tics of myelomatosis.^{2,8,9} The exact diagnosis can be established through biopsy, thus leading to correct treatment.¹⁰

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Neurotoxicity of halogenated hydroxyquinolines

Sir: In 1979 we (Baumgartner *et al*, *J Neurol Neurosurg Psychiatr* 1979;42:1073) recorded the results of a clinical analysis of cases of suspected hydroxyquinoline neurotoxicity reported from outside Japan between 1935 and 1977. The criteria employed in this assessment were fully discussed. Of the 222 cases from 201 reports, 42 were considered probably to have been related to the administration of clioquinol or similar compounds. In 69 there was a possible relationship. In 42 a relationship was thought to be unlikely, and in 59 insufficient information was available for assessment.

We have subsequently analysed a further 137 cases reported between 1977 and April 1983, although in a number of these the drug had been administered considerably before 1977. In these further cases, a relationship to the administration of hydroxyquinolines was considered probable in 27, possible in 28 and unlikely in 17. Insufficient information was available in 31, and 14 were excluded from evaluation because the symptoms were not neurological or because documentation of hydroxyquinoline intake was not presented.

Combining the assessments from the two series yields a total of 359 reported cases with the following attributability distribution: probable 69, possible 97, unlikely 59, no relationship 30, insufficient information 104. As was emphasized in our previous report, it is striking that the number of cases reported from outside Japan is of quite a different order from the large numbers encountered in that country before clioquinol sales were stopped in 1970. The reason for this disparity remains uncertain, but the greater consumption of clioquinol in Japan is likely to have been the most important factor.

In our assessment of the cases reported since 1977, the semiological categorisation again included cases of acute fully reversible toxic encephalopathy with amnesia as a prominent feature. This usually occurred following the intake of a large amount of the product over a short period. Also included were cases of isolated optic atrophy of subacute or insidious onset, most commonly in children. Thirdly there were cases of myelopathy, usually of subacute onset, either in isolation or accompanied by optic neuropathy. It is of interest that no cases of peripheral neuropathy were identified in the probable category, either in isolation or associated with optic neuropathy or myelopathy. It is now clear that the neurotoxic effects of the halogenated hydroxyquinolines are substantially confined to the central nervous system. The term subacute myelo-optic neuropathy (SMON) is therefore probably a misnomer.

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Isolated ictal autonomic symptoms in complex partial seizures

Sir: A variety of autonomic changes may occur at the onset of complex partial seizures.¹⁻⁴ Autonomic disturbances may frequently comprise the seizure aura, and may occur independently after the remission of generalised seizures.²

A 46-year-old right-handed man was hospitalised for transurethral prostate resection. Eighteen years earlier he had fallen from a vehicle travelling at 50 miles per hour, and had sustained a right frontotemporal extradural haematoma which was surgically evacuated. He recovered uneventfully from the prostate operation, but three days later was found seated in a chair with intermittent difficulty in comprehending what was said to him, although he remained alert and conversant. For periods of one to three minutes, the patient became flushed and had left-sided piloerection over the arm, leg, and trunk, with enlargement of the left pupil from 3 to 5 mm. The right pupil enlarged occasionally, but right-sided piloerection was not present. These episodes were separated by periods of five to six minutes during which his skin colour was normal, piloerection was absent, and pupillary size and reaction were normal and symmetric. On examination he was able to speak and comprehend, and speech and mentation did not change; flushing, pupillary dilatation, and piloerection subsided. He was afebrile and had no change from a normal pulse and blood pressure during the episodes. He had a mild left hemiparesis with increased tone, which did not change between episodes, and focal clonic jerking was not present. Deep tendon reflexes were increased on the left and left Babinski sign was present. Pathological reflexes and frontal release signs were otherwise absent. After approximately one hour of episodic autonomic changes, he developed focal clonic jerking of the left arm, followed by a generalised convulsion with incontinence and postictal confusion. He was given intravenous phenytoin and had no further seizures. An electroencephalogram several hours later showed polymorphic delta slowing in the right temporoparietal area, with frequent epileptiform discharges arising from the F₇ and T₇ electrodes. Computed tomography showed postoperative changes in the right frontal region and right frontotemporal low density, with enlargement of the right lateral ventricle. Cerebrospinal fluid was normal. The patient's family had observed no previous seizures.