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Electrophysiological evaluation of tremors secondary to space occupying lesions and trauma: Correlation with nature and sites of lesions☆

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ABSTRACT

Background: Electrophysiological evaluation of tremor secondary to intracranial space occupying lesions (SOL) and cranial trauma may provide information regarding pathophysiology of tremors.

Objectives: To compare the electrophysiological characteristics of tremor secondary to SOL and trauma and to correlate tremor characteristics with sites of lesion, and types of SOL.

Methods: Multi-channel tremor recording and MRI were performed in 18 patients with predominantly tremor secondary to SOL (F: M = 5:6; age ± SD: 26.6 ± 15.0 years) and following trauma (7 men; age: 27.3 ± 11.0 years).

Results: In both groups, there was a wide range in the frequency of tremor (2.5–7.5 Hz in the SOL group and 2–7.5 Hz in the post-trauma group) and a strong inverse correlation of the frequency with the duration of EMG bursts (SOL group: $r = 0.8$, $p = 0.004$; post-trauma group: $r = 0.9$, $p = 0.02$). While all the patients with SOL had regular EMG bursts (synchronous – 54.6%, alternating – 27.3%, mixed – 18.2%), 85.7% of post-trauma patients had irregular EMG bursts (synchronous – 42.9%, alternating – 14.3%, mixed – 42.9%). In SOL group, those with predominantly intrinsic destructive lesions of brainstem, thalamus, or basal ganglia ($n = 7$) had a statistically significant lower mean frequency of tremor than those ($n = 4$) with either extrinsic or intrinsic compressive lesions (3.5 ± 0.9 Hz vs 6.7 ± 0.6 Hz; $p = 0.0001$). In the post-trauma group, the patients with additional lesions in thalamus or striatum, apart from white and grey matter lesions had lower mean tremor frequency (3.7 ± 1.0 Hz vs 6.1 ± 1.5 Hz; $p = 0.05$).

Conclusions: The electrophysiological characteristics of tremor secondary to SOL and trauma differ and correlate with the nature and sites of lesions. This information, which need to be validated in larger cohort of patients, may be useful in understanding the pathogenesis of tremor.

1. Introduction

A variety of movement disorders, often labeled as secondary movement disorders (SMDs), can result from acquired insults to the brain. While a large number of patients with idiopathic or primary movement disorders have normal brain imaging, many of the SMDs give a unique opportunity to correlate clinical phenomenology with the sites of lesions. Functional imaging, while useful in understanding dysfunction of basal ganglia, is expensive and not routinely available. Alternatively, electrophysiological evaluation of the abnormal movements may provide useful information on the pathophysiology and sometimes prognosis of therapeutic interventions. Evaluation of tremor is the most common and easily available electrophysiological procedure. While there are many reports of SMD and several studies have attempted to critically look into the relationship of brain lesions with the clinical patterns of abnormal movements [1–12], there is a paucity of studies that have electrophysiologically evaluated SMDs.

In this study, we have clinically, radiologically and electrophysiologically evaluated 18 patients with secondary tremor, resulting from intracranial space occupying lesions (SOL) or following trauma. We have attempted to correlate the electrophysiological characteristics of tremor with the etiology, anatomical site of lesion on magnetic resonance imaging (MRI) and the nature of pathology in cases of SOL.

2. Subjects and methods

Eighteen patients presenting predominantly with tremor secondary to either intracranial SOL or following cranial trauma were recruited from the Neurology and

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Neurosurgery Departments. All patients underwent a detailed clinical evaluation and 1.5 Tesla MRI of brain. Details of onset, evolution, and the nature of tremor and other movement disorders, when present were recorded. Tremor was recorded using 8 channel Nihon Kohden machine. Surface electrodes were placed in a tendon-belly arrangement over the antagonistic muscles. The muscles were selected after visual inspection and palpation of the body part having tremor. Prolonged periods of continuous monitoring of EMG activity were often required when tremor bursts were intermittent. Tremor was recorded at rest, during action and sometimes during mental tasks, which brought out the tremor.

The Institutional Ethics Committee approved the protocol and written informed consent was taken from patients and from the legal guardian in case of children.

2.1. Data analysis

The frequency (number of distinct EMG bursts/second), duration and pattern (alternate vs synchronous contraction) were ascertained by visual inspection of long stretches of records. Analysis of MRI scans was done by a neuro-radiologist blinded to the clinical and electrophysiological data. The details of anatomical sites of lesions identified on MRI are given in Table 1.

Unpaired '*t*' tests were used to compare the electrophysiological parameters (frequency of tremor and duration of EMG bursts) between subgroups of patients in both patients with SOL and those having post-traumatic tremor. Fisher's exact test was used to compare the prevalence of different types (synchronous, alternating or mixed) and pattern of EMG bursts (regular or irregular) between the patients with SOL and those with post-traumatic tremor. The correlation between frequency and duration of bursts were determined using regression analysis.

3. Results

Eighteen patients (F: $M = 5{:}13$; age ± SD: 27.0 ± 13.2 years; age at onset of symptoms (AAO): 25.4 ± 13.1 years; duration of symptoms: 1.6 ± 2.6 years) of SMD were studied (Table 1). The etiology of the SMD was SOL in 11 (F: $M = 5{:}6$; age: 26.6 ± 15.0 years; AAO: 25.4 ± 14.7 years; duration: 1.1 ± 2.4 years) and trauma resulting from road-traffic accidents in 7 (all men; age: 27.7 ± 11.0 years; AAO: 25.3 ± 11.4 years; duration: 2.4 ± 3.0 years). In post-traumatic group, the latency from trauma to onset of tremor, based on reliable history from 6 patients was 0.4 ± 0.4 years. In the post-traumatic group 2 patients had seizures following head injury and they were on phenytoin sodium. In patients with SOL, 2 patients had intermittent jerky tremors which were interpreted by referring physicians as seizures and were given anti-epileptic drugs (phenytoin sodium, sodium valproate and clonazepam).

