

Occupational asthma due to stainless steel welding fumes

HELENA KESKINEN, P.-L. KALLIOMÄKI and K. ALANKO*

*Institute of Occupational Health, Helsinki, and * Lahti Central Hospital,
Department of Diseases of the Chest, Lahti, Finland*

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Summary

Seven patients with respiratory symptoms after welding were examined. Five had occupational asthma caused by fumes from manual metal arc stainless steel welding. The detailed clinical results of two patients with repeated challenge tests are reported.

One patient was nonatopic and had earlier had contact dermatitis due to chromium. He experienced a non-immediate type of asthmatic reaction after welding stainless steel. The other patient was atopic and showed an immediate-type reaction. Both reactions were repeated in the second provocation test after about 2 years. In addition both disodium cromoglycate and beclomethasone pretreatments inhibited the reaction of the two patients, but a placebo medication did not. A remarkable amount of chromium and nickel are detected in the fumes released during stainless steel welding but not in the fumes from mild steel welding, with which all provocation tests were negative. The chromium or the nickel in welding fumes might be the aetiological factor of the reaction.

Introduction

Several types of welding processes are currently in use. The subjects in this study had welded both mild and stainless steel using only the manual metal arc (MMA) welding technique.

Welding fumes consist of solid and gaseous pollutants. Solid particles are formed from workpiece metal and electrode coating. Gaseous products are mainly nitrogen oxides, ozone, fluoride compounds and carbon dioxide.

The concentration of solid particles in welding fumes varies considerably in welding work and depends upon the welding method (type of base material, type and dimension of the electrodes, current etc) and ventilation conditions. It can range between broad limits, e.g. between 2 and 100 mg/m³ (Her Majesty's Stationery Office, 1970).

Correspondence: Dr H. Keskinen, Institute of Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland.

Table 1. Average chemical composition of fumes from MMA mild steel welding (OK 48.15) (Stern, 1977) and stainless steel welding with coated electrode

Element	Concentration of element in mild steel welding (%, range in brackets)	Stainless steel welding	
		Concentration of acid soluble element (%)	Concentration of water soluble element (%)
Al	1.56		
Na	0.92 (0.20–1.20)	7.5	6.3
Mg	2.86 (2.5–3.3)	2.5	1.8
F	8.89 (6.6–11.8)	14.7	6.3
Si	0.28 (0.09–0.56)	7.3	—
K	14.31 (10.9–19.9)	22.1	19.5
Ca	12.92 (11.7–14.7)	1.4	0.44
Ti	0.809 (0.64–1.0)	2.12	
Cr	<0.1	3.8	3.6
Mn	3.34 (2.9–4.2)	2.7	0.25
Fe	24.34 (16.4–29.2)	6.3	0.008
Ni	<0.01	0.53	0.005
Cu	0.29 (0.025–0.36)	0.027	
Zn	0.01 (0.05–0.25)	0.16	0.007
Mo	<0.01	0.12	0.07
Ag	<0.005		
Cd	<0.005		
Pb	<0.005		
V		0.6	
Total		71.8	43.3

The composition of solid inorganic particles in welding fumes is dependent on the composition of the material being welded and the electrodes used. Normal MMA welding on mild steel produces fumes containing significant amounts of Al, Mg, F, Si, K, Ca, Ti, Mn and Fe, with trace amounts of Co, Zn and Pb (Table 1) (Stern, 1977). MMA welding on stainless steel contains significant amounts of all the elements already mentioned plus Cr and Ni (Table 1).

The total mass median diameter of MMA welding fume particles is on the order of 0.3–0.5 μm (Stern, 1977). According to transmission electron micrographs particles form long chains and rings with an amorphous shell.

Numerous papers have dealt with chronic lung diseases among mild steel welders. Siderosis is the oldest known occupational lung disease found among these workers. During the last 10 years non-specific lung diseases, including chronic bronchitis and emphysema, have also been connected with welding (Hunnicut, Cracovaner & Myles, 1964; Fogh, Frost & Georg, 1969; Peters *et al.*, 1977).

From the hygienic point of view of stainless steel welding fumes might represent a special problem. All stainless steel welding processes produce metals like chromium and nickel compounds.

Preliminary studies have indicated that especially MMA stainless steel welding fumes have mutagenic activity (Hedenstedt *et al.*, 1977). However, there are very few studies concerning real health risks caused by stainless steel welding fumes.

