

Review

# Scientific basis behind traditional practice of application of “shoe-smell” in controlling epileptic seizures in the eastern countries

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Received 4 August 2007; received in revised form 28 December 2007; accepted 6 February 2008

## Abstract

Epilepsy has been known for thousands of years and has been subjected to various forms of conventional and non-conventional therapies including a non-pharmacological conservative treatment known as aromatherapy, ever since. One commonly practiced form of aromatherapy that persists as an immediate first-aid measure even today in some parts of developing countries in the East is the application of “shoe-smell” during an epileptic attack. The questionable remedial role has intrigued neuro-scientists at least in these parts of the world. This brief paper attempts to provide an insight to the basis of persistence of this practice and to explore a possible scientific logic behind its unscientifically reported remedial effectiveness. The neurophysiology of olfactory stimulation from “shoe-smell” reveals a sound and scientific reasoning for its remedial efficacy in epilepsy; olfactory stimuli in this study have been found to possess significantly effective anti-epileptic influence which could have formed the basis for the use of application of “shoe-smell” in earlier times and also for its persistence even today in those parts of developing regions.

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*Keywords:* Aromatherapy; De-synchronization; Epilepsy; Olfaction; Shoe-smell; Synchronization

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## 1. Introduction

Epilepsy is a very common disorder ranking second among neurological disorders even in developed countries. People have known about epilepsy for thousands of years

and many explanations have been put forward concerning its etio-pathogenesis, which still remains a domain under intense exploration. In earlier times, seizures were supposed to be caused by demons attacking the person; different spirits were thought to cause the different kinds of seizures; the patient was supposed to be “seized” by evil spirits, hence the term “seizure”.

In Europe in the Middle Ages, epilepsy was called the falling sickness, and people looked to saints and relics for cures. There were different remedies suggested by various

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categories of wise people, for example “The three wise men and St. Valentine” were particularly important patrons of people with epilepsy; “if you had epilepsy you could wear a special blessed ring that would help control your seizures” was an advice in vogue in those days.

Many different methods have been tried and adopted by epileptic patients from learning and recognizing trigger factors and situations that precipitate their attacks. The advantage of these physical and psychological therapies is that the epileptic patient learns to control his/her attacks (to a variable extent), the feeling of which boosts confidence and the patient feels psychologically more secure and strong. This feeling is not only likely to improve the quality of life but can also actually reduce seizures.

Aromatherapy has been used since the beginning of civilization to treat a number of conditions in which treatments require the use of a variety of extracted fragrant essential oils. There are many different ways to use essential oils including inhalation, diffusion, massage, bathing, hot and cold compresses, and perfumes.

Aromatherapy has been used to help people who experience auras, which warn of an impending seizure. Use of certain oils may help to prevent or lessen the severity of an epileptic attack if inhaled gently before the seizure occurs as vigorous sniffing can in itself trigger a seizure. Some oils can trigger seizures and should therefore be avoided by people with epilepsy. These oils include rosemary, sage, hyssop, fennel and wormwood. In ancient medicine also, unpleasant odors like burning hartshorn were used to ward off impending seizure or even actual attacks.

Aromatherapy has been tried as a behavioral form of medicinal approach to control epilepsy [1,2]. Research carried out at the Queen Elizabeth Hospital, Birmingham by Dr. Tim Betts demonstrated that aromatherapy could help in controlling epilepsy [3]. In some people, whose seizures are preceded by an aura, breathing in the scent of the aromatic oils at the start of the warning can reduce the chance, or severity, of an epileptic attack. It has been suggested that smell can act as a countermeasure in epilepsy because of its property of evoking activity in the same cortical region where epileptic potentials arise very often. Efron showed [4] that odor not only acts as a countermeasure but can also be conditioned easily resulting in formation of smell-memory (odor-memory), which can help inhibit epileptic activity or its spread or both. Further, such memories are not easily de-conditioned or erased. EEG changes during olfactory stimulation have also been observed in several studies [5,6].

Some Eastern parts of the world like India have witnessed since time immemorial, a practice of application of “shoe-smelling” in an attempt to arrest the seizures. The practice consisted of bringing the sole of shoe near the nostrils of the patient during the epileptic attack by near-by attendants or passers-by in the event of the attack occurring in a public place. The practice has continued and still remains a form of first-aid treatment in developing countries especially in

countryside and rural areas. Although today, this age-old practice of “shoe-smell” may sound ridiculous apart from being most unscientific, its persistence as a remedy does tempt researchers to provide an insight to the reasons and basis for this continuing practice.

