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Clinical paper

Resting state EEG relates to short- and long-term cognitive functioning after cardiac arrest

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Abstract

Background: Approximately half of cardiac arrest survivors have persistent cognitive impairment. Guidelines recommend early screening to identify patients at risk for cognitive impairment, but there is no consensus on the best screening method. We aimed to identify quantitative EEG measures relating with short- and long-term cognitive function after cardiac arrest for potential to cognitive outcome prediction.

Methods: We analyzed data from a prospective longitudinal multicenter cohort study designed to develop a prediction model for cognitive outcome after cardiac arrest. For the current analysis, we used twenty-minute EEG registrations from 80 patients around one week after cardiac arrest. We calculated power spectral density, normalized alpha-to-theta ratio (nATR), peak frequency, and center of gravity (CoG) of this peak frequency. We related these with global cognitive functioning (scores on the Montreal Cognitive Assessment (MoCA)) at one week, three and twelve months follow-up with multivariate mixed effect models, and with performance on standard neuropsychological examination at twelve months using Pearson correlation coefficients.

Results: Each individual EEG parameter related to MoCA at one week (β_{nATR} = 7.36; P < 0.01; β_{peak} frequency = 1.73, P < 0.01; β_{CoG} = -9.88, P < 0.01). The nATR also related with the MoCA at three months ((β_{nATR} = 2.49; P 0.01). No EEG metrics significantly related to the MoCA score at twelve months. nATR and peak frequency related with memory performance at twelve months. Results were consistent in sensitivity analyses.

Conclusion: Early resting-state EEG parameters relate with short-term global cognitive functioning and with memory function at one year after cardiac arrest. Additional predictive values in multimodal prediction models need further study.

Keywords: Prediction, Cognitive outcome, Cardiac arrest survivors, EEG

Introduction

Approximately half of all cardiac arrest survivors experience enduring cognitive impairment in the long term. Most have been reported in the domains of memory, attention, and executive functioning.[1–5] Cognitive impairments have been associated with reduced quality of life[6] and societal participation.[2] Guidelines recommend screening for early identification of patients at risk for cognitive impairment.[7] However, validated predictive measures are not provided by the guidelines.[7]

Previous studies on potential predictors have focused on duration of coma, blood biomarkers and bedside screening batteries. Longer duration of coma,[8–10] high S-100B levels,[11,12] elevated neurofilament light chain (NFL) levels[13] and score of ≤ 94.5 on a self-developed bedside neuropsychological test battery[11,12] were associated with long-term cognitive impairment on the group level, but predictive values for individual patients are unknown. A recent analysis showed that the Montreal Cognitive Assessment (MoCA) is a valid screening instrument for detection of current cognitive impairment after cardiac arrest,[14] and also holds predictive value for long-term cognitive impairment.[15]

Electroencephalogram (EEG) holds potential to contribute to prediction of cognitive function after cardiac arrest. EEG is a sensitive measurement to detect hypoxic-ischemic brain damage[16] and can reliably contribute to prediction of gross neurological recovery of comatose patients after cardiac arrest.[17–20] In patients with neurodegenerative diseases, such as Alzheimer's and Parkinson's disease, EEG measures of brain activity have been associated with various measures of cognitive functioning. In general, more diffuse EEG

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slowing was associated with more cognitive deterioration in these populations.[21–25]

In this study we investigate the relation between early resting state EEG parameters and short- and long-term cognitive functioning in patients after cardiac arrest. We hypothesize that background slowing and poorer differentiation of the EEG are related to poorer cognitive functioning. Results will be used as input for future derivation and validation of multimodal prediction models for cognitive outcome after cardiac arrest.

