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Peak alpha frequency: an electroencephalographic measure of cognitive preparedness

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Abstract

Objective: Electroencephalographic (EEG) peak alpha frequency (PAF) (measured in Hz) has been correlated to cognitive performance between healthy and clinical individuals, and among healthy individuals. PAF also varies within individuals across developmental stages, among different cognitive tasks, and among physiological states induced by administration of various substances. The present study suggests that, among other things, PAF reflects a trait or state of cognitive preparedness.

Methods: Experiment 1 involved 19-channel EEG recordings from 10 individuals with traumatic brain injury (TBI) and 12 healthy matched controls, before, during, and after tasks of visual and auditory attention. Experiment 2 involved EEG recordings from 19 healthy young adults before and after a working memory task (WAIS-R Digit Span), repeated on 2 different days to measure within-individual differences.

Results: Experiment 1 showed significantly lower PAF in individuals with TBI, mostly during post-task rest. Experiment 2 showed PAF during pre-task baseline to be significantly correlated with Digit Span performance of the same day but not with Digit Span performance of another day. Moreover, PAF was significantly increased after Digit Span for those participants whose PAF was lower than the sample median before the task, but not for those who had it higher. Finally, both PAF and Digit Span performance were increased during the second day.

Conclusions: PAF was shown to detect both trait and state differences in cognitive preparedness, as well as to be affected by cognitive tasks. Traits are better reflected during post-task rest, whereas states are better reflected during initial resting baseline recordings.

Keywords: Electroencephalographic; Peak alpha frequency; Cognitive preparedness; Traumatic brain injury; Working memory; Trait; State; Resting baseline

1. Introduction

1.1. The EEG alpha rhythm and peak alpha frequency

One of the most prominent electroencephalographic (EEG) phenomena, and the first to be discovered, is the *alpha* rhythm, a bursting oscillation between 8 and 13 Hz. The spectral distribution of the alpha rhythm usually resembles a bell-shaped curve with an average peak of 10–11 Hz in healthy adults. This peak is slower (in Hz) in children and the elderly, but also varies among individuals (Klimesch, 1997; Posthuma et al., 2001).

Different methods have been used to quantify the variation of spectral distribution within the alpha range. Peak alpha frequency (PAF) measures the discrete frequency with the highest magnitude within the alpha range. Individual alpha frequency (IAF) measures the center of gravity, rather than peak, within the boundaries of alpha frequency for each individual, and has been used as a different, and possibly more accurate measure of spectral distribution than PAF (Klimesch, 1997). PAF (or IAF) is an EEG measure that reflects the wave frequency of the maximum (or center of gravity for IAF) magnitude within the alpha band (8–13 Hz), and is measured in Hz. Therefore, reference to 'lower' or 'higher' PAF should be understood as differences in frequency (same for 'increases' or 'decreases'). PAF is not a direct measure of magnitude, and should not be compared to EEG alpha magnitude measures. Higher (faster) PAF means that there is greater magnitude at the higher part of the alpha spectrum, and vice versa. An alternative way to observe spectral alpha distribution is to divide the alpha spectrum into two

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frequency bands above and below the average peak of 10–11 Hz, for example 8–10 Hz (low alpha) and 10–12 Hz (high alpha), and then observe the differential magnitude of these two sub-bands (e.g. Pfurtscheller, 1989; Doppelmayr et al., 1998). This is based on the assumption that alpha rhythms are generated by at least two independent neuronal populations, one oscillating below 10 Hz and another above 10 Hz.

1.2. PAF and cognitive performance in healthy individuals

Several studies have shown PAF to reflect performance in various cognitive functions, including attention, arousal, working memory, long-term memory, and reading. It has been suggested that PAF variations within individuals reflect attentional demands and/or arousal (Klimesch et al., 1990). Osaka (1984) reported that PAF showed lateralized increases according to the type of cognitive task in 10 healthy young adults. PAF increased more over the right hemisphere during a visuospatial task and more over the left hemisphere during an arithmetic task. In a different study, Osaka et al. (1999), found PAF to increase during an auditory working memory task as compared to a control task. Klimesch et al. (1990) reported that IAF during a memory task was correlated with performance in a group of healthy young individuals and suggested that IAF may be an index of memory ability. In another experiment, these researchers found that individuals with lower memory performance decreased their IAF during increasing memory demands, whereas individuals with higher memory performance held their PAF constant (and higher than that of the first group) during the same conditions (Klimesch et al., 1993). In a resting state immediately after reading, high alpha (10–12 Hz) has been found to increase in magnitude, relatively to reading, with no changes in low alpha (8–10 Hz), suggesting a post-task increase in either PAF or IAF (Angelakis and Lubar, 2002).

