was no evidence of spinal arterial thrombosis. The cord showed some degeneration of the medial dorsal columns in keeping with the dorsal root demyelination. There was no evidence of necrosis of the cord or of nerve roots. The findings were thought to be consistent with damage from intrathecal phenol.

Discussion

Haxton[4] reported one case of low spinal anaesthesia lasting for three hours after chemical sympathectomy and associated with unilateral quadriceps weakness persisting for three months. Our patient had an apparently permanent, severe neurological deficit in both legs. The unilateral vasodilatation suggests that the phenol was correctly placed. Extradural injection would be unlikely to produce such an extensive lesion. It was thought that there might have been damage to an unusually low artery of Adamkiewicz, producing low cord ischaemia, but this was not sustained by the necropsy findings. Reid *et al*[3] quoted three or four occasions on which cerebrospinal fluid was obtained during the procedure. Intrathecal injection of phenol is usually followed within minutes by paraesthesiae, anaesthesia, and paralysis, which usually last for only a few hours.[5]

We assume that, despite the failure to obtain cerebrospinal fluid from the needle, and the absence of immediate neurological symptoms, some of the injected phenol must have entered the subarachnoid space.

We thank Professor J A Strong for allowing us to report on his patient.

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Occupational exposure to petroleum products in men with acute non-lymphocytic leukaemia

Chronic exposure to benzene[1 2] and exposure to ionising radiation[3] are environmental hazards increasing the risk of acute non-lymphocytic leukaemia (ANLL). Otherwise, however, occupational factors are not known to favour its development. We have noticed that in their work several of our men patients have handled petrol-driven motors or were otherwise exposed to motor fuels.

Material, methods, and results

The occupational titles and information on professional exposure to motor fuel were recorded in all men patients (50 cases) aged 20-65 (median 42·5) with ANLL seen at the Department of Internal Medicine at the University Hospital at Lund from 1969 up to and including May 1977. Three clinical groups served as controls: (1) 100 consecutive men patients aged 20-65 (median 43) treated for non-malignant disorders at our outpatient department in 1977; (2) 100 consecutive men aged 20-65 (median 39) treated at our outpatient department of allergic diseases; (3) 22 men aged 20-62 (median 41·5) with chronic myeloid leukaemia (CML) and 10 men aged 41-63 (median 52) with chronic lymphocytic leukaemia (CLL) seen during the same period as the patients with ANLL.

The occupational groups of men aged between 16 and 64 living in the county where our hospital is situated were obtained from the Swedish population census of 1970 and the number of groups indicating occupational exposure to petroleum products in this age group was obtained from the county council for the same year. Eighteen out of 50 (36%) working men with ANLL were occupationally exposed to petroleum products or their combustion residues. Workers at filling stations, bus or truck drivers, operators of excavating machines or power saws, and road hauliers were typical occupations recorded for these patients. There was an especially large proportion of exposed patients in the age group 36-50 years—where nine out of 14 ANLL patients (64%) had a history of occupational exposure to petroleum products (see table).

Occupational exposure to petroleum products in men with ANLL

Age (years):	20–35	36–50	51-65
Exposed	4	9	5
Non-exposed	13	5	14
Total	17	14	19

In each of the outpatient control groups 10 out of 100 (10%) were exposed. The difference in exposure between the ANLL group and that in each of the control groups is significant ($P=0.0002$, Fisher's exact probability test). In the patients with CML three out of 22 (14%) were occupationally exposed and in the patients with CLL none of the nine patients. The difference between the patients with ANLL and the 31 patients with chronic leukaemias is significant ($P=0.006$) and there is also a difference when the patients with ANLL are compared with patients with CML ($P=0.04$) or patients with CLL ($P=0.03$).

In the county where our hospital is situated about 250 000 men were 16-64 years old in 1970. Nearly 27 000—that is, 10-11%—were occupationally exposed to petroleum products according to the occupational titles.

Chromosome analyses of bone marrow cells at diagnosis by the Giemsa banding technique were available in eight of the exposed patients and will be reported elsewhere.

Discussion

A large proportion (36%) of our men patients aged between 20 and 65 with ANLL were occupationally exposed to petroleum products or their combustion residues. The corresponding figure found in our three control series treated at the same clinic was about 10% and data from the Swedish population census of 1970 indicate that the frequency of occupational exposure to petrol products is similar in the healthy, working male population. We therefore conclude that occupational exposure to motor fuel is unduly common among working men who develop ANLL, and our results indicate that some leukaemogenic factor(s) must be considered in the exposed patients.

Chronic exposure to benzene will increase the risk of ANLL,[1 2] and petrol may contain 6-8% benzene.[1] The haematological hazards of benzene in petrol have been pointed out by Verwilghen *et al*[4] and Vigliani.[1] Nevertheless, other leukaemogenic agents must be considered in individuals exposed to petrol or its combustion products. For example, road tanker drivers show an increased frequency of lymphocyte chromosome breakage irrespective of whether they transport petrol or milk.[5]

Unlike the patients with ANLL, occupational exposure to petrol products was not unduly common among the patients with CML or CLL. This result is in line with the findings in benzene-induced leukaemia, which is usually acute.[1 2] Detailed environmental studies of patients with ANLL are needed to identify factors that may promote the development of the disease.

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