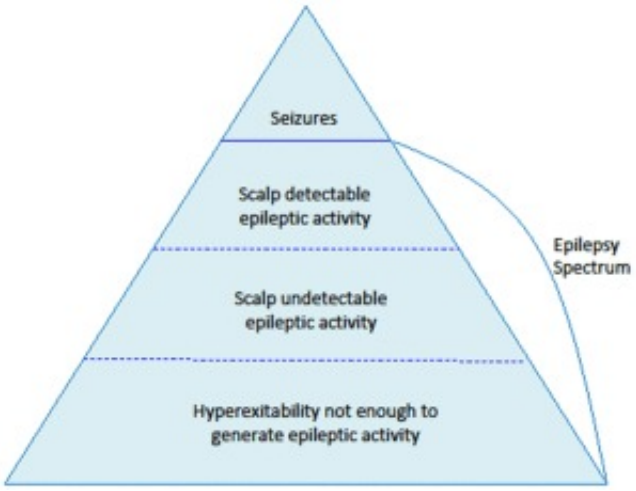
Epilepsy spectrum disorders

Symptoms of mental illness (like depression or hallucinations) tend to change slowly overtime (with or without treatment). At time individuals may experience episodic symptoms like sudden and brief panic attacks or sudden and brief violent episodes. While episodic psychiatric symptoms are not uncommon they have not been researched as extensively as other more enduring type of symptoms. Some evidence has accumulated over the years that at least in a subset of patients exhibiting these symptoms there may be evidence for the presence of an abnormally increased irritability of localized areas of neural (brain) tissue. These focal areas could be in the cerebral cortex (the outer layer of the brain or cortical areas) or in deeper brain structures (subcortical). This increased irritability reflects a dysbalance between the excitatory and inhibitory chemicals in the brain and leads to a state of hyperexcitability. In its extreme form this is the condition that leads to seizures (i.e, epilepsy). In these cases the condition could be conceptualized as an epilepsy spectrum disorder (ESD) with significant treatment implications. Seizures thus represent the tip of the iceberg of a pyramid of increasing degrees of hyperexcitability (Figure).



Tip of the Iceberg

There is currently no clear demarcation of this category of symptoms, their prevalence is unknown, there is minimal understanding of how these symptoms are produced by the brain and what is appropriate work up and possible treatments. We here propose that milder degrees of increased neural excitability (i.e., a subthreshold excitation insufficient to cause seizures) may nonetheless be capable of causing observable behavioral changes. The observable behavioral changes depend on the degree of hyperexcitability and the location of the hyperexcitable brain tissue. For example, a hyperexcitable area in the brain system that mediates emotions (limbic system) may manifest as a panic disorder whereas hyperexcitable brain tissue in the frontal lobe area (mediating self-control

and cognition) may manifest as rage attacks. The location of the abnormal brain tissue may dictate the initial manifestation of an attack resulting from activation of the hyperexcitable tissue, but the anatomical connectivity (brain areas connected to each other) of the abnormal region will dictate the breadth of the manifestations. The following criteria for diagnosing ESD are proposed:

A: 1) Probable ESD: Presence of any form of episodic symptoms. An examination by an expert epileptologist is likely useful at this stage to rule out clinically diagnosable epileptic attacks. Episodic symptoms can include but not limited to: panic and other dissociative attacks, episodic violence; and sudden dysphoric mood and/or 2) an elevated score on the Structured Clinical Interview for Complex Partial Seizure-like Symptoms (SCIPS) (we preliminarily recommend a score of 62.8 or higher). The SCIPS is a standardized symptom interview with 35 items describing cognitive, affective, and psychosensory symptoms associated with partial seizure disorders. The predictive value of this SCIPS score for a favorable response to anti-epileptic drugs (AEDs) must be tested in well-designed studies to avoid placing too many patients on potentially toxic AEDs.

B: Possible ESD: 1) Evidence from electrophysiological studies like the electroencephalogram (EEG) for epilepsy-like activity. Long-term monitoring in specialized epilepsy monitoring units could be able to detect abnormalities particularly during actual episodes; and/or 2) Evidence from positron emission tomography (PET) scanning for a localized abnormality suggestive of an epileptic process; and/or 3) Neuropsychology testing suggestive of focal deficit in a brain region relevant to the clinical presentation.
C) Definite: A significant clinical response to anticonvulsant medications when the above criteria are present.

Publication

Epilepsy spectrum disorders: A concept in need of validation or refutation.
Boutros NN, Bowyer S, Wang J, Urfy MZ, Loeb JA
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