Conduction Velocities in Methylmercury Poisoned Patients

by R. VON BURG

Department of Pharmacology and Toxicology
University of Rochester
Rochester, N.Y.
and
HUSSAIN RUSTAM
Department of Neurology
Medical City Hospital
Baghdad, Iraq

In contrast to the Japanese Minamata and Nigata epidemics, the 1972 Iraqi epidemic of methylmercury is significant in that it represents an acute pulse dose of methylmercury as compared to the slower accumulation in the Japanese episodes. Nevertheless, the signs and symptoms associated with this mode of intoxication were identical (McALPINE and ARAKI 1959; BAKIR et al. 1973).

The earliest symptoms to appear include paresthesia and fatigue. More severe CNS damage is indicated by ataxia, dysarthria, dysphagia and visual field constriction (TAKEUCHI 1972) and there appears to be a correlation between the incidence of reported symptoms and the concentration of mercury in the blood (BAKIR et al. 1973).

Due to the fact that the early symptoms of intoxication are reportedly similar to peripheral polyneuropathy (HUNTER et al. 1940) and electrophysiological techniques are useful for such a diagnosis (LENMAN 1968), an attempt was made to investigate the peripheral nervous system by these methods. It was speculated that such techniques would be useful to convert the subjective symptoms into objective signs and aid in the identification and early diagnosis of patients as well as assessing the extent of the epidemic.

METHODS

Fourteen patients with blood mercury levels between 1000 ng/ml and 3900 ng/ml at the height of exposure were examined seven months post-exposure when their blood mercury levels were between 100 and 800 ng/ml. Seven control individuals selected from the hospital personnel with normal blood levels were also examined. A Disa 3 channel clinical electromyograph was employed. Motor conduction velocity of the median nerve (MCV) and peroneal nerve (PCV) was determined by the method of McCOMAS et al. (1971). Sensory threshold (ST), sensory latency (SL) and sensory nerve conduction velocity of the median nerve (SCV) were measured by methods outlined by JOHNSON and MELVIN (1967) and DAWSON and SCOTT (1949). Threshold for the H reflex (HT), as well as conduction time and conduction velocity for this reflex (HR) were determined by suggested methods (MAYER 1963; STIMSON et al. 1969).

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TABLE 1A

CONDUCTION VELOCITY DETERMINATION--CONTROLS

Subject	Sex	Age	MCV M/sec	SCV M/sec	SL msec	SL M/sec	ST volts	PCV M/sec	HT volts	HR msec	HR M/sec
H. R.	M	37	68	68	2.2	50	35	60	27	30.1	45
R. H.	M	25	67	64	2.8	50	35	55	37	32.0	44
S. M.	F	18	58	68	2.4	50	37	53	25	26.8	49
F. Y.	M	23	62	58	2.9	45	45	50	40	28.6	45
M. I.	M	15	55	68	2.6	54	35	42	--	26.8	44
E. K.	F	15	60	72	2.4	50	36	--	13	25.9	51
H. D.	M	25	67	62	2.4	54	40	49	20	30.3	42
Mean		22.5	62.4	65.7	2.5	50.4	37.5	51.5	27	28.6	45.7
S.D.			4.6	4.3	0.2	2.8	3.5	6.3	9.3	2.1	2.9

TABLE 1B

CONDUCTION VELOCITY DETERMINATION--METHYLMERCURY INTOXICATED PATIENTS

Patient number	Sex	Age	MCV	SCV	SL		ST	PCV	HT	HR	
			M/sec	M/sec	msec	M/sec	volts	M/sec	volts	msec	M/sec
37	M	12	60	--	2.6	40	45	61	--	26.8	39*
49	M	14	59	72	2.6	47	25	39	14	26.1	52
50	M	15	72	67	2.8	53	26	79	33	29.0	52
51	M	10	73	--	--	--	--	--	--	33.5	36*
52	M	8	56	65	2.4	39	42	49	15	20.7	41
95	M	8	53	66	2.4	50	20	52	10	21.5	49
96	M	6	43	65	2.3	49	23	29	--	19.0	50
119	M	6	50	67	2.0	50	38	45	7	18.5	39*
130	F	25	65	66	2.4	50	40	51	22	27.7	44
141	F	6	56	45	2.3	41	27	37	12	17.9	47
146	F	10	56	62	2.1	54	50	40	14	21.6	42
147	M	10	56	60	2.4	42	34	60	6	21.6	49
151	M	5	57	52	2.6	38	45	44	5	21.0	45
155	M	10	36*	66	2.3	26*	45	47	8	25.1	38*
Mean		10.4	56.5	63.4	2.2	41.2	35	48.5	10.4	22.8	43
S.D.			9.5	7.2	0.2	8.4	9.4	12.2	8.2	3.4	5.2

*Abnormal values.

RESULTS AND DISCUSSION

The results of the electrophysiological determinations are summarized in Tables 1A and 1B. The values obtained are in good agreement with previously published values (MAYER 1963). As can be seen, there is a large overlap between the experimental and the control groups with no statistical significance between groups except for the determination of the threshold for the H reflex, which according to the Wilcoxon and Wilcox Rank Sum Test (WILCOXON and WILCOX 1964) indicates a confidence level of .05.

These results indicate that, in humans, clinical electrophysiological testing does not support the concept that methylmercury poisoning resembles peripheral polyneuropathy since this mode of testing could not detect any consistent abnormalities even in severely afflicted patients.

The positive observation of a reduced threshold for the H reflex is intriguing because it suggests the possibility of lower brain stem damage. STIMSON et al. (1969) point out that lesions in specific inhibitory centers of the lower brain stem might explain easily elicited H reflexes and WINDLE et al. (1962) report that monkeys exposed to neonatal asphyxia suffered damage to the medial and gracile cuneate nuclei, inferior colliculus, ventral posterior thalamic nuclei, basal ganglia and vestibular nuclei. The animals were free from cerebral or cerebellar cortical damage but demonstrated behavior strikingly similar to that seen in the Iraqi poisoned patients. That is, they exhibited ataxia and tremor, dysmetria, difficulty in righting and tendency to flex the arms and fist the hands, and had emotional outbursts. It is suggested that this aspect of electrophysiological investigation be pursued either experimentally or in subsequent cases of methylmercury poisoned patients.

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