

## Short reports

### Neuropathy in a petrol sniffer

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**SUMMARY** A 4 year old boy developed a profound motor neuropathy after repeated deliberate inhalation of petroleum vapour. The condition was characterised by extreme slowing of the nerve conduction velocity. He made a gradual recovery over six months. The neuropathy was attributed to the N-hexane component of petroleum.

The deliberate sniffing of petrol (gasoline) to produce euphoria is known to cause acute neurological dysfunction, but chronic complications of this practice are rarely reported. The case described here is remarkable both for the youthfulness of the sniffer and for the pattern of neurological damage.

#### Case report

The patient was a Sudanese boy living in Khartoum. His family history and previous health were unremarkable.

At age 4 years 1 month a disturbance of gait and balance were noted but the symptoms were mild and initially ignored. His mother observed that he sometimes smelt of petrol. At age 4 years 4 months he was found comatose beside an open can of petrol that was stored in an outbuilding. Dyspnoea,

hypoxia, and signs of encephalopathy persisted for 48 hours. When he recovered he was profoundly weak, hypotonic, and unable to stand or walk, but intellect, speech, and social behaviour were unimpaired.

Two months later, when he was seen in London, he could sit but was unable to stand. There was mild symmetrical weakness of the facial muscles and of all limbs. The legs were more affected than the arms. All four limbs were hypotonic and areflexic with some muscle wasting.

No sensory or cerebellar signs could be shown. The features were those of a predominantly motor neuropathy.

**Investigations.** Immediately after the acute episode, a blood lead concentration measured in Sudan was 2.1  $\mu\text{mol/l}$ .

Two months later the following investigations were normal: blood count and film, erythrocyte sedimentation rate, urea, sodium, potassium, calcium, phosphate, and random glucose concentrations, liver function tests, urine reducing substances, creatine kinase activity, urinalysis, x ray films of skull, chest, and legs, blood lead concentration (0.5  $\mu\text{mol/l}$ ; normal = <1.8), visual acuity, visual evoked responses, audiogram.

The only abnormal investigation was the motor nerve conduction velocity (see Table).

**Progress.** When last seen, seven months after the acute illness, he could walk and there was only minimal weakness.

#### Discussion

The acute effects of petrol inhalation are those of an encephalopathy with euphoria, lethargy, anorexia, speech disturbances, blurred vision, and seizures.<sup>1</sup> Sniffing is primarily an activity of teenagers, though in communities where the practice is widespread 7 and 8 year olds may become involved.<sup>2</sup> It is most unusual for a 4 year old to discover and indulge the habit in isolation.

Table Results of investigations of motor nerve conduction

Nerve	Distal latency (milliseconds)	Conduction velocity (m/sec)	Amplitude ( $\mu\text{v}$ )
Right medial popliteal, knee-ankle	49.7	0.5	50
Right lateral popliteal	38.4	0.5	50
Right median, elbow-wrist	14.6	0.5	450
Right ulnar, elbow-wrist	10.2	0.5	100

The alkyl leads (commonly tetraethyl lead and tetramethyl lead in the United Kingdom) and triorthocresyl phosphate along with lead scavengers are added to petroleum as anti-knock agents. These, along with the hexane isomers, are usually assumed to be the main toxic constituents of gasoline. The alkyl leads can be absorbed by inhalation but triorthocresyl phosphate is less volatile. The composition of the gasoline that the child was exposed to in Sudan is not known.

Lead poisoning is more likely to cause a mononeuropathy and the nerve conduction is only slightly reduced. In our patient, however, there was gross slowing of the conduction velocity. Furthermore, lead concentrations, x ray films, and the blood picture was normal. Karani<sup>3</sup> reported a case of toxic neuropathy in a young man who regularly drank petrol and attributed this to the additive triorthocresyl phosphate,<sup>3</sup> but this substance is unlikely to be absorbed in large quantities by sniffing petrol vapour.

We suggest that the profound motor neuropathy in our patient was probably caused by the N-hexane component of petroleum.<sup>4</sup> There are many reports of a similar neuropathy occurring in glue sniffers and in workers exposed to industrial sources of N-hexane or petroleum benzene. N-hexane seems to be the main toxic agent in organic solvent mixtures; toluene and other hydrocarbons, though potentially neurotoxic, seem to exert some protective effect

against the nerve damage caused by N-hexane. The clinical features depend to some extent on the length and level of exposure. A motor neuropathy is usually the predominant finding, though in some cases there are also some sensory symptoms. Deterioration often continues after exposure has ceased. The motor nerve conduction velocity typically is grossly reduced and histological examination reveals a primary axonal neuropathy with secondary effects on the myelin and pronounced nodal widening.<sup>5</sup>

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#### References

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## Hypovitaminosis E induced neuropathy in exocrine pancreatic failure

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**SUMMARY** A 4 year old girl with congenital nerve deafness and pancreatic insufficiency had incapacitating ataxia. Electrophysiological studies of the median nerve and the brain stem evoked response were abnormal. Serum vitamin E concentration was low. After intramuscular injections of vitamin E the ataxia disappeared and electrophysiological variables reverted to normal.

This report describes a 4 year old girl with a variant of the pancreatic deficiency and congenital nerve deafness syndrome. As a consequence of the accompanying malabsorption she had hypovitaminosis E,

incapacitating ataxia, and abnormal electrophysiological studies of the nervous system. Treatment with intramuscular vitamin E resulted in clinical and functional improvement.

#### Case report

The patient was a 4 year old girl, the oldest of three sisters born to non-consanguineous Arab parents. Her birth weight was 2700 g. At the age of 1 year she was noticed to be suffering from abdominal distension, frequent loose smelly stools, and failure to gain weight in spite of a ravenous appetite. Early in life she had been found to have impaired hearing, with a resultant delay in speech development. At 13