Neurobiology of Aging 69 (2018) 261–273

Contents lists available at ScienceDirect

Neurobiology of Aging

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

Aging of human alpha rhythm

Maria G. Knyazeva [a,b,*], Elham Barzegaran [a,b], Vladimir Y. Vildavski [c], Jean-François Demonet [b]

[a] Department of Clinical Neurosciences, Laboratoire de recherche en neuroimagerie (LREN), Lausanne University Hospital and University of Lausanne, Lausanne, Switzerland
[b] Department of Clinical Neurosciences, Leenaards Memory Centre, Centre Hospitalier Universitaire Vaudois and University of Lausanne, Lausanne, Switzerland
[c] Department of Psychology, Stanford University, Stanford, CA, USA

ARTICLE INFO

Article history:
Received 9 October 2017
Received in revised form 11 May 2018
Accepted 12 May 2018
Available online 22 May 2018

Keywords:
Alpha rhythm slowing
Component structure of alpha rhythm
Oscillations
PARAFAC
Resting state
Source localization

ABSTRACT

Alpha rhythm (AR) changes are the most pronounced electroencephalogram phenomenon in the aging brain. We analyzed them based on the inherent AR structure obtained by parallel factor analysis decomposition in the cortical source space. AR showed a stable multicomponent structure in 78% of sixty 20- to 81-year-old healthy adults. Typically, it consists of 2 components. The distribution of the higher frequency occipito-parietal component widens with age, with its maximum moving from BA18/19 to BA37. The low-frequency component originating from the occipito-temporal regions in young adults also moves anteriorly with age, while maintaining its maximum within BA37. Both components slow down by 1 Hz over the adult lifespan. The multicomponent AR is more common in younger subjects, whereas a single-component AR in older subjects. This uneven occurrence as well as the increasing spatial and frequency overlaps between components suggest transformation of the multicomponent AR into the single-component AR with age. A detailed knowledge of AR component structure would be useful to monitor age-related neurodegenerative processes in humans.

1. Introduction

The changes of the posterior alpha rhythm (AR) in electroencephalogram (EEG) are among the most pronounced phenomena related to brain aging in humans. They include AR slowing (Başar, 2012; Clark et al., 2004; Gaál et al., 2010; Hubbard et al., 1976; Lodder and van Putten, 2011; Markand, 1986; Peltz et al., 2010; Shigeta et al., 1995; Van Sweden et al., 1999; Wang and Busse, 1969), reduction of its power (Lodder and van Putten, 2011; Vysata et al., 2012), a shift of AR sources in the posterior-to-anterior direction (Babiloni et al., 2006; Niedermeyer, 1997; Rossini et al., 2006), and the declining AR reactivity (Gaál et al., 2010; Hong et al., 2015; Vaden et al., 2012) observed in middle-aged and older participants.

The description of age-related AR changes has been largely based on the analysis of the entire AR band or AR peak (Caplan et al., 2015; Davidson and Davidson, 2012; Peltz et al., 2010). Alternatively, some studies reported differential effects of aging on *pre-selected* high- and low-frequency sub-bands of AR (Babiloni et al., 2015; Moretti et al., 2004). However, these findings have only limited explanatory potential, as the correspondence of fixed sub-bands to separate rhythmic components remains an open question.

The concept of individual AR peak frequency (Klimesch, 1999) became an essential step toward improved AR analysis. The evidence-supported alpha sub-bands (i.e., having borders dependent on the individual peak frequency) correlated better with neurodegeneration in the older adults than the fixed ones (Angelakis et al., 2007; Moretti et al., 2011). Yet, this procedure cannot reveal the inherent structure of the AR. The variation in the number (2 or 3) of analyzed sub-bands among published reports once again emphasizes the subjective nature of such segmentation.

Meanwhile, it is essential to consider the number, origin, and functionality of the rhythms that contribute to the posterior AR in the surface EEG for understanding its evolution with age. Indeed, in the presence of more than 1 component, characterized by individual frequency, source, and temporal dynamics, the age-related AR changes, stated previously, can be explained by different scenarios. For instance, the observed slowing down of the posterior AR with age may result from a decrease in the frequency of all AR components, or from a decrease in the power of the high-frequency component, leading to different interpretations of aging processes in the brain.

* Corresponding author at: Department of Clinical Neurosciences, Lausanne University Hospital and University of Lausanne, 1011 Lausanne, Switzerland. Tel.: +41 21 314 32 31.
E-mail address: mariagknyazeva@gmail.com (M.G. Knyazeva).

0197-4580/$ – see front matter © 2018 Elsevier Inc. All rights reserved.
https://doi.org/10.1016/j.neurobiolaging.2018.05.018

An inherent structure of the AR can be demonstrated by its decomposition with a multidimensional technique that takes into account all 3 natural EEG dimensions—time, space, and frequency. To this end, by using a 3-way parallel factor analysis (PARAFAC) method, we found a stable multicomponent AR structure in about 90% of *young healthy adults* (Barzegaran et al., 2017). In addition to the predominant occipito-parietal component of AR, well known from the literature, we identified a weaker occipito-temporal component, largely left unattended in previous studies. These 2 components comprised the most typical configuration of the posterior AR, recordable with noninvasive surface EEG in young adults.

