Scand J Work Environ Health 13 (1987) 56–61

Lead exposure during demolition of a steel structure coated with lead-based paints

II Reversible changes in the conduction velocity of the motor nerves in transiently exposed workers

by Hans Muijser, PhD, Elisabeth MG Hoogendijk, PhD, Jacob Hooisma, PhD, Divera AM Twisk, PhD (Psych)[1]

MUIJSER H, HOOGENDIJK EMG, HOOISMA J, TWISK DAM. Lead exposure during demolition of a steel structure coated with lead-based paints: II Reversible changes in the conduction velocity of the motor nerves in transiently exposed workers. *Scand J Work Environ Health* 13 (1987) 56–61. In a group of workers exposed to high levels of lead during five months nerve conduction velocity parameters were evaluated at the termination of exposure, and also three and fifteen months later. At the termination of exposure the mean blood lead level was 4.0 μmol/l, and motor conduction velocities in the median and the ulnar nerves were slower and the distal latencies in the median nerve were longer compared to the values measured 15 months later. Sensory conduction velocities, measured distally in the same nerves, were not depressed compared to the values measured three or fifteen months later. It was tentatively concluded that the effect of lead on the conduction velocity of the motor nerves has an initial reversible phase, dependent on the duration of exposure.

Key terms: demolishment workers, median nerve, subclinical neuropathy, ulnar nerve.

The toxic effect of chronic exposure to lead on the peripheral nervous system in man has been studied extensively in epidemiologic investigations (2, 3, 4, 5, 6, 8, 10, 11, 12, 13, 14, 15, 16, 17, 18, 20). On the basis of such investigations it is generally agreed that chronic exposure to lead can reduce the conduction velocity of the fastest fibers in the motor nerves in both the arms and legs, although some disagreement persists about the minimum level of lead exposure leading to such an effect.

Investigations on the effects of transitory exposure to lead on the peripheral nervous system are scarce. To the best of our knowledge only studies tangentially connected to this subject have been published. (See references 1, 7 and 9). In a recent study, Spee & Zwennis (19) reported that workers involved in the demolition of a large steel bridge showed a sharp rise in blood lead levels. Thus it was decided to evaluate the peripheral nerve conduction of these workers. The present report describes the results of these measurements.

Subjects and methods

Demolition workers

The bridge was demolished by a working party composed of eight men. Initially the party was assisted by a ninth worker, number 8 in the paper of Spee & Zwennis (19), but only for a short period, and that particular worker was therefore not included in the study. All eight consented to take part in the investigation. The mean age was 38.6 (SD 8.1) years. (See table 3 in the Results section.) Exposure to lead originated from the lead-based paint which was burnt in the process of cutting the bridge into parts. The demolition of the bridge, and thus the exposure to lead, lasted five months. Towards the completion of the hob two workers complained about gastrointestinal symptoms indicative of lead intoxication. Further details regarding exposure have been presented by Spee & Zwennis (19).

Referents

Nine volunteers (male laboratory workers) comprised a reference group. They were not occupationally exposed to lead nor did they suffer from any known diseases predisposing to peripheral neuropathy. The mean and standard deviation of age in the reference group, 38.3 years and 8.0 years, respectively (table 1), were similar to those of the lead-exposed group.

Electrophysiology

The nerve conduction velocity of the fastest motor fibers of the median and ulnar nerves were measured from the elbow to the wrist. The distal motor latencies of both nerves were recorded as well. In addition the conduction velocity of the fastest sensory fibers of the median and ulnar nerves was measured antidromically from the wrist to the ring finger. Convention-

[1] Medical Biological Laboratory TNO, PO Box 45, 2280 AA Rijswijk, The Netherlands.

Reprint requests to: Dr H Muijser, Medical Biological Laboratory TNO, PO Box 45, 2280 AA Rijswijk, The Netherlands.

al methods, using skin electrodes, were employed. The nerves were stimulated with constant current pulses from a unit (IS/C, Medelec) triggered by the electromyograph (MS 91, Medelec). Conduction velocity parameters are dependent on the temperature of the nerves. When the *skin* temperature is above 30°C on the lower arm and above 27°C on fingertips, however, conduction velocity parameters are not dependent on *skin* temperature (17). Ashby (2) gives slightly higher values, above 31°C at the wrist and above 33°C at the elbow. To assure this condition, the hand was warmed up to the wrist in water of 39°C for 3 min before the measurement. During the measurement the hand and the lower arm were prevented from cooling by the heat delivered by two infrared heaters.

