

Effects of allyl chloride on occupationally exposed subjects¹

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HE F, ZHANG SL. Effects of allyl chloride on occupationally exposed subjects. *Scand J Work Environ Health* 11 (1985): suppl 4, 43–45. Effects of allyl chloride on occupationally exposed subjects were studied in two factories manufacturing sodium allyl sulfonate. Twenty-six subjects in factory A and 27 workers in factory B were exposed to allyl chloride at levels of 2.6–6 650 mg/m³ for 2.5 months–6 years and 0.2–25.13 mg/m³ for 1–4.5 years, respectively. Most subjects of factory A had weakness, paresthesia, and numbness in extremities with sensory impairment in the glove-stocking distribution, as well as reduced ankle reflexes. Electroneuromyography showed neurogenic abnormalities in 10 of the 19 subjects examined, the prevalence of neuropathy therefore being 52.6 %. Similar symptoms of workers in factory B were clinically much milder, and there were few abnormal neurological signs — yet electromyographic findings indicating mild neuropathy were found in 13 of the 27 subjects. No significant abnormalities of other organs were noted. Possible etiologic factors other than exposure to allyl chloride were excluded. All the evidence obtained indicates that chronic exposure to allyl chloride mainly causes toxic polyneuropathy. The neurotoxicity of allyl chloride has also been confirmed by experimental neuropathological studies.

Key terms: electroneuromyography, gas chromatography, neurological examinations, polyneuropathy.

Allyl chloride (3-chloropropene) is a volatile chlorinated hydrocarbon liquid at room temperature. It is commercially used as one of the raw materials for manufacturing epichlorohydrin, epoxy resin, glycerin, pesticides such as Cartap (Padan), and sodium allyl sulfonate, which is one of the monomers of polyacrylonitrile.

Previous observations on industrial exposure and animal studies reported in the literature indicate that allyl chloride causes irritation of the eyes and upper respiratory tract and produces a mild narcotic effect at high concentrations, while chronic exposure results in liver and kidney damage (4). No neurotoxic effect had been reported until two outbreaks of polyneuropathy without liver and kidney dysfunction occurred among workers exposed to allyl chloride in China in the early 1970s (2). The neurotoxicity of allyl chloride on the peripheral nervous system was then further confirmed by neuropathological studies in rabbits and mice (1, 2, 3).

This paper presents the effects of allyl chloride at different exposure levels on two groups of occupationally exposed subjects studied in two factories manufacturing sodium allyl sulfonate.

Material and methods

In both factories allyl chloride and sodium sulfite were mixed and heated at 40°C to synthesize sodium allyl

sulfonate. The production in factory A was begun in 1970 and was automated between 1975 and 1977, when production completely ceased. The atmospheric concentration of allyl chloride determined by gas chromatography ranged from 2.6 to 6 650 mg/m³. Twenty-six exposed subjects were examined in 1976, and follow-up studies were done for 10 of them in 1977 after the cessation of exposure. Factory B was built in 1978 with much better work conditions resulting in atmospheric concentrations of allyl chloride ranging between 0.2 and 25.13 mg/m³. Twenty-seven subjects from this factory were observed in 1982. The sex, age, and exposure duration of both groups of subjects are shown in table 1.

All the subjects were given detailed questionnaires about occupational history, symptoms, past illness, and family history. Physical and neurological examinations, visual acuity and visual field measurement, skin temperature measurement, rheography of the extremities, and electrocardiography were done. Electroneuromyography (ENMG) was carried out with a Disa 1500 type electromyographic system recording muscle potentials from the hands and legs and measuring the motor conduction velocity of the median, ulnar, and lateral popliteal nerves and the sensory conduction velocity of the median, ulnar, and sural nerves. Laboratory tests included routine blood and urine tests, liver function [serum glutamic pyruvic transaminase (SGPT), thymol turbidity test (TTT), and thymol flocculation test (TFT)], blood glucose, electrolytes, nonprotein nitrogen, erythrocyte sedimentation rate and the phenolsulfonphthalein test. Electroencephalography, the determination of basal metabolic rate and blood lactic dehydrogenase, and serum urine protein electrophoresis were also done for some of the subjects.

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Table 1. Age, sex, and exposure of the two exposed groups.

Group	Number of subjects	Age (years)	Sex		Exposure level of allyl chloride (mg/m ³)	Exposure duration
			Male	Female		
Factory A	26	20—51	—	26	2.6—6 650	2.5 months—6 years
Factory B	27	18—41	14	13	0.2—25.13	1 year—4.5 years

Table 2. Symptoms and signs found in the two exposed groups.

Symptom or sign	Factory A (26 subjects)		Factory B (27 subjects)	
	Number	Percentage	Number	Percentage
Symptoms				
Weakness of extremities	24	92.3	18	66.7
Cramping pain	17	65.3	5	18.5
Tingling sensation	15	57.6	9	33.3
Numbness in hands and feet	10	38.5	11	40.7
Coldness in hands and feet	8	30.7	8	29.6
Dizziness	6	23.1	10	37.0
Insomnia	7	26.9	5	18.5
Anorexia	5	19.2	1	3.7
Signs				
Distal sensory impairment				
Pain	17	65.3	4	14.8
Light touch	16	61.5	—	0
Vibration sensation	16	61.5	1	3.7
Position sensation	6	23.1	—	0
Decreased muscular strength	15	57.6	—	0
Diminished ankle reflexes	11	42.3	2	7.4
Tenderness in calf muscles	5	19.2	2	7.4
Enlarged liver	2	7.6	—	0

Table 3. Electromyographic changes found in the two exposed groups.

