

Peripheral nerve conduction velocity in workers occupationally exposed to lead

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CHEN ZQ, CHAN QI, PAR CC, QU JY. Peripheral nerve conduction velocity in workers occupationally exposed to lead. *Scand J Work Environ Health* 11 (1985): suppl 4, 26–28. The peripheral nerve conduction velocity between the wrist and elbow of the right upper limb and the relationship between this parameter and biochemical parameters for workers occupationally exposed to lead were studied. The results showed that nerve conduction velocity could be altered by lead exposure. The nerve conduction velocity was decreased when the exposure level was 0.0652 mg/m³ (time-weighted average), and it slowed further in proportion to increasing exposure levels. There was a negative correlation between the nerve conduction velocity and the length of exposure. A decrease in the nerve conduction velocity was noted after less than three years of exposure to lead. There was a close relationship between the nerve conduction velocity and the level of lead in the blood; the nerve conduction velocity decreased proportionally with increasing blood lead levels. The blood lead level associated with a change in the nerve conduction velocity was < 40 µg/100 ml (< 1.9 µmol/l). No correlation between the nerve conduction velocity and the amount of metabolites of porphyrin was found. The measurement of nerve conduction velocity can be used as one of the sensitive criteria for the early detection of the toxic effects of lead.

Key terms: blood lead, delta-aminolevulinic acid, free erythrocyte protoporphyrin, median nerve, ulnar nerve, zinc protoporphyrin.

Lead is a common industrial chemical with affinity for the nervous system. It has been reported that peripheral nerve conduction velocity is significantly decreased in patients with subclinical lead poisoning and in workers occupationally exposed to lead. The determination of nerve conduction velocity seems to be a sensitive method for detecting subneuropathy (1, 3, 5, 6). However few studies dealing with this issue have been published. In the present paper the changes in the nerve conduction velocity of workers and the relationship between this parameter and other parameters of the effects of lead on occupationally exposed workers were studied.

Subjects and methods

The exposure group comprised 98 lead workers from a battery plant who did not have symptoms of clinical neuropathy. Thirty-nine persons (students, clerks and architecture workers) not exposed to any chemical agent were used as a reference group. The average age of the two groups was 36.79 (SD 9.29) and 35.67 (SD 10.32) years, respectively.

Atmospheric area samples of the worksites were collected with DK60-2 type samplers each day for 2 d. The time-weighted average (TWA) concentration, according to the actual exposed time of the workers per day, was calculated as the exposure level of the work-

ers. A dithizone method was used for the determination.

The blood lead (PbB) level was measured with atomic absorption spectrophotometry.

The level of free erythrocyte protoporphyrin was determined with a 930 fluorometer, the level of zinc protoporphyrin (ZPP) in the blood was analyzed with a ZPP hematofluorometer, and the level of delta-aminolevulinic acid was measured with the routine method.

An electrophysiological examination was performed on all the subjects with a JD-II electromyograph made in Shanghai. Motor conduction velocity (MCV) of the median and ulnar nerves and the sensory conduction velocity (SCV) of the median nerve between the elbow and wrist of the right upper limb were measured. The motor conduction velocities were measured with an ordinary method, and the sensory conduction velocity was measured with an antidromic method (4). The room temperature was kept at 24–25°C. The skin temperature at the wrist and elbow was 31 and 33°C, respectively.

Results

The neurophysiological findings of the two groups are given in table 1. The nerve conduction velocity was lower in the exposed group than in the reference group. Statistical differences were found between the motor and sensory conduction velocities of the median nerve

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of the two groups ($p < 0.01$), but the differences between the ulnar measurements of the two groups were not significant.

The lead workers were divided into three groups according to their length of exposure. The results of the neurophysiological measurements for the three groups are presented in table 2. Both the motor and sensory conduction velocities of the median nerve were decreased in the three groups when compared with the corresponding values of the reference group, and the amplitude of the decrease was proportional to the increase in the duration of exposure. A significant difference in these values was seen even for the group exposed less than three years. Further analysis showed that the motor conduction velocity of the median nerve and length of exposure had a significantly negative correlation ($p < 0.01$). No significant difference was found for the measurements of the ulnar nerve, either within the exposure group or between the exposed and reference groups.

