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ELECTROPHYSIOLOGICAL OBSERVATIONS ON METHYLMERCURY POISONED PATIENTS IN IRAQ

by

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The earliest signs and symptoms associated with methylmercury intoxication suggest a neurological dysfunction similar to peripheral polyneuropathy (1). Classically, such a condition is amenable to electrophysiological confirmation. However, this technique failed to confirm such a disorder in the patients of the Iraqi methylmercury epidemic of 1972 (2). Instead, the data suggest two alternative sites of damage.

One of these areas could be the lower brain stem since intoxicated patients demonstrated lower thresholds for eliciting the H reflex as well as clinical signs normally associated with this type of damage.

The other site of damage could be at some point in the neuromuscular linkage. Some of the patients exhibited myoneural transmission failure similar to that seen in myasthenia gravis. This failure responded to intramuscular injection of neostigmine. Subsequent neostigmine therapy significantly improved the clinical condition and clinical strength of the patients tested during the course of a procedure that was interspersed with placebo administration (3).

Although the exact mode of action of methylmercury in this regard is still unknown, preliminary investigations on isolated rat phrenic nerve-diaphragm preparations indicate that methylmercury can hasten muscle fatigue and increase the tolerance to a fixed doxe of neostigmine

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