Many metals, including platinum, chromium, nickel and also cobalt, are known to

cause contact dermatitis, but there is very little data on their role in the pathogenesis of asthma.

Occupational asthma caused by different chemicals has proved to have different mechanisms (Popa *et al.*, 1969). The complex salts of platinum are known to cause immediate type asthma (Pepys, Pickering & Hughes, 1972). Immediate type asthma is also suspected to be caused by chromic acid (Joules, 1932) and nickel sulphate (McConnel *et al.*, 1973). Both cases first developed contact dermatitis from exposed metal. Williams (1969) described well the different hygienic hazards of chromium; he also reported two cases, one of asthma due to fumes of chromic acid and one of asthma due to zinc chromate primer paint.

Aluminium sulphate has been reported to cause a late asthmatic reaction (K. Alanko, personal communication; Simonsson *et al.*, 1977). Mostly the typical signs of immediate type allergy are lacking in cases of asthma due to different chemicals, but positive patch tests indicate altered cell-mediated immunity (Popa *et al.*, 1969).

In this communication we report two re-examined cases of occupational asthma due to welding fumes of stainless steel.

Material

During 1976–1978 seven patients complaining of asthmatic symptoms after welding were examined. They all came to the Institute of Occupational Health in Helsinki for the examination. Two of them had other reasons for their symptoms. One had chronic maxillary sinusitis, and the other simple chronic bronchitis.

A preliminary examination showed that five patients developed asthmatic symptoms after MMA stainless steel welding during welding provocation tests. The two earlier mentioned patients were also given the provocation test, and the results were negative. Welding mild steel did not cause bronchial obstruction.

The five patients with positive results drew our attention to the hygienic conditions of the welding procedure, which were then investigated more closely. After about 2 years the five patients were requested to attend a re-examination to see if the result could be repeated; three of the five did. One patient no longer reacted to the welding provocation. He had not been exposed to welding fumes nor experienced any symptoms since the first examination. Two patients could not leave work to attend the re-examination.

Methods

The basic clinical examination for occupational asthma was performed as described by Keskinen, Alanko & Saarinen (1978). Contact allergy for nickel, chromium, cobalt, etc. was tested with the chamber method (Pirilä, 1975).

A clinical examination was carried out before both sets of provocation tests.

The following bronchial provocation challenge tests were made in 1978, the first three having also been performed about 2 years earlier: (1) placebo test with lactose powder; (2) MMA/mild steel welding (30 min); electrode OK48 was used; (3) MMA/stainless steel welding (30 min) (except for the first test of case 1, which was 1 hr); electrode AVESTA 832 SK-PW was used; (4) MMA/stainless steel welding (30 min) with beclomethasone pretreatment; (5) MMA/stainless steel welding (30 min) with disodium chromoglycate pretreatment; (6) MMA/stainless steel welding (30 min) with placebo medication; (7) stainless steel welding with the metal-inert gas (MIG) tech-

nique (1 hr). [Concentrations of chromium and nickel in MIG welding fumes are about 10 and 7%, respectively; most of their compounds are acid soluble (Stern, 1977).]

The respiratory function was followed by the measurement of peak expiratory flow (PEF) values with a Wright peak flow meter every 15 min during the challenge, and afterwards every 1–4 hr until the end of 24 hr. The normal diurnal PEF curves were used for reference.

Case reports

Case 1

A.K., aged 51 years was initially employed by a foundry, but in 1966 he left to become a cement worker, a job which exposed him to cement dust containing chromium. At that time his main occupation was manually grinding concrete surfaces. After developing occupational eczema from contact with chromium in 1968, he moved to maintenance work. Then he welded mild steel using coated electrodes for about half of the workday. During that period he had not been exposed to dust other than mild steel welding fumes. Finally in 1973 he began work in a building factory, where he welded both mild steel (50%) and stainless steel (50%) for about 4 hr daily. He used several types of basic coated and rutile electrodes made by ESAB, Avesta and Philips. He had never done shield gas welding. He has been on a pension because of occupational asthma ever since 1976.