Table 3 presents the neurophysiological measurements in relation to the content of lead in the air of

the workplace. The group exposed to even the lowest lead level (0.0652 mg/m^3) showed a decrease in the motor and sensory conduction velocities of the median nerve when compared to the reference group.

The change in nerve conduction velocity in relation to the blood lead level was also examined (table 4). A significant decrease in the motor conduction velocity of the median nerve was found in the group with a blood lead level of $< 40 \text{ } \mu\text{g}/100 \text{ ml}$ ($< 1.9 \text{ } \mu\text{mol/l}$) when the group was compared to the reference group ($p < 0.05$). The significant difference in the decrease in the motor and sensory conduction velocities of the median nerve was stronger in the group with a blood lead level of $> 70 \text{ } \mu\text{g}/100 \text{ ml}$ ($> 3.4 \text{ } \mu\text{mol/l}$) ($p < 0.01$). Further analysis showed that there was a negative correlation between the motor conduction velocity of the median nerve and the blood lead level ($p < 0.05$), but not between the corresponding measurement of the ulnar nerve and the blood lead level.

In our study no correlation was found between nerve conduction velocity and the levels of selected metabolites of porphyrin.

Table 1. Comparison of the nerve conduction velocities of the exposed and reference groups.

Group	Number of workers	Motor conduction velocity (m/s)				Sensory conduction velocity (m/s)	
		Median nerve		Ulnar nerve		Mean	SD
		Mean	SD	Mean	SD		
Exposed	98	53.81	5.83	58.89	5.72	55.85	6.09
Reference	39	58.36	5.39	60.14	5.74	59.55	5.15

Table 2. Relationship between nerve conduction velocity and length of exposure.

Group	Number of workers	Motor conduction velocity (m/s)				Sensory conduction velocity of the median nerve (m/s)	
		Median nerve		Ulnar nerve		Mean	SD
		Mean	SD	Mean	SD		
Exposed ^a							
<3	29	55.52	5.14	59.46	6.18	56.51	5.24
3–6	35	53.89	6.00	57.78	5.31	55.47	5.84
6–8	34	52.26	5.94	59.19	5.44	55.67	7.06
Reference	39	58.36	5.39	60.14	5.74	59.55	5.15

^a Subgroups divided according to length of exposure in years.

Table 3. Relationship between nerve conduction velocity and lead in the air of the workplace.

Group	TWA ^a (mg/m ³)	Number of workers	Motor conduction velocity				Sensory conduction velocity of the median nerve	
			Median nerve		Ulnar nerve		Mean	SD
			Mean	SD	Mean	SD		
Exposed								
I	0.0652	26	54.73	5.58	58.99	4.88	55.09	6.64
II	0.2991	38	55.14	8.51	57.71	6.68	56.85	5.86
III	0.8251	34	52.06	4.74	60.06	5.36	52.29	5.93
Reference	0.0033	39	58.36	5.39	60.14	5.74	59.55	5.14

^a TWA = time-weighted average.

Table 4. Relationship between nerve conduction velocity and lead in blood (PbB).

Group	PbB ^a ($\mu\text{g}/100\text{ ml}$)	Number of workers	Motor conduction velocity				Sensory conduction velocity of the median nerve	
			Median nerve		Ulnar nerve		Mean	SD
			Mean	SD	Mean	SD		
Exposed								
I	<40	20	54.28	5.50	60.00	6.61	56.32	7.15
II	40–70	57	54.21	6.44	57.99	5.33	56.23	6.02
III	>70	21	52.27	4.02	60.28	5.63	54.40	5.62
Reference	19.42 (SD 7.36)	39	58.3	5.39	60.14	5.74	59.55	5.15

^a 1 $\mu\text{g}/100\text{ ml}$ = 0.048 $\mu\text{mol/l}$.

