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CLINICAL AND ELECTROPHYSIOLOGICAL FEATURES OF POLYNEUROPATHY PRODUCED BY A NEW ORGANOPHOSPHATE COMPOUND

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This paper analyses the clinical and electrophysiological findings in 10 patients (age 14-28 years ; 6 males) who developed polyneuropathy after exposure to the organophosphate (OP) insecticide " Tamaron " marketed in Sri Lanka. The illness is characterised by two phases ; an initial phase of cholinergic crisis which responds to atropine and pralidoxine and a delayed phase of paralysis of limbs developing 2-4 weeks after the poisoning. Paralysis first affects the distal muscles of the lower limbs and 2-4 days later, the muscles of the hand and the forearm are affected. On examination the affected muscles show weakness and wasting of varying severity. Four patients seen late in the course of the disease had evidence of pyramidal tract dysfunction in addition. The late development of pyramidal tract signs has also been reported in poisoning due to another OP compound, tricresyl phosphate.

Electromyography of the distal limb muscles showed evidence of denervation to varying degrees. Motor nerve conduction was impaired in the distal segments of the nerves, while the conduction in the proximal segments remained unaffected until the muscles were completely denervated. Sensory conduction was unaffected.

An unusual feature of the polyneuropathy caused by " Tamaron " was the asymmetry of the neural involvement. In all patients the right hand was worse affected than the left hand clinically as well as electrophysiologically. This observation that in all the cases the dominant limb was affected more severely raises the possibility whether factors such as excessive use and fatiguability of muscles has a bearing on the pathogenesis of the neuropathy in OP poisoning.