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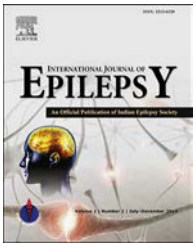

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Less invasive disconnection surgery using advanced image guidance for wide spread cortical malformations

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Purpose: Cortical dysplasia (CD) is the important pathogenesis in the pediatric intractable epilepsy. The surgical treatment is extremely effective if the epileptogenic zone is adequately detected and resected. The extent of CD is, however, usually obscure even with careful MR imaging. In widespread or multilober CD, localization of epileptogenic zone is more difficult because of multifocal and synchronous electrophysiological abnormalities. In those cases, the eloquent cerebral tissue is involved frequently inside the CD tissue in mosaic pattern, and it should be preserved intact in the surgical intervention. For better seizure control and less invasive surgery, we have introduced subcortical disconnection with techniques including intraoperative ECoG, and advanced image-guidance.

Method: Thirty-nine CD patients with intractable epilepsy were operated. Numbers of involved cerebral lobes were; one in 6 cases, two in 9 cases, three in 6 cases and hemispheric in 18 cases. Among them, 15 cases were diagnosed as symptomatic West syndrome.

Results: The surgical procedures were; focus resection in 12 cases, multilober disconnection in 12 cases and functional hemispherotomy in 15 cases, respectively. Engel Class I (no disabling seizure after the surgery) was attained in 33 cases and rare seizures in 3 cases. No serious permanent complication was experienced. Considerable amelioration in development was observed in 28 patients.

Conclusion: Less invasive disconnection surgery using advanced image guidance was successful for wide spread cortical malformations The intervention at earlier age would be recommended for better seizure control and psychomotor development.

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Vagus nerve stimulation – Mechanism of action and usefulness of its combination with corpus callosotomy for palliation of refractory epilepsy

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Vagus nerve stimulation (VNS) is indicated as an adjunctive therapy for refractory epilepsy patients who are not suitable for resective surgery (adults: grade A; children: grade C recommendation). It is effective to various seizure types regardless of their pathology both acutely and chronically. Early studies revealed a mean seizure frequency reduction of 24–31% over 3 months of follow-up. And its effects are enhanced over time (median seizure reduction of 45% at one year, with 20% of patients achieving a greater than 75% reduction).

Its mechanism of action (MOA) is not established yet. Theories include direct activation, neurotransmitter and neuropeptide modulation influencing ictal discharge, preictal changes and arousal. VNS is thought to have an effect on EEG synchronization which may prevent establishing epileptic discharge in the neural circuits and act as the acute effect. In VNS effective patients, PET scanning showed increased blood flow in the thalamus, hypothalamus, and the insular cortex with decreased blood flow in the amygdala, hippocampus, and posterior cingulate. Animal studies have looked into various possible mechanisms. In a maximal electroshock rat epilepsy model, VNS therapy was no longer effective when noradrenergic pathways were depleted by lesioning of the

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locus coeruleus. These data suggest complex MOA of VNS in both acute and chronic phases.

In recent years, we have studied the combination of VNS and corpus callosotomy, and found the combination of both techniques in selected patients achieves better results than both techniques separately. In this paper we would discuss our tentative experience and indications.

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Experience with short video EEG in small town (yield and cost effectiveness)

Nashik Anand Diwan

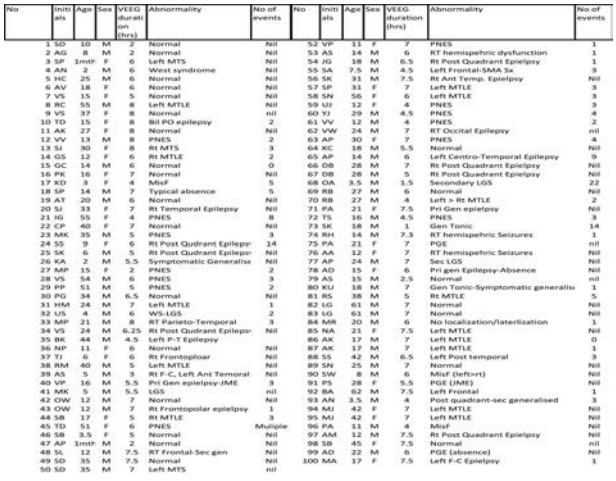
Background: Semiology, type of seizure, true or pseudo-seizure (PNES – psychogenic non epileptic seizures) are often hard to differentiate clinically. Accurate diagnosis is essential for the optimum medical or surgical treatment and outcome for the patient. Most of the times, diagnosis requires inpatient video telemetry, which is both time consuming and expensive. Short video electroencephalography (SV-EEG) has been described previously and was shown to be a useful diagnostic tool in other specialist centres.

