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Reversible nerve lesions after accidental polychlorinated biphenyl exposure

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SEPPÄLÄINEN AM, VUOJOLAHTI P, ELO O. Reversible nerve lesions after accidental polychlorinated biphenyl exposure. *Scand J Work Environ Health* 11 (1985) 91–95. Several capacitors containing polychlorinated biphenyls (PCB) exploded in a cardboard plant. During the accident, and in the clearing work, several workers were in probable contact with PCB and/or its degradation products. Nausea, intense perspiration, and headache were acute symptoms, which cleared quickly. The 15 men with the greatest exposure were studied neurophysiologically twice, namely, two and six months after the explosion. Motor conduction velocities (MCV) of the right median, ulnar, and peroneal nerves; sensory conduction velocity (SCV) of the right sural nerve; and distal SCVs of the right median and ulnar nerves were measured with skin electrodes. Thirty male workers with a similar age distribution served as referents. Two months after the explosion all SCVs and distal SCVs were slower in the exposed men, and still six months after the explosion the distal SCV of the ulnar nerve and the SCV of the sural nerve were slightly slower among the exposed. However, clear improvement occurred in the distal SCVs during the follow-up. As in many toxic distal axonopathies, the distal SCVs were reversibly impaired after an accidental exposure to PCB fumes. PCB seem to exhibit neurotoxic properties in humans.

Key terms: nerve conduction velocity, neuropathy.

On 3 August 1982, at 0010, capacitors exploded in a cardboard plant in southeastern Finland. There was a total electricity block in the whole plant, and the machinery stopped. There were 96 capacitors in the explosion area, and 15 of them were destroyed. The capacitors contained polychlorinated biphenyls (PCB), namely, Clophen A 30 (43 % chlorine), mainly di-, tri- and tetrachlorobiphenyls. It is estimated that, at the most, 300 l of PCB escaped and 100 of the 300 l underwent pyrolysis degradation. The first air concentrations were measured about 5.5 h after the explosion and the highest concentrations at that time were 8 000–16 000 $\mu\text{g}/\text{m}^3$. Detailed information about the exposure has been published elsewhere (4). Black smoke and soot spread to the neighboring rooms. The firemen of the plant had to search for the explosion site for half an hour within the smoke. The clearing workers became nauseated in an hour and a half after the explosion. Intense perspiration and headache were other symptoms. Ten of the workers were admitted to the local hospital. The symptoms disappeared quickly without treatment. Later some of the workers complained of odd feelings, pin and prick sensations, itching, or odd temperature feelings in their arms or legs.

Neurotoxic effects of PCB have not been extensively studied, although central nervous system symptoms have been mentioned in a study of PCB-exposed workers (14). Quite recently neuropathic symptoms and signs were reported among patients

poisoned by ingested PCB (3, 8). Therefore we thought it relevant to determine if PCB exposure affects peripheral nerves.

Subjects

Altogether 16 men were considered to have been extensively exposed to PCB. Some had been working in the adjacent areas, and most had participated in the clearing work. All these workers were invited to neurophysiological tests conducted in the plant clinic on two occasions, on 1 October 1982 and 9 February 1983, consequently about two and six months after the explosion, respectively. All the 16 men participated in the second examination; one could not participate in the first.

Methods

Several nerve conduction velocities were measured with a portable Disa electromyograph and skin electrodes, namely, the maximal motor conduction velocity (MCV) of the median, ulnar, and deep peroneal nerves on the right (15) and the distal sensory conduction velocity (dSCV) of the right median and ulnar nerves. The latter measurements were made antidromically with strip electrodes around the second and fifth fingers for recording. The sensory conduction velocity (SCV) of the sural nerve was measured by stimulating at the midcalf and recording at the lateral malleolus. The latencies were read directly from the oscilloscope and the scale of the electromyograph, and the amplitudes of the sensory action potentials were measured from photographic recordings.

