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STUDIES ON THE PULMONARY REACTION OF WORKERS EXPOSED TO COBALT IN THE TUNGSTEN CARBIDE INDUSTRY

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ABSTRACT

Groups of workers from a tungsten carbide industry with defined exposure to cobalt in the air and control groups matched with respect to sex, age, smoking habits and length of employment have been studied. The exposure was described in terms of cobalt concentration in the air, in the blood and in the urine. The examination included subjective symptoms, spirometry and closing--volume determination. An association between the degree of exposure and findings has been demonstrated.

Rare cases of pneumoconioses have been observed in the tungsten carbide producing industry, especially during earlier years[3,5]. With progressing improvement of the hygienic conditions these cases have become extremely rare. Other complaints, e.g. irritation of the respiratory organs, breathing difficulties[2] etc., have attracted less attention until recent years. We have studied this problem in a group study.

SUBJECTS AND METHODS

Examined groups

Three groups of workers from the tungsten carbide producing industry with different degrees of exposure to cobalt have been studied and, for comparison, a non-exposed control group from the same industry matched with regard to sex, age, length of employment, smoking habits. All exposed workers handling the material used could be exposed to dust in mixing, pressing and grinding.

Group A: the average content of cobalt in the air was determined at 0.005–0.01 $mg \cdot m^{-3}$ during the last years; Group B: average exposure during the last years 0.06 $mg \cdot m^{-3}$; Group C: average exposure to cobalt during the last years 0.01 $mg \cdot m^{-3}$; Controls: non-exposed workers or clerks from the same industry.

Methods

The methods used included: clinical routine examination, pulmonary X-ray examination, dynamic spirometry, closing-volume determination of nitrogen concentration in exhaled air during slow exhalation after a maximal inhalation of oxygen, determination of cobalt in urine and blood by means of atomic absorption-spectrometry using an electrothermic atomizer. In order to determine very low values of cobalt we used an ion exchange separation step. With the separation step the detection limit is 2–3 $nmol \cdot l^{-1}$ for urine and blood. Without the separation step the detection limit is 70 $nmol \cdot l^{-1}$ in the blood and 40 $nmol \cdot l^{-1}$ in urine[1].

The cobalt content of the atmosphere was determined with the aid of a personal air sampler and atomic absorption spectrometry.

RESULTS

X-ray examination of the lungs

No gross changes or signs of pneumoconiosis could be detected.

Symptoms from the respiratory tract

The frequency of reports of certain symptoms in the different groups is shown in Table 1. Chronic bronchitis and tightness of the chest were reported more frequently among the exposed workers in Group B than among their controls. In that group there was no increase of reports on cough without sputum. In Groups A and C no such differences appeared in comparison with the controls.

TABLE 1

Per cent frequency of different symptoms in the exposed groups and their controls. P-values for the differences.

	Group A		Group B		Group C	
	n	P	n	P	n	P
Cough, no sputum	20/3	0.125	8/10	1	8/8	1
Chronic bronchitis	0/0	–	11/0	0.016	0/0	–
Chest tightness	10/0	0.250	24/0	0.001	16/0	0.125
Number of pairs	30		63		27	

Lung physiology, comparison between exposed and control groups

When the exposed groups were compared with their controls on Monday morning there were marked differences in Group B (Table 2). The results indicate obstructive changes. In the other groups there are no significant changes. Closing-volume values on Monday morning before work did not indicate any differences between the exposed groups and their controls (Table 3).

TABLE 2
Spirometric results on Monday morning before work. Mean difference between pairs of exposed persons and their matched controls. P-values for the differences.

	Group A		Group B		Group C	
	Difference	P	Difference	P	Difference	P
FVC	0.04	0.742	0.10	0.320	0.22	0.080
$FEV_{1.0}$	−0.05	0.700	−0.19	0.048	−0.22	0.117
$FEV_{\%}$	− 1.4	0.582	−3.0	0.006	−0.5	0.780
MMF	− 0.08	0.734	− 0.42	0.022	− 0.36	0.261
Number of pairs	30		63		27	

TABLE 3
Per cent closing-volume in exposed and control workers on Monday morning before work. Mean difference between pairs. P-value for mean differences.

	n	Closing-volume		Difference	P
		Exposed	Controls		
Group A	9	19.3	16.0	3.28	0.340
Group B	14	22.7	20.7	2.00	0.545
Group C	19	19.5	18.5	1.04	0.678

Lung physiology, changes during a working shift/week

When spirometry was performed before and after a working shift, an impairment of some parameters was found in Group B, but not in Groups A and C (Table 4). In Group B, a decrease was found in FVC, $FEV_{1.0}$ and MMF. Determination of the closing-volume revealed an increase in Group B over a working shift and no significant changes in Groups A and C (Table 5).