Thus, this short article attempts to shed light on the reasons for the earlier usage of this practice as well as to explore a possible scientific basis behind its persistence even today.

## 2. Discussion

### 2.1. Author's explanation

The fact that this purported remedy of “shoe-smell” continues to remain in vogue raises possibility of existence of an underlying scientific basis although admittedly, some rituals and superstitions persist even without factual base. Various degrees of relief have also been noticed and reported albeit not recorded scientifically, which also therefore leads one to assume there could be a possible sound scientific logic to explain the “relief” in epilepsy by this time tested practice. The author attempts to provide an explanation as follows.

Temporal lobe epilepsy (TLE) still remains one of the commonest forms of epilepsy even today. In olden times too, it was most likely to have been the commonest epilepsy on account of difficult child births leading to birth asphyxia, febrile seizures and head injuries not having been paid attention to, not to mention the states of poorly treated brain infections; for the reasons cited above this assumption may not be viewed as a mere speculation. Temporal lobe is known to be very susceptible to head injury, hypoxia–ischemia effects and febrile seizures during childhood, and the association of TLE with febrile seizures is commonly observed [7].

In earlier times, at least in case of temporal seizures with secondary generalization, strong olfaction (in the form of an old shoe) is likely to have succeeded in halting the progress of the seizure and aborting its generalization. People may have learnt this remedial effect from the above fortuitous observation. In those days, shoes were commonly made from leather and the other contaminants like sweat, dust, mud, etc., may have contributed to the strong smell emanating from the shoes; further, the easy availability of the shoes as a first and handy aid coupled with difficult accessibility to medical aids (both the physician and the drugs) aided in the evolution and development of the application of “shoe-smell” as an important first-aid treatment for epileptic seizures.

Existence of an inherent relation between smell and TLE especially uncinat seizures has been known for a long time, uncus being phylogenetically a part of olfactory brain. Olfactory hallucinations and auras often accompany temporal lobe seizures [8,9]. Olfactory areas are in close proximity as well as directly connected to regions where seizures develop in TLE and neuronal activity generated by olfaction can thus prevent the spread of synchronous activity responsible for the epileptic attack.

Epileptic seizures are no longer considered as random events. Despite the presence of damaged neurons that fire in the epileptic mode continuously and are called pacemaker cells, seizures arise only spasmodically [10]. Attacks occur in focal seizures when the pools of neurons surrounding the epilepsy focus are sufficiently excited for seizure activity to spread. Generalized seizures occur when the level of cortical excitability, or corticoreticular excitation, has reached a point at which thalamic recruiting volleys generalize and start to spread [11].

Rajna and Lona demonstrated that external stimulation can inhibit epileptic seizures. They found a strong inhibitory effect of acoustic stimuli on absence seizures in 16 of 19 patients proved by EEG. The peripheral sensory stimulation in the form of vagus nerve stimulation has emerged as a well-accepted technique for the treatment of intractable epilepsy. If external stimuli can inhibit seizures, it is reasonable to assume that patients or their attendants can intentionally inhibit their seizures. Regular seizure inhibition experience and reduction in seizure frequency has been observed in patients suffering from milder forms of epilepsy which suggests that intentionally inhibiting seizures can be recommended in treatment of mild forms of epilepsy [12].

Shoe-smell was applied at the time of initiation or warning signs of the attack, even during the full-blown state, as the laymen had no idea of the physiological basis behind an epileptic attack. The application appears to have become a stereotyped immediate first-aid remedial measure irrespective of the phase of the epileptic attack. The author does not intend to state that the application was effective (even to a variable extent) in all the epileptic patients, but does emphasize that it must have been effective in a definite proportion of the epileptic population and this observation has been instrumental in the sustenance of this form of remedy.

## 2.2. *Neurophysiological basis*

Synchronization and de-synchronization of cortical activity are known to play a basic role in seizure susceptibility and resistance, respectively. It is well documented that EEG synchronization forms the main underlying basis for development of an epileptic state whereas de-synchronization exerts anti-epileptic influence. It is believed that epileptic seizures occur due to hypersynchronous discharges [13]. Spikes itself are caused by synchronization of population group 1 neurons [14] and depict interictal epileptiform activity and their correlation with intracellular recordings shows that the former are associated with firing of action potentials [15]. Anterior thalamic stimulation (low frequency) is found to lead to cortical synchronization and increased susceptibility to epileptogenesis, whereas high frequency stimulation leads to de-synchronization and resistance to epilepsy [16].