Building upon our findings regarding AR structure in young people and the aforementioned phenomenology of AR changes in the older adults, we suggest that each of the AR components has its own evolution, leading to the distortion of AR structure with age. We test this hypothesis by identifying the AR structure of middle-aged to older normal adults with the PARAFAC method, comparing it to that of young participants and, finally, analyzing the age-related trends over the adult lifespan.

2. Methods

2.1. Participants

In this study, we analyzed EEG data of 60 participants aged from 20 to 81 years. The data from healthy 20- to 45-year-old participants, representing AR structure in early adulthood, have recently been reported (Barzegaran et al., 2017) and are used here only to characterize AR evolution over the adult lifespan (see Section 2.9).

Thirty-two middle- to old-aged community-dwelling adults were de novo enrolled in the study. Potential participants underwent a brief clinical interview that included the Montreal Cognitive Assessment (MoCA) test. The description of AR structure in the aging population is based on EEG data from these individuals (12 men and 20 women aged 45–81 years) with a MoCA score $\geq$26 (mean group score was 27.4) and without cognitive complaints, past or present neurological or psychiatric illness including depression, psychoactive drug use or alcohol use disorders, head trauma, chronic systemic illnesses or other conditions that interfere with cognition (for details see Supplementary materials, Section I).

According to the Hospital Anxiety and Depression scale (Zigmond and Snaith, 1983), none of the recruited participants showed symptoms of depression as indicated by the individual scores all being <8. Independent living skills of all the potential participants aged 65+ years had been assessed with the Lawton Instrumental Activities of Daily Living Scale (Lawton and Brody, 1969). At the time of experiment, all the actual participants functioned independently in the 8 (women) or 5 (men) domains of function included in the Instrumental Activities of Daily Living scale.

The sample selected using abovementioned tests represents adults without clinically significant evidence of brain pathology, that is, those in the course of *normal aging* in contrast to *healthy or successful aging*. The latter is characterized by the uncommon structural and functional preservation of the brain that can be shown with extended neuropsychological and neurological examinations and brain imaging (Harrison et al., 2012; Rogalski et al., 2013, 2018). Our choice of tests and inclusion/exclusion criteria for participants of this study was motivated by the objective to explore *typical age-related changes of the inherent AR structure* rather than exceptional neuroprotective mechanisms that stand against destructive aspects of aging.

All methods and procedures in this study conform to the Declaration of Helsinki (1964) of the World Medical Association concerning human experimentation (Rickham, 1964). It was also approved by the local Ethics Committee (Commission cantonale d'éthique de la recherche sur l'être humain). We provided the essential information about the research to each potential participant and obtained written informed consent from all actual participants.

2.2. EEG recording and preprocessing

Here, we briefly summarize methods used for collecting and analyzing EEG data. For detailed technical description, the reader is referred to Barzegaran et al. (2017). The recording sessions of 90–120 minutes included several cognitive tasks and Rest with Eyes Open (REO) interleaved with the Resting state with Eyes Closed (REC). Detailed description of the recording timeline is presented in Supplementary materials, Section II. We concatenated the REC or REO episodes (each $\leq$3 minutes) into 4 consecutive REC periods and 1 REO period of 8–10 minutes. This report is mainly based on the data from all 4 REC periods (REC1–REC4). The REO condition was used to analyze the AR reactivity (Supplementary materials, Section III).

The EEGs were collected on a 64-channel mobile EEG system eego sports using waveguard original electrode caps (ANT Neuro, Enschede, The Netherlands) with a CPz reference and a high-cutoff filter set to 100 Hz. The electrode impedances were kept under 30 kΩ (under 10 kΩ in most cases), which was well below the recommended maximum of 50 kΩ for high-impedance eego amplifiers. The EEGs were digitized at 500 Hz with a 24-bit resolution and band-pass filtered within a 1- to 45-Hz band by a phase preserving digital filter. We marked the artifacts based on the off-line visual inspection and used the data without visible artifacts for further analysis. We removed the 2 mastoid electrodes, which contained low-quality EEG in many participants from the data before re-referencing the signals from CPz to the common average of the remaining electrodes.

2.3. General design of EEG analysis

A multidimensional 3-way PARAFAC, breaking the source-space EEGs into a number of AR components (ARCs), lies at the core of the data processing in this study. We calculated the time-varying spectra of the scalp EEGs by taking short-term Fourier transforms over a large number of epochs (see Section 2.4), and then converted the spectra to the cortical space by the low-resolution electromagnetic tomography technique (Section 2.5). We applied the first PARAFAC decomposition to the sensor-space spectra, yielding reliable estimates of the frequency, space, and time features of the components (Section 2.6). In the second application of the PARAFAC to the cortical-space spectra, we took the frequency and time features as fixed parameters from the sensor-space decomposition, thus reducing the number of estimated parameters and obtaining robust estimates of the cortical distribution of the components. We then characterized the ARCs statistically (Section 2.7) including the assessment of their temporal stability (Section 2.8), as well as age-related trends (Section 2.9). For more details and the block diagram of the analysis design, we refer the reader to Barzegaran et al. (2017).

2.4. Spectral analysis of EEG

We calculated the time-frequency representation of multichannel EEG signals from all 4 REC periods by the discrete short-time Fourier transform of 5-second artifact-free epochs multiplied by the Hann window and overlapped by 50%. For the REC condition, the number of time windows constituted 782 $\pm$ 114 (mean $\pm$ standard deviation), while for REO it was 182 $\pm$ 63 per participant.