Review Article

Neuropsychological deficits in temporal lobe epilepsy: A comprehensive review

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Abstract

Temporal lobe epilepsy (TLE) is the most prevalent form of complex partial seizures with temporal lobe origin of electrical abnormality. Studies have shown that recurrent seizures affect all aspects of cognitive functioning, including memory, language, praxis, executive functions, and social judgment, among several others. In this article, we will review these cognitive impairments along with their neuropathological correlates in a comprehensive manner. We will see that neuropsychological deficits are prevalent in TLE. Much of the effort has been laid on memory due to the notion that temporal lobe brain structures involved in TLE play a central role in consolidating information into memory. It seems that damage to the mesial structure of the temporal lobe, particularly the amygdale and hippocampus, has the main role in these memory difficulties and the neurobiological plausibility of the role of the temporal lobe in different aspects of memory. Here, we will cover the sub-domains of working memory and episodic memory deficits. This is we will further proceed to evaluate the evidences of executive function deficits in TLE and will see that set-shifting among other EFs is specifically affected in TLE as is social cognition. Finally, critical components of language related deficits are also found in the form of word-finding difficulties. To conclude, TLE affects several of cognitive function domains, but the etiopathogenesis of all these dysfunctions remain elusive. Further well-designed studies are needed for a better understanding of these disorders.

Key Words

Neuropsychological deficits, temporal lobe epilepsy, neurological correlates

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Introduction

Temporal lobe epilepsy (TLE) is the most common type of complex partial seizures (CPS). In addition to seizures, this condition also presents with several varied forms of notorious clinical features. A more concerning aspect of TLE is its cognitive sequelae. Recently, several studies have shown that recurrent seizures affect all aspects of cognitive functioning including attention, language, praxis, executive function (intelligence), judgment, insight, and problem solving.[1-3]

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The aim of this review is to highlight and elaborate these cognitive impairments in TLE patients in a comprehensive manner as it is impossible to detail all these deficits in a single review. We will thus take an ariel view on the main neuropsychological findings and will try to address only the relevant and landmark studies conducted in this context. For the purpose of this review, we will divide the abnormal cognitive findings into:

1. Memory impairments
2. Executive function impairments
3. Language impairments and
4. Other cognitive impairments.

Memory impairments

Memory deficits in TLE have been the most studied among all the neuropsychological domains. This seems to be because of the neurobiological implication of the temporal lobes, for memory, and related functions.[4] There are several domains of memory which are affected by TLE. Here are we will address two of these sub-domains of memory, which have been

explored the most scientifically. These are the working memory (WM) and episodic memory related deficits.

Working memory deficits

Among all the sub-domains of memory, WM has received much attention as evident by the plethora of studies in this field. Short-term memory is a transient trace of information temporarily stored that requires consolidation by the support of the medial temporal lobe (MTL) to be converted into a more stable status of long-term memory (LTM). WM impairment carries immense clinical importance not only because of the disability that it leads to, but also because of its association with LTM. This association has been highlighted in the recent models of WM in the role of the "episodic buffer" thus linking it to LTM systems[5] [Figure 1]. Thus consequently, LTM will also be affected if STM is impaired in TLE. As we proceed to the next section of autobiographical memory, this association will become clearer from several neuropsychological evidences.

Several neuropsychological studies provide evidence of WM dysfunction in TLE [Table 1].[6-9] The WM impairments affect both visuospatial[6] as well as verbal WM abilities in such patients.[9] The clinical and pathological correlates of these deficits will be dealt in next sub-section.