Discussion

The determination of nerve conduction velocity is a tool for detecting the early effect of toxic agents. It has been reported that there is no significant difference in nerve conduction velocity between the limbs and between the sexes of healthy adults (9). Room and skin temperatures are believed to affect the result of such determinations however (7). In our experience, it was not a big problem if the room and skin temperatures could be controlled between the range of 20–60°C and 31–33°C, respectively (1, 2).

Ashby (1) and Bordo (2) reported that changes occur in nerve conduction velocity within two years of the initial exposure to lead and that the decrease in nerve conduction velocity and length of exposure has a negative correlation. Our results agreed with those of the two authors, and they indicate that special attention should be given to those workers who have been exposed to lead more than two years.

The average level of lead in the air of the workplace studied was 0.0652–0.8251 mg/m^3 . The value was similar to the level determined by Wang et al (8) using personal samplers one year earlier, and therefore we can assume that the level of lead in the air of the workplace did not fluctuate much.

Our results show that the higher the exposure to lead, the more notable the decrease in nerve conduction velocity. It was suggested that the measurement of lead in the air of the workplace is still a good indicator for environmental monitoring.

The changes in nerve conduction velocity were associated with the level of lead in the blood of the workers. Seppäläinen et al (4) reported that the decrease in nerve conduction velocity could occur at blood lead levels of < 70 $\mu\text{g}/100\text{ ml}$ (< 3.4 $\mu\text{mol/l}$). The nerve conduction velocity in the exposed group with a blood lead level of 60 $\mu\text{g}/100\text{ ml}$ (2.9 $\mu\text{mol/l}$) was statistically slower than that of the reference group. There was a negative correlation between nerve conduction velocity and the blood lead level. Recently Bordo (2) reported that the nerve conduction velocity of workers with blood lead levels of < 50 $\mu\text{g}/100\text{ ml}$ (< 2.4 $\mu\text{mol/l}$) was significantly decreased. The corresponding decrease in workers with a blood lead level

of > 70 $\mu\text{g}/100\text{ ml}$ (> 3.4 $\mu\text{mol/l}$) was more notable however. Our results are in general agreement with the findings of Seppäläinen et al (4) and Bordo (2). The blood lead level of 70 $\mu\text{g}/100\text{ ml}$ (3.4 $\mu\text{mol/l}$), previously considered the exposure limit for nerve conduction velocity effects, is therefore in question.

The lack of correlation between changes in nerve conduction velocity and biological indices of lead workers has been reported before (1, 6). The findings in the present paper are consistent with these reports. They suggest that the mode of the toxic effects of lead on the nervous system differs from effects on the metabolism of porphyrin.

The mechanism of the toxic effects of lead on the nervous system is not completely understood. It is considered to be segmental demyelination with partial damage of the nerve trunks, as well as axonal degeneration (1, 5). Because nerve lesions are often irreversible and the electrophysiological changes reflect the early lesions in the nerve, measuring nerve conduction velocity has played an important role in the early detection and prevention of lead poisoning.

References

1. Ashby JAS. A neurological and biochemical study of early lead poisoning. *Br J Ind Med* 37 (1980) 133–140.
2. Bordo B. Electrophysiologic changes in workers with low blood lead levels. *Am J Ind Med* 3 (1982) 23–32.
3. Hammand PB. Exposure of human to lead. *Ann Rev Pharmacol Toxicol* 17 (1977) 197–214.
4. Melvin JL, Harris DH, Johnson EW. Sensory and motor conduction velocities in the ulnar and median nerves. *Arch Phys Med Rehabil* 47 (1966) 511–519.
5. Seppäläinen AM, Hernberg S. Sensitive technique for detecting subclinical lead neuropathy. *Br J Ind Med* 29 (1972) 443.
6. Seppäläinen AM, Tola S, Hernberg S, Kock B. Subclinical neuropathy at safe level of lead exposure. *Arch Environ Health* 30 (1975) 180–183.
7. Simpson JA. Fact and fallacy in measurement of conduction velocity in motor nerves. *J Neurol Neurosurg Psychiatry* 27 (1964) 381–394.
8. Wang YL, Lu PK, Chen ZQ, Liang YX, Lu QM, Pan ZQ, Shao M. The risk of occupational lead exposure [in Chinese]. *Chin J Ind Hyg Occup Dis* 2 (1984) 129–133.
9. Wu ZM. Normal values of peripheral nerve conduction velocity. *Chin Med J* 62 (1982) 422–424.