Neural generators of synchronous EEG oscillations during non-rapid eye movement (NREM) sleep can promote electrographic seizure propagation while the neural generators of asynchronous neuronal discharge reduce electrographic

seizures during rapid eye movement (REM) sleep and wake alert state [17]. Hypersynchrony observed during NREM sleep facilitates both initiation and propagation of partial seizures [18]. Hyperventilation also causes synchrony and can precipitate epilepsy. On account of their EEG-synchronization property, NREM sleep and hyperventilation are both used as provocative techniques routinely during EEG recording.

REM sleep, characterized by low voltage fast activity (de-synchronization) is well documented to be associated with marked reduction, at times total absence of seizures, generalized bursts and abnormal discharges in EEG [18,19]. Some researchers consider REM sleep to be the most anti-epileptic state in the human wake–sleep cycle.

Similarly, wake alert state, which is also associated with EEG de-synchronization, not only reduces seizure frequency [14] but has been also found to inhibit epileptic seizures [20].

Vagal nerve stimulation (VNS) has emerged as an alternative therapy for intractable seizures, amongst which TLE accounts for a significant number of cases. VNS was approved in 1997 originally for partial seizures with secondary generalization. Although the exact mechanism of VNS action still remains to be elucidated, it has been proposed by me that a major mechanism of its anti-epileptic role is due to its widespread cortical de-synchronizing property via the reticular activating system (RAS) [21]; this mechanism has also been supported by the study of Marrosu et al. [22]. Olfactory stimuli can also cause widespread cortical de-synchronization in a somewhat similar manner. Olfactory stimuli have been found to control and inhibit epileptic attacks [23,24]. In 1956, Efron described a patient with temporal lobe seizures who also experienced stereotyped aura with secondary generalization; application of an unpleasant odor prevented spread of the seizure and even a subsequent conditioning of the olfactory stimulus to a bracelet also led to marked relief in the epileptic attacks [24]. Seizure propagation is also blocked when major part of involved neurons is rendered un-recruitable for spread of the epileptic discharge [25].

## 2.3. *Olfactory stimulation and seizure control*

Ebert and Loscher have reported that olfactory stimulation with toluene suppressed seizures in most of the kindled rats [26]. Olfactory stimulation with toluene or ammonia was found to increase the epileptic threshold. They also suggest that strong physiological stimulation like olfaction can interfere with ongoing seizure activity in the limbic system.

Efron has also reported a study in which the seizures could be inhibited by an olfactory stimulus [4,24]. Moreover, olfactory stimulus is one of the most frequently used methods for seizure inhibition [27,28].

It has been suggested that olfaction is likely to lead to widespread de-synchronization, akin to vagal nerve stimulation in exercising its seizure-reducing property. In one study, Moncrieff JW found that EEG data, collected dur-

ing presentation of odors, revealed a decrease in 8–13 Hz band (alpha) denoting widespread de-synchronization and suggesting increased activity in reticular activation system (RAS), a part of brain intimately related to thalamus. Anterior thalamic stimulation is also a recent emerging alternative technique for intractable epilepsy. The newer olfactory pathway passing through the thalamus can be responsible for the reticular excitation leading to widespread cortical de-synchronization.

De-synchronization can even arrest the ongoing epileptic activity and thus halt the progress of an epileptic attack, the explanation put forward is the alteration in the level of excitability in the area of epileptic focus that prevents seizure activity [14]. Olfactory stimulation induced de-synchronization (widespread or in the region of generation of epileptic activity in TLE patients) can thus play a major key role in arrest of seizure activity as explained earlier.

Some smells have been found to exacerbate epileptic attacks, suggesting that certain specific chemicals possess seizure-reducing properties; this subject however remains a topic for further investigation and assertion.

### 3. Conclusion

Thus, the above explanation does indicate that strong olfaction can aid in halting the progress of an epileptic seizure and/or abort the generalization of a partial seizure especially of temporal origin although more prospective studies are required to establish a clear and firm relation between the two, i.e. strong odor and seizure control. It may not therefore be incorrect to believe that in olden days too, strong olfaction applied in the form of “shoe-smell” did definitely play a suppressive role and thus exerted an inhibitory influence on epilepsy especially TLE, which was likely to have been the commonest form of epilepsy as even today.

This short paper is not intended to promote the use of “shoe-smell” as any form of remedy for epilepsy, but has been an endeavor to explore the scientific basis of its apparent remedial effect observed since long.

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