This patient developed severe eczema after working with cement for 1.5 years. A positive patch test proved that the eczema was caused by chromium, and the worker received compensation for an occupational disease. Later he did stainless steel welding, for 1.5 years before he started experiencing dyspnoea while welding stainless steel with electrodes in 1974. He associated his asthmatic symptoms with the stainless steel welding every time. The respiratory symptoms appeared about 2 hr after the beginning of the welding, and the obstruction continued for about 10–12 hr after the end of exposure. The symptoms during welding became progressively worse. Finally the attacks were so severe that the patient had to be hospitalized after attacks. Since his retirement in 1976 he has not suffered asthmatic attacks; in other words, he has not had any symptoms when not welding. He smoked about twenty cigarettes daily for 20 years but stopped in 1975. There had been no atopy in his personal or family history.

Case 2

E.R., aged 50 years had worked since 1947 for the same employer producing machines for processing and agricultural needs. He worked in the beginning as a mending welder, first welding only mild steel for about 5 years. From 1952 the percentage of time spent on stainless steel MMA welding gradually increased. Between 1962 and 1972 more than 90% of his work was stainless steel welding (about 5 hr daily). He had used only the MMA welding procedure with various types of basic coated and rutile electrodes; he had never done shield gas welding. Because of respiratory symptoms caused by the welding fumes in 1972, he stopped welding completely and began working as a mechanic repairing machines in order to avoid exposure to welding fumes or other respirable dusts.

He had started to experience dyspnoea in about 1968 while welding stainless steel in confined spaces. The dyspnoea appeared about 1 hr after the welding began and continued for 24 hr. However, he had also experienced similar dyspnoea attacks,

although seldom, after mild steel MMA welding when working on plated surfaces. During welding the symptoms became progressively worse. Chronic rhinitis symptoms appeared in about 1970, at the same time as the more severe dyspnoea attacks connected with stainless steel welding. Asthmatic attacks have not occurred since 1972 when exposure to welding fumes was eliminated and the subject no longer needs medication. He had smoked about ten cigarettes daily for 25 years, but he stopped 10 years ago. There were no symptoms of atopy in his family history nor in his former personal history. He had never suffered from eczema.

The occupational history of both cases is reported in Table 2.

Results

Case 1

A.K. was in good health, except for mild dermatitis on his hands and feet during the first examination in 1976. The results of his clinical findings are presented in Table 3. There was no exercise-induced asthma. The skin prick tests were negative, but the chamber tests resulted in a positive reaction to chromium. His diurnal PEF values were within normal limits. The results of his challenge tests were as follows: (1) placebo test with lactose powder: negative; (2) MMA/mild steel welding: negative (Fig. 1a); (3) MMA/stainless steel welding: in 1976 a severe bronchial obstruction immediately after the challenge test (duration of the test 1 hr); in 1978 a late bronchial obstruction of 30% after exposure of 30 min (Fig. 1a). (4) MMA/stainless steel welding with beclomethasone pretreatment: negative; (5) MMA/stainless steel welding with disodium cromoglycate pretreatment: negative; (6) MMA/stainless steel welding with placebo

Table 2. The nature and length of various jobs and the types of exposure of both patients

Subject	Age (years)	Work	Exposure time (years)	Type of exposure
A.K.	51	Construction worker	10	No exposure
		In iron foundry		
		—annealing of casting	2	Sand dust, iron fumes
		—masonry of furnace	6	Sand, clay dust, iron fumes
		In concrete products industry		
		—manual grinding of precast concrete panels	4	Cement dust, including chromium
		Maintenance man in building factories		
		—mild steel MMA welding (for about 4 hr daily)	3	Mild steel welding fumes
		—mild steel and stainless steel MMA welding (for about 4–5 hr daily)	3	Mild steel and stainless steel MMA welding fumes
		—retired for 2 years		
E.R.	50	Mending welding in repair shop of metal industry		
		—mild steel MMA welding	5	Mild steel welding fumes
		Mild steel and stainless steel MMA welding	20	Mild steel and stainless steel welding fumes
		As a fitter and mechanic repairing machines	6	No exposure to metal dust