Apart from the movement disorders mentioned below, other associated neurological dysfunctions in varied combination were observed in the two groups. In the SOL group, pyramidal signs were present in 81%, cranial nerve involvement (combination of papilloedema, 7th and 3rd cranial nerve palsies) in 45.5%, cerebellar signs and mild limb weakness in 18.2% each, and hemisensory loss in 9.1% of the patients. In the post-traumatic group 57.1% of the patients had pyramidal signs and 28.6% had cerebellar signs.

3.1. Types of SMD

Among patients with SOL, 6 had pure tremor, 3 had parkinsonism with predominant tremor, and 2 had tremor with mild dystonia. All patients had rest as well as action tremor (postural and intention). In the post-traumatic group 3 had only tremor (only action in 2 and rest as well as action tremor in one), 3 had tremor with dystonia (2 had rest and action tremor with dystonia of limbs, one had dystonic tremor of head) and one patient had features of parkinsonism with predominant rest tremor.

3.2. MRI findings

The details of MRI findings in each patient are given in Table 1. In SOL group, 4 had glioma, 2 had cysts, and one each had convexity meningioma, 4th ventricular cyst, cavernoma, haematoma, and granuloma. Seven patients had lesions in thalamus with or without involvement of adjacent structures. In post-traumatic group all patients had evidence of multiple old lesions in white matter. In addition, grey matter, thalamus and basal ganglionic structures were variably involved. Some representative MRI scans of patients are shown in Fig. 1.

Table 1
Clinical, electrophysiological and radiological characteristics of the patients.

Pt. no.	Age (yrs)	Sex (yrs)	AAO (yrs)	Dur (yrs)	Etiology	MD	Trm Freq (Hz)	EMG Bursts Contr	Durn (ms)	Pat	MRI lesion[a] site
1	5	M	4.75	0.25	SOL (Glioma)	RT/AT/DYS	2.5	Syn, Alt	200–250	R	T, GP, MB
2	55	F	54	1	SOL (Cavernoma)	RT/AT	2.5	Syn	300	R	MB, PO
3	30	F	22	8	SOL (Intrinsic heterogenous lesion suggestive of cavernoma)	RT/AT	3.5	Syn	200	R	T, MB
4	25	M	24.8	0.2	SOL (Glioma)	RT/AT	3.5	Alt	100–120	R	T, ST, MB
5	30	M	45	0.2	SOL (Granuloma)	RT/AT/DYS	3.5	Proximal-Syn Distal-Alt	125	R	MB, PO
6	14	M	13.8	0.2	SOL (Glioma)	RT/AT	4	Syn	150	R	T
7	27	F	26.96	0.04	SOL (Glioma)	RT/AT	5	Alt	80	R	T
8	42	F	40	2.0	SOL (Meningioma)	PAR/RT/AT	6	Syn	75	R	LFP, P, GM, WM
9	38	M	37.8	0.2	SOL (4th ventricular cyst)	PAR/RT/AT	6–7	Alt	75–80	R	PO, CR
10	8	M	7.7	0.3	SOL (Bleed in underlying choroid plexus carcinoma)	RT/AT	6.5–7	Syn	100	R	T
11	18	F	17.9	0.1	SOL (Extrinsic Cyst)	PAR/RT/AT	7.5	Syn	80	R	T, MB
12	29	M	27.5	1.5	Post-trauma	DT	2–3	Syn	200–400	IR	T, MB, WM
13	16	M	15.5	0.5	Post-trauma	AT	3–3.5	Syn	200	IR	T, GP, PO, WM
14	19	M	18.5	0.5	Post-trauma	AT	4.5	Alt (Syn)	120	IR	P, WM
15	39	M	37	2	Post-trauma	RT/AT	4.5	Alt	200	R	STR, WM
16	22	M	20.5	1.5	Post-trauma	RT/AT/DYS	Action-6 Rest-3	Action-Alt Rest-Syn	150–200	IR	GM, WM
17	23	M	14	9	Post-trauma	RT/AT/DYS	6–6.5	Alt, Syn	100	IR	WM, SN
18	46	M	44	2	Post-trauma	PAR/RT/AT	7.5	Syn	100	IR	GM, WM

Abbreviations: Alt – Alternating, AAO – Age at onset of symptoms, AT – action tremor, CR – Cerebellum, Contr – Contraction, Dur – Duration of symptoms, Durn – Duration, DT – Dystonic tremor, DYS – Dystonia, F – female, GP – Globus pallidus, GM – Grey matter, IR – Irregular, L – Lentiform, LFP – Left frontoparietal, M – male, MB – Midbrain, MD – Movement disorder, P – Putamen, PAR – Parkinsonism, Pat – Pattern, PO – Pons, Pt. no – Patient number, R – Regular, RT – rest tremor, Syn – Synchronous, SN – Substantia nigra, SOL – Space occupying lesion, ST – Subthalamus, STR – Striatum, T – Thalamus, Trm freq – Tremor frequency, WM – White matter.

[a] In the SOL group, the MRI lesions were heterogenous, depending on the etiology. Contrast enhancement was seen in all except Patients 5 and 11, edema in all except Patients 2, 5 and 11, and mass effect in all except Patients 2, 5, 6 and 9. All the patients in the post-trauma group (Patients 12–18) had old lesions without contrast enhancement, edema or mass effect.