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Abstract

EEG and Clinical Neuroscience Society (ECNS) 2018. Background. Transient ischemic attack (TIA) is characterized by stroke-like neurologic signs and symptoms in the absence of demonstrable structural neuropathology. There is no test for TIA, with classification often reliant on subjective, retrospective report. Functional brain measures such as the electroencephalogram (EEG) may be helpful in objectively detecting and describing the pathophysiology of TIA, but this has not been adequately examined. Methods. EEG was obtained from a single electrode over the left frontal lobe during 3-minute resting-state and auditory oddball conditions administered to consecutive patients within 72 hours of admission to the acute stroke ward of a tertiary hospital. Separately, patients were classified by their treating team as having suffered either an ischemic stroke (n = 10) or a TIA (n = 10). Relative power of delta, theta, alpha, and beta EEG frequency bands were extracted for comparison between the 2 clinical groups and an existing normative sample of 10 healthy, age-, gender-, and education-matched older adults. Results. Analysis of variance with post hoc testing identified pronounced delta activity in stroke patients, while alpha and beta power were elevated in TIA patients. Both patient groups exhibited attenuated theta activity compared with healthy controls. Receiver operating characteristic curve analysis identified thresholds for each EEG frequency capable of distinguishing the 3 participant groups. Conclusions. TIA, ischemic stroke, and healthy aging are each associated with distinct electrophysiological profiles. These preliminary findings suggest that acute EEG may be helpful in elucidating the pathophysiology and reversibility of TIA symptoms, and further exploration of the value of this unique functional brain data is encouraged.

Disciplines
Education | Social and Behavioral Sciences

Acute EEG patterns associated with transient ischemic attack

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Abstract

Background: Transient ischemic attack (TIA) is characterized by stroke-like neurologic signs and symptoms in the absence of demonstrable structural neuropathology. There is no test for TIA, with classification often reliant on subjective, retrospective report. Functional brain measures such as the electroencephalogram (EEG) may be helpful in objectively detecting and describing the pathophysiology of TIA, but this has not been adequately examined.

Methods: EEG was obtained from a single electrode over the left frontal lobe during 3-min resting-state and auditory oddball conditions administered to consecutive patients within 72 hours of admission to the acute stroke ward of a tertiary hospital. Separately, patients were classified by their treating team as having suffered either an ischemic stroke (n=10) or a TIA (n=10). Relative power of delta, theta, alpha, and beta EEG frequency bands were extracted for comparison between the two clinical groups and an existing normative sample of 10 healthy, age-, gender-, and education-matched older adults.

Results: Analysis of variance with post-hoc testing identified pronounced delta activity in stroke patients, while alpha and beta power were elevated in TIA patients. Both patient groups exhibited attenuated theta activity compared to healthy controls. Receiver Operator Curve analysis identified thresholds for each EEG frequency capable of distinguishing the three participant groups.

Conclusions: TIA, ischemic stroke, and healthy aging are each associated with distinct electrophysiological profiles. These preliminary findings suggest acute EEG may be helpful in elucidating the pathophysiology and reversibility of TIA symptoms, and further exploration of the value of this unique functional brain data is encouraged.

Introduction

Approximately 200,000 to 500,000 patients are diagnosed every year with a transient ischemic attack (TIA) in the United States.[1, 2] An additional 300,000 to 700,000 individuals experience neurologic symptoms suggestive of a TIA, but delay or fail to seek medical attention for their symptoms.[3, 4] However, the temporary nature of TIA symptoms may not be as benign as previously presumed. TIA often heralds an upcoming stroke,[5] with approximately one in 10 TIA patients suffering a stroke within the next 90-days, half of which occur within the first 48 hours.