Recently, dissociation-theories have been proposed between WM and other domains of memory in TLE.[10] On the basis of an experiment involving face-recognition test hypothesized that a double dissociation of short-term and LTM exists in TLE and

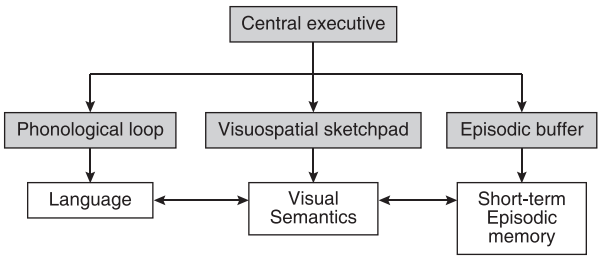

Figure 1: Schematic diagram of Baddeley's working memory model

Table 1: Studies on the effects of TLE on working memory

Author (year)	*n* (group)	Working memory assessment	Results
Cowey and Green[14]	12 (TLE) 12 (FLE) 12 (HC)	Visuospatial motor task, letter span	TLE patients were unimpaired on dual-task performance in comparison to FLE and HC
Duzel *et al*.[12]	20 (TLE)	Corsi block tapping, digit span	TMS over left temporal lobe induces recency effects in verbal working memory task
Grippo *et al*.[15]	29 (TLE) 26 (HC)	Delayed match-to-sample	Specific ERP abnormalities in memory impaired TLE is associated with reduced working memory capacity
Owen *et al*.[8]	32 (FLEx) 41 (TLEx) 19 (AHx) 91 (HC)	Matched verbal, visual and spatial	TLEx and AHx groups impaired on visual working memory compared to FLEx. Spatial working memory deficits evident in TLEx only at high demand
Krauss *et al*.[16]	8 (TLE)	Verbal and visuospatial	Mesial temporal spikes decreased working memory performance in 6/8 patients
Abrahams *et al*.[6]	47 (TLE)	Nine-box maze	Spatial working memory deficits in right TLE patients
Axmacher *et al*.[17]	11 (TLE) 23 (HC)	Delayed match-to-sample	iEEG revealed sustained MTL activity during multiple item maintenance in TLE. Confirmed by MTL fMRI activity in HC
Axmacher *et al*.[18]	13 (TLE) 23 (HC)	Delayed match-to-sample	MTL and inferior temporal lobe receive increasing top-down control as working memory load increase
Axmacher *et al*.[19]	8 (TLE) 19 (HC)	iEEG activation patterns	Working memory related hippocampal deactivation interferes with long-term memory formation
Campo *et al*.[20]	9 (LHS) 10 (HC)	MEG activity during verbal task	Reduced ipsilateral and increased contralateral MTL activity in TLE related to impaired performance
Cashdollar *et al*.[21]	6 (BHS) 6 (LHS) 8 (HC)	MEG activity during spatial working memory	Hippocampus dependent networks critical for spatial WM. BHS but not LHS group showed reduced performance
Wagner *et al*.[9]	96 (TLE) 30 (HC)	Matched verbal and visual supraspan tasks	Material specific deficits in working memory. Left TLE showed relatively more verbal deficits, right TLE showed relatively more visuospatial deficits
Black *et al*.[7]	207 (TLE) 216 (PES)	Working memory index of WAIS-R	Earlier age of TLE onset predicts poorer outcome
Vlooswijk *et al*.[22]	36 (Cryp) 21 (HC)	Delayed match-to-sample fMRI	Reduced prefrontal connectivity in patients compared to controls

LHS = Left hippocampal sclerosis, BHS = Bilateral hippocampal sclerosis, TLE = Temporal lobe epilepsy, FLE = Frontal lobe epilepsy, PES = Psychogenic non-epileptic seizures, FLEx = Frontal lobe excision, TLEx = Temporal lobe excision, AHx = Selective amygdalohippocampectomy, Cryp = Cryptogenic focal epilepsy, HC = Healthy controls, ERP = Event-related potential, TMS = Transcranial magnetic stimulation, MEG = Magnetoencephalography, iEEG = Intracranial electroencephalogram, WM = Working memory, MTL = Medial temporal lobe, fMRI = Functional magnetic resonance imaging, WAIS = Wechsler adult intelligence scale

idiopathic generalized epilepsy. Similar dissociation theories exist for the connections between WM and meta-memory. For example,[11] found intact meta-memory in TLE patients in spite of impairments in WM.