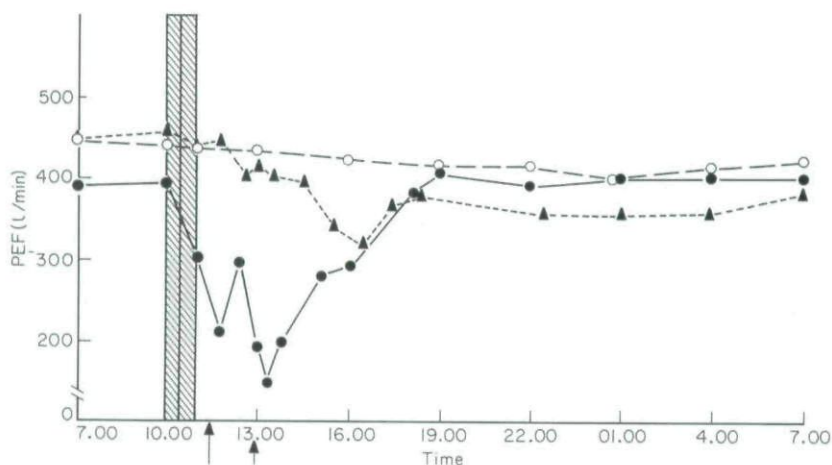


Fig. 1a. Results of the challenge tests (case 1, A.K.) with manual metal are (MMA) welding on stainless (SS) and mild steel (MS). Arrows show point of isoprenaline inhalation followed by i.v. theophyllamine and hydrocortisone. The test with MMA/MS gave similar response in both years. ●—●, MMA/SS exposition 1976; ▲---▲, MMA/SS exposition 1978; ○---○, MMA/MS exposition.

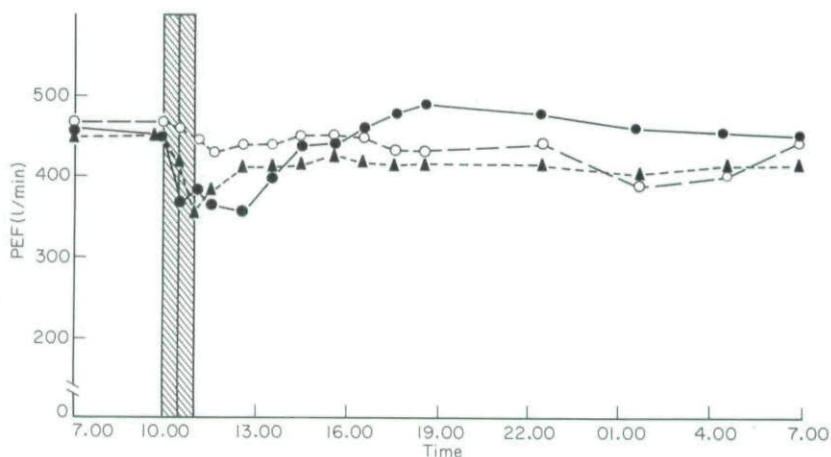


Fig. 1b. Results of the challenge tests (case 2, E.R.) with manual metal are (MMA) welding on stainless (SS) and mild steel (MS). The test with MMA/MS gave similar responses in both years. ●—●, MMA/SS exposition 1977; ▲---▲, MMA/SS exposition 1978; ○---○, MMA/MS exposition.

medication: bronchial obstruction reaction as above; The reduction in the PEF values was 28%; (7) MIG/stainless steel welding: negative.

Case 2

E.R. had a good clinical status; no râles were heard. His clinical findings are summarized in Table 3.

In the skin prick tests there were positive reactions to several pollens. However the RAST tests for corresponding pollens were negative. The patch tests were negative. Histamine provocation produced mild hyperactivity. There was no exercise-induced asthma. His diurnal PEF values were within normal limits. The results of the challenge

Table 3. Clinical findings of the welders examined in 1976 and 1978

Subject	Laboratory findings	Respiratory function				Skin tests	
		Spirometry		Transfer factor	Histamine challenge tests	Prick	Patch
		VC (%)	FEV ₁ (%)				
A.K.	Normal	71	70	71 (1976)	No bronchial hyperreactivity (1976)	Negative	Positive for Kalium dichromium
	IgE 21 No eosinophilia	73	79	81 (1978)	Mild bronchial hyperactivity (1978)		Negative for nickel
E.R.	Normal	91	91	120 (1977)	Slight bronchial hyperactivity (in 1977, 1978)	Positive for pollens with negative RASTs	Negative
	IgE 25 no eosinophilia	91	87	119 (1978)			

tests were as follows: (1) placebo test with lactose powder: negative; (2) MMA/mild steel welding: negative (Fig. 1b); (3) MMA/stainless steel welding: in both tests immediate bronchial obstruction occurred and was strongest 30 min after the end of exposure; the reductions in PEF values were 30 and 22% respectively (Fig. 1b); (4) MMA/stainless steel welding with beclomethasone pretreatment: negative; (5) MMA/stainless steel welding with disodium cromoglycate: negative; (6) MMA/stainless steel welding with placebo medication: immediate bronchial obstruction as above; The reduction in the PEF values was 22%; (7) stainless steel welding: negative.