Effects of cadmium on cadmium smelter workers

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LIU YZ, HUANG JX, LUO CM, XU BH, ZHANG CJ. Effects of cadmium on cadmium smelter workers. *Scand J Work Environ Health* 11 (1985): suppl 4, 29–32. The effects of cadmium on 65 workers in a cadmium-refining plant in the People's Republic of China were investigated. The average airborne cadmium oxide concentrations ranged from 0.004 to 0.187 mg/m³ at most worksites. Except for some vague symptoms and reports of anosmia, no significant abnormalities were found in the clinical examinations. The level of cadmium in the blood and urine was increased in the majority of the cadmium-exposed subjects. Nine cases (13.8 %) with suspected or mild renal tubular damage were found in this study.

Key terms: beta-2-microglobulin, blood cadmium, cadmium concentration, SDS-PAGE, tubular damage, urinary cadmium.

Cadmium refining is one of the important sources of occupational exposure to cadmium, but relatively few data about the effects of cadmium due to this kind of exposure are available in the People's Republic of China. The purpose of the present paper was to report the findings of a study on the effects of cadmium on workers in a metal-smelting factory.

Subjects and methods

The survey of occupational cadmium hazards was carried out in March 1982 on workers from the cadmium-refining plant in a metal-smelting factory in Hunan Province, the People's Republic of China. The plant produces about 300 t of metallic cadmium yearly as a by-product of zinc refining. The cadmium-containing materials are dissolved in sulfuric acid and then purified and filtered. The solution containing cadmium is then pumped to the electrolytic tank house, and the cadmium plates are transferred to the foundry where they are melted and cast into bricks of highly purified cadmium (99.99 %).

The air from the breathing zones of workers at most worksites in the plant was sampled with personal sampling pumps and membrane filters. The cadmium concentrations were measured by atomic absorption spectrophotometry (9).

Sixty-five cadmium-exposed subjects (47 men and 18 women) were examined. Their ages ranged from 16 to 48 years with a mean of 35.3 years. The subjects

had been working as cadmium smelters for 0.4 to 21.8 years with a mean of 7.8 years. Their job categories included dissolving (30 persons), purification and electrolysis (19 persons), foundry (8 persons), and others (eg, foremen, maintenance workers, etc — 8 persons).

Apart from questionnaires (on job history, past illnesses, present symptoms, etc) physical examinations, chest radiography, and laboratory tests were performed which included (i) blood hemoglobin, (ii) the determination of the blood cadmium content sampled from earlobe capillary blood and measured with an anodic stripping voltametric method (5), and (iii) tests for urinary protein determined by the trichloroacetic acid method (7) and for creatinine by quantitative analysis (Folin-wu method), urinary cadmium content measured by the anodic stripping voltametric method, and protein measured by a modified biuret method (3) in morning urine collected before breakfast. Protein electrophoresis was carried out on the prebreakfast urine sample by modified sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) (1). Urinary beta-2-microglobulin was determined, after adjustment of the pH to 5.6 or more, by enzyme immunoassay with the Phadezym β_2 -micro test (Pharmacia Diagnostics, Uppsala, Sweden).

Results

Exposure

The production procedures took place indoors and were semimechanized. The steps from dissolving to electrolysis were operated under wet conditions, but there was some cadmium oxide dust eliminated while the raw materials were fed. The casting was handled manually, but mechanical ventilation was available in the room. A layer of 2–3 cm of sodium hydroxide covered the surface of the liquid metallic cadmium to prevent the dispersion of cadmium fumes.