Materials and methods

Design

This is an analysis of the first 80 patients with EEG data included in the Brain Outcome after Cardiac Arrest (BROCA)-prediction study; NL9451). In short, BROCA-prediction is a prospective longitudinal multicenter cohort study designed to develop a prediction model for long-term cognitive outcome and societal participation of survivors after out-of-hospital cardiac arrest. Demographic, clinical, EEG, and MRI measures are collected during hospital stay and patients are followed for one year. For the current analysis, we used resting state EEG data collected during the protocolized inclusion window of 4 ± 3 weeks after cardiac arrest (generally during hospital stay after recovery of consciousness) and data on cognitive functioning, collected at 4 ± 3 weeks after cardiac arrest (during hospital stay) and at three- and twelve months after cardiac arrest. Patient inclusion started in November 2019 and is ongoing in six hospitals in the Netherlands. For this analysis we used data from patients included between November 11st, 2019 and May 15th, 2022. The medical Ethical Committee Arnhem/Nijmegen approved the protocol (NL69767.091.19).

Patients

Consecutive, adult, out-of-hospital cardiac arrest patients, after successful resuscitation and recovery of consciousness (Glasgow Coma Scale (GCS) score > 8), admitted to the cardiac care unit or cardiology department, were included in our study. We included patients after recovery from coma, after temporary admission on an intensive care unit, and patients that had not been admitted to an intensive care unit. Exclusion criteria were preexistent brain damage with modified Rankin Scale (mRS) >2, progressive neurodegenerative disease, life expectancy of less than three months because of another medical condition, need of intravenous sedative medication, and insufficient knowledge of the Dutch language to fill out questionnaires.

Written informed consent was obtained from the patients. In cases where patients were unable to give consent, a legal representative granted permission. As soon as patients recovered and were able to comprehend the information, informed consent was also obtained from the patients themselves.

Cognitive outcome

Our outcome measures for this analysis comprised global cognitive functioning as measured by the Montreal Cognitive Assessment (MoCA) at 4 ± 3 weeks after cardiac arrest (during hospital stay), and at three and twelve months after cardiac arrest. The MoCA is a 30-point scale screening instrument for cognitive impairment,[26] validated in patients after cardiac arrest.[14] To reduce the risk of classifying low-educated subjects as cognitively impaired, an extra point is given to those with 12 or fewer years of formal education. Patients with a score below 26 are considered cognitively impaired.[26] At twelve months after cardiac arrest, patients underwent additional neuropsychological examination consisting of cognitive tests for memory (Rey Auditory Verbal Learning Test (RAVLT)), attention (Trail Making Test A & B (TMT-AB), and Stroop Color and Word Test), and executive functioning (TMT-AB, Stroop Color and Word Test, short Raven's progressive matrices test, and letter fluency). Z-scores were calculated for the RAVLT, TMT-AB, and Stroop using Maasnorms, corrected for age, gender and educational level.[27] Norms for the Short Raven were based on previous research[28] and for the letter fluency on the Netherlands institute for Psychologists (NIP) norms.[29] Per cognitive domain a composite z-score was computed by dividing the sum of the individual z-score per cognitive domain by the number of subtests (supplementary material S1).

EEG recordings and analyses

Twenty-minute EEG was recorded at 4 ± 3 weeks after cardiac arrest. Twenty-one silver-silverchloride cup electrodes were placed on the scalp according to the international 10–20 system. Recordings were made using a Neurocenter EEG recording system (Clinical Science Systems, Leiden, The Netherlands), a Nihon Kohden system (VCM Medical, Leusden, The Netherlands) or a Brain RT system (Micromed, Mogliano, Veneto, Italy). Recordings contained alternating conditions of eyes open (EO) and eyes closed (EC). The following scheme was used: two minutes EO, six minutes EC, two minutes EO, six minutes EC, one-minute EO, three minutes EC. None of the patients received sedative medication during the EEG recording. No clinical EEG reports were made and no information from the EEG was shared with other professionals. All EEG analyses were prespecified and performed offline, after the registrations, using MATLAB R2020a.

Epoch selection and preprocessing

We selected eyes-closed, artifact-free epochs for each patient, assessed by visual analysis. In addition to artifacts, patterns indicative of the onset of sleep such as slow eye movements (SEM), the disappearance of the alpha rhythm, and the appearance of vertex waves were excluded from this analysis. The minimum epoch length was four seconds. EEGs with a sampling frequency other than 256 Hz were resampled to 256 Hz with the MATLAB resample function. All EEGs were bandpass filtered in the frequency range 1–40 Hz.

