

Serum neutralisation test for this type of virus carried out two months after the patient was first seen was positive in a 1/4 dilution.

### Discussion

Despite full immunisation, poliomyelitis may still occur, though it is comparatively rare. Margoffin *et al*<sup>1</sup> found, however, that 25 of the 497 patients clinically classified as suffering from paralytic poliomyelitis excreted various types of Coxsackie viruses. (A9 in three, B2 in six, B1 in one, B4 in eight, B5 in six, and B4 combined with B5 in one case) and that, with exception of two patients in this subgroup—in whom the evidence was equivocal—serological and other inquiries were against a polio infection. The findings in this early survey were extended shortly afterwards by Lenette *et al*,<sup>2</sup> who added Echo type 9 and mumps viruses as potential causes of paralytic manifestations. More recently Sells *et al*<sup>3</sup> have stated that “with the virtual elimination of poliomyelitis, the central nervous syndromes associated with Coxsackie and Echo viruses have attracted increasing attention.” They listed Coxsackie A 1-11, 14, 16-18, B types 1-6, and at least 24 of the recognised Echo viruses as having been implicated. Their review supported the conclusion of previous investigations that neurological sequelae of these non-polio enteroviral infections become less damaging with increasing age, the immature brain being principally vulnerable.

Paralysis of the palate in the patient described is likely to have been caused by a Coxsackie A9 infection, the source of which may have been river water, which was much depleted by a severe drought. The excellent spontaneous recovery which took place in this boy is in keeping with the good prognosis expected at his age.

I thank Mr Hazeley Anderson, consultant ear, nose, and throat surgeon, for referring the patient to me, and Dr T H Flewett, consultant virologist, for his help.

<sup>1</sup> Margoffin, R L, Lenette, E H, and Schmidt, N J, *Pediatrics*, 1961, **28**, 602.

<sup>2</sup> Lenette, E H, Margoffin, R L, and Knouf, E G, *Journal of the American Medical Association*, 1962, **179**, 687.

<sup>3</sup> Sells, C J, Carpenter, R L, and Ray, C G, *New England Journal of Medicine*, 1975, **293**, 1.

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## Unusual case of tetrahydrocannabinol intoxication confirmed by radioimmunoassay

The major manifestations of acute tetrahydrocannabinol (THC) intoxication include paranoia, hallucinations, confusion, restlessness, and excitement. Additionally there may be delirium, disorientation, and impaired consciousness.<sup>1 2</sup> These symptoms sometimes occur after quite small doses, particularly in “naive” users of the drug. Rarely, however, do toxic symptoms last more than a day.

### Case report

A 19-year-old student on holiday in London was found by friends in his hotel room collapsed and unresponsive with arms flexed and legs extended. On admission to hospital he was found to be in grade III coma and sweating (axillary temperature 37.5°C). His eyes were open but “not seeing.” The pupils were constricted and equal in their reaction to light, and gaze was downwards to the right. He exhibited the features of decorticate rigidity, with flexed arms and extended legs. Muscle tone in all limbs was increased and tendon reflexes were all brisk (plantar responses equivocal). Neck movement was stiff in all directions but did not show tonic reflex patterns. There was also sustained right ankle clonus. Respiration was irregular but pulse and blood pressure were normal. Radiography of the cervical spine and lumbar

puncture showed nothing abnormal, and Kernig's sign was absent. Haematological and biochemical values were all normal. Drug intoxication was suspected and samples of blood and urine were sent for toxicological analysis.

Twelve hours after admission the patient was less rigid and began to respond to pain. Over the next two days he was unable to speak coherently and suffered hallucinations, becoming difficult to control and at times violent. Chlorpromazine was prescribed and his condition improved, so that four days after admission he was responding normally. He was discharged next day. Subsequently he admitted to having smoked a quantity of material that he called “THC” a few hours before the onset of his symptoms.

Toxicological analysis of blood and urine obtained on admission included tests for alcohol, barbiturates, benzodiazepines, glutethimide, methaqualone, methadone, phenothiazines, tricyclic antidepressants, and other miscellaneous hypnotic and psychotropic drugs. All gave negative results. Owing to the persistence and nature of the symptoms and the circumstantial evidence of drug ingestion a blood sample obtained on admission was analysed by radioimmunoassay<sup>3</sup> for cross-reacting cannabinoid (CRC) concentration, a result of 180 µg/l being obtained.

### Comment

Reports of serious cannabis or THC intoxication resulting in loss of consciousness are rare, and the present case therefore represents a severe toxic episode of this kind. The persistence and nature of the symptoms were serious, particularly with regard to the hallucinatory changes.

The plasma CRC concentration of 180 µg/l some eight hours after intake may be compared with one of 70 µg/l in a volunteer immediately after smoking a cigarette impregnated with 5 mg of pure THC.<sup>4</sup> The volunteer experienced moderate effects attributable to cannabis. A specimen of blood taken from a driver killed in a motor-car accident had a CRC concentration of 315 µg/l.<sup>5</sup>

This case illustrates some of the problems in diagnosing an unusual type of drug intoxication, but one that may become more common should the illicit use of refined cannabis material increase.

<sup>1</sup> World Health Organisation Scientific Group, *Report*, No 478. Geneva, WHO, 1971.

<sup>2</sup> Graham, J D P, editor, in *Cannabis and Health*, p 271. London, Academic Press, 1976.

<sup>3</sup> Teale, J D, *et al*, *Journal of Pharmacy and Pharmacology*, 1975, **27**, 465.

<sup>4</sup> Teale, J D, *et al*, *Lancet*, 1974, **2**, 553.

<sup>5</sup> Teale, J D, and Marks, V, *Lancet*, 1976, **1**, 884.

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## Routine nortriptyline levels in treatment of depression

An important question about tricyclic antidepressant drugs is whether doses that lead to high plasma levels are ineffective or even prevent recovery. Several studies have indicated this possibility with nortriptyline,<sup>1-4</sup> and a therapeutic range of 50-150 µg/l has been recommended.<sup>3</sup> High plasma levels can result from low doses and it has been argued that therapeutic success could be improved if more attention were paid to plasma concentrations.

### Patients, methods, and results

Thirty-six depressed inpatients (16 men, 20 women) at two hospitals were treated with a constant dose of 75-150 mg nortriptyline daily, as decided by the treating psychiatrist. The average age of the men was 51 (range 23-70) and of the women 57 (range 21-74). Five further patients were excluded