Recognition of the increased risk of early adverse events after TIA has prompted a reconsideration of the clinical definition of the condition.[6, 7] TIA has historically been regarded as a temporary disruption of a specific arterial distribution, with the neurologic signs and symptoms considered to be brief and reversible,[8, 9] and damage to affected brain cells expected to be minimal.[10] This perspective has been increasingly challenged by evidence that TIA symptoms are not transient at the tissue level, with computed tomography (CT) and conventional magnetic resonance imaging (MRI) revealing the presence of neuroanatomically relevant ischemic brain infarcts in nearly one- to two-thirds of TIA patients.[11-13] However, even more sensitive diffusion weighted MRI (DWI) may fail to detect micro-ischemia after TIA, or to differentiate between acute and chronic infarction.[14] Furthermore, the "robustness"[15] or "ischemic tolerance"[16] of affected neuronal circuitry likely plays a role in the pathogenesis and prognosis of clinical symptoms in TIA patients, which cannot be readily assessed with anatomical tests. In addition to structural neuroimaging investigations, functional brain measures may therefore be helpful in detecting and describing the pathophysiology of TIA.

The electroencephalograph (EEG) is a well-established tool for detecting, describing, and monitoring brain function in healthy individuals and clinical populations.[17] EEG

measures the summation of electrical currents maintained in the networks of neurons throughout the brain, which are made up of excitatory and inhibitory post-synaptic potentials occurring in individual neurons.[18] Brain changes can be identified through the amplification or attenuation of electrical activity within specific, commonly-examined frequency bands (e.g. delta, theta, alpha, beta). Quantitative EEG refers to the conversion of EEG waveforms into a digital signal, and may offer improved detection and description of brain activity, as compared to visual inspection of analog EEG outputs.[17]

Ischemia is associated with immediate brain changes resulting from the suppression of oxygen and glucose supply, including a biochemical cascade that can ultimately lead to cell death and cerebral infarction.[19, 20] EEG is sensitive to the effects of these acute changes,[21, 22] capable of detecting voltage oscillations resulting from the toxic production of lactic acid[23] and free radicals,[24] calcium accumulation,[24] protein degeneration,[24] and loss of transmembrane gradients.[19] In particular, ischemic stroke is associated with the rapid appearance of globally distributed slow wave delta frequency activity.[25-30]

To date, few studies have applied EEG to the examination of TIA. Compared to a group of age-matched healthy controls, Madkour and colleagues[31] reported that 80% of TIA patients exhibited resting-state EEG abnormalities, most often in the form of increased theta activity in the ipsilateral hemisphere. Theta abnormalities were inconsistently accompanied by alpha and beta activity suppression, and increased delta activity. There was no CT evidence of focal cerebral ischemic lesions in any of the TIA patients. While these results provide an initial demonstration of the unique information provided by EEG, not available from purely anatomical tests, this study was not without its limitations. First, patients were examined up to two weeks after TIA, an unnecessarily variable and delayed assessment window. Second, three-quarters of the TIA group had a history of previous TIAs, introducing the confounding influence of previous or accumulated neurologic insult. Third,

the study did not include ischemic stroke participants, and it remains unclear whether the two ischemic syndromes possess unique EEG profiles.

In sum, the transient neurologic dysfunction occurring in TIA appears to be associated with objective EEG changes. However, this finding would benefit from replication in an independent sample, examined acutely, following a first episode of TIA, and compared with both healthy controls and ischemic stroke patients. It was hypothesized that the transient ischemic event associated with TIA would manifest as acute alterations in theta, relative to healthy controls. Excess delta activity, characteristic of ischemic stroke, was not expected to be present in the acute stages following TIA.

Methods

Participants

Ischemic stroke and TIA participants were recruited from the acute stroke ward of a tertiary hospital in Sydney, Australia. Patients admitted to the unit within 72 hours of first-ever stroke-like symptoms were eligible for participation. Time of onset was defined as the time the participant was last seen without symptoms, as documented in the medical records. Individuals with a previous history of neurologic or psychiatric disorder, non-English speaking, or under 18 years of age, were excluded. Stroke nurses assisted the study team in identifying eligible candidates.