Power spectrum analysis

We removed the prefrontal and frontal electrodes from the data to minimize the influence of eye movements. EEGs were reconfigured into an anterior-posterior montage. Subsequently, we estimated the power spectral density (PSD) for each channel pair in the anterior-posterior montage using Welch's method with a window length of 4 s and 50% overlap, resulting in a frequency resolution of 0.25 Hz. We obtained an average spectrum by calculating the median value of all frequencies, as this reduces sensitivity to outliers. This average spectrum is used for further analyses.

Normalized alpha-to- theta ratio

We calculated the absolute power of the theta band (4–8 Hz) and alpha band (8–13 Hz) on a whole brain level from the median PSD. The normalized alpha-to-theta ratio (nATR) was calculated as

$$nATR = \frac{(P_{alpha} - P_{theta})}{(P_{alpha} + P_{theta})},$$

where P_{alpha} and P_{theta} are the absolute powers calculated for the alpha band and theta band, respectively. By construction, nATR is in the range [−1,1], where nATR > 0 indicates a dominancy of frequencies in the 8–13 Hz range and a nATR < 0 a dominancy of frequencies in the 4–8 Hz range.

Peak frequency

The peak frequency was defined as the dominant frequency in the median PSD in the 4 to 13 Hz range.

Center of gravity

The center of gravity (CoG) reflects the space distribution of the EEG power in the brain.[30] Since we do not expect a left–right asymmetry after cardiac arrest, we focused on the distribution of the power of the previous defined peak frequency in the anterior-posterior direction. To calculate the CoG, we used the previously selected EEG epochs and removed only the prefrontal electrodes to minimize the influence of eye movements. For each other electrode, the frequency distribution was calculated with a frequency resolution of 0.5 Hz. Subsequently, we weighted the computed Fourier coefficients with its Euclidian distance from the Cz electrode, defined as the center of the brain, in the anterior-posterior direction. These results reflected the gravity of the spectral power as function of the frequency. Eventually, we defined the location of the peak frequency on the anterior-posterior line as the CoG. The CoG is normalized in the range −1 to 1, where −1 represents the posterior region of the brain and 1 the anterior part of the brain.

Statistical analyses

Demographic, baseline, EEG, and MoCA data are presented in a descriptive way. We used multivariate mixed models to examine the relationship between the EEG parameters and the continuous MoCA score at three different timepoints (during hospital stay and at three and twelve months follow up). We created four multivariate mixed models with random intercept in which the MoCA score at the three different timepoints was the dependent variable. We studied the relation between the MoCA score and one of the EEG parameters nATR, peak frequency, or CoG in models one, two and three respectively. In model four, we added all three EEG parameters as independent variables. Patient ID was used as a random effect. As sensitivity analyses, multivariate mixed analyses were repeated for the patients who had MoCA scores at all three timepoints. Predictive values of the models were evaluated using regression coefficients. Finally, we correlated EEG parameters with the composite score on the cognitive tests for the domains of memory, attention, and executive functioning from our neuropsychological examination using Pearson's correlation coefficients.

P-values < 0.05 were assumed statistically significant. Statistical analyses were performed with either MATLAB R2020a (The MathWorks, Inc.) or RStudio (RStudioTeam).

Data availability

The data that support the findings of this study are available from the corresponding author upon request, for verification of results or new relevant research questions, conditionally. Conditions include optimal data safety, adequate methodology, mutual appointments on collaboration, and approval of all collaborators.

Results

We included 80 patients in this analysis. At one year follow up, 51 patients were alive and consented to cognitive screening (Fig. 1).

Baseline characteristics of the 80 included patients are presented in Table 1. Mean age (61.5 years), proportion of male (88%), and proportion of shockable rhythms (96%) are as expected.[15,31] Median MoCA score increased from 24 to 27 between hospital stay and three months after cardiac arrest and remained essentially stable between three and twelve months (Fig. 2).

