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Temporal Lobe Epilepsy in Childhood – A Study Model of Auditory Processing

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Abstract

TLE in infancy has been the subject of varied research. Topographical and structural evidence is coincident with the neuronal systems responsible for auditory processing of the highest specialization and complexity. Recent studies have been showing the need of a hemispheric asymmetry for an optimization in central auditory processing (CAP) and acquisition and learning of a language system. A new functional research paradigm is required to study mental processes that require methods of cognitive-sensory information analysis processed in very short periods of time (msec), such as the ERPs. Thus, in this article, we hypothesize that the TLE in infancy could be a good model for topographic and functional study of CAP and its development process, contributing to a better understanding of the learning difficulties that children with this neurological disorder have.

Keywords: Auditory processing; Temporal lobe epilepsy (TLE); Childhood; Event-related potential (ERP); Model

Introduction

Central Auditory Processing (CAP) assumes a set of mechanisms and processes carried out by pathways and neuronal centres, enabling the human sound localization and lateralization, auditory discrimination, auditory pattern recognition, temporal resolution, temporal masking, temporal integration, temporal ordering, auditory performance in competing acoustic signals (including dichotic listening), and auditory performance with degraded acoustic signals [1-8]. According to the most heuristic model of CAP, this type of processing is defined as a series of processes that occur in time and enable acoustic and metacognitive analyses skills of the sound [2-8].

The first years of life are crucial to the proper development of those processes, depending on brain maturation and cerebral organization relatively, only developed in humans, parallel to a ubiquitous neuroplasticity, allowing corrections and improvements during the brain growth and cognitive development [9-11].

The sound transformed into nervous impulse in the cochlea is transmitted by the VIII cranial pair to the cochlear nuclei in the brainstem, and from here to the central auditory nervous system (CANS). The ascending auditory pathway is divided into ipsi and contralateral, starting in the cochlear nuclei complex located on the back of the pontomedullary junction, where the first sound analysis on the intensity, frequency and duration occurs [5,6]. Throughout the course of ascending auditory pathway, several neuronal centres will contribute to the auditory processing and forwarding of the message: (1) the upper olivary complex (UOC), considered the anatomical basis for the binaurality; (2) the reticular formation responsible for processing and integrating other sensory modalities (visual, vestibular and somesthetic information); (3) the lateral lemniscus nuclei and the inferior colliculus (midbrain) that have neurons sensitive to interaural phase differences and to the spatial location of the sound; (4) the medial geniculate nucleus complex (posterior caudal thalamus) which processes both the dynamic characteristics of sound (intensity, frequency and amplitude modulation) and the analysis of phonetics composition (auditory discrimination specific to speech), sending the verbal information to

the dominant hemisphere and nonverbal one to the non-dominant hemisphere that is involved both in the selective attention processes and the integration process, the interhemispheric communication occurring through the corpus callosum [6-8,10-12]. This information, after reaching the cortical level in the temporal lobes, the hippocampus stands out with an important role in declarative memory and in learning, the Heschl gyrus of the auditory primary cortex and the corner of the anterior temporal region being both responsible for impulse inhibition from a temporal lobe to another. In 1930, von Economo and Horn performed the first measurements of the Heschl gyrus, showing that the left hemisphere is usually bigger and particularly containing more white matter [13]. This anatomical differentiation is compatible with the hypothesis that the superior left temporal lobe has neurons capable of analyzing highly overlapping sequences of post-synaptic potentials, and of detecting minimum temporal variations, making this lobe more specialized for hearing discrimination and language processing. On contrary, the right temporal lobe is more sensitive to frequency variations, and thus being specific to spectral resolution processing, as shown in Figure 1 [12-15]. This specialization requires working memory processes, being highlighted in some studies on interactions with the anterior regions of the frontal lobes [16-19].

This hemispheric asymmetry suggests the existence of a functional adaptation and optimization in auditory processing of different acoustic environments. Any amendment or interference in this complex dynamic neuronal network, depending on the severity and location, can be reflected in one or several processes inherent to the

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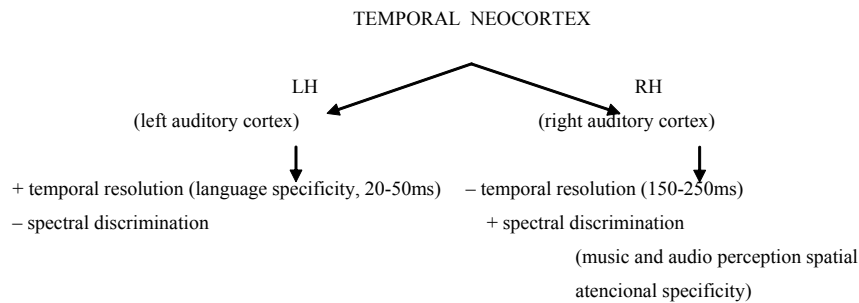


Figure 1: Temporal Neocortex neurofunctional specialization model (LH: left hemisphere; RH: right hemisphere).

acquisition and learning of a language system (verbal and nonverbal sequential memory, declarative memory, selective attention, auditory discrimination and phonological awareness) [20-22]. Certainly this linearity of CAP, somehow reductionist, can be tested by various methods even when faced with neurophysiological changes of neurological disorders, particularly in the course of brain maturation and the concomitant development of language.

