tive of myelomatosis. The exact diagnosis can be established through biopsy, thus leading to correct treatment.  

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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 or uncertain, possibly in 27, probably 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 attributable 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 chloquinol sales were stopped in 1970. The reason for this disparity remains uncertain, but the greater consumption of chloquinol in Japan is likely to have been the most important factor.

In our assessment of the cases reported since 1977, the semeiological 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, mostly common 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 myeloptic neuropathy (SMON) is therefore probably a misnomer.

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Letters

Isolated ictal autonomic symptoms in complex partial seizures

Sir: A variety of autonomic changes may occur at the onset of complex partial seizures. \(^ 4\) Autonomic disturbances may frequently comprise the seizure aura, and may occur independently after the remission of generalised seizures. \(^ 5\)

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 paresis 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 paresis was not present. These episodes were separated by periods of five to six minutes during which his skin colour was normal, paresis 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 paresis subsided. He was afibrile 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 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 F, T, and 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.
This patient suffered severe trauma to the right frontotemporal region, with residual neurologic deficit but no seizures for eighteen years. During his postoperative recovery he had a prolonged period of intermittent flushing, pupillodilation, and pilocrection contralateral to his area of injury, and he subsequently had complex partial seizures with secondary generalisation and an active right temporal epileptogenic focus on EEG. The episodes of autonomic changes were ictal in origin, but were not associated with impaired consciousness, nor with focal motor or sensory manifestations. Gowers\textsuperscript{1} encountered visceral symptoms in 17.7% of his patients, chiefly as the aura of a generalised seizure. Of the patients of Lennox and Cobb\textsuperscript{2}, 14-6% experienced visceral symptoms, and such symptoms sometimes persisted after the remission of generalised seizures. The majority of these autonomic manifestations related to the gastrointestinal tract. Mulder, Daly, and Bailey\textsuperscript{3} studied visceral manifestations in 100 patients, and in 83 found them due to cerebral structural lesions, principally tumours, with isolated autonomic symptoms in four patients. Visceral manifestations often occurred independently of epileptiform EEG activity or had no EEG concomitants. Twelve patients with ictal pupillary dilatation were reported by Pant, Benton, and Dodge;\textsuperscript{4} these cases yielded a variety of causative lesions, and pupillary changes occurred during or after seizures but not as isolated ictal manifestations. Van Buren and Ajmone Marsan\textsuperscript{5} monitored autonomic parameters and EEG during pentylenetetrazol-induced seizures in 20 patients. A stereotyped sequence of hypotension, tachycardia, decreased skin resistance, esophageal peristalsis and cessation of gastric motility, and inhibition of respiration was observed, but these changes were consistently preceded by diminished responsiveness.

Although isolated autonomic symptoms are infrequently observed clinically, stimulation of various brain regions in humans produces independent autonomic changes.\textsuperscript{6} The frequent absence of EEG changes during clinically-observed autonomic disturbances may reflect the deep location of such areas, and marked differences between scalp EEG and depth electrode recordings have been observed during such autonomic phenomena.\textsuperscript{7} Isolated ictal autonomic manifestations are likely to occur infrequently in complex partial seizure patients. Paroxysmal autonomic phenomena may nevertheless provide a clue to the ictal character of episodes reported by patients suspected to have complex partial seizures. Continuing autonomic symptoms may also represent seizures in patients with diagnosed complex partial seizures who are under medical therapy, and whose seizure control remains inadequate.

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