Letter to the Editor

Meditation: Epileptogenic versus antiepileptic influence

To the Editor:

Meditation is a widely adopted stress–relieving mental exercise associated with altered cognition and behavior. Recently, there has been debate [1,2] over the influence of meditation, and transcendental meditation (TM) in particular, on epilepsy. This debate was seemingly triggered by a view suggesting a potential epileptogenic influence of meditation [3], implying that long-term meditation can potentially cause development of an epileptic state de novo or predispose the brain to epileptogenesis (epileptogenic influence) with no consideration of the frequency, intensity, or duration of epileptic seizures in otherwise already established case(s) of epilepsy. Comprehension of the distinction between epileptogenic influence and antiepileptic influence is essential as the proponents of meditation have claimed and reported anecdotal experiences of a decrease in frequency and even control of seizures by meditation in established cases of epilepsy (antiepileptic role). The epileptogenic influence is essentially based on meditation-induced alterations [3] in neurophysiology (hypersynchrony and increased coherence of brain activity) and neurochemistry (release of glutamate and serotonin).

Lansky and St. Louis [4] describe a patient with new-onset epilepsy, who they claim is the first well-documented case reported in the literature of epilepsy caused by long-term meditational practice (epileptogenic influence).

Synchronized gamma activity, which has been found to correlate clinically and in animal models of epilepsy [5], has also been observed to occur as a self-induced neurophysiological alteration in long-term meditators [6].

It is documented that in established cases of epilepsy, the occurrence of seizures is the result of excitatory versus inhibitory influences in the epileptic region and/or its surround. A number of factors are known to trigger an epileptic seizure(s). Several studies have reported self-perception of trigger factors/seizure precipitants in patients with epilepsy. The commonest epileptic precipitant that has emerged consistently from these studies is stress, with sleep deprivation closely behind. Although no convincing explanation or mechanism for its antiepileptic influence has been presented, it can be postulated that meditation, which is recognized as a powerful and effective stress reliever, can effectively reduce the frequency, intensity, and duration of or even control epileptic seizures by alleviating stress.

Pagano et al. [7] observed non-REM sleep stages II, III, and IV in individuals during meditation, and it is found that sleep deprivation is a common trigger in patients with epilepsy. Therefore, the contribution of the sleep-inducing effect of meditation to its anti-epileptic influence cannot be ruled out in sleep-deprived patients with epilepsy.

The biochemical basis of the antiepileptic influence of meditation in established cases of epilepsy can be briefly outlined as follows: (1) The glutamate released by the prefrontal cortex during meditation is converted into GABA (potent inhibitory neurotransmitter) via the glutamate decarboxylase pathway [8]. (2) Serotonin exerts a complex effect on epilepsy, both facilitating and inhibiting seizure occurrence [9]; this can be assumed to be due to its differential electrophysiological effects on 5-HT receptor subtypes. In brain, activation of 5-HT1A, 5-HT2A receptors causes hyperpolarization of neuronal membrane through increased K+ conductance, whereas activation of 5-HT2A and 5-HT4 receptors causes slow depolarization via decrease in K+ conductance; fast depolarization is mediated by 5-HT3 receptor activation [10]. Thus, meditation does appear to exert dual effects on epilepsy in a complex manner, the behavioral outcome of which could be development of an epileptic state in normal or epilepsy-prone individuals, on the one hand, and exertion of an antiepileptic influence in established cases of epilepsy, on the other hand.

References


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