Objective: To determine the usefulness of SV-EEG in the diagnosis and management of various seizure types.

Method: After start of SV-EEG facility in Nashik, first 100 cases were selected, 1–55 years done over last 15 months.

Results: SV-EEG done on OPD basis for period of 1–8 h. Age – 1–62 years, M = 64:F = 36. Abnormal SV-EEG was reported in 75 patients. A positive SV-EEG supporting a diagnosis of true seizures occurred in 62 of patients (generalised epilepsy = 15, focal epilepsy = 47). PNES was diagnosed in 13 pts. Attacks recorded in these patients were 1–14. No attack or no interictal abnormality was noted in 25% of patients ($n = 25$), resulting in an inconclusive SV-EEG. One patient had undergone anterior temporal lobectomy surgery based on this SV-EEG.

Conclusion: The positive rates of attacks from SV-EEG were comparable and even better to previously published results and show that SV-EEG is easily implemented in small town centres. It is cost effective method with very good diagnostic yield.



No	Initials	Age	Sex	VEEG duration (hrs)	Abnormality	No of events	No	Initials	Age	Sex	VEEG duration (hrs)	Abnormality	No of events
1	SD	10	M	2	Normal	Nil	52	VP	11	F	7	PNES	1
2	AG	8	M	2	Normal	Nil	53	AS	14	M	6	RT hemispheric dysfunction	1
3	SP	1mth	F	6	Left MTS	Nil	54	JG	18	M	6.5	Rt Post Quadrant Epilepsy	1
4	AN	2	M	6	West syndrome	Nil	55	SA	7.5	M	4.5	Left Frontal-SMA Sx	3
5	HC	25	M	6	Normal	Nil	56	SK	31	M	7.5	Rt Ant Temp. Epilepsy	Nil
6	AV	18	F	6	Normal	Nil	57	SP	31	F	7	Left MTLE	3
7	VS	15	F	5	Normal	Nil	58	SN	56	F	6	Left MTLE	3
8	RC	55	M	8	Left MTLE	Nil	59	UJ	12	F	4	PNES	3
9	VS	37	F	8	Normal	nil	60	YJ	29	M	4.5	PNES	4
10	TD	15	F	8	Bil PO epilepsy	2	61	VV	12	M	4	PNES	2
11	AK	27	F	8	Normal	Nil	62	VW	24	M	7	RT Occital Epilepsy	nil
12	VV	13	M	8	PNES	2	63	AP	30	F	7	PNES	4
13	SJ	30	F	8	Rt MTS	3	64	KC	18	M	5.5	Normal	Nil
14	GS	12	F	6	Rt MTLE	2	65	AP	14	M	6	Left Centro-Temporal Epilepsy	9
15	GC	14	M	6	Normal	0	66	DB	28	M	7	Rt Post Quadrant Epilepsy	Nil
16	PK	16	F	7	Normal	Nil	67	DB	28	M	5	Rt Post Quadrant Epilepsy	Nil
17	KD	3	F	4	Misf	5	68	DA	3.5	M	1.5	Secondary LGS	22
18	SP	14	M	7	Typical absence	5	69	RB	27	M	6	Normal	Nil
19	AT	20	M	6	Normal	Nil	70	RB	27	M	4	Left > Rt MTLE	2
20	SJ	33	F	7	Rt Temporal Epilepsy	Nil	71	PA	21	F	7.5	Pri Gen epilepsy	Nil
21	IG	55	F	4	PNES	8	72	TS	16	M	4.5	PNES	3
22	CP	40	F	7	Normal	Nil	73	SK	18	M	1	Gen Tonic	14
23	MK	35	M	5	PNES	3	74	RH	14	M	7.3	RT hemispheric Seizures	1
24	SS	9	F	6	Rt Post Qudrant Epilepsy	14	75	PA	21	F	7	PGE	nil
25	SK	6	M	5	Rt Post Qudrant Epilepsy	Nil	76	AA	12	F	7	RT hemispheric Seizures	Nil
26	KA	2	M	5.5	Symptomatic Generalise	Nil	77	AP	24	M	7	Sec LGS	Nil
27	MP	15	F	2	PNES	2	78	AD	15	F	6	Pri gen Epilepsy-Absence	Nil
28	VS	54	M	6	PNES	3	79	AS	15	M	2.5	Normal	nil
29	PP	51	M	5	PNES	2	80	KU	18	M	7	Gen Tonic-Symptomatic generalis	1
30	PG	34	M	6.5	Normal	Nil	81	RS	38	M	5	Rt MTLE	5
31	HM	24	M	7	Left MTLE	1	82	LG	61	M	7	Normal	Nil
32	US	4	M	6	WS-LGS	2	83	LG	61	M	7	Normal	Nil
33	MP	21	M	8	RT Parieto-Temporal	1	84	MR	20	M	6	No lateralization/laterlization	1
34	VS	24	M	6.25	Rt Post Qudrant Epilepsy	Nil	85	NA	21	F	7.5	Left MTLE	Nil
35	BK	44	M	4.5	Left P-T Epilepsy		86	AK	17	M	7	Left MTLE	0
36	NP	11	F	6	Normal	Nil	87	AK	17	M	7	Left MTLE	1
37	TJ	6	F	6	Rt Frontoploar	Nil	88	SS	42	M	6.5	Left Post temporal	3
38	RM	40	M	5	Left MTLE	Nil	89	SK	42	M	7	Normal	Nil
39	AS	5	M	3	Rt F-C, Left Ant Temoral	Nil	90	SW	8	M	6	Misf (left>rt)	Nil
40	VP	16	M	5.5	Pri Gen epilepsy-JME	3	91	PS	28	F	5.5	PGE (JME)	Nil
41	MK	5		5.5	LGS	nil	92	BA	62	M	7.5	Left Frontal	1
42	OW	12	M	7	Normal	Nil	93	AN	3.5	M	4	Post quadrant-sec generalised	3
43	OW	12	M	7	Rt Frontopolar epilepsy	1	94	MJ	42	F	7	Left MTLE	Nil
44	SB	17	F	5	Rt MTLE	3	95	MJ	42	F	7	Left MTLE	Nil
45	TD	51	F	6	PNES	Multiple	96	PA	11	M	4	Misf	Nil
46	SB	3.5	F	5	Normal	Nil	97	AM	12	M	7.5	Rt Post Quadrant Epilepsy	Nil
47	AP	1mth	M	2	Normal	Nil	98	SB	45	F	7.5	Normal	nil
48	SL	12	M	7.5	RT Frontal-Sec gen	Nil	99	AD	22	M	6	PGE (absence)	Nil
49	SD	35	M	7.5	Normal	Nil	100	MA	17	F	7.5	Left F-C Epilepsy	1
50	SD	35	M	7	Left MTS	nil							