Blood lead levels

The blood lead content was determined by atomic absorption spectrophotometry. For further details the reader is referred to the report of Spee & Zwennis (19).

Design of the experiment

Nerve conduction velocity was monitored at the termination of exposure and at three and fifteen months postexposure. Preexposure values were not available. Since the number of people was rather small for the detection of changes in a comparison with a reference group, only longitudinal comparisons within the exposed group were carried out. In addition the stability of the nerve conduction velocity parameters over time was investigated. Therefore, the nerve conduction velocities of the subjects in the reference group were measured twice with an interval of one year.

The Wilcoxon signed rank test (two-tailed, $p<0.05$) was used to evaluate the significance of the results. A distribution-free test was used because exposure was not the same for each worker, so that, at least in principle, violation of the normality of the distribution of the parameters could be expected.

Results

Unexposed reference group

The individual results and the mean values of the nerve conduction velocities measured on the first occasion in the unexposed reference group are presented in table 1. Moreover the value at which a detoriation deviates by 2 SD units from the mean value is given for each parameter. Only one value exceeded these limits. One year later (table 2) four values exceeded these limits. Except for the sensory conduction velocity of the median nerve no statistically significant differences emerged in the two series.

Workers exposed to lead

The number of individual values exceeding the limits, as determined by the measurements of the reference group, decreased in the group exposed to lead after the exposure, ie, there were 11 at the termination of exposure (table 3), six at three months after the termination (table 4) and five at 15 months after the termination (table 5). A comparison between the values obtained at the termination of exposure (table 3) and three months later (table 4) did not show any significant differences with regard to the conduction velocity parameters. However, a comparison between the values obtained at the termination of exposure (table 3) and 15 months later (table 5) revealed a statistically significant increase in the conduction velocity of the motor fibers of the median and ulnar nerves, being 2.8 and 3.4 m/s, respectively, and a significant decrease (which is also an improvement) in the distal latency of the median nerve (by 0.37 ms). Significant changes in the sensory conduction velocity of the median and ulnar nerves and in the distal motor latency of the ul-

Table 1. Individual values and the means of the conduction velocities and distal latencies of the referents. The value in italics deviate by more than 2 SD units of the mean.

Subject	Age (years)	Median nerve			Ulnar nerve		
		Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)
1	39	61.4	2.96	56.6	57.4	2.56	51.0
2	54	56.4	3.52	51.8	51.3	*2.96*	54.9
3	25	61.8	2.64	60.5	66.7	2.32	56.8
4	39	57.0	3.12	54.9	54.6	2.64	56.8
5	43	65.6	3.04	52.7	63.2	2.40	52.7
6	42	59.6	3.44	46.5	53.2	2.64	50.7
7	34	61.7	3.28	50.0	61.3	2.64	53.3
8	33	63.2	3.76	49.0	57.4	2.56	53.3
9	36	64.4	3.36	53.6	54.3	2.56	54.9
Mean	38.3	61.2	3.24	52.8	57.7	2.59	53.8
SD	8.0	3.1	0.33	4.2	5.1	0.18	2.2
Deviation (deterioration) by 2 SD units from the mean		55.0	3.90	44.4	47.5	2.95	49.4

Table 2. Individual values and means of the conduction velocities and distal latencies of the referents one year after the measurements displayed in table 1. The values in italics deviate by more than 2 SD units from the means in table 1.

Subject	Median nerve			Ulnar nerve		
	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)
1	64.0	2.80	52.9	57.7	2.24	53.6
2	56.25	3.20	48.2	*45.5*	*3.04*	50.3
3	64.7	2.48	52.4	63.9	2.24	54.2
4	56.5	2.96	54.4	52.9	2.72	53.6
5	62.7	3.04	45.2	59.9	2.40	51.2
6	61.25	3.52	*41.7*	55.6	2.88	*48.1*
7	63.4	3.28	46.8	62.5	2.56	54.3
8	66.1	3.52	47.8	57.8	2.24	52.4
9	60.2	3.28	59.4	55.9	2.56	52.3
Mean	61.7	3.12	48.9	56.9	2.54	52.2
SD	3.5	0.34	4.1	5.5	0.29	2.1

Table 3. Individual values and the means of the conduction velocities and distal latencies of the lead-exposed workers, measured at the termination of exposure. The values in italics deviate by more than 2 SD units from the reference means (table 1).