Clinical and pathological correlates of working memory impairments

Table 2 shows that there are three factors that emerges from our literature review which is associated with impaired WM in TLE patients. The most important one of them seems to be the number seizures/early age of onset. More and more studies are making it evident that the severity of WM impairment in TLE is associated with number of seizures. More recently, Black *et al*.[7] investigated the impact of the duration of epilepsy and lifetime seizure load on frontal lobe function in 207 TLE patients and later conducted a multivariate regression analyses to find that age at onset was the strongest predictor of WM and executive function so that earlier onset of TLE was predictive of poorer performance on the subtests of WM and executive functions. The second important neuropathological correlate of WM deficits is the side of brain originating the TLE, popularly known as lateralization. In the only study to look specifically at material-specific lateralization of WM in TLE, Wagner *et al*.[9] studied a group of 96 patients with unilateral MTL damage (24 preoperative and 72 postoperative) on matched verbal and nonverbal supra span tasks. There was a significant interaction between material type and side of pathology although both groups showed reduced span sizes for both material types. The left TLE group made significantly more errors on the verbal span task, but not on the visuospatial task when compared with controls and right TLE patients. These results were considered to argue for material-specific lateralization of WM dysfunction arising from unilateral left MTL damage.[9] On the other hand, the works by found that spatial memory deficits across both working and reference memory conditions were found in patients with a right epileptogenic focus. Düzel *et al*.[12] induced changes in verbal WM performance in patients with left TLE using transcranial magnetic stimulation over the temporal lobe. The authors argued that the phonological loop, responsible for the short-term storage of verbal information, has a functionally and anatomically multi-modular structure including both frontal and temporal areas.

Finally, the third important neuropathological correlate for WM decline in TLE patients seems to be hippocampal sclerosis (HS). Earlier, Abrahams *et al*.[6] they found that patients with right hippocampal damage had impairments on spatial WM task, and that hippocampal and parahippocampal gyri volume negatively correlated with the number of spatial memory errors. In a recent study by Campo *et al*.,[13] it was shown that HS alters the functional connectivity needed for maintaining and executing the WM function. Especially, MTL damage (sclerosis) weakened backward connections from left MTL to left inferior temporal cortex, which was accompanied by strengthening of bidirectional connections between inferior frontal cortex and MTL in the contra-lesional hemisphere.

Table 2: Three important factors related with severity of impaired working memory in TLE patients

Name of factor related to severity
Left temporal lobe epilepsy
Early onset TLE/more number of TLE attacks
Hippocampal sclerosis

TLE = Temporal lobe epilepsy

Autobiographical memory deficits

In recent years, a subset of patients with TLE has been identified and subsequently explored who display normal, or even above normal performance at standard delays of recall (i.e., ~30 min), but impaired performance over longer periods of retention (i.e., days or weeks), suggesting an alteration of memory consolidation mechanisms.[23-25] This memory loss has been extensively studied in transient epileptic amnesia, a subtype of TLE where memory deficit is the core symptom of ictal and interictal manifestations.[25-30] In this syndrome, patients typically present with transient episodes of amnesia and most of them also disclose accelerated forgetting rate along with an isolated autobiographical memory deficit.[25,28-30] In such patients, autobiographical memory deficits are due to selective long-term consolidation deficits for autobiographical memories as evidenced by accelerated rate of forgetting.

A number of recent group and case studies[31,32] provide ample evidence indicating that TEA patients have deficits for autobiographical event memory. For instance, Butler *et al*.,[29] who recruited 50 patients diagnosed with TEA from across the UK as part of the TIME project, found that 70% of the patients complained of autobiographical memory deficits. In another report, Kopelman *et al*.[33] found that the autobiographical memory impairments extended back several decades. More recently, Milton *et al*.[30] tested 14 patients and 11 age and intelligent quotient matched controls using the autobiographical interview, which is currently the most sensitive test of autobiographical memory performance. This revealed autobiographical memory deficits across the entire lifespan. In addition, these impairments were present for all the different types of contextual information that were examined (event, time, place, perceptual, and thought/emotion details).

