Clinical Neurophysiology 128 (2017) 1810–1819

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

The neural bases of ictal tachycardia in temporal lobe seizures

Florian Chouchou [a,*], Romain Bouet [b], Vincent Pichot [c,d], Hélène Catenoix [e], François Mauguière [a,e,f], Julien Jung [b,e]

[a] NeuroPain Lab, Lyon Neuroscience Research Center – Inserm U 1028/CNRS UMR 5292, University of Lyon, France
[b] Dycog Lab, Lyon Neuroscience Research Center – Inserm U 1028/CNRS UMR 5292, University of Lyon, France
[c] Clinical Physiology Department, CHU Nord, Saint-Etienne, France
[d] EA 4607 SNA-EPIS Lab, University of Jean Monnet, University of Lyon, Saint-Etienne, France
[e] Epilepsy and Functional Neurology Department, Neurological Hospital Pierre Wertheimer, Hospices Civils de Lyon, Bron, France
[f] Claude Bernard Lyon 1 University, Lyon, France

See Editorial, pages 1772–1773

ARTICLE INFO

Article history:
Accepted 2 June 2017
Available online 27 June 2017

Keywords:
Epilepsy
Tachycardia
Autonomic nervous system

HIGHLIGHTS

- The neural bases of tachycardia that accompanied temporal lobe seizures remain elusive.
- The study suggests that hippocampal and amygdalar ictal activity play a pivotal role in tachycardia.
- The present study also suggests that ictal tachycardia is independent of ictal insular activity.

ABSTRACT

Objective: Due to limited information from scalp electroencephalographic (EEG) recordings, brain areas driving changes in cardiac rhythm during Temporal lobe (TL) seizures are not clearly identified. Using stereotactic EEG (SEEG) recordings, we aimed at identifying which of the brain regions involved in autonomic control trigger ictal tachycardia.
Methods: The neural activity of several mesial temporal lobe structures including amygdala, hippocampus, insula, and lateral temporal lobe recorded with SEEG were collected during 37 TL seizures in 9 patients, using indices based on High Frequency Activity (HFA). R-R intervals (RR) monitoring and time-frequency spectral analysis were performed to assess parasympathetic (High frequency power (HF)) and sympathetic (Low frequency/High frequency (LF/HF) ratio) reactivities.
Results: Tachycardia was associated with a significant increase in LF/HF ratio and decrease in HF. Autonomic cardiac changes were accompanied by simultaneous SEEG signal changes with an increase in seizure-related HFA in anterior hippocampal formation and amygdala, but not in insula.
Conclusion: In our sample, TL seizures are thus accompanied by an early decrease in parasympathetic control of cardiac rhythm and by an increase of sympathetic tone, concomitant to seizure activity in anterior hippocampus and amygdala.
Significance: These results support a pivotal role of hippocampus and amygdala in tachycardia occurring during TL seizures.
© 2017 International Federation of Clinical Neurophysiology. Published by Elsevier Ireland Ltd. All rights reserved.

1. Introduction

Temporal lobe epilepsy (TLE) can lead to intense changes in cardiac autonomic functions during both interictal and ictal periods (Lotufo et al., 2012; Eggleston et al., 2014). Tachycardia, defined as a decrease in the ECG R-R intervals (RR) (increase in heart rate), is the most common cardiac change occurring during epileptic seizures (Opherk and Hirsch, 2002; O'Regan and Brown, 2005; Toth et al., 2010) whereas bradycardia occurs in only 2% of them (Moseley et al., 2010). Tachycardia was proposed as a biomarker providing a somatic indicator of temporal lobe (TL) seizure onset (Osorio, 2014; Schiecke et al., 2014; Osorio and Manly, 2014,

* Corresponding author at: NeuroPain – Central Integration of Pain in Humans, Lyon Neuroscience Research Center, Hôpital Neurologique, Unité hypnologie, Rdj, 59 bvd Pinel, 69 677 Bron cedex, France.
E-mail address: florianchouchou@gmail.com (F. Chouchou).

http://dx.doi.org/10.1016/j.clinph.2017.06.033
1388-2457/© 2017 International Federation of Clinical Neurophysiology. Published by Elsevier Ireland Ltd. All rights reserved.

2015; Jeppesen et al., 2015; Behbahani et al., 2016; Van de Vel et al., 2016), opening the way to automatized and noninvasive seizure detection and treatment delivery prior to, or at the onset of, a TL seizure.