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The airborne cadmium oxide concentrations in the plant are shown in table 1. The average cadmium oxide concentration in the breathing zone of the operators ranged from 0.004 to 0.187 mg/m³ at most worksites; these values ranged below and above the maximal allowable concentration of 0.1 mg/m³ for fumes and dusts used in the People's Republic of China.

However the airborne cadmium oxide concentrations were even higher in some places of the plant when they were measured in 1973 and 1978. For example, the concentration in the foundry was 0.074 mg/m³ in 1973 and 0.13 mg/m³ in 1978, and the concentration over the mouth of the precipitating tank ranged from 0.21 to 0.95 mg/m³ in 1978 while wet cadmium-containing materials were being fed.

Symptoms and signs

The symptoms and signs of the cadmium smelter workers (table 2) were usually very mild and showed no significant relationship with the length of exposure. A history of renal calculi was found in one case. Yellow discoloration was seen on incisor teeth in a few cases, but the typical so-called "cadmium-ring" was not detected.

Table 1. Airborne cadmium oxide concentrations in the plant.

Site	Concentration (mg/m ³)	
	Range	Mean
Beside the smelting furnace (operating)	0.025—0.043	0.034
Over the mouth of the precipitating tank (feeding wet cadmium-containing materials)	0.005—0.075	0.027
Over the electrolytic tank (covered)	0.005	..
Over the electrolytic tank (uncovered)	0.159—0.178	0.168
Over the mouth of the dissolving tank (feeding raw materials)	0.165—0.209	0.187
Over the mouth of the dissolving tank (operating)	0.003—0.006	0.004

Hypertension and hypotension was found in two cases and one case, respectively. The liver was palpable in 35.4 % of the group. Because of frequent changes in jobs and little difference in the airborne cadmium oxide concentration in various places, no significant differences in the frequency of symptoms and signs were noted between different job groups.

Cadmium content in blood and urine

The results of the determination of cadmium in the blood and urine of the workers are shown in table 3. When determined by the same analytical methods, the geometric means and upper limits of normal subjects in Beijing were 0.35 and 1.00 µg/g of creatinine, respectively, for the urinary cadmium concentration and 0.50 and 1.6 µg/l (4.5 and 14.2 nmol/l), respectively, for the blood cadmium level (4). As shown in table 3, all the blood and urinary values of cadmium were higher than the limits, except for three workers in the case of urinary cadmium and one worker in the case of the blood cadmium. There were 13 cases with urinary cadmium levels between 5 and 10 µg/g of creatinine and 20 cases with corresponding values higher than 10 µg/g of creatinine (the highest being 42.6 µg/g of creatinine). The level of cadmium in the blood was between 5 and 10 µg/l (44.5 and 89.0 nmol/l) in seven cases, and 52 cases showed blood levels higher than 10 µg/l (89.0 nmol/l), the highest being 92.5 µg/l (823.0 nmol/l). The differences in the urinary and blood levels of cadmium were not significant for the subjects with different kinds of jobs. The excretion of cadmium seemed to increase with the length of exposure, but there was no significant correlation between the cadmium blood content and exposure duration. It was suggested by these findings that the urinary cadmium level may reflect the body burden of cadmium and that the level of cadmium in the blood may be an indicator of recent exposure.

Among the 65 subjects the mean urinary and blood level of cadmium was 5.61 (range 0.90—25.56) µg/g

Table 2. Symptoms and signs of the cadmium-exposed workers.

Symptom or sign	Exposure <5 years (25 persons)		Exposure ≥5 years (40 persons)		Total (65 persons)	
	Number of cases	Percentage	Number of cases	Percentage	Number of cases	Percentage
Headache	7	28.0	11	27.5	18	27.7
Dizziness	6	24.0	23	57.5	29	44.6
Fatigue	5	20.0	8	20.0	13	20.0
Sleep disturbance	2	8.0	7	17.5	9	13.8
Cough	4	16.0	7	17.5	11	16.9
Breathlessness	—	0.0	3	7.5	3	4.6
Lumbago	5	20.0	10	25.0	15	23.1
Pain in extremities	8	32.0	16	40.0	24	36.9
Hepatomegaly	6	24.0	17	42.5	23	35.4
Yellow discoloration on the teeth	1	4.0	4	10.0	5	7.7
Anosmia	3	12.0	11	27.5	14	21.5

