S2-1 From precipitation to inhibition of seizures: rationale of a therapeutic paradigm

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Epileptic seizures can be triggered by both non-specific facilitating factors such as sleep withdrawal, fever or excessive alcohol intake, and specific reflex mechanisms. These consist in sensory or cognitive inputs activating circumscribed cortical areas or functional anatomic systems which, due to some functional instability, respond with an epileptic discharge. Interruption of seizure activity at the stage of the aura (i.e. locally restricted discharge) can also be achieved by non-specific (e.g. relaxation or concentration techniques or vagal nerve stimulation) or by specific focus-targeted sensory or cognitive inputs. The latter, again, activate circumscribed cortical areas.

Intriguingly, in some patients it is the same stimulus which can both precipitate or abort a seizure. The response depends upon the state of cortical activation: seizure precipitation occurs in the resting condition, seizure interruption when epileptic discharge has begun close to the activated area. These relations can be understood on the background of experimental data showing that an intermediate state of neuronal activation is a precondition for the generation of paroxysmal depolarisation shifts, whereas a hyperpolarized neuron will remain subthreshold, and a depolarised neuron which already produces action potentials is not recruitable for other activity.

Sensory input meeting an immediately activated pool of potentially epileptic neurons is adequate to produce a seizure. In another condition, the same stimulus can depolarise a neuron pool in the same area sufficiently to block the further propagation of near-by epileptic activity.

S2-2 Geographically specific reflex epileptic syndromes in India: precipitation and inhibiting factors

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Sensory evocation plays a critical role in seizure induction in about 5-6% of epileptic patients. There are geographically specific reflex epileptic seizures precipitated by certain specific stimulus in India- Hot water epilepsy and eating epilepsy. Hot water epilepsy (HWE) though reported from many parts of the world, most frequently reported from India. We have published one of the largest series of HWE. We have developed a rat model mimicking human HWE and hypothesized that human HWE is a 'hyperthermic seizure'. We have postulated that aberrant thermoregulatory system in genetically susceptible individual is responsible for this type of reflex epilepsy. Hyperthermia with probable somatosensory factors could be the triggering factor. We have successfully demonstrated 'hyperthermic kindling' in the rat model of HWE. Recently we have studied interictal/ictal SPECT scan using 99m ECD in human HWE, demonstrating functional derangement in the hypothalamus and medial temporal structures during ictus. Knowing these factors led us to change the concept in the management of HWE. Eating epilepsy more frequently reported from Sri Lanka and parts of India is a symptomatic localization related epilepsy. This is probably due to the excitation of the critical mass of the epileptogenic cortex by various afferent stimuli located in the lingual, buccal and pharynx activated by extensive sensory input. These geographically specific reflex epileptic syndromes have probably multiple trigger factors such as genetic susceptibility with environmental factors playing an important role. Further work in this direction is in progress to delineate these factors.