Subject[a]	Age (years)	Median nerve			Ulnar nerve			Blood lead concentration (μmol/l)
		Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	
1	41	58.9	3.28	56.8	49.0	*2.96*	53.6	4.98
2	40	58.0	3.76	*44.0*	52.4	*3.12*	*48.5*	4.46
3	48	60.5	*4.48*	48.6	57.2	2.64	*48.1*	4.34
4	30	*52.5*	*4.32*	47.3	56.5	2.56	50.0	4.19
5	34	56.3	3.20	58.9	55.3	2.40	54.1	4.95
6	41	60.2	3.68	54.0	58.1	2.88	54.3	3.33
7	49	57.9	*3.92*	48.1	51.9	2.88	53.6	3.17
9	26	55.9	*4.24*	55.6	49.0	*3.12*	54.1	2.45
Mean	38.6	57.5	3.86	51.7	53.7	2.82	52.0	3.98
SD	8.1	2.6	0.47	5.3	3.6	0.26	2.7	0.91

[a] The numbers correspond to the numbers of the subjects in the paper of Spee & Zwennis (19).

Table 4. Individual values and the means of the conduction velocities and distal latencies of the lead-exposed workers, measured three months after the termination of exposure. The values in italics deviate by more than 2 SD units from the reference means (table 1).

Subject[a]	Median nerve			Ulnar nerve			Blood lead concentration (μmol/l)
	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	
1	55.9	3.36	52.1	*44.5*	*4.08*	51.3	2.60
2	60.9	3.84	52.9	50.7	2.8	54.9	3.01
3	56.4	3.28	52.1	55.8	*3.00*	*48.4*	2.37
4	61.9	3.60	48.1	55.8	2.32	55.4	2.40
5	59.4	3.52	57.3	54.7	2.56	54.3	3.04
6	58.6	*3.92*	57.3	57.1	2.80	53.1	2.11
7	57.2	3.84	48.1	57.1	2.72	54.7	2.31
9	63.5	*3.92*	60.7	60.3	2.56	62.5	1.58
Mean	59.2	3.66	53.6	54.5	2.86	54.3	2.43
SD	2.7	0.26	4.5	4.9	0.54	4.0	0.48

[a] The numbers correspond to the numbers of the subjects in the paper of Spee & Zwennis (19).

nar nerve were not detected. The mean values of both the reference and the exposed group on all occasions are summarized in table 6. Three months after the exposure the measures for which a significant improvement had been established were about intermediate between the values at the termination of exposure and the values 15 months after exposure. Notwithstanding the small number of subjects involved, these latter values were strikingly similar to the mean values of the reference group.

Table 5. Individual values and the means of the conduction velocities and distal latencies of the lead-exposed workers, measured 15 months after the termination of exposure. The values in italics deviate by more than 2 SD units from the reference means (table 1).

Subject[a]	Median nerve			Ulnar nerve			Blood lead concentration (μmol/l)
	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	Motor conduction velocity (m/s)	Distal motor latency (ms)	Sensory conduction velocity (m/s)	
1	61.4	3.28	46.0	54.2	*2.96*	*49.2*	1.30
2	60.5	3.60	50.3	54.7	2.72	52.1	1.54
3	62.3	3.68	50.0	56.0	2.40	*47.3*	1.37
4	60.1	3.20	*43.4*	59.9	2.48	50.8	1.06
5	59.2	3.44	50.8	55.3	*3.04*	50.8	2.66
6	62.5	3.44	51.5	59.7	2.64	52.7	1.24
7	57.6	3.68	45.1	56.8	2.64	50.8	1.29
9	58.7	3.60	56.8	60.5	2.56	58.8	0.70
Mean	60.3	3.49	49.2	57.1	2.68	51.6	1.40
SD	1.7	0.18	4.3	2.5	0.22	3.4	0.57

[a] The numbers correspond to the numbers of the subjects in the paper of Spee & Zwennis (19).

Table 6. Means and standard deviations of the measured values.

	Number	Median nerve						Ulnar nerve						Blood lead concentration (μmol/l)	
		Motor conduction velocity (m/s)		Distal motor latency (ms)		Sensory conduction velocity (m/s)		Motor conduction velocity (m/s)		Distal motor latency (ms)		Sensory conduction velocity (m/s)			
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Referents															
At first examination	9	61.2	3.1	3.24	0.33	52.8	4.2	57.7	5.1	2.59	0.18	53.8	2.1	..	..
One year after first examination	9	61.7	3.5	3.12	0.34	48.9	4.1	56.9	5.5	2.54	0.29	52.2	2.1	..	..
Exposed workers															
At termination of exposure	8	57.5	2.6	3.86	0.47	51.7	5.3	53.7	3.6	2.82	0.26	52.0	2.7	3.98	0.91
Three months after exposure termination	8	59.2	2.7	3.66	0.26	53.6	4.5	54.5	2.7	2.86	0.26	54.3	4.5	2.43	0.48
Fifteen months after exposure termination	8	60.3	1.7	3.49	0.18	49.2	4.3	57.1	2.5	2.68	0.22	51.6	3.4	1.40	0.57