Diagnosis was determined by the participant's treating team (see Procedure), which was independent from the study investigators. Patients subsequently diagnosed with hemorrhagic stroke or other stroke- and TIA-mimics (e.g. migraine, seizure, syncope, vertigo, anxiety)[32] were excluded from analysis. Finally, a gender-, education-, and age-matched, healthy older adult (>55 years old) control group was created from the dataset of a previously recruited study cohort.[33]

Tasks and measures

Continuous EEG was collected during both a 3-min eyes-closed resting-state, and a 3-min eyes-closed auditory oddball condition. The oddball task consisted of 500 Hz non-target and 1000 Hz target tones at 1-sec intervals, with non-target tones occurring with a probability of 0.8. Tones were presented binaurally through the inbuilt headphones of the MindSet headset device (see below) at an intensity of 65dB. Participants were not required to respond actively to the task, as electrophysiological markers of mental processing can be reliably evoked in a passive oddball paradigm.[34, 35]

The National Institute of Health Stroke Scale (NIHSS)[36] and Modified Rankin Scale (mRS)[37] were collected to provide information on the severity of neurologic symptoms and disability, respectively, in the stroke and TIA participants. The NIHSS is an 11-item scale used in assessing level of consciousness, visual, motor, sensory, and language function. Higher scores (max = 42) indicate a higher degree of stroke severity.[38] The mRS measures global disability[39] on a scale of 0 to 6, with higher scores reflecting poorer outcome. The Oxfordshire Community Stroke Project Classification[40] was used to classify stroke into four categories: Total Anterior Circulation Stroke, Partial Anterior Circulation Stroke, Posterior Circulation Stroke and Lacunar Stroke. In TIA patients, the 90-day incidence of stroke was also recorded.

EEG data acquisition and analysis

Continuous EEG was obtained with the MindSet device (NeuroSky, San Jose, California), a single channel, wireless headset with demonstrated validity[41] and reliability.[33] To facilitate bedside data acquisition in an acute neurologic population with potentially limited tolerances for psychophysiological assessment, the rapid fitting and calibration procedures of the portable device were preferred over conventional recording systems. [30, 41, 42]

The MindSet device consists of a ThinkGear microchip and embedded firmware, and 10 mm dry stainless steel active, material reference, and ground electrodes contained within a set of headphones. The reference and ground electrodes are housed within the left ear pad, while the EEG electrode is embedded in a flexible arm extending from the left headband, positioned at the International 10–20 system site FP1. Electrical potentials at the active and reference electrodes are subtracted through common mode rejection to derive a single EEG channel signal which is amplified 8000 times. Sampling and amplification of the raw 128 Hz data are carried out within the embedded microchip and transmitted wirelessly by Bluetooth© to a computer for recording and subsequent off-line quantitative analysis.

Using SCAN Edit version 3 software (NeuroscanTM, USA), the raw EEG waveform data was band-pass filtered (0.5-30 Hz), and manually inspected to identify any movement or muscle artifact. Identified sections of artifact were excluded from further processing. Remaining epochs containing amplitudes in excess of $\pm$ 100 µV were removed using the rejection filter included in the SCAN software. The artifact-free 4-sec EEG epochs (1/4 Hz resolution) were submitted to Fast Fourier Transforms, with 10% Hamming to extract the absolute power in the following frequency bands: delta (1.5–3.5 Hz), theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz), and beta (12.5–25 Hz). Relative power was calculated by summing absolute power across the four bands to compute total power, and then dividing the absolute power for each individual band by the total power, expressed as a percentage.

Procedure

For the clinical populations, continuous EEG recordings were obtained at hospital bedside within 72-hours of hospital admission. The control group had previously completed the EEG recording session in a university laboratory. In all participants, the single electrode was located at the International 10-20 system position FP1. After minimizing signal

impedance, participants were asked to close their eyes and relax for the two 3-min recording sessions, with the order of presentation counterbalanced between subjects. NIHSS and mRS ratings and Oxfordshire classifications were also completed at this time. The 90-day incidence of stroke in the TIA group was obtained from a subsequent review of the medical record. As a token of appreciation for their time, participants received $10 gift vouchers.

All study instruments were administered following specialist training with experienced clinicians. Rating scales and EEG recordings were completed by study investigators blinded to clinical diagnosis. Clinical diagnoses were based on a standard battery of medical investigations, including CT and MRI, transthoracic echocardiogram, fasting bloods and glucose, and carotid doppler ultrasound or computerized tomographic angiography. TIA was defined using clinical criteria of symptoms of neurological deficit persisting for <24 hours from onset, and no abnormalities detected on neuroimaging evidence. This research was approved by the Australian Catholic University and South Eastern Sydney Local Health District Human Research Ethics Committees, and each participant (or their carer/substitute decision maker) provided written informed consent for voluntary participation.