of creatinine and 22.44 (range 5.00—79.00) $\mu\text{g/l}$ [199.7 (range 44.5—702) nmol/l], respectively, for the 36 smokers and 4.30 (range 0.51—42.60) $\mu\text{g/g}$ of creatinine and 17.75 (range 1.00—92.50) $\mu\text{g/l}$ [157.9 (8.9—823.0) nmol/l], respectively, for the 29 nonsmokers. The somewhat higher levels of the smokers did not differ statistically significantly from those of the nonsmokers.

Other findings

All the hemoglobin values of the examined subjects were in the normal range, and no significant abnormalities related to cadmium exposure were found in the radiographic examination.

Nine workers had proteinuria, and one had glycosuria, as determined in a routine urinalysis. The mean value of the total protein in the urine of the 65 cadmium-exposed subjects was 106.3 (range 36.5—777.6) mg/g of creatinine; this value was higher than that of normal subjects in Beijing (83.0 mg/g of creatinine). Two workers had a total protein level in their urine (340.8 and 777.6 mg/g of creatinine) that was higher than the normal level. The first had worked with cadmium for 16 years and had a blood cadmium level of 26.25 $\mu\text{g/l}$ (233.6 nmol/l), a positive (+ +) protein qualitative analysis of the urine, and an increased beta-2-microglobulin level, but the SDS-PAGE pattern was normal. This case was diagnosed as cadmium-induced renal tubular damage. The second worker had suffered from hypertension for years, and, although his blood and urinary levels of cadmium were much higher than normal, his renal lesion was not a tubular type because he had a positive (+ + +) urinary protein reaction, middle-molecular weight proteinuria in the SDS-PAGE pattern, and a normal beta-2-microglobulin level.

In this study the SDS-PAGE patterns showed high-molecular weight proteinuria in one case in which the total protein and beta-2-microglobulin values in the urine were normal; middle-molecular weight proteinuria in one case, as has already been mentioned; low-molecular weight proteinuria in one case with an increased beta-2-microglobulin level but a normal total urinary protein value; and marginal low-molecular weight proteinuria in six cases, three of which also showed an increased beta-2-microglobulin level.

The range of beta-2-microglobulin was between 58 and 1 444 $\mu\text{g/g}$ of creatinine, with a mean value of 224.1 $\mu\text{g/g}$ of creatinine, which was significantly higher than that of normal subjects in Beijing (128 $\mu\text{g/g}$ of creatinine, $p < 0.01$). There were eight cases with values (481—1 444 $\mu\text{g/g}$ of creatinine) which exceeded the upper normal limit in Beijing (420 $\mu\text{g/g}$ of creatinine) (6), but in only one of these cases was the total urinary protein level high. Five of the eight cases had an SDS-PAGE pattern that was of the low- or marginal low-molecular weight type.

Discussion

Cadmium occurs naturally in association with zinc. The ratio of cadmium to zinc is about 1 : 100—1 : 1 000 in ore and soil. Cadmium is not mined directly, but generally metallic cadmium is the by-product of zinc, copper, and lead smelting. The cadmium-refining procedures reported in this paper are the same as those used in other plants in the People's Republic of China.

Because cadmium is an element with a long-term biological half-time in the body, its hazardous effects in the work environment cannot be evaluated by the present hygienic conditions only. Though the airborne cadmium concentrations in this plant were not very high, they were higher than the health-based occupational exposure limit for long-term exposure (10 $\mu\text{g/m}^3$) recommended by the World Health Organization (WHO) (8), and they had been much higher earlier.