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For a comparison of the results we chose 30 male workers from the reference series of previous studies conducted at the Institute of Occupational Health, Helsinki (10, 11, 12). The referents were chosen so that their age at the test date was within one year of

that of the exposed. For the youngest exposed worker (18 years) we could find only referents with the age of 22 years. The exposed worker who participated in only one examination was omitted from the final statistics. The mean age of the exposed workers was

Table 1. Motor (MCV), sensory (SCV), and distal sensory (dSCV) conduction velocities in 15 workers accidentally exposed to polychlorinated biphenyl fumes two months earlier and in 30 age-matched referents. (NS = not significant)

Nerve	Exposed (N = 15)		Unexposed (N = 30)		t_{14}	p-Value
	Mean	SD	Mean	SD		
Median nerve						
MCV	57.9	4.2	58.8	3.8	— 0.7	NS
dSCV	46.2	3.2	49.5	4.8	— 3.0	0.01
Ulnar nerve						
MCV	58.2	5.0	58.1	4.1	0.0	NS
dSCV	41.8	4.8	54.9	6.5	—10.6	0.001
Peroneal nerve						
MCV	49.9	5.9	53.0	5.8	— 1.6	NS
Sural nerve						
SCV	37.6	3.8	44.2	4.0	— 5.6	0.001

Table 2. Motor (MCV), sensory (SCV), and distal sensory (dSCV) conduction velocities in 15 workers accidentally exposed to polychlorinated biphenyl fumes six months earlier and in 30 age-matched referents. (NS = not significant)

Nerve	Exposed (N = 15)		Unexposed (N = 30)		t_{14}	p-Value
	Mean	SD	Mean	SD		
Median nerve						
MCV	57.8	4.3	58.8	3.8	—1.2	NS
dSCV	51.3	6.4	49.5	4.8	1.1	NS
Ulnar nerve						
MCV	59.4	4.6	58.1	4.1	0.8	NS
dSCV	44.7	4.0	54.9	6.5	—7.6	0.001
Peroneal nerve						
MCV	52.5	4.3	53.0	5.8	0.3	NS
Sural nerve						
SCV	40.0	3.2	44.2	4.0	—3.4	0.01

Table 3. Comparison of nerve conduction velocities and sensory action potential amplitudes (A) measured on two occasions in 15 workers accidentally exposed to polychlorinated biphenyl fumes. (MCV = motor conduction velocity, dSCV = distal sensory conduction velocity, SCV = sensory conduction velocity, NS = not significant)

Nerve	Two months after exposure		Six months after exposure		t_{14}	p-Value
	Mean	SD	Mean	SD		
Median nerve						
MCV	57.9	4.2	57.8	4.3	—0.01	NS
dSCV	46.2	3.2	51.3	6.4	2.9	0.02
A	27.3	8.8	35.3	14.9	2.8	0.02
Ulnar nerve						
MCV	58.2	5.0	59.4	4.6	0.8	NS
dSCV	41.8	4.8	44.7	4.0	2.5	0.05
A	16.3	7.9	28.6	11.2	5.1	0.001
Peroneal nerve						
MCV	49.9	5.9	52.5	4.3	2.3	0.05
Sural nerve						
SCV	37.6	3.8	40.0	3.2	1.7	NS

42.1 (range 18–57) years, and that of the unexposed 42.1 (range 22–56) years.

Statistical methods for comparing the exposed and unexposed men included the t-test for matched samples with an 1:k matching ratio (7). In comparing the results of the two consecutive tests performed on the exposed workers, we applied the t-test for mean paired difference. Amplitude measures were not sufficiently available from the referents. Statistical comparisons of these measures could thus be performed only concerning the two consecutive tests of the exposed workers.

Results

All the nerve conduction velocities were measurable for all the exposed subjects on both occasions. The amplitudes of the sensory action potential were at times low, especially at the first measurement, but always distinct enough to allow reliable measurement. The MCVs of the arm nerves were similar among both groups in both measurements. The MCV of the peroneal nerve was slightly slower among the exposed men in the first measurement, but the difference was not statistically significant (table 1). The dSCVs of the median and ulnar nerves and the SCV of the sural nerve were slower for the exposed, and the difference was statistically highly significant for the first measurement (table 1). Even in the second measurement the dSCV of the ulnar nerve and the SCV of the sural nerve continued to be slower for the exposed workers (table 2). When comparing the results of the two consecutive measurements, we noted that the MCVs of the median and ulnar nerves did not change, whereas the MCV of the peroneal nerve increased (table 3). Similarly, the dSCVs of the median and ulnar nerves increased significantly (table 3, figure 1), and the amplitude of the sensory action potential also increased (figures 2 and 3). The increase

Figure 1. Means and standard deviations of the distal sensory conduction velocity (dSCV) of the median nerve of 30 referents (R) and 15 workers two (X/82) and six (II/83) months after an explosion exposing the workers to polychlorinated biphenyls. The statistically significant differences are marked.

Figure 2. Sensory action potentials of the median nerve of a 43-year-old worker two (X/82) and six (II/83) months after acute polychlorinated biphenyl exposure.