If the basic defect behind these LTM impairments is in storage or retrieval still remains unclear. Evidence from patient R.G.,[34] mentioned earlier is consistent with the notion that these autobiographical memory deficits in TEA reflect a storage problem. Whereas, R.G.'s performance did not benefit from salient verbal and visual cues, a recent case study showed that a patient with TEA recovered previously lost, personally significant memories following episodes of déjà vu[35] raising the possibility that these deficits may, at least partially, be due to a retrieval deficit. One explanation often given for the autobiographical memory deficits in TEA is that clinical or subclinical activity propagates from the MTL to neocortical regions of the autobiographical memory network and this disrupts the memory trace (e.g.,[25,30]).

Tramoni *et al*.[36] took a step ahead in understanding the episodic memory deficits in TLE patients by specifically evaluating the cognitive processes for consolidation of LTM. They specifically investigated long-term consolidation of memory in a group of five adult-onset pharmacosensitive patients with TLE, exhibiting severe episodic memory complaints despite normal

performance at standardized memory assessment in a two-step experiment. In the first step of the experiment, the magnitude of autobiographical memory loss was evaluated using retrograde personal memory tasks based on verbal and visual cues. In both conditions, results showed an unusual U-shaped pattern of personal memory impairment, which was suggestive of long-term impairments in consolidation of personal episodes, adequately consolidated over "short-term" delays but gradually forgotten thereafter. To explore further, they conducted the second step, where patients were specifically investigated for short and long-term consolidation of contextually-bound experiences (episodic memory) and context-free information (semantic knowledge and single-items). In the short-term (1 h), performance of both contextually-free and contextually-bound memory tasks was intact. After a 6-week delay, however, contextually-bound memory performance was impaired while contextually-free memory performance remained preserved. This effect was independent of task difficulty and the modality of retrieval (recall and recognition). For pathological understanding of this finding, neuroimaging was also done for these patients who revealed the presence of mild metabolic changes within MTL structures. Authors concluded that mild MTL dysfunction can impede the building and stabilization of episodic memories, but leaves long-term semantic and single-items mnemonic traces intact.

Executive function impairments

Interestingly, although most studies have demonstrated the impairment of executive function deficits in TLE [Table 3], the explanations that they have hypothesized for this impairment have differed to a wide extent. These variations are rooted in the fact that there is no clarity on the specific role of temporal lobe in the context of executive functions. Hermann *et al*.[37] for example found that 44% of patients exhibited a clinically relevant executive dysfunction on Wisconsin card sorting test (WCST). The authors suggested that deficits in executive function may be associated with propagation of temporal lobe seizure activity to relevant areas of executive skill. Another neuropathological explanation was provided by Strauss *et al*.[38] who assessed 77 TLE patients with the WCST and found that set shifting ability was most impaired by the presence of left temporal lobe dysfunction, but only if damage occurred before the age of 1 year. Deficits in right TLE set shifting ability were less severe, but occurred independent to the age of onset. In contrast to these neurological perspectives there have also been some cognitive explanations for these deficits. Corcoran and Upton[39] found that HS, a pathological hallmark of mesial TLE (MTLE), compromised performance on the modified WCST (MWCST) such that HS patients completed fewer categories and made more preservative errors compared to frontal lobe epilepsy (FLE) and nonHS TLE groups. Authors argued that their results provide evidence for a dissociation between executive subsystems[39] as discussed before. A very different argument was given by Giovagnoli[40] who investigated the contribution of the hippocampus to performance on the MWCST. The performance of TLE patients with left HS was significantly impaired. There was also a trend for left TLE patients without HS to perform poorly. The authors argued that HS patients were compromised in their ability to form associations and register new information, two processes that are critical for the successful completion of the task. Several other studies have also found performance on the WCST to be compromised in TLE[2,41-44] and specifically those with HS.[40,45,46] Their explanations have varied from neurobiological to cognitive perspectives as mentioned above.