Statistical analysis

Between group differences in clinical and demographic variables were analyzed with univariate Analysis of Variance (ANOVA) or the likelihood ratio chi-square test (to correct for small sample sizes), depending on the nature of the data. ANOVAs were used to test between group differences in EEG data, with Bonferroni *post hoc* tests and 95% Confidence Intervals (95% CIs) used to identify significant interaction effects. Non-overlapping CIs reflect a statistically significant difference of $p < 0.01$, while CIs where the overlap is no more than half the average margin of error reflect a p-value < 0.05.[43] All analyses were

completed with IBM SPSS Statistics for Windows version 23 (IBM Corp., Armonk, N.Y).

Finally, Receiver Operator Curve (ROC) analyses were conducted using GraphPad Prism

version 7 for Windows (GraphPad Software Inc, La Jolla, CA) to determine optimal EEG

criterion cut-offs for classifying group membership.

Results

Demographic and clinical information for each participant group is presented in Table

1. The final sample included 10 patients diagnosed with ischemic stroke, 10 patients

diagnosed with TIA, and 10 healthy controls. Age ($p = 0.89$), gender distribution ($p = 0.86$),

and years of education ($p = 0.55$) were equivalent between groups. The stroke group had

greater global disability on admission (mRS score; $p = 0.02$), and more severe neurologic

symptoms (NIHSS score; $p = 0.02$) than TIA patients. On the basis of the NIHSS score,[36]

70% of stroke patients had suffered a *mild* (≤ 8) and 30% a *moderate* (9-15) stroke. The

majority of strokes had occurred in the right hemisphere (70%). Over the 90-days following

admission, no TIA patients experienced a subsequent stroke.

EEG was obtained from stroke and TIA patients on average within the first two days

(range 18-72 hours) after symptom onset (Table 1). There was no difference between clinical

groups in the time interval to EEG collection ($p = 0.90$). In the eyes-closed resting condition,

there was a significant main effect of group on relative delta power (Table 2). Post-hoc

testing identified that delta was significantly elevated in stroke patients, relative to TIA

patients ($p < 0.01$) and healthy controls ($p = 0.03$). ROC analysis identified that a delta value

of 0.34 provided the optimal threshold for discriminating stroke patients from TIA patients

and healthy controls (Table 3).

In the auditory oddball condition, ANOVAs identified a significant effect of group for

all four EEG bands (Table 2). Post-hoc testing and inspection of the 95% CIs (Figure 1) were

used to identify significant group x EEG component interactions. Relative delta power was significantly elevated in stroke patients, compared to both the TIA ($p < 0.01$) and control groups ($p = 0.03$), with an optimal ROC threshold of 0.35 (Table 3). Relative theta power was significantly diminished following stroke ($p < 0.01$) and TIA ($p = 0.03$), compared to the control group, with an optimal ROC threshold of 0.34. Relative alpha power was elevated in TIA patients, compared to both the stroke ($p < 0.01$) and control groups ($p = 0.02$), with an optimal ROC threshold of 0.24. Similarly, relative beta power was elevated in TIA patients, compared to both the stroke ($p < 0.01$) and control groups ($p = 0.02$), with an optimal ROC threshold of 0.13.

Discussion

The current study presents a small, consecutive series of previously-well ischemic stroke and TIA patients. All 10 TIA patients experienced good neuroradiologic (i.e. no ischemic brain infarcts on CT or MRI) and neurologic outcomes (i.e. no 90-day incidence of stroke). The 90-day incidence of progression from TIA to stroke was lower than the early adverse event risk rates reported in the literature,[5, 44] and is likely owing to the positive impact of the timely inpatient care these patients received.[45, 46] However, despite these good outcomes, acute EEG recordings from a single left pre-frontal region identified a number of electrophysiological features in TIA patients that were distinct from those recorded during eyes-closed resting and auditory oddball conditions in age-, education-, and gender-matched ischemic stroke patients and healthy older adult controls. These unique EEG profiles are discussed below.