Relation between EEG parameters and MoCA scores

Results of multivariate mixed model analyses relating EEG parameters with MoCA scores are presented in Table 2 and Fig. 3. There was a statistically significant relation between each individual EEG parameter and the MoCA score during hospital stay (β_{nATR} = 7.36, p < 0.01; $\beta_{peak\ frequency}$ = 1.73, p < 0.01; β_{CoG} = -9.88, p < 0.01). When all three parameters were entered in the model (model 4), only relations with nATR and dominant peak frequency remained statistically significant (β_{nATR} = 3.93, p = 0.01; $\beta_{peak\ frequency}$ = 0.84, p = 0.02).

At three months after cardiac arrest, only nATR showed a statistically significant relation with the MoCA score (β_{nATR} = 2.49, p = 0.01; Table 2). None of the other EEG parameters showed significant relations with the MoCA score at three or twelve months after cardiac arrest. When all EEG parameters were entered (model 4),

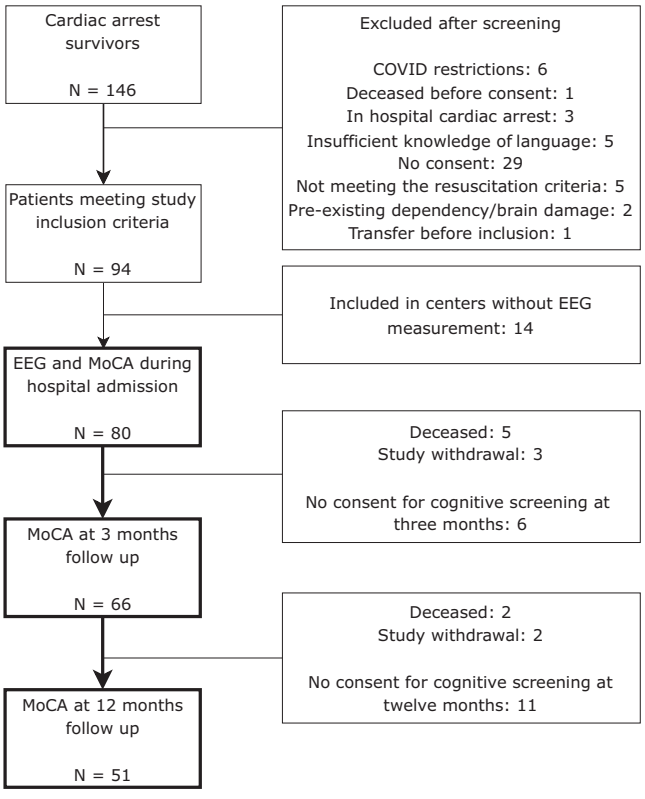

Fig. 1 – Flow of patients through the BROCA prediction study in the period of November 1st 2019, and May 15th 2022. Bold squares indicate flow of patients considered for this EEG analysis.

Table 1 – Demographic and clinical characteristics of the study population (N = 80).

Characteristic	Mean ± sd/number (%)/median [IQR]
Age in years	61.5 ± 11.4
Male sex	70 (88)
Resuscitation delay in minutes *	0 [0–1]
ROSC in minutes**	10 [8–15]
Initial rhythm	
Non-shockable	1 (1)
Shockable	77 (96)
Unknown	2 (3)
Admission to ICU (yes)	61 (76)
Stay on ICU in days	2.2 [1.5–5]
Recovery of consciousness on ICU in days	1 [1–3]
Presence of delirium	17 (21)
Days of delirious state	3 [2–5]
Time from cardiac arrest until EEG in days	6.6 [3.9–11.8]
Time from cardiac arrest until first MoCA in days	7.8 [3.4–11.3]
EEG parameters	
nATR (N = 80)	0.22 [-0.14–0.48]
Peak frequency in Hz (N = 76)	8.8 [8.0–9.7]
CoG (N = 74)	−0.30 [−0.45 – −0.17]
MoCA	
Hospital stay (N = 80)	24 [20–26]
3 months (N = 66)	27 [25–28]
12 months (N = 51)	27 [25–28]
MoCA score < 26	
Hospital stay (N = 80)	55 (69)
3 months (N = 66)	25 (38)
12 months (N = 51)	16 (31)
Z-scores per cognitive domain at 12 months	
Attention	−0.46 [−1.14–0.21]
Executive function	−0.41 [−0.86–0.19]
Memory	−0.72 [−1.33 – −0.21

ROSC = Return of spontaneous circulation; nATR = normalized alpha-to-theta ratio; CoG = center of gravity; MoCA = Montreal Cognitive Assessment. *Estimated delay from cardiac arrest to start resuscitation; **Estimated time to return of spontaneous circulation.