	Factory A (19 subjects)		Factory B (27 subjects)	
	Hands	Legs	Hands	Legs
Number of muscles sampled	20	34	39	27
Insertional activity				
Normal	20	34	39	27
Prolonged	—	—	—	—
Spontaneous potentials				
Fibrillation	3	5	—	—
Positive sharp waves	—	3	—	—
Motor unit potentials				
Duration				
Normal	20	34	12	25
Prolonged	—	—	27	2
Polyphasic potentials				
Normal	18	30	17	25
Increased	2	4	22	2
Recruitment of motor units				
Interference pattern	3	4	35	26
Reduced interference pattern	16	23	4	1
Discrete pattern	1	7	—	—

Results and discussion

All the subjects exposed to allyl chloride in factory A at 2.6—6 650 mg/m³ for 2.5 months—6 years complained of lacrimation and sneezing when they were initially exposed. Then the sensation of irritation of the eyes and upper respiratory tract gradually diminished. But, as shown in table 2, most of them developed weakness, a tingling sensation, cramping pain,

and numbness in the distal part of the extremities with the shortest latency period of two months.

On examination, two-thirds of this group showed symmetrical distal sensory deficits which mainly involved pain, light touch, and vibration sensation in the glove-stocking distribution. Distal muscular strength decreased to grade 3—4 of the MRC scale in 57 % of these subjects. Ankle reflexes were also diminished in 42 % of this group. No muscular atrophy was noted.

Table 4. Comparison of nerve conduction between a reference group and the two exposed groups.

Nerve examined	Reference group			Factory A			Factory B		
	Number examined	Mean	SD	Number examined	Mean	SD	Number examined	Mean	SD
<i>Motor conduction velocity (m/s)</i>									
Median nerve									
Elbow-wrist	46	60.28	5.27	5	57.36	4.19	27	56.3*	7.3
Lateral popliteal nerve									
Popliteal-ankle	50	55.68	5.32	9	46.24***	1.67	26	51.09**	6.65
<i>Motor distal latency (ms)</i>									
Median nerve									
Wrist-musculus abductor pollicis brevis	46	3.63	0.56	5	5.16**	0.22	27	3.96*	0.67
Lateral popliteal nerve									
Ankle-musculus extensor digitorum brevis	50	4.53	0.7	9	6.38**	0.37	26	4.35	1.28

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

ENMG abnormalities manifested as denervation potentials or as increased polyphasic motor unit potentials, reduced interference pattern or discrete pattern of recruitment, and delayed distal latency of motor nerves gave evidence of polyneuropathy in 10 of the 19 subjects examined from factory A, the prevalence being 53 %. Three of the subjects showing fibrillation, positive waves, and a discrete pattern of recruitment in the electromyogram had clinically more severe findings. Their recovery did not occur until 11 months after the cessation of exposure. This occurrence suggested that the denervation potentials probably resulted from axonal degeneration of peripheral nerves, a phenomenon which might be similar to that seen in allyl chloride intoxicated rabbits and mice (1, 2).

Similar but much milder symptoms appeared in some of the examined subjects exposed to allyl chloride at 0.2–25.13 mg/m³ for 1–4.5 years in factory B (table 2). Reports of cramping pain were much less frequent than in the workers from factory A ($p < 0.01$). Few abnormal neurological signs were found. However an increase in the number of polyphasic potentials and a prolongation of the duration of the motor unit potentials without any denervation potentials in the ENMG was noted in 13 of the 27 subjects examined in factory B. The ENMG abnormalities [partial denervation and reinnervation in the muscles (1)] indicated the existence of mild neuropathy (tables 3 and 4).

Insomnia, dizziness, and anorexia were rare in both groups. The liver was palpable in two workers with normal liver function from factory A. One subject in factory B had impaired liver function (SGPT 354 units, TTT 18 units, TFT ++) without enlargement of the liver. No significant abnormalities of the kidney, heart,

or other organs were found in either group in the physical examination or the laboratory tests. Other possible etiologic factors of neuropathy than exposure to allyl chloride were excluded.

The results of this study indicate that the main risk of industrial exposure to allyl chloride is damage to the peripheral nervous system. Polyneuropathy is thought to be the main clinical manifestation of chronic allyl chloride poisoning, and electroneuro-myography is essential and valuable for early diagnosis and biological monitoring. Moreover, it is necessary for the factories concerned to carry out effective preventive measures to minimize the exposure to allyl chloride among occupationally exposed subjects.

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References

1. He F, Jacobs JM, Scaravilli F. The pathology of allyl chloride neurotoxicity in mice. *Acta Neuropathol* 55 (1981) 125–133.
2. He F, Shen DG, Guo YP, Lu BQ. Toxic polyneuropathy due to chronic allyl chloride intoxication: A clinical and experimental study. *Chin Med J* 93 (1980) 177–182.
3. Lu BQ, Dong SW, Yu AR, Xian YL, Geng TB, Chui T. Studies on the toxicity of allyl chloride. *Ecotoxicol Environ Saf* 6 (1982) 19–27.
4. National Institute for Occupational Safety and Health. Occupational exposure to allyl chloride, criteria for a recommended standard. US Government Printing Office, Washington, DC 1976.