Group differences in slow frequency activity

As expected, stroke patients exhibited increased slow wave delta activity, relative to both TIA patients and healthy older adult controls. Increased activity in the delta band of the EEG has been repeatedly reproduced using a resting state paradigm,[25-30, 47] and the current study demonstrates that this characteristic EEG feature of acute stroke is also present during a passive auditory oddball condition. The ROC curve cut-offs identified for both the resting-state and oddball conditions were highly similar (0.34 c.f. 0.35), and provided reasonable accuracy for distinguishing stroke patients from TIA patients and healthy controls (80-90% sensitivity, 80-85% specificity). A previously reported delta threshold of 0.51, derived from a 19-channel, lab-based recording montage,[29] may be overly stringent, and was associated with a true positive rate of only 30% in the current study. In sum, the current results add to a growing body of evidence that acute delta abnormalities after stroke can be reliably detected from a more limited frontal recording montage.[30, 47] Finally, increased delta activity was specific to stroke patients only. During both resting-state and oddball conditions, there was no evidence of increased delta activity in TIA patients, whose average relative power in this frequency band was equivalent to healthy older adults.

In contrast, both stroke and TIA patients exhibited acute reductions in oddball condition theta activity, which accurately distinguished these clinical groups from healthy controls (85% sensitivity, 90% specificity). Reduced theta activity after stroke has previously been reported,[30] but has not been described after TIA. A previous EEG investigation of TIA, in fact, reported enhanced theta activity,[31] which the authors speculated may be a secondary effect of alpha frequency suppression. However, this explanation is incompatible with the pattern of results obtained from clinical participants in the current study, in which alpha activity either was within normal limits in the stroke patients (as compared to healthy controls), or was increased in TIA patients (discussed in the next section). Reduced frontal

theta has been previously described in older adults exhibiting cognitive inefficiencies,[48-51] and may reflect dysfunction in cortical circuitry linked to the frontal lobe.

The observed pattern of acute theta attenuation after both TIA and ischemic stroke, and delta enhancement after stroke only, may be explained by the concept of "viability thresholds."[19, 52] EEG is sensitive to changes in the metabolic and electrical activity of neurons that occurs when cerebral blood flow is compromised.[53, 54] Two critical perfusion thresholds have previously been identified, an "ischemic threshold" associated with reversible functional failure within the ischemic penumbra, and an "infarction threshold" associated with irreversible failure and structural damage within the ischemic core.[55, 56] If sufficient blood flow is restored within a suitable time frame, areas within the ischemic penumbra recover without residual morphologic damage.[55] The observed theta attenuation in the current study may reflect acute alterations occurring in a brain experiencing hypo-perfusion below the ischemic threshold, which can persist following TIA,[57] and results in temporary functional changes but not structural injury. In contrast, the observed acute delta enhancement may emerge when the crucial perfusion threshold for irreversible failure is met. Ischemic stroke crosses the infarction threshold,[19] at which point an excitotoxic ischemic cascade is triggered that leads to cell death and produces signs of infarction within the ischemic core.[58]

Group differences in faster frequency activity

TIA patients in the current study also demonstrated group differences in higher frequency alpha and beta activity during the passive auditory oddball condition. The activity in these frequencies was significantly elevated, with ROC analysis identifying cut-offs for alpha and beta that distinguished TIA patients from both healthy controls and ischemic stroke patients with reasonable accuracy (80-90% sensitivity, 80-90% specificity). This relationship

has not previously been reported, and the implications for brain function and symptomatology are not clear. Results from studies examining methods for improving outcomes after acquired brain injury may provide a possible explanation. Specifically, increases in alpha[59, 60] and beta[60, 61] EEG activity have been associated with functional recovery after brain injury, and may represent ongoing improvements within functional cerebral processing networks. In the current study, the observed amplification of faster EEG frequencies in TIA patients may also represent functional recovery processes, initiated following the transient deterioration in perfusion rates below the ischemic threshold. While providing a potential marker of the mechanism underlying the reversibility of symptomatology in TIA, this hypothesis remains untested.

