Neuropathy after organophosphorus compounds poisoning

Sir: I read with interest the report by Senanayake: Tricresyl phosphate neuropathy in Sri Lanka: a clinical and neurophysiological study with a three year follow up. However, in the table there is an error: terminal latency is not measured in m/s, but in ms. In fig 2, either the diagram or the legend should specify the terminal latency values measured in ms. As the author shows, the cases presented display a series of important differences from tricresylphosphate neuropathy (TOCP) neuropathy.

In our 12 patients who had accidentally ingested TOCP-polluted alcohol, the paralysis began slowly in the lower limbs (15–30 days) and later (5–10 days) spread to the upper limbs. In the acute phase the patients displayed flaccid paralysis mainly in the lower limbs. After 2–3 months there were signs of both peripheral and central nervous system lesions, i.e., pyramidal signs. The patients showed a good recovery from the peripheral nerve lesions, 1–2 years after TOCP ingestion, but not from the deficits due to the pyramidal lesions. The latter not only failed to improve, but even extended more distally, especially in the lower limbs. We presume that these signs of CNS lesions were present in the distal zones before examination (1–2 years after intoxication), but became evident only after improvement of the peripheral nerve lesions, which had masked them. On reexamination of two patients 13 years after TOCP ingestion, we found a tendinous retraction of toes I–V and fingers II–V in flexion, and of knees both in flexion and adduction. The gait was spastic. At the same time there was a marked weakness distally, especially in the feet.

Two to three months after intoxication all the patients showed concurrent peripheral and central nervous system signs of lesions, predominantly in the distal portions of the axons, especially the longer ones, which indicated that the process characterising neuropathy by TOCP is of the “dying-back” type, as described first by Cavanagh. However, the clinical picture in five patients was of a mixed, particularly motor and distal, polyneuropathy. Reexamination performed 13 years later in two of five patients with a predominantly neuropathic disorder showed mainly pyramidal signs. There was marked improvement of peripheral nerve lesions and poor improvement of central lesions in both cases which made the initial predominantly neuropathic form pass into the predominantly spinal form of neuropathy with a clinical and electrophysiological picture resembling amyotrophic lateral sclerosis. This finding is in agreement with the observation of Morgan and Penovich who showed that the TOCP lesion described as delayed neurotoxicity was not a neuritis but rather a spinal cord syndrome. Our electrophysiological findings suggest that a mixed process of axonal degeneration and secondary demyelination underlies this predominantly neuropathic form. We assume that in our patients the secondary demyelination occurred in fibres which had already undergone degeneration followed by regeneration, as shown by Dyck et al in uraemic neuropathy. In the remaining seven patients, pyramidal signs prevailed, objective sensibility was not disturbed, and motor conduction velocity was normal. These patients thus had a predominantly spinal form of neuropathy. The electrophysiological data suggest that this form of neuropathy was mainly an axonal degeneration.

We also studied two cases of poisoning with organophosphorus insecticides, Dipexor and Divipan, in which there was footdrop, distal weakness especially in the lower limbs, abolishing of the Achilles reflex, and mild pyramidal signs. Electrophysiological findings suggest that this neuropathy also was a distal axonopathy.

C VASILESCU
Institute of Neurology and Psychiatry
Berceni 10, CP 61-80
R-75622 Bucharest, Romania

References