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Displaced aggression predicts switching deficits in people with temporal lobe epilepsy

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ABSTRACT

This study examined the relationship between task-switching abilities and displaced aggression in people with temporal lobe epilepsy (PWE). Participants (35 PWE and 35 healthy controls) performed emotion and gender classification switching tasks. People with temporal lobe epilepsy showed larger switch costs than controls. This result reflected task-switching deficits in PWE. People with temporal lobe epilepsy reported higher anger rumination, revenge planning, and behavioral displaced aggression compared with controls. Displaced aggression was a significant predictor of the task switch costs. It is suggested that displaced aggression is a significant marker of task-switching deficits.

1. Introduction

1.1. Temporal lobe epilepsy and aggression

Temporal lobe epilepsy (TLE) is one of the most common types of epilepsy in which seizures occur in temporal lobes and are often characterized by memory impairments and sensory changes. Seizures can be either complex or partial depending upon the symptoms. Complex partial seizures spread through a large area of the temporal lobe, which results in loss of consciousness. Simple partial seizures are confined to small areas in the temporal lobe such as the amygdala and hippocampus and do not disturb consciousness. Etiological factors range from traumatic injury, brain infections, mesial temporal sclerosis, to genetic syndromes [1].

Mood fluctuations and associated behavioral changes appear frequently in epilepsy, which have adverse effects on quality of life and functional capacities [2–5]. Changes in mood are a stronger predictor of quality of life than seizures [6,7]. Seizures are also associated with violent acts and aggressive behavior [8–10]. People with temporal lobe epilepsy (PWE) often report anger, low mood, irritability, and aggression towards others [11–15]; however, better coping mechanisms and social support can improve health-related quality of life [7].

Aggression in PWE can be categorized on the basis of its relationship with seizures: ictal, interictal, and postictal aggression. The ictal and postictal forms of aggression often complicate epilepsy where psychotic symptoms such as delusions, hallucinations, mood changes, and aggression may arise [16,17]. Interictal aggression is independent of ictal activity and may be related to antisocial personality disorder [16]. These symptoms may lead to self-destructive acts; episodic dyscontrol; and antisocial, catastrophic, and serious assaultive acts [17,18].

Significant associations have been found among several neuroanatomical regions/structures and aggressive behaviors, such as reduction in prefrontal gray matter which contributes to aggressive impulses [19]. Regions of the prefrontal cortex (PFC) are involved in aggression as well as in other cognitive functions such as inhibition of emotions and reduced activations of PFC, particularly the medial and orbitofrontal regions which are associated with violent, aggressive, and antisocial acts [20]. Brain areas such as the hypothalamus and the periaqueductal gray matter of the midbrain are also critical for aggressive expression. Areas of the midbrain have a connection with the amygdala and prefrontal cortex. Electrical stimulation of the hypothalamus generates aggressive acts, and receptors in the hypothalamus determine aggression in coordination with neurotransmitters, such as low levels of serotonin, which explain vulnerability to impulsiveness and potential aggression [21,22].

Aggression is linked with social learning [23]. Angry feelings and recurrent thoughts about anger predict aggressive behavior [24]. Anger regulation is related to well-being and health [25]. Aggression is displaced towards innocent individuals when a person is unable to strike back to the original provocateur [26]. Individuals may ruminate about the original provocateur and maintain an aggressive mood. As a

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result, cognitive representation becomes highly accessible and increases affect and arousal [27–31]. Aggression disturbs an individual on the affective (rumination), cognitive (revenge planning), and behavioral (displacement) levels [32]. Angry people show a tendency to blame another person's behavior rather than the circumstances. They are inclined to stereotypes and show narrow attention that results in greater attention to superficial aspects and less attention to details [33]. People tend to remember and pay attention to emotion-related things in the state of anger [34].

The cognitive neoassociation theory of aggression suggests that an aversive stimulus produces a negative affect that automatically stimulates thoughts, memories, emotional expression, and physiological response [31]. Cues associated with the event can trigger cognitive, emotional, and physiological responses [35]. Aggression-related thoughts, emotions, experiences are closely interlinked in memory [36]. Concepts with similar connotations are activated simultaneously that can be reappraised on the cognitive level to downregulate frustration [27]. Regular activation of a concept results in its greater chronicle accessibility, whereas a sudden situational activation results in its short-time accessibility [37,38]. Aggression can interrupt higher-order cognitive functions that may influence decision-making, for example, people may consciously avoid committing antisocial acts though they experience high levels of aggression. However, failure to do so may involve delinquent and violent behavior [30].