There has been limited research using other measures of executive functions in TLE. These assessments have their own merits in assessing EFs. In fact, WCST may be inadequate on some fronts of EF assessments. For example, Rzezak *et al*.[47] evaluated 35 children with TLE and 25 healthy controls with the WCST and with a more comprehensive battery. Among the children with TLE, 77.14% showed impairment on the WCST. On other tests (Wechsler Intelligence Scale for Children-Digit Forward, Matching Familiar Figures Test, Trail Making Test, Word Fluency, Finger Windows, and Number-Letter Memory), impairment was demonstrated in 94.29%. The authors concluded that the WCST is a good paradigm to measure executive impairment in children with TLE; however, it may be not enough. Evaluation performed only with the WCST not only underestimated the number of patients with erectile dysfunction (ED), but also missed relevant information regarding the type of ED.

Labudda *et al*.[48] assessed feedback based decision-making in 20 TLE patients using the Iowa gambling task.[49] The test requires subjects to select a card (typically with monetary value) from one of four decks, two decks provide short-term gain and long-term loss (disadvantageous decision), and two provide short-term loss but long-term gain (advantageous decision). Subjects are assessed on their ability to utilize the immediate feedback from each deck in order to make greater advantageous than disadvantageous decisions. Their results depicted that compared with controls, TLE patients were significantly impaired in their decision-making, and those with a preference for disadvantageous decisions performed less well on other tests of executive functions as well.

While these studies provide convincing evidence for executive dysfunction in TLE, there have also been some contradictory findings. McDonald *et al*.[50] administered the Trail Making Test, a measure of mental flexibility, to FLE patients, TLE patients, and healthy controls. FLE patients showed significant impairment in both speed and accuracy compared to TLE and controls in the more cognitively demanding set switching condition. Fewer studies have taken up the task of exploring the clinic-pathological correlates of these EF impairments and the details of all of them are out of scope here. However, two of these studies warrant special mention here. Findings of the retrospective study by Thompson and Duncan[51] suggested that as duration of epilepsy and the number of CPSs increase, executive functions decline. Similarly,[7] found through their multivariate analysis that earlier age at seizure onset, longer duration, and higher lifetime seizure frequency affect cognitive functioning in both TLE.

Language impairments in temporal lobe epilepsy

In addition to the cognitive domains as mentioned above, language impairments have been consistently observed in TLE. Word finding difficulties have been the most studied

Table 3: Studies of executive function in TLE

Author (year)	*n* (group)	Executive-function assessment	Results
Hermann *et al*.[37]	64 (TLE)	WCST	44% exhibited clinically relevant executive dysfunction
Corcoran and Upton[39]	16 (HS)	MWCST, VF Stroop	HS reduced performance on MWCST
	13 (TLEo)		
	18 (FLE)		
Strauss *et al*.[38]	77 (TLE)	WCST	Poor performance related to left sided pathology and early age of onset
Horner *et al*.[42]	38 (TLE)	WCST	50% TLE patients showed clinical executive dysfunction as measured by perseverative responses
Jokeit *et al*.[52]	96 (TLE)	"Frontal" battery including TMT and digit span	26% patients showed reduced prefrontal hypometabolsim, significantly effecting performance on frontal measures
Allegri *et al*.[45]	50 (MTLE)	WCST, VF, TMTb WCST	MTLE (particularly HS) showed reduced executive performance across all tasks
	20 (HC)		
	16 (TLE)		
Drake *et al*.[41]	12 (PGE)		75% TLE patients showed Reduced performance compared to 12% PGE
Giovagnoli[40]	112 (TLE)	MWCST	Left FLE and Left HS sig. impaired on MWCST
	53 (FLE)		
	36 (HC)		
Oddo *et al*.[53]	71 (HS)	WCST, Stroop, TMTb	25% showed impaired WCST performance
McDonald *et al*.[50]	23 (FLE)	TMTb, D-KEFS	TLE performance equal to controls
	20 (TLE)		
	23 (HC)		
Schacher *et al*.[54]	27 (MTLE)	Faux-pas test	MTLE impaired in recognizing social faux-pas
	27 (TLEo)		
	12 (HC)		
Takaya *et al*.[55]	21 (MTLE)	MWCST, TMT	Patients with frequent seizures more impaired in set-shifting; related to prefrontal hypometabolism
Hermann *et al*.[2]	96 (TLE)	WCST, Stroop, TMTb	Cluster analysis revealed 29% TLE belonged to a memory, executive and speed impaired group
	82 (HC)		
	32 (TLE)		
Wang *et al*.[56]	42 (HC)	VF, Stroop	TLE reduced performance on all measures
Cahn-Weiner *et al*.[57]	29 (TLE)	Executive daily living test	Both groups within normal limits
	9 (FLE)		
Keller *et al*.[58]	43 (TLE)	Stroop, VF	Volume atrophy of dorsal prefrontal cortex related to poorer executive performance
	30 (HC)		
Labudda *et al*.[48]	20 (TLE)	IGT, Game of Dice	TLE, in particular HS patient show reduced performance in IGT
	20 (HC)		
Black *et al*.(2010)[7]	207 (TLE)	WCST, Stroop, VF	Early age of TLE onset predicts poorer outcome for each measure
	216 (PES)		
Garcia Espinosa *et al*.[46]	42 (TLE)	WCST	Reduced WCST performance related to increased depressive symptoms