TABLE 4
Spirometry. Mean difference between morning and afternoon values. P-values for the differences.

	Group A		Group B		Group C	
	Difference	P	Difference	P	Difference	P
FVC	0.04	0.256	0.08	0.050	0.06	0.109
$FEV_{1.0}$	0.01	0.562	0.06	0.022	0.04	0.552
$FEV_{\%}$	− 0.4	0.222	0.3	0.592	0	–
MMF	− 0.05	0.624	0.23	0.016	− 0.01	0.289
N	36		73		39	

TABLE 5
Per cent closing-volume before and after a working shift. P-values for the differences.

	n	Closing-volume		Difference	P
		Monday	Friday		
Group A	9	19.1	20.6	− 1.5	0.197
Group B	14	22.9	26.7	− 3.82	0.002
Group C	16	18.3	20.1	− 1.78	0.184

When spirometry results from Monday and Friday morning before working shift were compared, the FVC in Group B had decreased during the week. In the other groups no changes were revealed (Table 6).

TABLE 6
Spirometry. Mean differences between Monday morning and Friday morning before work. P-values for the differences.

	Group A		Group B		Group C	
	Difference	P	Difference	P	Difference	P
FVC	0.01	0.960	0.06	0.012	0.01	0.800
$FEV_{1.0}$	0.01	0.872	0.04	0.222	− 0.01	0.765
$FEV_{\%}$	0.4	0.982	− 0.5	0.224	0.1	0.906
$MMF^{\%}$	0.16	0.114	0.06	0.522	− 0.03	0.790
N	36		73		39	

Lung physiology, changes over 4 weeks vacation

Groups of workers were examined on Monday morning before work, before and after 4 weeks of vacation. No significant changes in the spirometry results could be seen (Table 7).

TABLE 7
Spirometry. Mean differences between values on Monday morning before and after 4 weeks of vacation. P-values for the differences.

	Group A		Group B		Group C	
	Difference	P	Difference	P	Difference	P
FVC	− 0.05	0.617	0.04	0.588	0.03	0.800
$FEV_{1.0}$	− 0.07	0.752	0.03	0.634	− 0.01	0.911
$FEV^{\%}$	0.88	0.580	− 0.06	0.893	− 0.86	0.757
MMF	0.03	0.839	0.09	0.120	− 0.11	0.757
N	16		50		7	

The closing-volume before and after vacation was studied only in Group B. On an average it was 0.13 l smaller after vacation than before. This difference is not significant ($p = 0.193$; $n = 15$). All observed effects were more pronounced in smokers than in non-smokers. The results indicate that exposure to cobalt, 0.06 $mg \cdot m^{-3}$, induces an irritative reaction in the respiratory tract with symptoms of bronchitis and mainly obstructive changes in the spirometry findings. The reactions could be demonstrated in the course of a working shift and there was an improvement overnight and still more so over the weekend. We could see no further improvement during a 4 weeks' vacation. In groups exposed to 0.005–0.01 $mg \cdot m^{-3}$ no such reactions could be observed.

Cobalt in blood and urine

In a group of non-exposed persons ($n = 25$) we found an average of 0.5 ± 0.1 ng $Co \cdot g^{-1}$ in the blood and 0.4 ± 0.1 $ng \cdot ml^{-1}$ in urine.

The resorption of cobalt in the lungs is very rapid[4]. We followed the concentration in urine from Friday afternoon till Monday morning, the first sample voided at the end, and the last before, the working shift. The results showed a rapid decrease of the urinary concentration of cobalt during the first 20–30 hours after exposure when the exposure was relatively high (Fig. 1). The