Auditory Neuroplasticity

The Temporal Lobe Epilepsy in infancy (TLE) being a neurological disorder that affects directly the cortical areas of CANS, could be a good model for topographic and functional study of CAP and its development process, contributing to a better understanding of the learning difficulties that children with this disorder have and the paradoxical processes of neuronal plasticity interfered by epileptogenesis.

The relationship between changes in EEG, linguistic deficits and seizures is not yet well clarified. Notwithstanding, there is a bias resulting from the several possible aetiologies of ELT, which are inconsistent with the proposal of a single neurofunctional model not considering the possibility of different topographic locations for the epileptogenesis. These results, on one hand indicate different structures and systems with highly specific functions in auditory processing that might be “epileptically” changed, justifying the existence of different and varied neuropsychological deficits; on the other hand, is increasingly documented that a higher frequency and longevity of seizures are associated with worst neuropsychological deficits. This is undoubtedly a very important fact, especially if we consider the absence of neurophysiopathology and neuropsychology data which could contribute to sustain or even strengthen the theoretical basis on which findings are based on.

Finally, the use of research methodologies is very ambitious but little specific to study a phenomena of high temporal resolution, resulting in non unanimous conclusions such as linguistic or cognitive deficits are dependent on the structural lesions identified in MRI, on the seizures, or are secondary to attention deficits or both. Thus, the demand of “why” led to the search of a responsible element – whether it is a gene, organ, structure or neurotransmitter – that can always escape. A more profitable demarche, would be trying to answering to the “how” question, especially if we know that in TLEs with spontaneous resolution of the seizures in adolescence, occurs a simultaneous improvement in neuropsychological deficits [20,23-25]. In other words, what we really need is a new paradigm that allows us to explore the consequences of different etiologies. The following hypothesis is inscribed in this new

research paradigm. Not downplaying the importance of research lines concerning the functional activity mapping, it is obvious that we are dealing with mental processes that require methods of cognitive-sensory information analysis processed in very short periods of time (msec). This is the case of event-related potentials (ERP).

The Hypothesis

The TLE in childhood may be cryptogenic or have several etiologies, being the most frequent of the mesial temporal sclerosis, cortical dysplasia, brain injuries, extrahippocampal lesions and/or hippocampal sclerosis, always with progression and expansion to the temporal cortex [24,26]. Regardless of the neuronal origin of epileptic focus, neuropsychological deficits are common among children and adults with TLE, being more serious incidents as higher the frequency of the seizures and the time that linger [23,27-29].

This fact sustains that neuropsychological expression can be seen as a final common pathway of the different types of injury or dysfunction. The purely neuropsychological approach leaves us far from understanding the genesis of these phenomena in children. In addition, the methods widely used in brain imaging, namely the fMRI, have the temporal resolution bias, besides the non-evidence based assumptions usually made by neuroradiologists. The temporal resolution of the order of second (sec) is manifestly insufficient to give an account of the nature of the neuronal processes inherent in the processing of information in those cortical areas, which is of the order of milliseconds (msec). The assumptions underlying the paradigm used in neuroimaging studies are based on the reductionist conviction that the activation of a particular area of the cerebral cortex is linked to a particular cognitive function, and sometimes even to a behavioural function. This type of assumption, beyond reductionist, not only gives importance to the inhibitory and the related phenomena (e.g., the activation of a cortical area by an inhibitory step from other areas, which may, once inhibited, desinhibit other areas and so on), as it leaves aside the hypothesis according to which a substantial part of the information processing in the brain occurs via anaerobic processes.

The use of high temporal resolution techniques with the possibility to register several measures of the electrophysiological response (e.g., frequency resolution techniques, phase correlation, event related potential studies, signal location, among others), applied to the right and left auditory cortex in children with TLE, would allow answering more adequately to the following questions:

1. The auditory cortical organization always presupposes a lateralization for language development? If so, is this lateralization genetically determined?

2. How will be the cortical auditory organization and the response of their functional specialisations in TLE?

Implications and Further Studies

If future research validates these hypotheses, important implications and an expansion of knowledge may be derived for a better understanding of the consequences of epileptic seizures and the correlated neuropsychological deficits, as well as the type of neurological disorder that affects the thalamic auditory pathways and cortical areas. The creation of new lines of research, involving techniques such as fMRI, MEG and dichotic electrophysiological evaluation response seem to be the most promising.

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