Discussion

With regard to the results of the exposed workers, it was found that 15 months after the termination of exposure the conduction velocities of the fastest motor fibers of the median and ulnar nerves and the distal motor latency of the median nerve had improved in comparison with the values obtained immediately after the termination of exposure. An obvious interpretation is that, because of exposure to lead, the conduction velocities deteriorated and that, in the following period without exposure, the values returned to normal. However, the existence of a small irreversible change cannot be excluded. On the basis of the data reported by Corsi et al (7), obtained in a group of seven patients formerly intoxicated with lead for less than three and a half months, such an irreversible change cannot be excluded either. Yet both in the results of Corsi et al (7) and in the present results (table 2 and table 5) the mean values of the workers, long after the termination of exposure, and those of the referents are strikingly smilar. These similarities should not simply be dismissed, but should be taken instead as a hint that an irreversible effect of transitory exposure is less likely.

Interestingly, in a different group of patients exposed to lead for at least six months, Corsi et al (7) could still demonstrate a significant motor conduction deficit after exposure had been terminated for at least three years and lead had been removed by chelation from the body at least two years before the measurement. The deficit was 4.9 m/s and 3.8 m/s in the ulnar and peroneal nerves, respectively.

It can tentatively be concluded that the toxic effect of lead on the peripheral nervous system has two phases possibly caused by different mechanisms. Exposure to lead during less than a few months leads to an at least partly, if not completely, reversible decrease in motor conduction velocity. However, exposure to lead for at least six months can lead to a permanent decrease in motor conduction velocity.

Araki et al (1) have reported that chelation therapy leads to an increase of the nerve conduction velocity

in some, but not all, patients with lead intoxication. In most cases the increase in the conduction velocity was recorded within a month after chelation. It can be inferred that, if a decrease in the nerve conduction velocity due to exposure to lead can be reversed, the reverse is rather fast. It seems plausible that the delay found in the improvement of the nerve conduction velocity in the present study was simply due to the time course of the removal of lead from the body, rather than it being associated with structural repair in the nerves.

Feldman et al (9) have investigated workers dismantling an elevated train network. Because of the burning of lead-based paint, the blood lead content increased to a mean of 4.0 μmol/l in 32 burner workers and to a mean of 2.4 μmol/l in 12 nonburner workers. A mean decrease of 5.8 m/s was found in the motor conduction velocity of the peroneal nerve in a comparison of 13 burner workers with six nonburner workers. The decrease was larger than the changes found in the present study for the motor fibers of the median (2.8 m/s) and ulnar (3.4 m/s) nerves. Feldman et al (9) did not state clearly how long exposure lasted; however the authors did report that symptoms of increased bodyburden appeared for lead after a period of exposure as short as one month. Workers with such symptoms were chelated. Unfortunately Feldman et al (9) did not report on the recovery of the nerve conduction velocity after chelation nor after the termination of exposure.

The reproducibility of the motor conduction velocity parameters and the distal sensory conduction velocity of the ulnar nerve in the reference group was satisfactory, ie, the differences were not significant. However the reproducibility of the distal sensory conduction velocity of the median nerve was poor in that a statistically significant difference existed. The lack of a difference in the other distal parameters indicates that a difference in temperature of the hand can be ruled out as a cause. Except chance, no other cause seems present.

In our experiment the distal conduction velocity of the sensory fibers did not increase in the recovery period after the short-term, but relatively high exposure. (The biological limit recommended for blood lead content by the countries of the European common market is 3.4 μmol/l.) Singer et al (18) found that, in a group of workers exposed to lead for less than 10 years, only a decrease in the (distal) conduction velocity of the sensory nerves could be found, whereas in a group of workers in the same factory, exposed longer than 10 years, a decrease in the conduction velocity of the motor nerves could be demonstrated as well. Such findings suggest that a decrease in conduction velocity develops earlier in sensory fibers than in motor fibers. Seppäläinen et al (17) also found evidence supporting this possibility. Our results indicate that a reversible decrease in the distal conduction velocity of the sensory nerves after exposure to lead during months rather than years cannot be demonstrated at all, but a limited irreversible decrease cannot be excluded.

