

EEG in CLINICAL PSYCHIATRY

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VALUE OF DIAGNOSTIC TESTS

- Cost &
- Inconvenience
- VS.
- Information Obtained

IS IT REALLY DEMENTIA?

56 Y/O woman diagnosed with Alzheimer's Disease and has been in a nursing home, and wheel chair bound, for 4 years. She stopped eating and was brought to the County Psychiatric Center ER. She was admitted to the Neuropsychiatry ward. In reviewing her history a complete work up was never performed. A comprehensive evaluation was started including blood work, CT and EEG. There was nothing significantly abnormal on any test. EEG was perfectly normal.

Given the advanced stage of AD a normal EEG was incompatible with the picture. A DST was performed and was highly non-suppressor.

An anti-depressant was started. Nothing happened for two weeks. During the third week the patient started speaking. Her speech was completely disorganized with severe psychotic content. Haloperidol was added and the³ patient was able to leave the hospital ambulatory after two more weeks.

IS IT REALLY DTs?

44 Y/O man with a long history of alcohol dependence. He presented to the ER intoxicated with an alcohol level of 0.42. He was admitted for detox. As his level started coming down he started becoming confused. Benzodiazepine dose was increased. He progressively became agitated and was placed in 4-point restraints. A Neurology consult was called. The consultant strongly suggested increasing the benzodiazepine. Patient continued to escalate despite increasing doses of the medication. Later on that same day a Medicine consult was called. They suggested that the patient was in "iatrogenic Delirium". They recommended stopping all meds.

An EEG was obtained and revealed a moderate degree of diffuse slowing consistent with encephalopathy and not the fast low-voltage picture expected with DTs.

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Medications were rapidly tapered and patient progressively recovered

IS IT REALLY ACUTE MANIA?

A 24 Y/O woman, single and mother of 3 children. No past psychiatric history. She partied with her friends for two nights (out all night dancing). On Sunday evening she was unable to sleep. Monday AM she was agitated and having religious delusions and some hallucinations. She was brought to the ER by her mother because "something was wrong with her". ER urine and blood works were normal including drug screens.

Patient was agitated, hyperactive and with severely pressured speech. Patient oriented only to place and person. Memory for three words was deficient at 3 minutes. **Diagnosis of acute mania was made.**

Patient admitted and over the next three days Valproic acid started, olanzapine, ativan, and eventually low dose haloperidol were added with no effects. Actually patient continued to esclate and had to be in restraints or secluded. In the fourth day she had diffuculty breathing and was trasnferred to ICU where she experienced a generalized tonic clonic seizure and lapsed into coma.

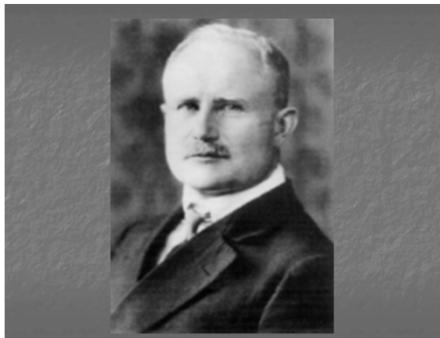
Autopsy, with detailed brain examination for all possible rare diseases, proved negative.

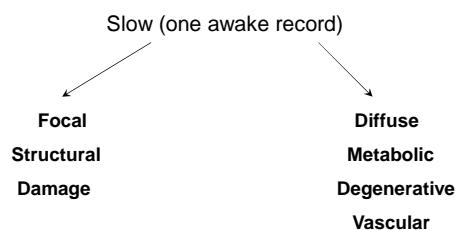
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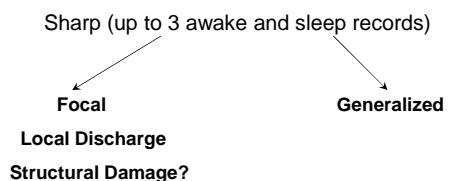
Would an EEG, performed early in the course, have made a difference ?

ANSWER IS YES

- A Normal EEG supports functional disorder.
- Diffusely slow EEG suggests an encephalopathy and would have resulted in a work-up and a Neurology consult.
- Status epilepticus (ambulatory-Non convulsive). Dx made and Tx instituted.