HS = Hippocampal sclerosis, TLE = Temporal lobe epilepsy, MTLE = Mesial temporal lobe epilepsy, TLEo = Temporal lobe epilepsy without hippocampal sclerosis, FLE = Frontal lobe epilepsy, cTLE = Children with temporal lobe epilepsy, PGE = Primary generalized epilepsy, HC = Healthy controls, WCST = Wisconsin card sorting test, MWCST = Modified WCST, VF = Verbal fluency, IGT = Iowa gambling task, TMTb = Trail-Making-Test part B, D-KEFS = Delisambling task, Tve function system test, TMT = Trail-Making-Test

among them. Multiple studies have reported that up to 40% of patients with TLE and a speech dominant focus have a clinically significant deficit in naming abilities.[59,60] Word finding difficulties that interfere with daily life activities are frequently reported by epileptic patients whose seizures originate in the language dominant cerebral hemisphere.[61] Recently, in a study of Spanish speakers with pharmacoresistant TLE, Lomlomdjian *et al*.[62] observed that almost one-third of patients reported frequent and severe word finding problems during spontaneous speech. In naming tests, the patients exhibited delayed time durations for finding words. Even if the target word was identified and semantically activated, there was a difficulty with lexical access, which improved when a phonetic cue was given. Left TLE patients derived a lower benefit from phonetic cues in accessing words, even when the word was known and recognized semantically. Word finding abnormalities in TLE were originally investigated with the picture naming tasks. Hamberger *et al*. however used oral definitions instead of pictures for eliciting patients responses. The definition task has proved to be more sensitive than the picture task when it comes to detecting preoperational TLE language deficits.[59,63,64] For example, hamburger and Tamny used both auditory and visual naming tasks in TLE patients. They found that left

TLE group obtained significantly lower scores than other groups on auditory naming, whereas their performance on visual naming was indistinguishable from that of right TLE patients and normal. Furthermore, whereas cut-off scores on the auditory naming task predicted seizure focus laterality in 85% of patients, performance on the visual naming task predicted laterality in only 60% of patients.

Neurobiology of naming defects in temporal lobe epilepsy

It is well-established that naming function is mediated by the perisylvian cortex in the language dominant hemisphere. Healthy speakers producing words during functional imaging show widespread activation of left perisylvian and extrasylvian cortex. During picture naming, distribution of activity in the left anterior, inferior and posterior middle/superior temporal cortex, posterior inferior frontal and inferior parietal cortex has been shown.[65-71] More recently, evidence has been accumulating from cortical stimulation studies[72] as well as functional magnetic resonance imaging (fMRI) studies that hippocampus is directly involved in naming functions. These findings support the notion that separate components of the common network supporting naming are required differentially for distinct cognitive processes[69,73]