Implications of EEG results

There is no test for TIA, and the diagnosis relies heavily on the patient's account of their history and on expert interpretation of that history.[32] Inter-rater agreement regarding the diagnosis of TIA has historically been poor,[62] even among stroke trained neurologists,[63] and the identification of quantitative biomarkers may enhance the objectivity of clinical assessment. The identification of objective biomarkers may also facilitate the accurate and appropriate deployment of early treatment and secondary prevention interventions to reduce the risk of further events, once an ischemic cause of symptoms is confirmed.

While previous research has suggested it is difficult to detect a TIA by EEG,[64, 65] the current study identified unique electrophysiological profiles distinguishing TIA patients from both ischemic stroke patients and healthy controls. In particular, an excess of frontal delta activity (> 0.35) recorded from a single pre-frontal channel provides a potential marker of a brain that has surpassed a crucial infarction threshold, and is passing into a state of irreversible injury following ischemic stroke. Reductions in frontal theta (< 0.34) appear to

provide a sensitive but non-specific biomarker of temporary functional deterioration, as the brain surpasses an ischemic threshold following either TIA or ischemic stroke. An excess of higher frequency alpha (> 0.24) and beta (> 0.13) activity may be biomarkers of the recovery processes that drive the reversibility of neurologic signs and symptoms following a TIA.

Of note, the group differences in EEG activity following TIA were not detectable during a resting-state condition. The modestly increased demands of the passive oddball condition are associated with changes in the alpha[66, 67] and beta[68, 69] frequency of the EEG, reflecting stimulus processing and cognitive control, respectively. As such, resting-state tasks may be insufficient to elicit the subtle and reversible changes occurring after a TIA, and may provide an explanation for the conflicting results between the current study and past electrophysiological examinations of TIA patients.[31, 64] The recording of EEG during a passive oddball paradigm is encouraged for further exploration of cerebrovascular diseases, owing to the potential superior sensitivity to a range of ischemic syndromes, compared to resting-state conditions.

Limitations of the current study

The current study used a relatively small sample size (n=30), which can reduce the chance of detecting a true effect.[70] To overcome this, visual inspection of 95% CIs was utilized to detect significant interactions, and the number of *post-hoc* analyses was limited. Furthermore, the effect sizes of significant results were all *large* ($d > 0.70$), as specified by the guidelines of Cohen,[71] suggesting the study was adequately powered. Regardless, the current results should be regarded as preliminary, with further research needed to confirm and extend these findings.

The current study relied upon CT and conventional MRI for visualization of the brain in TIA and stroke patients. However, DWI has superior resolution and contrast

enhancement,[72] and is recommended as the preferred method of imaging in patients with TIA.[7] Reliance in the current study on CT and conventional MRI techniques may have failed to detect clinically relevant acute ischemia in the TIA sample.[73]

Furthermore, the collection of clinical (i.e. mRS and NIHSS) and EEG data typically occurred on the second day after symptom onset. As the average duration of TIA symptoms is less than one hour,[7] it may be preferable to acquire data sooner after symptom onset. Longitudinal clinical and EEG follow-up (e.g. 90- or 180-days) would also be informative, to examine the prognostic value of the observed electrophysiological findings.

Finally, clinical participants were recruited solely from a hospital setting. While this is a typical recruitment strategy for stroke patients, many individuals experiencing a TIA do not present to hospital,[1, 4] and the TIA patients included in the current study may reflect a unique sub-group of this clinical population.

Conclusions

For a condition as common and serious as TIA, there remains diagnostic uncertainty, and the pathophysiology of the condition requires further investigation. In particular, the term "ischemic" in TIA is an indefinite diagnostic categorization, often decided by a clinician's best judgement as to whether neurologic signs and symptoms are consistent with a specific arterial territory.[74] However, due to the "transient" nature of the condition, the semiologic information clinicians use to localize the clinical features to a vascular territory is often historical, provided by the patient or other observers, and susceptible to the reliability and knowledge of the informant.[74] The current preliminary results identified sensitive and specific EEG characteristics associated with TIA, which may help to objectively characterize and categorize the clinical features of the condition, and ultimately to understand the features of TIA that permit rapid recovery. The prognostic value of the current EEG findings is

unknown, and further work examining the relationship between acute electrophysiological

markers of brain function and outcomes after TIA is encouraged.