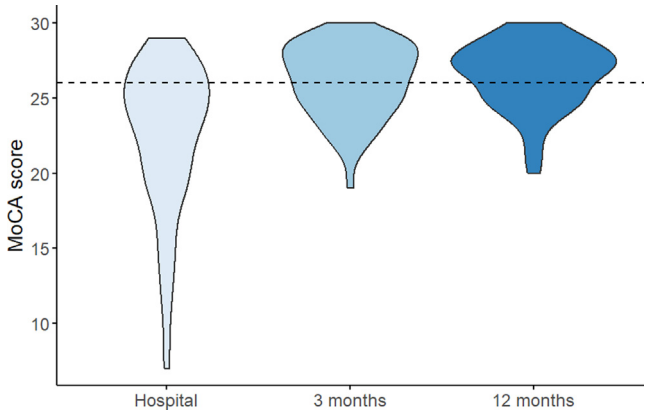

Fig. 2 – Violin plot showing the MoCA score on the three different timepoints. Median MoCA increased from 24 to 27 between hospital stay and three months after cardiac arrest and remained essentially stable between three and twelve months. The dashed line indicates a MoCA score of 26; a lower score reflects cognitive impairment.

none of the parameters showed significant relations with the MoCA score at three or twelve months after cardiac arrest.

Results were essentially similar in sensitivity analyses in the 50 patients with MoCA data on all three timepoints (Supplementary Material S2). Only the relation between dominant peak frequency and MoCA during hospital stay was no longer statistically significant in multi-parameter analyses in these 50 patients.

Correlation between EEG parameters and performance on neuropsychological examination at twelve months

More negative nATR and lower peak frequency during hospital stay were significantly correlated with a poorer memory performance at twelve months (Pearson R = 0.30, $p = 0.04$; Pearson $R = 0.48$, $p < 0.01$, respectively; supplementary material S3), but not with performance on attention and executive functioning. There were no significant correlations with CoG. See Table 3 for all correlations.

Discussion

We found a distinct relation between resting state EEG measures and global cognitive functioning of survivors in the first month after cardiac arrest: a negative alpha-to-theta ratio, a lower dominant peak

Table 2 – Results of multivariate mixed effects regression models relating individual EEG parameters with cognitive functioning measured during hospital stay, and at three and twelve months after cardiac arrest. The table shows estimates, 95% confidence intervals, and p-values.