However, the time course and the neural bases of tachycardia in TL seizures is still incompletely understood. Firstly, the timing of the change in cardiac rhythm relatively to seizure onset is still debated. Previous results are mainly based on scalp EEG, which implies some uncertainty regarding time relationship between brain structures involved in seizure and tachycardia (Weil et al., 2005; Kato et al., 2014). Secondly, the central autonomic network includes several brain regions such as amygdala, hippocampus and insula, which are commonly involved in temporal lobe seizures, (Critchley and Harrison, 2013; Beissner et al., 2013) but the respective role of those three structures in ictal tachycardia is not yet established. Many studies in animals support a major role of the insula in the cardiac autonomic control (Zhang et al., 1998; Oppenheimer and Cechetto, 2016; Marins et al., 2016). In Humans, Oppenheimer and colleagues showed in five epileptic patients that insular stimulations induce tachycardia (Oppenheimer et al., 1992), suggesting a potential role of insula in ictal tachycardia, which remains to be demonstrated by coupling ECG with intra-cerebral recordings during spontaneous seizures. Thirdly, if some studies suggested that right seizure lateralization is associated with an earlier and higher cardiac response this finding is not consistent across all studies (Massetani et al., 1997; Leutmezer et al., 2003; Di Gennaro et al., 2004; Mayer et al., 2004; Adjei et al., 2009; Kato et al., 2014). Lastly, some animal experiments and human studies suggest that autonomic control is lateralized in the brain, the right hemisphere being involved in sympathetic control and the left one in parasympathetic control (Oppenheimer et al., 1992; Wittling et al., 1998; Zhang et al., 1998); thus an exhaustive evaluation of cardiac changes during temporal lobe seizures should not only assess heart rate, but also autonomic cardiac control, which has been evaluated in very few studies in epilepsy (Sevcencu and Struijk, 2010; Jaychandran et al., 2016). Evaluating RR variability (heart rate variability) is a common noninvasive method for assessing sympathetic and parasympathetic activities well suited to probe the influence of the autonomic nervous system on heart activity during seizures. The power of high-frequency (HF) variations of the RR interval is considered as a marker of parasympathetic control of cardiac rhythm, while that of the low frequency (LF) RR variations and the ratio of low-to-high-frequency powers of RR variations (LF/HF ratio) are markers of the sympathetic tone (Malliani et al., 1991).

The main objective of the present study was to assess which cerebral regions, engaged in the central autonomic control including insula, amygdala, and hippocampus (Beissner et al., 2013), are involved in ictal tachycardia. To this end, we analyzed simultaneously intracranial EEG and ECG recordings in 9 patients. The temporal relationships of quantitative stereotactic EEG (SEEG) and RR changes were evaluated. Secondly, we also evaluated autonomic cardiac changes using a time-frequency analysis of heart rate variability (HRV) (Pichot et al., 1999, 2016) to study the influence of sympathetic and parasympathetic systems on ictal tachycardia.

2. Methods

2.1. Patients

Nine patients (6 women, mean age 30.4 ± 9.4 (mean ± standard deviation (SD)) years, range [21–50]) who suffered from long lasting partial drug refractory epileptic seizures (range [4–26] years) were enrolled during presurgical evaluation of their epilepsy. Clinical features of patients are reported in Table 1. Criteria of inclusion

Table 1
Demographic and clinical features of the 9 patients suffering from TLE.

Patients	Gender, Age	Epilepsy onset (years)	Ictal semiology	MRI	N° and side of seeg electrodes	Epileptogenic zone	Surgery	Engel class	Follow-up (months)
Patient 1	M, 32	26	Pallor, stop activity, loss of consciousness	Right hippocampal sclerosis and right anterior frontal flair hypersignal	12R, 2L	Right mesio-temporal structures	Anterior temporal lobectomy	1a	26
Patient 2	F, 37	8	Elementary visual hallucination, oroalimentary automatisms, left arm dystonic	Right ventricular dilatation and posterior cortical flair hypersignal	11 R, 2L	Right temporo-occipital region	No		
Patient 3	F, 22	8	Tachycardia, pallor, cold sensation	Right hemispheric atrophy and hippocampal sclerosis	13R, 1L	Right mesio-temporal structures	Anterior temporal lobectomy	1a	48
Patient 4	M, 26	11	Cephalic aura, abdominal constriction, hypersalivation, nausea, laryngeal striction, vomiting. Postictal aphasia	Normal	10L, 2R	Left amygdala and temporal pole	Amygdalectomy and polectomy	1a	69
Patient 5	F, 27	16	Loss of consciousness, amnesia, postictal confusion	Normal	11R, 3L	Right mesio-temporal structures	Anterior temporal lobectomy	2b	52
Patient 6	M, 21	8	Left oculo-cephalic version, left facial clonus, secondary generalization	Right temporal cortical malformation	11R, 3L	Right posterior temporal neocortex	No		
Patient 7	F, 50	17	Epigastric aura, oroalimentary automatisms, left arm dystonic, postictal aphasia	Left parietal cavernoma, left hippocampal sclerosis	8L, 5R	Left temporo-mesial structures	No		
Patient 8	F, 23	4	Epigastric aura, oroalimentary automatisms, postictal aphasia	Bilateral hippocampal sclerosis	9L, 1R	Left temporo-mesial structures	Anterior temporal lobectomy	2b	45
Patient 9	F, 36	23	Mutism, stop activity	Right periventricular heterotopy	12L, 3R	Left anterior temporal neocortex	Temporal polectomy	2a	21

Download English Version:

https://daneshyari.com/en/article/5627236

Download Persian Version:

https://daneshyari.com/article/5627236

Daneshyari.com