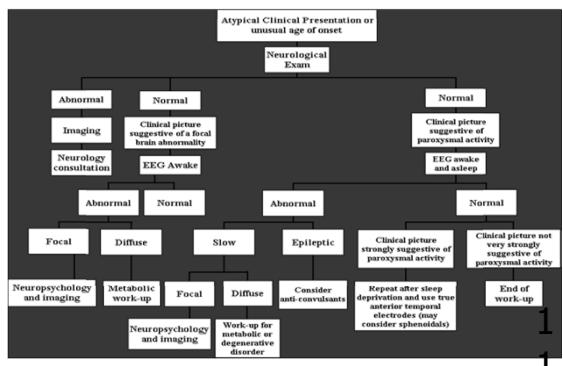




- Non-invasive
- Inexpensive
- Widely available

- High false negative rate.
- Criteria for abnormality varies among clinicians and researchers.
- Qualitative.

EEG in Clinical Psychiatry



What do EEG abnormalities tell us?

- 1) Epileptiform discharges suggest anticonvulsants could be useful.
- 2) Diffuse slowing in the absence of medications suggests a diffuse metabolic or degenerative disorder. Needs work up.
- 3) Focal slowing suggests early damage. Aberrant behavior may be compensatory or adaptive?

Specific Disorders

- Autistic Spectrum Disorders (ASD)
- Panic Attacks
- Aggression
- Treatment unresponsive ADHD
- Atypical Bipolar Disorder or cyclothymia.

EEG abnormalities in ASD

Findji et al (1979) described an 8-year-old child whose behavior alternated between excitation and autism with stereotypes. The EEG showed 5c/sec temporo-parietal sharp wave discharges lasting from 1 second to 20 minutes. These discharges (even the prolonged ones) were not associated with any clinical signs of epilepsy. Gillberg & Schaumann (1983) described two cases of infantile autism without clinical seizures, where EEG abnormalities were not discovered until **relatively late** in the course of the psychiatric disorder. Anticonvulsant medications led to the complete disappearance of psychotic symptoms and to simultaneous disappearance of the pathological EEG changes.

- Hashimoto et al. (2001) examined the EEGs (during sleep) of 86 autistic children. Forty-three % (37 cases) had epileptic discharges.

- Rossi and colleagues (1995)
- 106 autistic patients without any evidence of a congenital or acquired encephalopathies were examined. They reported a prevalence of 18.9% of EEG abnormalities in children without epilepsy. Epileptiform activity was focal and multifocal.

Screening EEGs in ASD (Kagan-Kushnir et al 2005) ture.

- 1) They concluded that **seizures are common** occurring in 20-30%.
- 2) **Subclinical EEG abnormalities (i.e., no epilepsy) was found in 6.1 to 31%.**
- 3) Evidence for the **effectiveness of anticonvulsants and corticosteroids** in reducing seizures and/or autistic symptoms is based primarily on **case series, with only one published randomized trial.**
- 4) They concluded that, as of the time of the report, **there is insufficient evidence to recommend against the use of screening EEGs in autistic patients.**
- 5) They also conclude that given the high frequency of seizure disorders in this population that a high index of clinical suspicion should be maintained for subtle symptoms of seizures.

Response to anticonvulsant treatment.

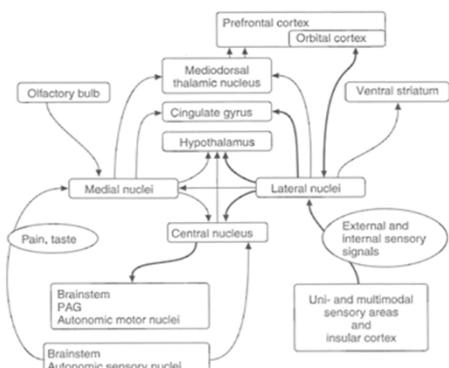
- Hollander et al (2001) conducted a retrospective pilot study to determine whether divalproex sodium was effective in treating core dimensions and associated features of autism.
- They included 14 patients with ASD. Subjects were included irrespective of h/o seizures or EEG abnormalities. Ten of the 14 patients who completed the trial (71%) were rated as responsive to treatment (mean dose 768mg/day and range of 125-2500 mg/day).
- Improvement was noted in core symptoms of autism as well as the associated features of affective instability, impulsivity, and aggression. **Of note is that all patients with abnormal EEGs were rated as responders.**

Response to anticonvulsant

- Hollander et al, 2006.
- Double-blind, Placebo-controlled.
- 13 ASD patients.
- C-YBOX
- Significant differences in repetitive behavior with a large ES (1.6).
- Int. J. Neuropsychopharm 9:209-213.