		MoCA hospital		MoCA 3 months		MoCA 12 months	
	Predictors	*Estimates*	*P*	*Estimates*	*P*	*Estimates*	*P*
Model 1	Intercept	21.36 [20.63; 22.10]	<0.01	25.51 [24.68; 26.34]	<0.01	26.10 [25.18; 27.03]	<0.01
	nATR	**7.36 [3.28; 5.01]**	**<0.01**	**2.49 [0.51; 4.47]**	**0.01**	1.74 [−0.54; 4.02]	0.13
	Δ hospital			**4.87 [2.80; −6.94]**	**<0.01**	**5.60 [3.26; 7.98]**	**<0.01**
	Δ 3 months	**−4.87 [−6.94; −2.80]**	**<0.01**			0.75 [−1.68; 3.18]	0.54
	Δ 12 months	**−5.62 [−7.98; −3.26]**	**<0.01**	−0.75 [−3.18; 1.68]	0.54		
Model 2	Intercept	7.75 [3.60; 11.91]	<0.01	22.14 [17.42; 26.86]	<0.01	23.75 [18.05; 29.46]	<0.01
	Peak frequency	**1.73 [1.26; 2.21]**	**<0.01**	0.46 [−0.08; 0.99]	0.09	0.31 [−0.34; 0.96]	0.34
	Δ hospital			**1.28 [0.72; 1.84]**	**<0.01**	**1.42 [0.75; 2.09]**	**<0.01**
	Δ 3 months	**−1.28 [−1.84; −0.72]**	**<0.01**			0.14 [−0.55; 0.83]	0.68
	Δ 12 months	**−1.42 [−2.09; −0.75]**	**<0.01**	−0.14 [−0.83; 0.55]	0.68		
Model 3	Intercept	19.46 [17.92; 21.00]	<0.01	24.92 [23.25; 26.60]	<0.01	25.82 [23.93; 27.71]	<0.01
	CoG	**−9.88 [−13.96; −5.80]**	**<0.01**	−3.33 [−7.75; 1.10]	0.14	−1.62 [−6.37; 3.13]	0.50
	Δ hospital			**−6.55 [−11.10; −2.00]**	**<0.01**	**−8.26 [−13.13; −3.39]**	**<0.01**
	Δ 3 months	**6.55 [2.00; 11.00]**	**<0.01**			−1.71 [−6.69; 3.28]	0.50
	Δ 12 months	**8.26 [3.39; 13.13]**	**<0.01**	1.71 [−3.28; 6.69]	0.50		
Model 4	Intercept	13.81 [7.72; 19.89]	<0.01	23.41 [16.99; 29.83]	<0.01	24.87 [17.23; 32.52]	<0.01
	nATR	**3.93 [0.82; 7.03]**	**0.01**	0.61 [−2.79; 4.02]	0.72	0.95 [−3.33; 5.22]	0.66
	Peak frequency	**0.84 [0.14; 1.54]**	**0.02**	0.23 [−0.50; 0.91]	0.53	0.16 [−0.73; 1.05]	0.73
	CoG	−2.96 [−7.49; 1.57]	0.20	−1.78 [−6.80; 3.25]	0.49	0.06 [−5.78; 5.89]	0.99

Model 1:Relation between normalized alpha-to-theta ratio (nATR) and MoCA score. Model 2: Relation between peak frequency and MoCA score. Model 3: Relation between center of gravity (CoG) and MoCA score. Model 4: Relation between all three EEG parameters and MoCA score. Bold font indicates statistical significance at $p < 0.05$.