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Figure 1. 95% Confidence Intervals for the auditory oddball task EEG frequencies of interest for each participant group. Asterisks identify the significantly different group for each EEG component.
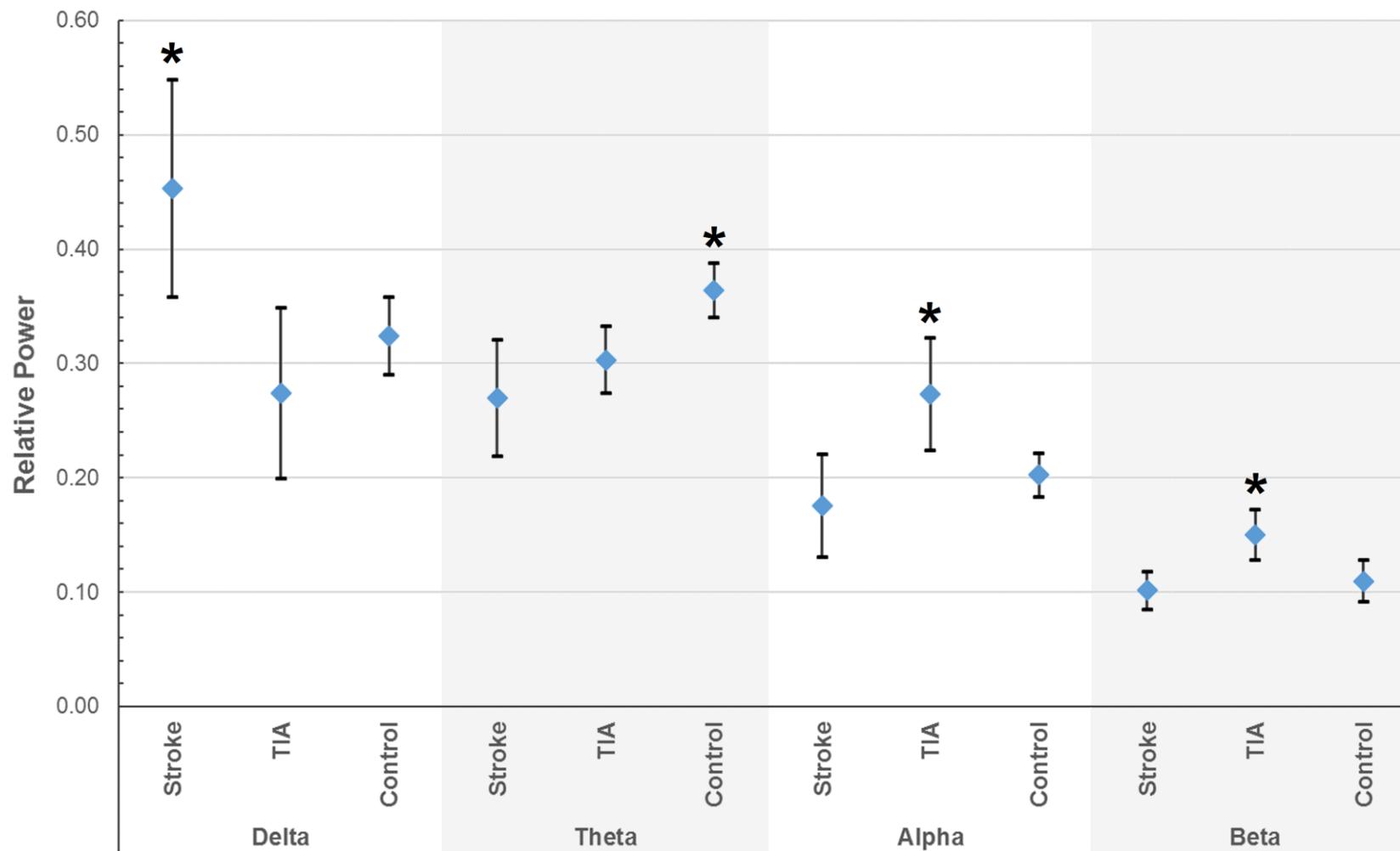
Table 1. Demographic and clinical information for the ischemic stroke, TIA, and healthy control groups

