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Clinical Neurophysiology 118 (2007) 1341–1347

www.elsevier.com/locate/clinph

Analysis of the generators of epileptic activity in early-onset childhood benign occipital lobe epilepsy

Alberto J.R. Leal [a,b,*], Sofia Nunes [c], Ana I. Dias [b], José P. Vieira [b], Ana Moreira [b], Eulália Calado [b]

[a] *Department of Neurophysiology, Hospital Júlio de Matos, Av. do Brasil nr 53, 1749-002 Lisbon, Portugal*
[b] *Department of Pediatric Neurology, Hospital Dona Estefânia, Lisbon, Portugal*
[c] *Department of Pediatrics, Hospital Fernando Fonseca, Amadora, Portugal*

Accepted 7 February 2007
Available online 29 March 2007

Abstract

Objective: The Panayiotopoulos type of idiopathic occipital epilepsy has peculiar and easily recognizable ictal symptoms, which are associated with complex and variable spike activity over the posterior scalp areas. These characteristics of spikes have prevented localization of the particular brain regions originating clinical manifestations. We studied spike activity in this epilepsy to determine their brain generators.
Methods: The EEG of 5 patients (ages 7–9) was recorded, spikes were submitted to blind decomposition in independent components (ICs) and those to source analysis (sLORETA), revealing the spike generators. Coherence analysis evaluated the dynamics of the components.
Results: Several ICs were recovered for posterior spikes in contrast to central spikes which originated a single one. Coherence analysis supports a model with epileptic activity originating near lateral occipital area and spreading to cortical temporal or parietal areas.
Conclusions: Posterior spikes demonstrate rapid spread of epileptic activity to nearby lobes, starting in the lateral occipital area. In contrast, central spikes remain localized in the rolandic fissure.
Significance: Rapid spread of posterior epileptic activity in the Panayitopoulos type of occipital lobe epilepsy is responsible for the variable and poorly localized spike EEG. The lateral occipital cortex is the primary generator of the epileptic activity.

Keywords: Epilepsy; Panayiotopoulos syndrome; Occipital lobe; sLORETA; Idiopathic; ICA

1. Introduction

The Panayiotopoulos type of idiopathic occipital lobe epilepsy (IOLE) has been a focus of research in epilepsy in recent years (Panayiotopoulos, 2002) but major issues still remain unsolved such as the particular brain area originating the seizure clinical symptoms and also the significance of the variability of posterior spike activity (Ohtsu et al., 2003). In this respect there is a sharp contrast with the syndrome of idiopathic epilepsy with rolandic spikes, which is associated with a consistent neurophysiological picture, pointing to the brain area of seizure onset in the rolandic fissure (Wolff et al., 2005).

The source analysis methods in EEG (Yoshinaga et al., 2005, 2006) and MEG (Kanazawa et al., 2006), using the equivalent current dipole, suggest origin of the epileptic activity near the calcarine sulcus and parieto-occipital areas. These results do not provide a good explanation to the typical ictal clinical manifestations and also fail to explain why the visual symptoms are so rare in the syndrome.

In this work we perform a neurophysiological study of posterior EEG spikes in cases of the Payionotopoulos type

* Corresponding author. Present address: Department of Neurophysiology, Hospital Júlio de Matos, Av. do Brasil nr 53, 1749-002 Lisbon, Portugal. Tel.: +351 969851734; fax: +351 217819809.
E-mail address: a.leal@netcabo.pt (A.J.R. Leal).

1388-2457/$32.00
doi:10.1016/j.clinph.2007.02.017

Table 1
Clinical data

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age of onset (Y)	6	3	6	2	4
Development	Normal	Normal	Normal	Normal	Cerebral palsy
Ictal symptoms					
Vomiting	no	Yes	Yes	Yes	Yes
Eye deviation	Yes	No	Yes	Yes	No
Decreased reactivity	Yes	No	No	Yes	Yes
Other		Nistagmus, sialorrea	Pallor		Decreased tonus
Seizure duration		>20 min	5–10 min	>15 min	>20 min
Number of seizures	1	4	2	4	3

of IOLE and a comparison is made with rolandic spikes also present in some of these patients. Our main goal is to determine the usefulness of the interictal EEG analysis to highlight the localization of the primary epileptic foci in this syndrome.

2. Methods and subjects

Five patients with the diagnosis of early onset IOLE (Panayiotopoulos type) were selected from the epilepsy outpatient clinic of Hospital Dona Estefânia. All demonstrated clinical (Table 1) and neurophysiological (Table 2) data compatible with the ILAE criteria for the diagnosis (Covanis et al., 2005).

Patients were studied in two moments, with a temporal interval of 3 years. In the early study, a special cap with 30 electrodes (10–20 plus Fp_z, $P_{9/10}$, $PO_{3/4}$, $PO_{7/8}$, O_z, $O_{9/10}$ and PO_z) was used to perform a sleep study with 1-h duration. For the repeated study a 24-h ambulatory EEG was performed with 30 electrodes individually glued to the scalp (10–20 plus $F_{9/10}$, $T_{9/10}$, $P_{9/10}$, $PO_{3/4}$, $O_{9/10}$ and O_z). The electrodes were photographed in several planes and their position in the scalp reproduced in a 3D rendering of a standard average brain. The sampling rate for both recordings was 256 Hz with high- and low-pass filters at 1 and 70 Hz, respectively.

The EEG recordings were visually reviewed by a clinical neurophysiologist (AL) and the peak of individual spikes marked. Because some of the patients had spikes with different topographies, we selected a minimum of 40 spikes (average of 91 ± 26) for each spike type. This proved possible because all patients had abundant spikes during the sleep period.

The EEG spikes with no artifacts associated were cut in epochs (−100 to 300 ms) around the voltage peak, using the Scan 4.3.1 (Neuroscan, El Paso) software. Data were exported to the EEGLAB 4.515 package (Delorme and Makeig, 2004) for decomposition of the raw spike groups in independent components, using the Infomax algorithm (Bell and Sejnowski, 1995) and methods detailed elsewhere (Leal et al., 2006). The spatial components with consistent activation at the time of spike peak were selected and submitted to cross-coherence analysis in order to obtain data on their temporal dynamics.

The independent component analysis (ICA) components were sorted by time of activation, providing information on the spreading patterns of the spike activity, and later submitted to source analysis to improve localization of the cortical areas involved.

Source analysis was done using the sLORETA software package (Pascual-Marqui, 2002) available at http://www.unizh.ch/keyinst/NewLORETA/LORETA01.htm. The maximum of the current density obtained at a given moment was taken as the source of the particular component, and because the spatial components recovered from

Table 2
Neurophysiological data

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Early study					
Age	7	7	8	9	9
Background	Normal	Normal	Normal	Normal	Normal
Interictal	Left occipital spikes	Parietal spikes	Right central and left occipital spikes	Right central and occipital spikes	Left central and occipital spikes
Early generator	Left occipital	Parietal-occipital	Left occipital	Right occipital	Left occipital
Spread areas	Left temporal		Left temporal and parietal	Right Parietal	Left parietal
Second study					
Age	10	10	11	11	12
Background	Normal	Normal	Normal	Normal	Normal
Interictal	Left occipital spikes	Parietal spikes	Right central spikes	Right central and parietal spikes	Left central spikes
Early generator	Left occipital	Parietal-occipital		Right parietal	
Spread areas	Left temporal			Right central	