicate that asthma in response to welding fume may occur during employment as a welder and that this condition is not confined to apprenticeship. There does not appear to be a difference in the incidence of asthma between stainless steel and mild steel welders [19], and the 1997 review of stainless steel welding and asthma by the UK Health and Safety Executive determined that there was not enough evidence to specifically label stainless steel welding fume as a cause of asthma under the European Union criteria for respiratory sensitisers [20]. The welder with occupational asthma may develop an asthmatic response to one type of welding but not others [21-23].

Known causes of asthma that may be of relevance in welders include nickel and chromium [24-26], but skin tests for allergy to metals are negative in most [27-30] but not all [31-33] studies. Some have suggested that gases (such as ozone or nitrogen dioxide) rather than the particulates may be the cause of some cases of asthma in welders [34], but other case reports cast doubt on this [22, 23]. It is clear, therefore, that occupational asthma due to welding is not a homogeneous clinical entity, and distinguishing asthma from irritant effects can be difficult [35-37].

The longitudinal study by Lillienberg [38] using a health questionnaire indicated that the long-term respiratory problems of established welders were due to chronic obstructive pulmonary disease (COPD) rather than asthma, but there is a problem with the medical diagnosis of asthma and COPD which overlap in both symptoms and to a certain extent signs and investigatory findings. The two conditions can coexist. Put simply, the COPD is the irreversible element of the airway disease and the asthma the reversible part. Population reporting or work questionnaire surveys are not a robust mechanism of ascertaining the prevalence of asthma on the basis of wheeze or cough or even a medical diagnosis of asthma. The only robust mechanism of making the diagnosis of occupational asthma due to welding is to undertake a respiratory challenge test in the laboratory to assess changes in lung function in a controlled environment in which the subject is exposed to welding fume. It is then incumbent on the clinical investigator to elucidate, if possible, which component of the welding fume or gases is the cause of the asthma.

A single acute exposure to various agents may cause reactive airway dysfunction (RADS) which is an irritant-induced airway condition. This has been reported in welders [39, 40], but a recent systematic review indicated that there is no conclusive evidence that RADS is caused by welding fume [41].

2.2 Large airways—cross-sectional studies of large airway function

There have been many cross-sectional studies of lung function in welders. The advantage of such studies is that they are relatively easy to undertake, but they have the disadvantage that they are more likely to be biased or unable to control for confounding than longitudinal studies and do not measure change with time. Many cross-sectional studies have indicated reduced lung function in welders [42-54], but some have not demonstrated any difference [14, 55-65]. A few papers have reported on welders who have never smoked, thereby removing the confounding adverse effect of smoking on lung function. Those that have studied only welders who have never smoked have demonstrated evidence of pulmonary changes when compared with nonsmoking controls [51, 53, 66, 67]. Metal fume fever is associated with subsequent lung symptoms but not with changes in lung function on follow-up [68].

2.3 Large airways—longitudinal studies of large airway function

To date, ten longitudinal studies [15, 69-77] have been published on exposure to welding fume and decline in lung function with time. A systematic review of the suitable longitudinal studies published up to 2011 has been undertaken by Szram et al. [78]. The meta-analysis found that annual rates of decline in FEV_1 are greater in welders than in workplace controls and especially so in welders who smoke, but none of the pooled differences reached statistical significance. The pooled difference in annual decline in lung function in the five studies that were suitable for meta-analysis was a 9 ml/year greater decline in welders compared to controls (95 % CI −22.5 to +4.5 ml/year; $p=0.193$), and the pooled difference between welders who smoked and welders who did not smoke was 13.7 ml/year greater loss of FEV_1 in the welders who smoked (95 % CI −45.9 to −1.7; $p=0.179$). There was no difference between welders who did not smoke and controls (a loss of −3.8 ml/year in the welders greater than controls with a 95 % CI of −20.2 to 12.6; $p=0.650$).

2.4 Small airways and welding

Several cross-sectional [43, 45, 47, 49, 56, 57, 59, 61, 63, 64, 66, 67, 79-83] and four longitudinal studies [69, 70, 74, 76] have assessed small airway function in welders mainly as part of wider studies investigating the potential effect of welding fumes in the lung. This is technically more difficult to do and more difficult to obtain a valid result. Most studies have found an effect on small airway function in welders [43, 45, 47, 57, 59, 67, 69, 70, 76, 80, 81, 83] although some have not [49, 56, 61, 63, 64, 66]. With regard to welders who have never smoked, most have not found any adverse effect on small airways [49, 56, 57, 59, 61, 66, 76, 83] although a few have [47, 67, 69, 81]. There is, however, universal agreement in the literature that smoking has an independent adverse effect on small airway function and that welding increases this effect.