It might be contended that the present result, ie, a transitory decrease in motor conduction velocity in the exposed workers, was caused by a difference in the mean temperature of the upper extremities at the time of the subsequent measurements. However the absence of a change in the distal sensory conduction velocity does not support such a contention.

In conclusion, it was found that after the termination of a transitory exposure to lead the motor conduction velocity and the distal motor latency, but not the distal sensory conduction velocity, improved in the workers involved. It was argued that a reversible component was present in the decrease of motor conduction velocity resulting from transitory exposure to lead and that sensory conduction velocity is probably not affected under these conditions.

References

1. Araki S, Honma T, Yanagihara S, Ushio K. Recovery of slowed nerve conduction velocity in lead-exposed workers. Int Arch Occup Environ Health 46 (1980) 151–157.
2. Ashby JAS. A neurological and biochemical study of early lead poisoning. Br J Ind Med 37 (1980) 133–140.
3. Baker EL, Feldman RG, White RA, Harley JP, Niles CA, Dinse GE, Berkey CS. Occupational lead neurotoxicity: A behavioural and electrophysiological evaluation. Study design and year one results. Br J Ind Med 41 (1984) 352–361.
4. Bordo BM, Filippini G, Massetto N, Musicco M, Boeri R. Electrophysiological study of subjects occupationally exposed to lead and with low levels of lead poisoning. Scand J Work Environ Health 8 (1982): suppl 1, 142–147.
5. Buchthal F, Behse F. Electrophysiology and nerve biopsy in men exposed to lead. Br J Ind Med 36 (1979) 135–147.
6. Catton MJ, Harrison MJG, Fullerton PM, Kazantzis G. Subclinical neuropathy in lead workers. Br Med J 2 (1970) 80–82.
7. Corsi G, Bartolucci GB, Fardin P, Negrin P, Manzoni S. Biochemical and electrophysiological study of subjects with a history of past lead exposure. Am J Ind Med 6 (1984) 281–290.
8. Englert N. Periphere motorische Nervenleitgeschwindigkeit bei Probanden mit beruflicher Blei-Exposition. Arbeitzmed Sozialmed Präventivmed 15 (1980) 254–260.
9. Feldman RG, Lewis J, Cashins R. Subacute effects of lead-oxide fumes in demolition workers. Lancet 1 (1977) 89–90.
10. Jeyaratnam J, Devathasan G, Ong CN, Phoon WO, Wong PK. Neuropsyhiological studies on workers exposed to lead. Br J Ind Med 42 (1985) 173–177.
11. Nielsen CJ, Nielsen VK, Kirkby H, Gyntelberg F. Absence of peripheral neuropathy in long-term lead-exposed workers. Acta Neurol Scand 65 (1982) 241–247.
12. Paulev PE, Gry C, Dossing M. Motor nerve conduction velocity in asymptomatic lead workers. Int Arch Occup Environ Health 43 (1979) 37–43.
13. Rosén I, Wildt K, Gullberg B, Berlin M. Neurophysiological effects of lead exposure. Scand J Work Environ Health 9 (1983) 431–441.
14. Seppäläinen AM, Hernberg S. Sensitive technique for detecting subclinical lead neuropathy. Br J Ind Med 29

(1972) 443 – 449.
15. Seppäläinen AM, Hernberg S. Subclinical lead neuropathy. Am J Ind Med 1 (1980) 413 – 420.
16. Seppäläinen AM, Hernberg S. A follow-up study of nerve conduction velocities in lead exposed workers. Neurobehav Toxicol Teratol 4 (1982) 721 – 723.
17. Seppäläinen AM, Hernberg S, Vesanto R, Kock B. Early neurotoxic effects of occupational lead exposure: A prospective study. NeuroToxicology 4 (1983) 181 – 192.
18. Singer R, Valciukas JA, Lilis R. Lead exposure and nerve conduction velocity: The differential time course of sensory and motor nerve effects. NeuroToxicology 4 (1983) 193 – 202.
19. Spee T, Zwennis WCM. Lead exposure during demolition of a steel structure coated with lead-based paints: I Environmental and biological monitoring. Scand J Work Environ Health 13 (1987) 52 – 55.
20. Triebig G, Weltle D, Valentin H. Investigations on neurotoxicity of chemical substances at the workplace: V Determination of the motor and sensory nerve conduction velocity in persons occupationally exposed to lead. Int Arch Occup Environ Health 53 (1984) 189 – 204.

Received for publication: 12 September 1986