WHO (8) has recommended a blood (whole) cadmium level of 10 $\mu\text{g/l}$ (89.0 nmol/l) and a urinary cadmium concentration of 10 $\mu\text{g/g}$ of creatinine as the individual critical level if the exposure is regular and long-term. Since the aforementioned blood and urinary values of cadmium are levels at which renal effects have been observed, and in view of the long biological half-time of cadmium, WHO recommends that control measures be applied as soon as the individual concentrations of cadmium in the urine and/or blood exceed 5 $\mu\text{g/g}$ of creatinine and 5 $\mu\text{g/l}$ (44.5 nmol/l), respectively. In our study 80 and 31 % of the workers had blood and urinary levels of cadmium of more than 10 $\mu\text{g/l}$ (89.0 nmol/l) and 10 $\mu\text{g/g}$ of creatinine, respectively, and 91 and 51 % had corresponding values of more than 5 $\mu\text{g/l}$ (44.5 nmol/l) and 5 $\mu\text{g/g}$ of creatinine, respectively. According to the results of the urinary and blood determinations used as indicators for biological monitoring, the possibility of a potential cadmium hazard in this plant is apparent. It is generally believed that the systemic chronic effects of cadmium are better evaluated by complementary determinations of the blood and urinary cadmium levels, which mainly reflect exposure and body burden, respectively.

Pulmonary emphysema and renal tubular proteinuria are the main clinical manifestations of chronic occupational cadmium poisoning. Epidemiologic studies have shown that pulmonary changes are usually minimal in long-term low-level exposure to cadmium (2, 8). No significant changes were found in the lungs of the workers in the present study.

Friberg et al (2) pointed out that the best way to evaluate individual renal damage accurately may be to determine the total amount and electrophoretic pattern of protein, as well as the beta-2-microglobulin level in the urine, simultaneously. In our study there were nine cases (13 % of the total) with low- or marginally low-molecular weight patterns in the SDS-PAGE and/or high beta-2-microglobulin values which

suggested mild tubular damage. These nine cases had been exposed to cadmium for 2.5—17.2 years with a mean exposure duration of 11.6 years. The blood and urinary cadmium levels ranged from 15.75 to 70.00 $\mu\text{g/l}$ (140.1 to 622.8 nmol/l) and from 4.92 to 13.50 $\mu\text{g/g}$ of creatinine, respectively. Urinary cadmium values of $< 10 \mu\text{g/g}$ of creatinine were seen in four cases. According to this study in medical monitoring, the level of 5 $\mu\text{g/g}$ of creatinine should be regarded as a critical value for cadmium in urine.

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References

1. Chen MF, Zhang QY, Hu MS, Huang MX, Xu LF, Zhang HZ. An investigation of urinary protein excretion with sodium dodecyl sulfate polyacrylamide gel electrophoresis [in Chinese]. *Chin J Intern Med* 18 (1979) 326—329.
2. Friberg L, Nordberg GF, Vouk VB, ed. Handbook on the toxicology of metals. Elsevier/North-Holland, Amsterdam 1979, pp 356—381.
3. Huang JX, Liu YZ. Studies on urinary protein determination by improved biuret method [in Chinese]. *J Inst Health* (in press).
4. Huang JX, Liu YZ, Wang BY. Significance of blood and urine cadmium determinations in assessment of occupational cadmium hazard [in Chinese]. *J Inst Health* 13 (1984): 2, 17—20.
5. Huang JX, Wang BY. A study on determining cadmium in urine and blood by ASV method [in Chinese]. *Occup Med* 10 (1983): 1, 22—25.
6. Huang JX, Wang BY, Liu YZ. A preliminary survey on urinary β_2 -microglobulin concentration of 60 normal adults [in Chinese]. *Chin J Ind Hyg Occup Dis* 1 (1983) 74—75.
7. Piscator M. Proteinuria in chronic cadmium poisoning: II The applicability of quantitative and qualitative methods of protein determination for the demonstration of cadmium proteinuria. *Arch Environ Health* 5 (1962) 325—332.
8. World Health Organization. Recommended health-based limits in occupational exposure to heavy metals. Geneva 1980, pp 21—35. (Technical report series 647).
9. Xu BH, Han KQ, Xiao HR. Sampling with micro-pore filter and determination of metal compounds in air by atomic absorption spectrometry [in Chinese]. *J Inst Health* 13 (1984): 1, 5—10.