Panic Disorder

- Panic symptoms carry a significant resemblance to symptoms induced by temporo-limbic epileptic activity particularly those originating from the Sylvian fissure. Fear, derealization, tachycardia, diaphoresis, and abdominal discomfort are characteristic symptoms of simple partial seizures with psychiatric and autonomic symptomatology.



EEG and ADHD

- Franks (1993), 31% abnormal EEGs (21 of 64). Of the 21, 84% had epileptiform activity.
- Philips (1993) 9% definite abnormalities. Excluded controversial patterns and considered findings to be "negative".
- Hughes (2000), examined EEGs of 176 ADHD children. 30.1 % with definite abnormalities.

ADHD

- Hughes (2000) 18.8% additional ADHD children with the 14 & 6 positive spikes.
- Boutros 1998, correlated PS with ADHD.
- Millicap 2000, 7% strongly suggestive of epilepsy, additional 19% abnormal but not epileptiform.
- Wojna (2000), one third abnormal.
- Gustafson (2000) one third abnormal.

Seizure risk in ADHD with stimulant therapy (Hemmer SA. et al, 2001)

- 234 Children with ADHD
- 36 (15%) epileptiform abnormalities.
- Rolandic spikes accounted for 40% of abnormalities.

*Seizure incidence with NORMAL EEG .6%
 Seizure incidence with abnormal EEG 6%
 2 of 12 with Rolandic spikes had seizures (16.7%).

Mood Disorders

- Atypicality of presentation
- History of seizures
- History of head injury
- Abnormal EEG
- All predict good response to valproate in Bipolar patients.
- Stoll al, et al. J Clin Psychopharmacology, 14:311-313, 1994.
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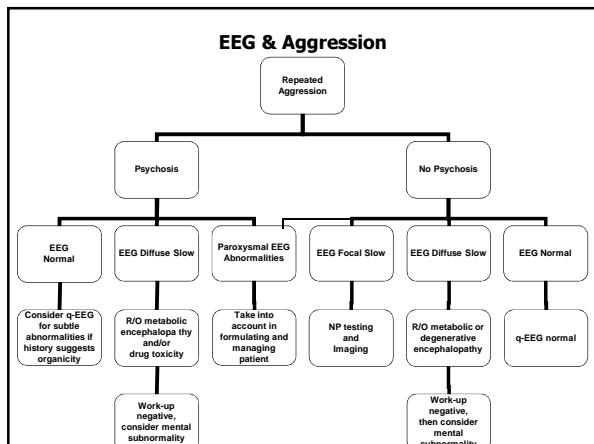
EEG & Aggression

The prevalence of abnormal EEGs in clinical populations with aggressive symptoms vary widely among studies ranging from as low as 6.6% in patients with rage attacks and episodic violent behavior (Riley, Niedermeyer, 1978) to as high as 53% in patients diagnosed with antisocial personality disorder (Harper et al, 1972).

Hill and Watterson (1942) examined the EEGs of 151 subjects with psychopathic personalities. They reported 48% of this group to exhibit abnormal EEGs as compared to 15% of a non-patient control group.

- *65% of aggressive patients and only 32% of non-aggressive subjects to exhibit abnormal EEGs.
- *Significant relationship between history of head injury and presence of EEG abnormalities.
- *The more aggressive the patient the more likely the EEG to be abnormal.

It has also been shown that among groups of prisoners convicted of murder, the highest incidence of EEG abnormalities (74%) occurred in individuals whose crimes were apparently motiveless or had minimal motives (Stafford-Clark and Taylor, 1949).



Psychiatric Indications for a Standard EEG

- Atypical presentations
- Atypical symptoms
- Panic attacks and other dissociative Sx
- Repetitive violence (more if unmotivated)
- Medically un-responsive ADHD
- ASD
- Acute confusional state (if cause unknown)
- DAMS
- Dementia vs Depression

EEG is a noninvasive and economic way of evaluating organicity

■BREAK