When welding fume is inhaled, it deposits in the distal airways and if not expectorated is taken up into the lung tissues surrounding the small airways. Such deposits of welding fume may remain in the tissues around the small airways causing fibrosis and distortion, and this may reduce the small airway calibre. This type of fibrosis also occurs in smokers and is then known as respiratory bronchiolitis (RB). With high cumulative doses of welding fume, more than 100 to 200 mg m^{-3} years [84, 85], significant pulmonary fibrosis may occur and is usually of a type known as desquamative interstitial pneumonia (DIP) [86, 87], a condition that is known to occur in other occupational exposures [88]. DIP and RB are related conditions. Pulmonary fibrosis in welders is currently the subject of a review by Commission VIII.

2.5 Potential causes of loss of lung function in welders

There are a number of possible reasons why welding might potentially cause an accelerated loss of lung function in welders, apart from tendency of welders to smoke tobacco:

- Long-term welding fume exposure may result in overload of the protective mechanisms within the lung together with inflammation. This may lead to obstructive airway disease.
- Welding fume particles may cause obstruction of the small airways by inducing fibrosis around and distortion of the small airways.
- Welding processes produce gases such as ozone and oxides of nitrogen which may have a toxic effect on the lung.
- Historically, welders have been extensively exposed to asbestos in the workplace which may cause pulmonary fibrosis.
- Loss of lung capacity may arise as a result of infective episodes, in particular, pneumonia, which is more common in welders [89-92].
- Finally, there is a synergistic toxic effect on the lung of a combination of tobacco smoke and welding fume which results in an accelerated loss of lung function [78].

2.6 Relationship to exposure

Total welding fume exposures in the developed world have gradually declined with time as the effects of welding fume have been recognised and controlled. It is apparent reviewing the medical literature that exposure assessment has been lacking and a failure to provide even basic information in the paper about the type of welding being undertaken hampers efforts to stratify the results of the cross-sectional studies.

Few longitudinal studies reported detailed measurements of exposure, but the paper by Christenssen, which included an exposure matrix derived from occupational histories and validated with air sampling [75], failed to find a relationship between cumulative welding particulate exposure (range 0–42 mg m^{-3} years) and decline in FEV_1. It is to be noted that this is considerably below the cumulative exposure required to cause moderate or significant pulmonary fibrosis [84].

In contrast, longitudinal studies that have assessed the effect of local exhaust ventilation and personal protective equipment have found a beneficial effect. Erkinjuntti-

Pekkanen and colleagues [74] reported that welders with no personal or environmental protection (mask, air purifier or local exhaust ventilation) had a significantly steeper decline in lung function, especially if they were smokers in comparison to nonsmoking, protected welders where there was no evidence of a significant decline in lung function. Similarly, in a study of British welders [70], the constant use of local ventilation was associated with a smaller decline in lung function.

3 Recommendations

Whilst it is difficult to come to any firm conclusions on the basis of the available epidemiological evidence as to whether exposure to welding fume and gases does or does not cause an accelerated decline in lung function, it seems sensible to take a precautionary approach. There is a consistent adverse effect of smoking and welding in all studies on lung function and also evidence of a benefit of using exhaust ventilation and personal protective equipment. It is also possible to conclude that occasional cases of asthma develop as a result of exposure to welding fume, but it is not possible to say which component of the fume or gases is the cause. It is likely that occupational asthma due to welding is not a homogeneous diagnosis.

Commission VIII strongly recommends that:

- 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 welder keeps 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/powered air purifying 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 GTAW/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 an air fed/powered air purifying helmet.
- The welder uses a technique of counting to five at the end of the welding process before lifting an air fed/powered air purifying 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 which should be lifted immediately.
- 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 to the lungs than would be the case with welding fume or smoking alone.

4 Conclusion

It is the conclusion of Commission VIII that arc welding is associated with occasional cases of asthma and may be associated with an accelerated decline in lung function, particularly in combination with smoking. Given the number of studies that have already been completed and the failure to fully address the question as to whether there is an accelerated decline in lung function, Commission VIII reinforces advice already provided to the welding industry on careful control of welding fume to minimise exposure at the workplace and reinforces the advice already provided by Commission VIII that welders should not smoke.

If clinicians and epidemiologists with an interest in welding consider any further study of the respiratory health effects of welding, it is strongly recommended that a multidisciplinary approach is used, involving welding engineers and industrial hygienists from the start, to ensure that there is a rigorous exposure assessment of both fume and gases in addition to the clinical measurements undertaken by the clinicians.

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