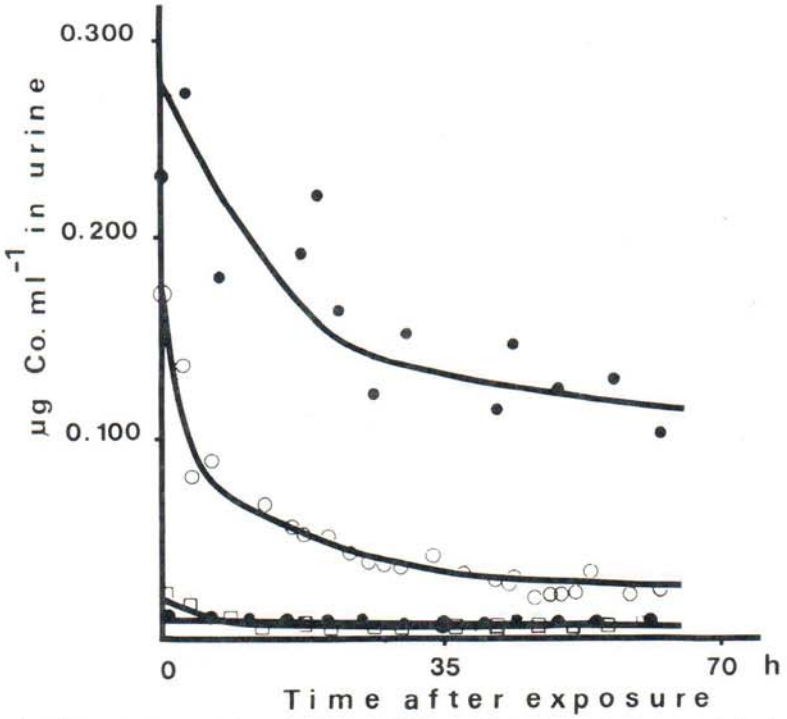

FIG. 1 – Urinary concentration of cobalt at different times after exposure to cobalt dust. Four persons with a different degree of exposure.

decrease was less pronounced when the exposure was lower. Then a period with prolonged excretion followed, with some persons not reaching the "normal zone" even after 4 weeks' vacation.

A group of exposed persons was followed for a whole week. The exposure of each person was determined by means of personal air sampler and the concentration in urine was checked every morning and afternoon. Each shift was found to lead to an increase in urinary cobalt concentration, the concentration decreasing consistently the following morning. When the average exposure for the whole week was compared with the cobalt concentration in urine, the correlation coefficient on Friday afternoon was 0.79 and on Monday morning 0.81. For blood the correlation coefficient was 0.87 on Friday afternoon and 0.76 on Monday morning.

The variations in cobalt concentrations in blood are less rapid but follow the exposure. When blood and urine concentrations of cobalt were compared on Friday afternoon the correlation coefficient was $r = 0.82$ ($N = 40$, $p < 0.001$).

DISCUSSION

From the results it would appear that an average exposure to 0.06 mg $Co \cdot m^{-3}$ of air may have an irritative effect on the respiratory tract and cause obstructive changes in the spirogram. This reaction can be demonstrated in the course of a working shift. It decreases overnight and even more over the week-end. However, regression in Group B was not complete, because there is a difference between exposed persons and their matched controls indicating a damage and this difference does not regress over a 4 weeks' vacation, indicating a stationary defect. This may be due to a higher earlier exposure or to a subsequent slow deterioration. From Table 8 it can be seen that the difference between the exposed men and their controls increases with the total time of exposure of up to 15 years. It must be kept in mind, however, that some exposed persons have left the job and that this factor is not under control. The reactions were more pronounced in smokers.

In the two groups with lower exposure, 0.005–0.01 mg $Co \cdot m^{-3}$, no such clear-cut effects were observed.

TABLE 8

Group B, mean difference (l) in $FEV_{1.0}$ between exposed persons and controls with different duration of exposure.

	Duration of exposure in years			
	1–4	5–9	10–14	15–19
n	14	20	14	6
$\bar{X}$	−0.07	−0.20	−0.30	−0.23
S.D.	−0.95	0.62	0.86	0.47

Methods for determining low cobalt contents in the blood and urine within the normal range have been worked out. The cobalt content was about 0.5 $ng \cdot g^{-1}$ in the blood and 0.4 $ng \cdot ml^{-1}$ in the urine of a non-exposed group. After exposure, cobalt was rapidly excreted in urine. When the exposure was relatively high, there was a rapid continuous decrease in excretion for 10–20 hours followed by a phase of slower excretion. When the exposure was low, the excretion of cobalt in urine was relatively constant, although 5–10 times higher than in non-exposed persons. The slow excretion continued for a long time, sometimes even for 4 weeks after exposure.

REFERENCES

1. *Alexandersson, R., Lidums, V. and Swensson, Å.* Cobalt content in blood and urine of exposed and non-exposed persons (in Swedish). Arbete and Hälsa, Stockholm, **8**, 1979.

2. *Coates, E. O. and Watson, J. H. L.* Diffuse interstitial lung disease in tungsten carbide workers. Ann. Intern. Med., **75** (1971) 709–716.

3. *Fairhall, L. T., Castberg, H. T., Carosso, N. J. and Brinton, H. P.* Industrial hygiene aspects of the cemented tungsten carbide industry. J. Occup. Med., **4** (1947) 371–378.

4. *Gustavsson, P., Lidums, V. and Swensson, Å.* Uptake, distribution and elimination of cobalt in the rat after single intratracheal administration (in Swedish). Arbete and Hälsa, Stockholm, **9**, 1979.

5. *Lundgren, K.-D. and Ohman, H.* Pneumoconiose in der Hartmetallindustrie. Virchows Arch., **325** (1954) 259–284.