

Chronic peripheral neuropathy in workers with previous exposure to carbon disulphide

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ABSTRACT Two groups of viscose rayon production workers were examined 10 years after discontinuation of exposure to CS₂. Group A comprised 20 subjects exposed to high CS₂ concentrations and group B 13 subjects with lower exposure. Clinical findings and the measurements of nerve conduction velocity of the slow fibres in the peripheral nerves were evaluated. Twelve subjects had both clinical and electromyographic evidence of neuropathy and 10 showed a diminution in motor conduction velocity. A relationship between the degrees of exposure to CS₂ and prevalence of polyneuropathy was found. On re-examining 12 subjects with neuropathy who had been examined four years before no significant electromyographic improvement was observed. These findings are consistent with a permanent axonal neuropathy caused by carbon disulphide.

Peripheral neuropathy due to carbon disulphide poisoning has been reported by many investigators,¹⁻¹¹ all of whom agree that electromyography is a sensitive method for its early diagnosis. In particular, there is a clear relation between exposure to CS₂ and alterations in the maximal motor conduction velocity (MCV), the conduction velocity of the slower motor fibres (SFCV), and the distal latency. There are, however, few data on the electrophysiological changes that occur after cessation of exposure. Manu *et al*³ observed an improvement in the electromyography pattern of subjects after a reduction in or the cessation of exposure to CS₂, and Giglioli *et al*¹⁰ found incomplete signs of neurophysiological recovery in subjects whose exposure to CS₂ had ceased eight years before.

The aim of this work, therefore, was to study the prevalence of CS₂ neuropathy in workers 10 years after exposure had ceased.

Subjects and methods

Thirty three viscose rayon plant workers were selected on the basis of the following criteria:

- (1) They had been exposed to CS₂ for more than eight years;
- (2) They had stopped working in the plant 10 or

more years before the present study was undertaken; and

(3) They had no concomitant factors that could cause peripheral neuropathy, such as alcohol consumption of more than 100 g/day, diabetes, dietary deficiencies, serious liver diseases, or cancer.

The patients were divided into two groups based on the degree of CS₂ exposure. *Group A* comprised 20 subjects who had been employed on the sulphidation and xanthation process, or in the fibre spinning and rayon washing departments. The 13 subjects in *group B* had not been continuously exposed to CS₂, having been either maintenance staff or employed in other jobs.

All the patients were submitted to a neurological examination and to electromyography (EMG), the latter performed on the abductor digiti V and extensor digitorum brevis, using a Medelec MS6 apparatus and coaxial needle electrodes. The MCV and the SFCV of the ulnar and peroneal nerves were thus determined. Twelve subjects with neuropathy who had previously been examined (on average four years before) using the same apparatus and method were also studied to determine whether any improvement could be noted.

Carbon disulphide concentrations in the sulphidation, spinning, and washing departments of the plant, measured in 1951,¹² were six to seven times greater than the TLV of 30 mg/ml¹³ and continuously exceeded this value later.

Table 1 Results of clinical and EMG investigations and relation between diagnosis of neuropathy and age

Diagnosis	No of subjects	Age (years)
Definite neuropathy	12	65.8 ± 7.0
Latent neuropathy	10	61.4 ± 6.6
Normal findings	11	64.1 ± 10.7

Results

Table 1 shows the results of clinical and EMG investigations, and the relation between diagnosis of neuropathy and age. Twelve subjects had definite neuropathy—that is, objective signs of peripheral alterations in the nervous system (distal sensory loss, altered tendon reflexes, diminished muscle power, or muscle atrophy) and reduction of motor conduction velocities. Eleven had latent neuropathy defined as a reduction of motor conduction velocities without objective signs. In 10 the findings were normal. No relation was found between age and the diagnosis of neuropathy. In fig 1 individual values MCV and SFCV of the peroneal nerve in groups A and B are reported.

The prevalence of neuropathy in groups A and B is shown in table 2: 95% of the subjects with the highest exposure have definite or latent neuropathy compared with 23% of subjects with discontinuous exposure. No difference between the two groups was found regarding age and years of exposure.