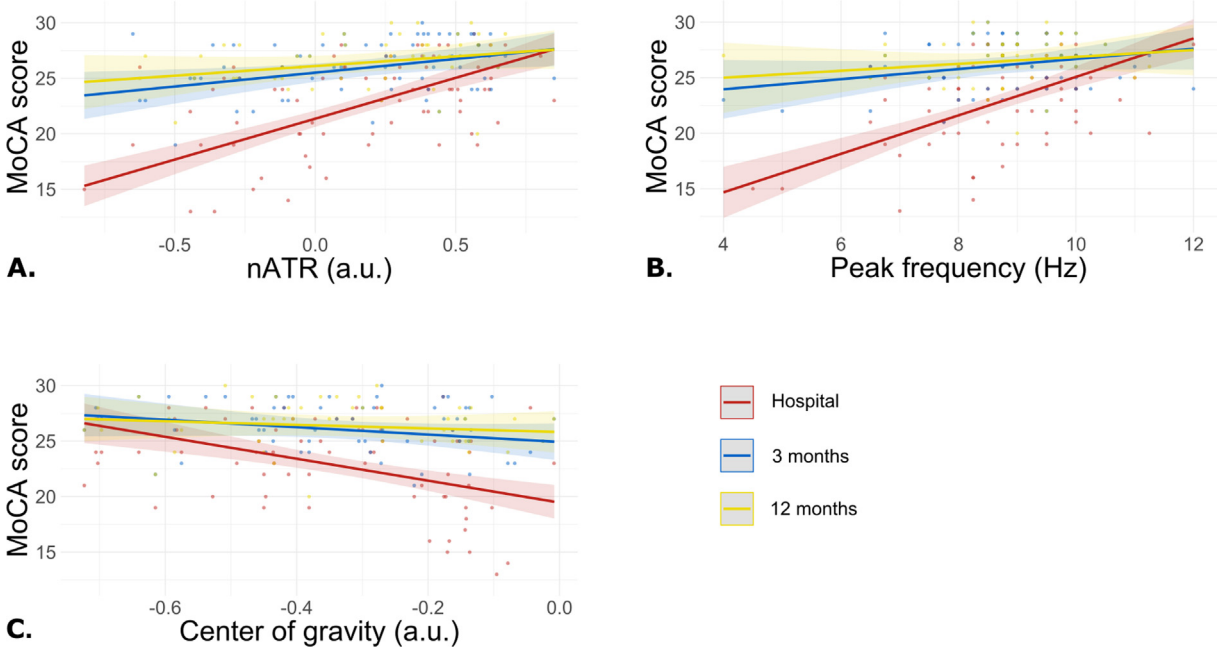

Fig. 3 – Visualization of the results of the multivariate mixed effects regression models of the three individual EEG parameters with MoCA scores during hospital stay, and at three and twelve months after cardiac arrest. It shows a clear relation between each of the individual EEG parameters and the MoCA scores during hospital stay (red lines) and a modest relation between normalized alpha-to-theta ratio (nATR) and the MoCA score at three months (blue line 3A), but not between the other EEG parameters and the MoCA scores at three months (blue lines 3B&C). None of the EEG parameters related with the MoCA score at twelve months yellow lines). The dispersion reflects the 95% confidence interval. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 3 – Correlations between EEG parameters and performance on neuropsychological examination at twelve months. The table shows Pearson R coefficients and p-values.

EEG parameter	*Cognitive domain*	*Pearson R*	*P*
nATR	Memory	**0.30**	**0.04**
	Attention	0.22	0.14
	Executive functioning	0.26	0.13
Peak frequency	Memory	**0.48**	**<0.01**
	Attention	0.04	0.80
	Executive functioning	0.13	0.46
CoG	Memory	−0.14	0.36
	Attention	−0.05	0.76
	Executive functioning	−0.09	0.62

nATR = normalized alpha-to-theta ratio, CoG = center of gravity. Bold font indicates a significant value for $p < 0.05$.

frequency, and more anterior location of the dominant peak frequency were associated with poorer general cognitive function during hospital stay. In addition, a negative alpha-to-theta ratio and lower dominant peak frequency were related to poorer memory performance at twelve months after cardiac arrest.

To our knowledge, no other study focused primarily on the relationship between early resting state EEG parameters (recorded while patients are awake) and short- and long-term cognitive functioning in patients after cardiac arrest. From a different perspective, one study investigated the effect of hypothermia treatment versus no hypothermia treatment on cognitive functioning using quantitative EEG measures as potential biomarkers. This study showed a trend of better EEG parameters (more fast and less slow frequency activities in all brain regions) in the hypothermia group, but no significant relations with cognitive functioning.[32]

Previous studies have focused on the role of the 'acute' EEG in predicting neurological outcomes, as measured by the cerebral performance categories (CPC), in comatose patients after cardiac arrest. Suppressed EEG patterns at 12 and 24 h after cardiac arrest were invariably associated with a poor outcome (CPC score 3–5).[17,19] Continuous rhythms at 12 and 24 h were strongly associated with a favorable outcome (CPC score 1–2).[17,19] In these comatose patients, quantitative EEG analysis was often superior/more accurate than visual analysis.[33,34] Here, we investigated the relationship between 'subacute' EEG (4 ± 3 weeks after cardiac arrest) in awake patients and short- and long-term cognition. Classically, our population falls within the CPC 1 and 2 categories. Capturing the more subtle EEG abnormalities in awake survivors appeared to be more challenging than that of the prominent EEG abnormalities in the comatose population.

