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ABBREVIATIONS Measurements should be expressed in SI units (see *Journal of Clinical Pathology* 1974;**27**: 590-7; *British Medical Journal* 1974;**4**:490; *International System of Units* 1972. National Bureau of Standards, Special Publication 330. United States Bureau of Printing: Washington). For recognised abbreviations see *Journal of Neurology, Neurosurgery, and Psychiatry* 1975;**38**:5; and *Units, Symbols and Abbreviations,* Third Edition 1977, edited by DN Baron, Royal Society of Medicine: London.

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Coers C, Woolf AL. *The innervation of muscle. A biopsy study.* Oxford: Blackwell, 1951:16-24.

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Glue sniffing and movement disorder

Sir: There is sketchy evidence that toluene abuse in the form of glue sniffing may cause movement disorder; in 1961, Grabski described a man who had abused toluene for six years and appeared to suffer permanent cerebellar damage.[1] Since that time, there have been sporadic case reports of a similar nature, including a follow up in 1966 of Grabski's original patient.[2–6] While cerebellar dysfuntion with its concomitant movement disorder may be a common presentation, we now describe a case in which the movement disorder is not classically cerebellar and where there may be presumptive evidence of altered dopamine activity in the area of the basal ganglia.

A 27-year-old man with a 15 year history of uninterrupted glue sniffing presented to the emergency room having suffered two witnessed grand mal seizures. During previous psychiatric hospitalizations for substance abuse, his preference had been for a plastic cement containing toluene as its only solvent. Initial investigations included a normal haemoglobin and slightly elevated white blood cell count at 12,900 mm^{-3}. His serum sodium and potassium were normal but his chloride was elevated to 110 mmol/l; he had a matabolic acidosis with a pH of 7·26, pCO_2 39 mm Hg bicarbonate 17mmol/l pO_2 96 mm Hg and 0_2 saturation 96%. A routine screen for drugs of abuse, including alcohol, methanol, ethylene glycol and isopropanol was negative: an assay for toluene was not available. Over the next 24 hours, he remained unresponsive to any external stimuli and was noted to be opisthotonic at times. He would occasionally "wake up" spontaneously but was completely disoriented, incoherent, and extremely aggressive. Chest and skull radiographs were normal as were tests for renal and hepatic function. CT brain scan was also normal. An EEG demonstrated diffuse slow wave activity in the theta and delta range, with frequent sharp wave activity.

Over the course of the next two days, he became more responsive, though his level of consciousness continued to fluctuate and he remained disoriented for time and place. There was no evidence of cranial nerve abnormality nor nystagmus, but there was a generalised increase of muscle tone with slight rigidity, and the deep tendon reflexes were brisk but symmetrical. The most striking feature at this point was almost continual movements of the upper limbs occasionally extending to the entire trunk. These consisted of involuntary, random, smooth movements which varied in frequency, were accentuated when the patient was aroused or anxious, and disappeared when he fell asleep. He also had frequent involuntary movement of his mouth and jaw, accompanied by a mild dysarthria. The diagnosis of choreoathetotic movement of no clear aetiology was made.

For the first two days in hospital, he was given a continuous intravenous infusion of diazepam. On his third hospital day, this was discontinued and he was placed on oral phenytoin: no blood level was obtained. By the sixth day there was a modest improvement in his mental state but the movement disorder persisted. Phenytoin was then stopped and a combination of levodopa/carbidopa was started. The initial dosage was 125/12·5 mg twice daily and this was doubled to 250/25 mg two days later. Within three days of starting this medication, there was a marked reduction of the abnormal movements and he was reported to be much more cooperative and coherent. Despite this clinical improvement, a repeat EEG continued to show excess slow wave activity in all head regions. He remained on levodopa/carbidopa and recovered to the point of being discharged from hospital on the twelfth day, fully oriented and alert with no evidence of dysarthria, tremor, or other abnormal movements.

Even though an assay was not available, there seems little doubt that this man presented with acute manifestations of toluene inhalation: seizures, disorientation, fluctuations in consciousness, hyperchloraemia, and metabolic acidosis.[5,6] He differed from most cases reported to date in that he had a choreo-athetotic movement disorder which might have responded to levodopa/carbidopa, although the remission could have been spontaneous. The choreoathetotic nature of the movement disorder is compatible with abnormal function of the corpus striatum, particularly within the dopamine-rich caudate nucleus. Fuxe *et al.*[7] have recently reported alterations in dopamine turnover in various areas of rat caudate nucleus as a consequence of toluene inhalation. Low concentrations resulted in reduced dopamine turnover in the marginal zone, and in the medial and central parts of the anterior caudate: there was a similar reduction in the nucleus accumbens.

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Notice

The IIIrd International Brain Heart Conference will convene in Trier on 7–9 September 1985. Further Information may be obtained from Dr T Stober, Department of Neurology, Faculty of Medicine, University of Saarland D-6650 Homburg/Saar, FRG.