Figure 2 shows the changes in the peroneal nerve SFCV in the 12 subjects examined six (first control) and 10 (second control) years after discontinuation of exposure to CS₂. An improvement was found in two, one patient showed a reduction in SFCV, and no changes were observed in the remaining nine.

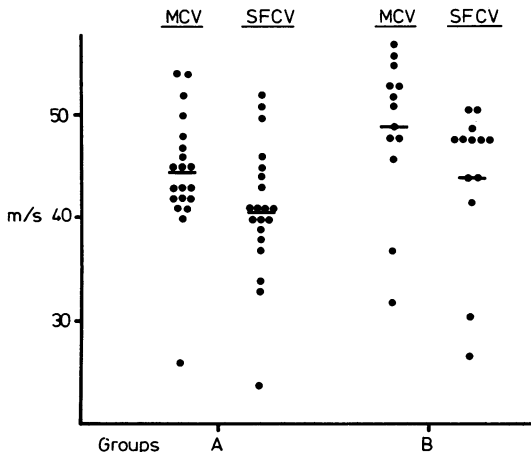


Fig 1 Individual MCV and SFCV values in peroneal nerve of groups A and B. Horizontal bars represent mean value.

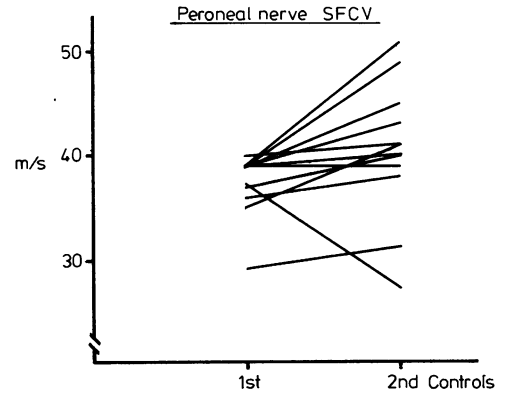


Fig 2 Changes in peroneal nerve SFCV in 12 subjects examined six (first control) and 10 (second control) years after discontinuation of exposure to CS₂.

Discussion

A high prevalence of neuropathy (67%) was found in this group of CS₂ workers, although it must be borne in mind that the men examined had had a long exposure and that SFCV is a more sensitive measure of nerve damage than MCV alone.^{6,10} Moreover, five of the 11 subjects with "normal" findings had minimal EMG alterations, such as a lengthening of distal latency, but we thought these signs insufficient to warrant a diagnosis of latent peripheral neuropathy.

In the neuropathic patients the reduction of motor conduction velocity was generally not severe (only five cases had SFCV of less than 35 m/sec) and was more frequent in the longer nerves (in half of the subjects the neuropathy was limited to the peroneal nerve alone and in four cases to the ulnar nerve).

As table 2 shows, the prevalence of neuropathy is related to the intensity of exposure to CS₂, whereas age and duration of exposure are not. Only one patient in group A had normal EMG findings, but he had shown signs of a latent polyneuritis four years previously (six years after cessation of toxic exposure). This patient had had milder exposure than the others in group A because he had worked in the fibre washing department only from 1962 to 1971, when efforts had been made to lower the CS₂ concentrations at the workplace.

In group B one of the neuropathic patients

Table 2 Prevalence of neuropathy in Groups A and B

	No of subjects	Age (years)	Exposure (years)	Neuropathy No	Neuropathy %
Group A	20	63.0 ± 7.8	22.0 ± 9.5	19	95
Group B	13	62.8 ± 8.1	25.5 ± 9.2	3	23

showed severe EMG alterations (pronounced loss of motor unit), although he had had only slight and irregular exposure to CS₂; the diagnosis of neuropathy as a consequence of exposure to CS₂ in this case is thus questionable.

In conclusion, a high frequency of toxic peripheral neuropathy was found in subjects who had been exposed to the highest levels of CS₂ 10 years previously. This disease was not severe but was extremely persistent. Moreover, as no change was observed between the re-examinations six and 10 years after cessation of toxic exposure, the alterations may be considered permanent. These findings are consistent with an axonal neuropathy as studies in animals have shown.¹⁴⁻¹⁶

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