Our current EEG parameters correlate well with general cognitive function during hospitalization, but less with cognitive function at three and twelve months follow up. This can be attributed to gradual cognitive improvement during follow up or to delirium during hospitalization. After regaining consciousness, some patients (33–100%) develop temporary states of confusion and agitation that may classify as delirium.[35,36] Delirium is a clinical syndrome, and we cannot unequivocally distinguish between "delirium" and "postanoxic encephalopathy" in patients with signs of inattention, disorganized thinking, and altered, fluctuating consciousness levels in the first weeks after cardiac arrest. The exact pathophysiology is largely unknown. It is likely a combination of different processes occurring simultaneously, including neuronal aging, neuroinflammation, oxidative stress, neuro-endocrine dysregulation, and circadian dysregulation.[37] In patients after cardiac arrest, post anoxic encephalopathy is probably the most important component. Diffuse slowing of EEG and increase of theta- and delta-power are common EEG phenomena in patients with a delirium.[38] An early study showed that improvement in cognition up to 19 months after a delirium was accompanied with significant increase in relative alpha power and reduction of theta and delta in follow up EEGs.[39] In our population, 17 patients (21%) were classified as delirious during median [IQR] 3 [2-5] days. It is plausible that the temporary clinical and EEG characteristics of delirium drive part of the relation between our EEG parameters and cognitive function during hospitalization, while recovery of these clinical signs explain the subsequent decline in this relation over the long term.

Our correlations suggest that early EEG abnormality could be a possible clinical predictor of long term memory dysfunction. However, due to the relatively weak correlations and limited sample size included in the current analysis, clinically relevant predictive values could not be derived from this study.

Strengths of our study include the prospective multicenter design, the use of validated cognitive screening and testing instruments, and state-of-the-art EEG analyses. Our study also has limitations. First, in our prospective protocol we stated that we would include patients at 4 ± 3 weeks after cardiac arrest during their hospital stay. However, more than half of our sample regained consciousness relatively early after cardiac arrest, and could be discharged within one week. Therefore, we deviated from our study protocol relatively often, with inclusion in the first week after cardiac arrest. Second, our sample was relatively small (N = 80) and our study lacks external validation. Also, cognitive testing at twelve month follow up was done in only 59% of the patients, due to mortality and dropping out. We cannot exclude that this introduced a certain bias, although sensitivity analysis confirmed our main findings. If mortality or dropping out mainly occurred in relatively severe encephalopathy, our established relations could be an underestimation. Third, we did not yet test the value of EEG parameters in addition to other potential or known predictors of cognitive outcome, such as demographic factors (e.g. age and higher education),[40] event variables (e.g. CPR prior to ambulance arrival)[41,42] and treatment.[10] Fourth, some patients probably

had a delirium during testing, which might have affected the strength of the relation between the EEG parameters and MoCA score during hospital stay.

Conclusion

In this prospective multicenter cohort study, early resting state EEG parameters from awake patients after cardiac arrest relate with early general cognitive function, as well as with memory performance at twelve months follow up. The additional predictive value of nATR for individual patients after cardiac arrest, in addition to potential clinical or MRI predictors of cognitive function, will be addressed in future multimodal analyses of the full BROCA dataset.

Conflict of interest disclosure

M.J.A.M. van Putten is co-founder of Clinical Science Systems, which is a supplier of EEG systems for two of the participating sites (Rijnstate Hospital and Medisch Spectrum Twente). Clinical Science Systems did not provide funding and was not involved in the design, execution, analysis, interpretation or publication of the study. The other authors do not report any conflicts of interest.

CRediT authorship contribution statement

A.B. Glimmerveen: Writing – original draft, Visualization, Software, Methodology, Investigation, Formal analysis, Conceptualization. **M. M.L.H. Verhulst:** Writing – review & editing, Software, Methodology, Investigation, Formal analysis. **N.L.M. de Kruijf:** Writing – review & editing, Software, Investigation, Formal analysis. **P. van Gils:** Writing – review & editing, Methodology, Investigation. **T. Delnoij:** Writing – review & editing, Methodology, Formal analysis. **J. Bonnes:** Writing – review & editing, Methodology, Formal analysis. **C.M. van Heugten:** Writing – review & editing, Methodology, Formal analysis. **M.J.A.M. Van Putten:** Writing – review & editing, Supervision, Software, Methodology, Formal analysis. **J. Hofmeijer:** Writing – review & editing, Supervision, Methodology, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.resuscitation.2024.110253.

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