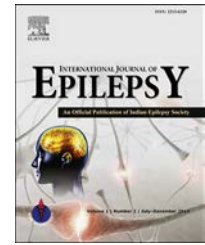


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## Scientific Abstracts: Asian Epilepsy Surgery Congress – Udaipur (India) October 23–25, 2015

### 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 multilobar 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, multilobar 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



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	Inti site	Age	Sex	V EEG Duration (hrs)	Abnormality	No of events	No	Inti site	Age	Sex	V EEG Duration (hrs)	Abnormality	No of events
1 SD	10	MA	2	Normal	Nil	52 VP	11	F	7	PNES	Nil	1	
2 AD	8	MA	2	Normal	Nil	53 AS	34	MA	6	RT Hemisphere dysfunction	Nil	1	
3 SP	1imp	F	6	Left MTL	Nil	54 JS	38	MA	6.5	RI Post Quadrant Epilepsy	Nil	1	
4 AN	2	MA	6	West syndrome	Nil	55 SA	7.5	MA	4.5	Left Frontal SMA Se	Nil	3	
5 HC	25	MA	6	Normal	Nil	56 SC	31	F	7.5	RI Ant Temp. Epilepsy	Nil	3	
6 AV	18	F	6	Normal	Nil	57 SP	31	F	7	Left MTL	Nil	3	
7 VS	15	F	6	Normal	Nil	58 SN	56	F	6	Left MTL	Nil	3	
8 WC	55	MA	6	Left MTL	Nil	59 UJ	32	F	4	PNES	Nil	3	
9 VS	37	F	6	Normal	Nil	60 VJ	29	MA	4.5	PNES	Nil	4	
10 TD	15	F	8	RI Post epilepsy	Nil	61 VV	32	MA	4	PNES	Nil	2	
11 AK	27	F	6	Normal	Nil	62 VV	24	MA	7	RT Occital Epilepsy	Nil	1	
12 VV	13	MA	8	PNES	2	63 AP	30	F	7	PNES	Nil	4	
13 SJ	30	F	6	RI MTL	3	64 KC	18	MA	5.5	Normal	Nil	1	
14 GS	12	F	6	RI MTL	2	65 AP	34	MA	6	Left Centro-Temporal Epilepsy	Nil	9	
15 GC	14	MA	6	Normal	0	66 DB	28	MA	7	RI Post Quadrant Epilepsy	Nil	1	
16 PK	16	F	7	Normal	Nil	67 DB	28	MA	5	RI Post Quadrant Epilepsy	Nil	1	
17 KD	3	F	4	Normal	5	68 GA	3.5	MA	3.5	Secondary LGS	Nil	22	
18 SP	14	MA	4	Typical absence	5	69 RB	27	MA	6	Normal	Nil	1	
19 AT	20	MA	6	Normal	Nil	70 RB	27	MA	4	Left > RI MTL	2	2	
20 SJ	33	F	7	RI Temporal Epilepsy	Nil	71 RA	21	F	7.5	RI Gen epilepsy	Nil	1	
21 HG	55	F	4	PNES	8	72 TS	18	MA	4.5	PNES	3	3	
22 CP	40	F	6	Normal	Nil	73 SK	18	MA	1	Gen Tonic	Nil	14	
23 MK	35	MA	5	PNES	3	74 RH	14	MA	7.3	RT Hemisphere Seizures	3	1	
24 SD	9	F	6	RI Post Quadrant Epilepsy	3	75 RA	21	F	7	PNES	Nil	1	
25 SK	6	MA	5	RI Post Quadrant Epilepsy	Nil	76 AA	12	F	7	RT Hemisphere Seizures	Nil	1	
26 SD	2	MA	6	Post Symptomatic Generalis	Nil	77 AS	24	MA	2.5	Normal	Nil	1	
27 RMP	15	F	2	PNES	2	78 AD	15	F	6	RI Gen Epilepsy-Absence	Nil	1	
28 VS	34	MA	6	PNES	3	79 AS	24	MA	2.5	Normal	Nil	1	
29 WP	51	MA	6	PNES	2	80 KJ	18	MA	7	Gen Tonic-Symptomatic generalis	Nil	1	
30 PG	34	MA	6.5	Normal	1	81 BS	38	MA	5	RI MTL	5	1	
31 NEM	24	MA	7	Left MTL	1	82 SJ	63	MA	7	Normal	Nil	1	
32 US	4	MA	6	MS-LGS	2	83 SJ	63	MA	7	Normal	Nil	1	
33 MP	23	MA	7	RI Centro-Temporal	3	84 NR	26	MA	6	No localization/lateralization	Nil	1	
34 VS	24	MA	6.25	RI Post Quadrant Epilepsy	Nil	85 NA	21	F	7.5	Left MTL	Nil	1	
35 MC	44	MA	6.5	Left RT Epilepsy	Nil	86 AK	17	MA	7	Left MTL	Nil	1	
36 AP	11	F	6	Normal	Nil	87 AK	17	MA	7	Left MTL	Nil	1	
37 JJ	6	F	6	RI Frontopolar	Nil	88 AS	22	MA	7.5	RI Post temporal	Nil	1	
38 RM	40	MA	5	Left MTL	Nil	89 SN	25	MA	7	Normal	Nil	1	
39 AS	5	MA	6	RI F-C Left Ant Temporal	Nil	90 SA	2	MA	6	Self (convuls)	Nil	1	
40 VP	16	MA	5.5	RI Gen epilepsy-IME	3	91 PS	28	F	5.5	PNES (LME)	Nil	1	
41 MK	5	MA	5.5	LGS	Nil	92 RA	62	MA	7.5	Left Frontal	Nil	1	
42 DW	17	F	7	Normal	1	93 AJ	3.5	MA	4	Post quadrant-sec generalisad	Nil	1	
43 CW	12	MA	6	RI Frontopolar epilepsy	1	94 MJ	42	F	7	Left MTL	Nil	1	
44 SB	17	F	6	Normal	3	95 AJ	42	F	7	Left MTL	Nil	1	
45 TD	51	F	6	PNES	Multifoc	96 PA	13	MA	4	MAL	Nil	1	
46 SB	3.5	F	6	Normal	3	97 AM	12	MA	7.5	RI Post Quadrant Epilepsy	Nil	1	
47 AP	1imp	MA	2	Normal	Nil	98 SB	45	F	7.5	Normal	Nil	1	
48 SJ	12	MA	7.5	RI Post-Quadrant-sec gen	Nil	99 AD	22	MA	6	PNES (Absence)	Nil	1	
49 SD	35	MA	7.5	Normal	Nil	100 MA	17	F	7.5	Left F-C Epilepsy	Nil	1	
50 SD	35	MA	7	Left MTL	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