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Uncommon lesions in the medial temporal lobe presenting with intractable epilepsy

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Introduction: Medial temporal lobe is a major site of seizure origin. Lesions present in the medial temporal lobe might predominantly present with epilepsy which might even be refractory to anti-epileptic drugs. We describe 8 uncommon lesions involving the medial temporal lobe which presented with intractable seizures.

Material and methods: 8 patients were included in the study from July, 2014 to July, 2015 who had presented to a tertiary care centre with seizures which were not controlled on medications. Complete clinical and radiological assessment of these cases was done. Treatment received and the seizure outcome (Engel's grade) were also noted.

Results: 6 cases presented with complex partial seizures out of which 5 had olfactory auras. 5 patients had right sided lesions and remaining 3 had left sided lesions. Among these 8 cases, 2 were tuberculomas and cavernomas each, 1 was epidermoid, 1 was ganglioglioma and 1 was a low grade glioma. All patients had a complete excision of the concerned lesion. Anterior medial temporal lobe resection (including amygdale and hippocampal resection) was performed in all these cases. 7 cases had Engel grade 1 seizure control and 1 had Engel grade 2 seizure control. No significant post-operative complication occurred in any of the patients.

Conclusion: Medial temporal lobe may harbour various pathologies and due to its location, it predisposes the patient for seizures. Lesionectomy when combined with AMTR gives good seizure control.

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RNA-Seq analysis of hippocampal tissues reveals novel candidate genes for drug refractory epilepsy in patients with MTLE-HS

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Array-based profiling studies shows aberrant gene expression patterns during epileptogenesis. We have performed RNAseq analysis of the hippocampal tissues resected from the patients with MTLE-HS to investigate the molecular basis of epileptogenicity and/or pharmacoresistance in MTLE. For non-epileptic control experiments, healthy tissues from tumour margins obtained during tumour surgeries were used. RNA sequencing was performed using standard protocols on Illumina HiSeq 2500 platform. Differential gene expression