Discussion

Both of the cases reported in this communication responded asthmatically after welding stainless steel using different electrodes. According to their occupational history, and also the challenge tests, welding mild steel with electrodes or shield-gas stainless steel welding (MIG), did not cause asthmatic symptoms. Since MMA welding of mild and stainless steel produces similar gaseous pollutants, the bronchial obstruction was not evidently caused by irritant gases. MMA stainless steel fumes consist of several inorganic compounds, e.g., chromium and nickel, which are practically non-existent in MMA mild steel fumes. A comparison of the chemical characteristics of MIG and MMA stainless steel welding fumes shows that a significant difference is a high fraction of water-soluble chromium in the MMA stainless steel fumes. In addition there are many differences in the morphological properties of the particles produced by these two welding processes; e.g. the mass median diameter of particles in MIG welding fumes is smaller (0.01–0.05 μm) than that of the MMA welding fume particles. According to the challenge tests it seems possible that the asthmatic reaction might have been caused by either the chromium or nickel compounds in MMA stainless steel welding fumes.

In many reported cases of occupational asthma due to chemicals contact dermatitis has preceded asthmatic symptoms (Popa *et al.*, 1969). Two of our five cases had also

previously had eczema due to chromium. This occurrence points to some link between cell-mediated and humoral immunity. In addition an interaction between humoral and cell-mediated allergy is a topic of current interest (Katz, 1978). However the immunological mechanism is not yet clear. A non-immunological mechanism is suspected in cases with exposure to aluminium sulphate and aluminium fluoride (Simonsson *et al.*, 1977). Different types of reaction following the same challenge test are quite usual when one tests with chemicals (Burge *et al.*, 1978). In 1978, after a challenge test the non-atopic patient showed a non-immediate reaction, and the atopic one an immediate type of asthmatic reaction of similar duration. It is remarkable that the reactions could be repeated after about 2 years without exposure. With occupational asthma due to cobalt dust, the challenge test becomes negative after 2–3 years without exposure. Case 1 showed mild bronchial hyperreactivity in the second histamine challenge test, while the first such test was negative. Case 2 reacted in the same way both times. Pain & Simons (1972) reported on seven patients with occupational asthma, all of whom had a positive methacholine test after 3 years of non-exposure.

During the challenge tests the exposure of the present two cases was standardized in the following respects only: the duration of exposure (except in case 1 in 1976); the types of electrodes, and the premedication. The concentration of pollutants in the inhaled area was not controlled nor were any chemical analyses of fumes made. Because the exact composition of the fumes is not known, more specific challenge tests to specify the actual agent causing the asthmatic reaction, cannot be performed.

The asthmatic reaction can be tested with pre-treatment before the specific bronchial provocation test. An immediate type reaction can be blocked with disodium cromoglycate, but not with beclomethasone, while non-immediate reactions can be blocked by both compounds (Pepys & Hutchcroft, 1972).

The beneficial effect of disodium cromoglycate pretreatment on both the immediate and non-immediate asthmatic reactions after the challenge tests was expected, but it was unusual that beclomethasone inhibited the immediate reaction of the atopic patient. However, this reaction resolving fully after 3–4 hr also had a prolonged non-immediate type component and so differs from the classical immediate reaction lasting maximally only 2 hr.

Exposure to different fumes and dusts of metals and their salts is usual in many types of industry; for example there are more than 100 different occupations in which exposure to chromium can occur. Our results show that bronchial asthma must be taken into consideration, when a worker complains of respiratory symptoms after welding. With bronchial challenge tests simulating working conditions, the reaction can be detected. However the mechanism of the reaction is still unexplained, although cell-mediated mechanisms may play an important role.

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