Variable	Stroke (n=10)	TIA (n=10)	Control (n=10)	Statistic	Significance
Age[a], yrs	67.80 (12.73)	70.40 (13.59)	69.20 (8.88)	$F(2, 27) = 0.12$	$p = 0.88$
Male[b]	7 (70)	6 (60)	7 (70)	$G(2) = 0.30$	$p = 0.86$
Education[a], yrs	11.60 (3.63)	11.40 (1.96)	12.60 (1.78)	$F(2, 27) = 0.62$	$p = 0.55$
Recording Admission Interval[a], hrs	44.40 (22.03)	43.40 (16.55)	-	$F(1, 18) = 0.02$	$p = 0.90$
NIHSS score[a]	5.20 (5.65)	0.60 (1.08)	-	$F(1, 18) = 6.39$	$p = 0.02$
mRS score[b]				$G(4) = 11.49$	$p = 0.02$
0	0 (00)	3 (30)	-		
1	4 (40)	6 (60)	-		
2	1 (10)	1 (10)	-		
3	3 (30)	0 (00)	-		
4	2 (20)	0 (00)	-		
Stroke Hemisphere[b]					
Right	7 (70)	-	-		
Left	3 (30)	-	-		
Stroke Location					
PACS	2 (20)	-	-		
TACS	1 (10)	-	-		
POCI	4 (40)	-	-		
LACS	3 (30)	-	-		

*Note: [a]Mean (SD); [b]n (%). *G* statistic: likelihood ratio chi-square test. LACS: Lacunar Stroke; mRS: modified Rankin Scale; NIHSS: National Institutes of Health Stroke Scale; PACS: Partial Anterior Circulation Stroke; POCI: Posterior Circulation Stroke TACS: Total Anterior Circulation Stroke

Table 2. EEG outcomes for the ischemic stroke, TIA, and healthy control groups

Variable[a]	Stroke	TIA	Control	*F* Ratio	Significance	Power
EC delta	0.43	0.23	0.28	$F(2, 27) = 7.26$	$p < 0.01$	0.91
	(0.12)	(0.15)	(0.07)			
EC theta	0.28	0.24	0.33	$F(2, 27) = 2.47$	$p = 0.10$	0.45
	(0.05)	(0.13)	(0.07)			
EC alpha	0.18	0.21	0.25	$F(2, 27) = 1.41$	$p = 0.26$	0.28
	(0.08)	(0.13)	(0.09)			
EC beta	0.11	0.11	0.13	$F(2, 27) = 0.39$	$p = 0.68$	0.11
	(0.04)	(0.07)	(0.05)			
OB delta	0.45	0.27	0.32	$F(2, 27) = 8.16$	$p < 0.01$	0.94
	(0.13)	(0.10)	(0.05)			
OB theta	0.27	0.30	0.36	$F(2, 27) = 8.64$	$p < 0.01$	0.95
	(0.07)	(0.04)	(0.03)			
OB alpha	0.18	0.27	0.20	$F(2, 27) = 8.40$	$p < 0.01$	0.94
	(0.06)	(0.07)	(0.03)			
OB beta	0.10	0.15	0.11	$F(2, 27) = 7.94$	$p < 0.01$	0.93
	(0.02)	(0.03)	(0.03)			

Note: [a]Mean (SD); EC: eyes-closed condition; OB: auditory oddball condition

Table 3. Optimal Receiver Operator Curve criterion cut-offs for the EEG frequencies

of interest

Criterion and Threshold		AUC	Sensitivity [95% CI]	Specificity [95% CI]
EC delta	> 0.34	0.86**	80.00 [44.39, 97.48]	80.00 [56.34, 94.27]
OB delta	> 0.35	0.87**	90.00 [55.50, 99.85]	85.00 [62.11, 96.76]
OB theta	< 0.34	0.93**	85.00 [62.11, 96.76]	90.00 [55.50, 99.75]
OB alpha	> 0.24	0.81**	80.00 [44.39, 97.48]	90.00 [68.30, 98.77]
OB beta	> 0.13	0.86**	90.00 [55.50, 99.75]	80.00 [56.34, 94.27]

Note: **$p < 0.01$; AUC: area under curve; CI: confidence interval; EC: eyes-closed condition; OB: auditory oddball condition