Jensen *et al*.[74] examined the lexical and semantic processing in left-sided TLE patients by comparing behavioral and neuroimaging data associated with words and nonwords (lexicality) or with concrete and abstract words (concreteness). In addition, brain activation was studied using fMRI. Although the control group showed greater activation associated with word stimuli than with nonword stimuli in a bilateral language network, TLE groups showed greater activation for nonword stimuli than word stimuli. The TLE groups also exhibited differential activation patterns during the processing of abstract and concrete words compared to controls. For abstract words, in particular, the patient having HS group showed activation of frontal areas typically associated with executive functions, whereas the nonlesional patients (NL group) showed activation of more posterior semantic processing regions.

Understanding this language supporting network is important in understanding and integrating various theories of language impairments in TLE. In TLE patients difficulties in word retrieval, most clearly seen in the definition task, are associated with more anterior temporal sites. This could apparently be due to fundamental specifics versus reorganization of the language system in pharmacoresistant epilepsy.[72,75] Alternatively, it could point to the need for characterizing naming performance and naming failure in TLE in a much more detailed manner, for example using the processing model. A central aspect of such a model is the distinction between concept identification and word form retrieval. The importance of this distinction was previously noted by Damasio *et al*.,[73] who distinguished tip-of-the-tongue states from correct naming scores. Drane *et al*. (2008) also distinguished recognition from naming processes in patients with anterior temporal lobectomy. In the latter study, patients with nondominant TLE exhibited an identification deficit, whereas patients with dominant lobe resection mostly suffered from word retrieval deficits.[77] Other studies showed that naming difficulties in TLE were more likely to be due to lexical retrieval problems associated with the temporal lobe network.[78]

The neural basis of these language deficits in left TLE patients has also been investigated using electricocortical stimulation mapping. For example, stimulation of brain areas during picture naming has been the task of choice for identifying language cortex based on positive naming sites during presurgical mapping (Ojemann *et al*., 1989). Within the temporal lobe, Hamberger *et al*. have shown that definition naming sites (i.e., sites at which stimulation impairs auditory but not visual naming) are generally located anterior to visual naming sites or so-called "dual" sites (i.e., sites at which stimulation disrupts both auditory and visual naming).[76,79] Hara *et al*.[80] used a phonetic oddball paradigm in patients with TLE to elicit the mismatch negativity (MMN) response at frontocentral sites and the mismatch positivity (MMP) response at mastoid sites. The MMN in 26 patients were compared with that of 26 age- and gender-matched healthy control participants. Electroencephalography responses were recorded during the presentation of speech sounds: The vowels "a" and "o" in alternation. Average waveforms were obtained for standard and deviant trials. They found that the MMP responses at bilateral mastoid sites were reduced, whereas the MMN response at frontocentral sites did not change significantly. These results support the view that the MMN is generated by separable sources in the frontal and temporal lobes and that these sources are differentially affected by TLE.

Laterality and speech in temporal lobe epilepsy patients

Laterality in TLE patients seems to contribute to the language related abnormalities. Recently, fMRI has been frequently used for assessing laterality related issues in language impairments in TLE especially because of its noninvasive nature and high reproducibility for localizing brain parts involved in processing language function.[81,82] fMRI studies using simple phonemic verbal fluency paradigms have reliably lateralized language dominance in healthy controls and TLE patients by showing left frontal lobe activation corresponding to Broca's area, and less prominent activation in the MTLs.[83,84] By combining language with fMRI and voxel-based morphometry in patients with left-sided mesial TLE and HS,[85] studied whether atypical language dominance is associated with temporal and/or extra-temporal cortical volume changes. They found that patients with atypical language lateralization had more volumes of grey matter, mainly within right-sided temporolateral cortex, and less significantly within frontal brain regions compared to patients with typical language lateralization. Patients with atypical language lateralization did not differ in terms of language performance from patients with typical language dominanc,e. Atypical language lateralization in patients with left-sided mesial TLE was also associated with increased grey matter volume within the nonepileptic right temporal and frontal lobe. The exact meaning of these findings in language-related impairments of TLE patients is not clear and requires further elaborate studies for clarification.