Figure 3. Means and standard deviations of the sensory action potential amplitude (SAP) of the median and ulnar nerves of 15 workers two (X/82) and six (II/83) months after acute polychlorinated biphenyl exposure. The amplitudes increased significantly (t-test).

Figure 4. The polychlorinated biphenyl (PCB) concentrations of serum from workers accidentally exposed to polychlorinated biphenyl fumes on 3 August 1982. The number of samples studied varied between 11 and 16 on the different occasions. (NG/G = nanograms per gram, SEPT = September, DEC = December, FEB = February, MAX = maximum, MED = median, MIN = minimum)

in the SCV of the sural nerve was not statistically significant. Only four individual workers did not show any increase in their conduction velocities. All of the four had perfectly normal conduction velocities already in the initial examination, whereas, of the 11 others, 7 had slightly abnormally slow conduction velocities in one or two nerves at the time of the initial examination. In the second examination all the individual conduction velocities were within the normal limits of the laboratory (10).

Blood was drawn from 11 persons from the present series during the first 3 d after the explosion, and later on from all 16 subjects. The PCB concentrations in the blood decreased quickly. The highest PCB concentrations were measured 3 d after the explosion; they decreased to baseline levels within one to two months (figure 4). The data on individual PCB levels are limited, and the variation of individual PCB levels, especially in the later measurements, is narrow. Consequently no clear correlations between the PCB levels and the nerve conduction velocities were found. Possible polychlorinated dibenzofurans were below the detection level in the blood samples drawn from seven workers in the morning after the explosion.

Discussion

A reversible impairment of the peripheral nerves was noted among workers accidentally exposed to PCB and their degradation products. The PCB com-

pounds contained mainly two to four chlorine atoms. The soot samples also contained products of pyro-synthesis, eg, tetrachlorodibenzofurans, monochloropyrenes, and dichloropyrenes (4). In the most polluted area the soot contained a tetrachlorodibenzofuran content of 90 $\mu\text{g/g}$, and the concentration of the most toxic 2, 3, 7, 8-isomer was 6.5 $\mu\text{g/g}$. The impairment was slight, and it was noted mainly in the distal portions of sensory nerves, where the conduction velocity was decreased and the amplitude of the sensory action potentials was also decreased. Although the MCV of the peroneal nerve did not differ statistically significantly from that of the referents, some reversible changes occurred also in that the MCV improved significantly during the follow-up period.

Earlier studies by Okumura (8) and Chia & Chu (3) on patients with PCB poisoning after the ingestion of contaminated cooking oil in Japan and Taiwan have reported symptoms and signs of mainly sensory neuropathy. Chia & Chu (3) also found an abnormal slowing of the MCV of the tibial and ulnar nerves in some of their patients. In their series the mean motor and sensory conduction velocities were slower for patients than for normal referents. These measurements were taken one to two years after the intake of the contaminated cooking oil, but the blood PCB levels were still high around that time, mean 34.6 parts per billion. In addition polychlorinated dibenzofurans were detected in the cooking oil samples in Taiwan, and these compounds were considered to play an important role in the etiology of the PCB poisoning (2).

The differences in nerve conduction velocities between the patients and the referents in the Taiwanese studies were of an order similar to that of the present study, performed two months after acute exposure. The reversibility of the findings was, however, remarkable in the present study. In the Seveso accident, where residents of the area were exposed to 2,3,7,8-tetrachlorodibenzodioxin (dioxin-TCDD), those subjects who developed chloracne also had significantly more often neuropathic signs in clinical and electrophysiological examinations than unexposed referents (5).

The impairment noted in the present investigation was typical of toxic distal axonopathy, in which there is symmetrical axonal degeneration beginning distally in long and large peripheral nervous system tracts (9). Similar electrophysiological findings have been described for patients with hexacarbon (1, 6), carbon disulfide (13), and acryl amide (16) neuropathy. Pathological studies of the peripheral nervous system in patients with PCB exposure have not come to our attention. PCB can cross the blood-brain barrier, since PCB have been detected in the brain of a still-born baby born to a Yusho mother, and reportedly also in the brain of adult Yusho patients (17). Various central nervous system symptoms have been reported among Yusho patients (17) and among American workers occupationally exposed to PCB (14). The subjects of the present study were exposed only for short periods (a few hours) although probably to a high peak exposure, and no permanent damage developed. PCB remained however in the body for some time, which evidently explains why some reversible nervous system impairment was detectable two months after the acute exposure.

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