1.2. Temporal lobe epilepsy and cognition

Structures within the temporal lobe are involved with various cognitive functions, such as the amygdala which is involved in memory modulation, decision-making, and emotion learning [39,40]. Bilateral damage to the amygdala impairs memory and recognition of certain basic emotions such as anger and fear [41,42]. People with temporal lobe epilepsy may show cognitive decline in domains of memory, executive function, and motor speed [43–46], which is related to generalized reduction in total brain tissue that extends into extratemporal regions over time [47]. These regions mediate executive system abilities and are adversely affected by the epileptogenic cortex, thereby resulting in cognitive deficits [48,49]. The hippocampus is also involved in executive functions, and deficits in registration and consolidation of new information are attributable to hippocampal pathology [50,51].

1.3. Task switching

Executive functions encompass a variety of cognitive functions such as working memory, planning, problem solving, task flexibility, execution, and reasoning to exert cognitive control and supervise the attentional system [52,53]. Prefrontal areas of the frontal lobe are responsible for carrying out these functions [54]. Dorsolateral areas of the prefrontal cortex are involved in set-shifting, response inhibition, reasoning, and problem solving [55]. The anterior cingulate cortex mediates inhibition of inappropriate response and decision-making. The orbitofrontal cortex monitors ongoing behavior, set maintenance, and impulse control [56]. Miyake and colleagues proposed that executive functions have three types of features: updating, inhibition, and shifting. Updating is the deletion and maintenance of working memory. Inhibition is the capacity to overcome an automatic response. Shifting is the cognitive flexibility to switch between tasks, known as task switching [57].

Task switching can be tested through experimental paradigms. The switching experiment involves shifting between two task sets. On switch trials, the task is changed, whereas on repeat trials, the task remains the same as the previous trial. Switching between tasks requires a rapid attentional shift to adopt the new task set [58]. Task set is defined as an intention to perform a task, accomplished by one's mental state (e.g., working memory and attention) to be in accordance with task demands. Task switching requires activation of the new task set in the working memory and inhibition of the currently irrelevant task set. An efficient processing of these mechanisms produces low switch cost [59,60]. The theory of task-set reconfiguration asserts that once a task is implemented, it remains in an activated state until it is reconfigured or such as when a new task set is executed [61]. Strongly activated tasks are difficult to reconfigure [62]. The idea of task-set inertia suggests that task sets remain in the state of perseveration and interfere proactively with the implementation of a new task set [61]. Switching between tasks involves the suppression of just completed task set; thus, incomplete inhibition is reflected in high switch costs [63].

1.4. The present study

Previous studies suggest that TLE is associated with impaired executive function and aggression [44,47,64,65]. Mood fluctuations in PWE are a strong predictor of quality of life and functional capacities [2–7]. Given that aggression disrupts the affective, cognitive, and behavioral functioning of an individual [32], it is expected that aggression might hinder the task-switching ability in PWE. To the best of our knowledge, none of the previous studies investigated whether task switching performance is influenced by frequent aggressive impulses in PWE. We formulated the following hypotheses for the current study: (i) PWE would show switching deficits compared with healthy individuals, (ii) PWE would display recurrent displaced aggression compared with controls, and (iii) displaced aggression would be a significant predictor of task-switching performance.

2. Method

2.1. Participants

Thirty-five PWE who took part in the study fulfilled the following criteria: (1) seizures of temporal origin as evidenced by interictal and ictal EEG monitored with scalp and sphenoidal electrodes and (2) unilateral lesions in the temporal lobes as shown by MRI. Thirty-five healthy individuals (controls) were contacted through an advertisement from the community. People with TLE and controls were matched for age, gender, socioeconomic status, education, and intellectual function (see Table 1). The presence of any signs of psychiatric and neurological disorders was considered as an exclusion criterion.

Table 1
Demographic and clinical characteristics of PWE and healthy controls.

Variables	PWE (n = 35)		Controls (n = 35)	
	M (SD)	(Min–mix)	*M* (SD)	(Min–mix)
Age at the time of testing	20.00 (0.60)	(18.00–26.00)	19.20 (1.00)	(18.00–25.00)
Age at epilepsy onset	11.00 (2.00)			
Anticonvulsant medication				
Monotherapy	16			
Polytherapy	19			
Seizure frequency per 4 weeks				
22 seizures	25			
20 seizures	05			
18 seizures	05			
Gender				
Female	17		16	
Male	18		19	
Economic status				
Lower	04		03	
Middle	19		20	
Higher	12		12	
Education				
Primary	–		–	
Secondary	10		12	
Higher	25		23	
Intellectual function (see 2.2.2)	45 (0.50)		46 (0.65)	