An important research, one of its kind was conducted by,[86] for word recognition in patients with long-standing, medically-intractable epilepsy localized to the left or right temporal lobe. Participants were asked to read words that varied in the frequency of their spelling-to-sound correspondences. For the right temporal lobe group, reaction times (RTs) showed

the same pattern across spelling-to-sound correspondence conditions as previously reported for normal participants. For the left temporal lobe group, however, the pattern of RTs suggested that performance was worse on words whose orthographic body was less frequent in the language.

Other cognitive impairments

As the evidence of various cognitive and language related impairments in TLE became stronger, the scientific committee's interest toward exploring the intactness of other cognitive domains also began to rise. Emotional cognition is one such domain, which includes sub-domains like facial recognition and theory of mind (ToM). A detailed description of all these cognitive aspects is out of scope of this review so we just make passing remarks on them. The amygdala has been implicated in the recognition of facial emotions, especially fearful expressions. Golouboff[87] investigated the recognition of facial emotions in children and adolescents, 8-16-years-old, in patients suffering from both TLE and ECE. Each was matched on age and gender with a control subject. Subjects were asked to label the emotions expressed in pictures of children's faces miming five basic emotions (happiness, sadness, fear, disgust, and anger) or neutrality (no emotion). All groups of children with epilepsy performed less well than controls. Authors also found that early seizure onset was associated with poor recognition of facial expression of emotion in TLE group, particularly for fear. Based on their findings, they concluded that early-onset TLE can compromise the development of recognizing facial expressions of emotion in children and adolescents and suggested a link between impaired fear recognition and behavioral disorders.

Another cognitive domain, although studied scarcely at the best is the ToM. ToM is a crucial aspect of social cognition and is mediated by a complex neural network. Studies on TLE suggest that its neuropathological involvement is not limited to temporal lobes as such and instead involves several other brain areas. Some of these regions seem to overlap the neural network responsible for ToM, and this overlap could possibly exist in TLE. Studies on whether TLE patients evidence ToM deficit, however, are scant and controversial. Li *et al*.[88] examined whether ToM deficit is evident in TLE. Thirty-one TLE patients and 24 matched controls were recruited and completed four tasks measuring different levels of ToM: False belief, faux pas recognition, processing of implied meanings, and cartoon ToM. The patients were impaired in both basic and advanced ToM. Right TLE had a more wide-ranging picture of deficit than left TLE. ToM appears to be vulnerable to TLE, especially on the right side. Since ToM might contribute to patients' psychosocial adjustment, a ToM measure should be included in regular neuropsychological assessments of such patients.

Other forms of social cognitive functions have also been studied. Social cognitive function refers collectively to the higher cognitive functions that are essential in our social lives, and its representative aspects are facial expression recognition and decision-making. Yamano *et al*.[89] conducted a study on the social cognitive function (decision-making) of patients with TLE, and found that this function is impaired, and that the right amygdalohippocampal complexes play an important role. Schacher *et al*.[54] investigated advanced ToM capacity in TLE through the detection of social faux pas, a sensitive indicator of higher-order deficits. The task requires the detection of social faux-pas (i.e., where someone makes a social blunder) from short scenarios. The performance of MTLE patients was compared to demographically matched nonmedial TLE patients and healthy controls. The MTLE group performed less well than both nonmedial TLE patients and healthy controls in detecting social faux pas. There was no significant difference in performance between nonmedial TLE and controls. Across both epilepsy groups, performance did not correlate with epilepsy related variables such as age at seizure onset, or duration of epilepsy. The authors suggested that MTL damage particularly that involving the amygdale was the specific cause of the deficit.[54]

Conclusion

Temporal lobe epilepsy affects several domains of cognitive functioning. A plethora of studies have established dysfunctions in the domains of memory, executive functioning, language, emotional cognition, and social cognition. However, other domains are affected as well. Neurobiological basis of all these impairments remain to be known and understood. In future, several of such studies are needed whose results will have enormous implications in treatment as well as in prognosis of such patients.

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