PAF has been also related to developmental differences in cognitive performance. A recent study found higher PAF in children with higher reading performance as compared to age-matched controls (Suldo et al., 2001). This study also found that PAF of children with higher reading performance was at the same level as older children of equal reading performance, and thus interpreted PAF as a maturational index of the brain. In a study with 120 individuals from 46 to 80 years of age, Li et al. (1996) found PAF to decrease with increasing age, and to be correlated with speed and performance in a number of cognitive tasks. In a study with 101 healthy males aged 20–45, Anokhin and Vogel (1996) found PAF to be positively correlated with verbal and non-verbal cognitive abilities assessed by Amthauer's Intelligence Structure Test and Raven's Standard Progressive Matrices, though PAF was not correlated with general (*g* factor), spatial, and arithmetic abilities. In a large twins study with 688 participants, Posthuma et al. (2001), found PAF and intelligence to be highly heritable, although PAF did not correlate with intelligence. However, their intelligence scale used raw scores that were not standardized for age.

1.3. PAF and cerebral blood flow in psychiatric and neurological diagnoses

Patients with Alzheimer's disease (AD) have been found to have reduced PAF or IAF when compared to age-matched controls. Klimesch et al. (1990) found that a group of 18 AD patients had 1.46 Hz lower IAF than that of an age-reference control group of 20 (mean IAF for the two groups: 7.58 and 9.04 Hz, respectively). Moreover, they found a within-group difference in IAF associated with memory performance. Both AD patients and controls with better memory performance on the Wechsler Memory Scale had higher IAF than those with worse memory performance, within groups. However, while the AD group showed significant correlations between IAF and memory performance, the control group did not. The authors did not clarify whether the IAF was calculated on an EEG recorded on the same or on a different day as the memory test.

Passero et al. (1995) also found reduced PAF in AD patients when compared to age-matched controls, mostly over temporal and parietal areas. These investigators also measured cerebral blood flow (CBF) and also found it to be lower in the temporal and parietal areas of AD patients. However, contrary to Klimesch and colleagues' findings, PAF was not significantly correlated with cognitive impairment, although CBF was. This may have been due to the difference between IAF and PAF, the former possibly being a more sensitive measure.

Other psychiatric syndromes that involve decreased PAF include schizophrenia, chronic fatigue syndrome, and hemispheric stroke. Patients with schizophrenia showed decreased PAF both before and following treatment with aripiprazole (Canive et al., 1998). In the case of chronic fatigue syndrome, PAF was negatively correlated with total fatigue and 'today fatigue' reports (Billiot et al., 1997). Hemispheric stroke patients showed PAF decreases over the affected hemisphere within 48 h of their acute episode, with PAF recovering after 2–4 weeks (Juhasz et al., 1997). Interestingly, children with mental retardation showed increases in PAF after 6 months of pineal hormone treatment (Psatta et al., 1991).

1.4. PAF, emotional states, and arousal

PAF has been shown to reflect emotional and/or autonomic states. Kostyunina and Kulikov (1995; Kostyunina, 1998) found PAF increases over right centro-temporal (C4, T4), left frontal (F3), and occipital (O1) locations during mental reproduction of joy and anger, and PAF decreases during fear and sorrow compared to a neutral baseline. In a different study, Tiffin et al. (1995) found that

people with poorer sleep and higher anxiety had higher PAF than controls.

Single doses of nicotine and caffeine have been shown to increase PAF. A single cigarette was found to increase PAF at the fourth puff (Knott, 1988). Intravenous administration of nicotine in regular smokers who abstained from smoking for 12 h produced a linear, dose-related increase of PAF due to increase in high alpha power with no changes in low alpha (Lindgren et al., 1999). Caffeine has also been associated with increases in PAF accompanied by a decrease in alpha power at occipital sites, in both patients with panic disorder and healthy controls (Newman et al., 1992).

1.5. Piracetam, CBF, and PAF

Piracetam is a drug that belongs to the nootropic class. Nootropics are defined as mind enhancing drugs that "exert the greatest cognition-enhancing effects under conditions of neural impairment, and … should generally not produce the kinds of behavioral effects associated with classic psychotropic drugs" (Feldman et al., 1997, p. 902). Piracetam appears to alter acetylcholine and norepinephrine levels, and to alter cellular brain metabolism by increasing the levels of brain adenosine triphosphate (Conners and Sparrow, 1999). Piracetam has been found to improve reading performance of healthy and dyslexic children and adults (for reviews, see Wilsher, 1994; Conners and Sparrow, 1999), as well as to improve the symptoms of 10–30% of patients with Alzheimer's disease (Pierlovisi et al., 1991; Feldman et al., 1997, p. 903).

A study with 10 healthy elderly volunteers in their 60's found that acute piracetam administration increased PAF (Saletu et al., 1984). Consequent studies with healthy young volunteers and geriatric patients showed single doses of piracetam to increase high alpha (above 9.5 Hz) magnitude (Kinoshita, 1990), and to prevent changes in the EEG caused by hypoxia without piracetam in healthy volunteers (Saletu et al., 1995).

Some studies have shown a positive relationship between PAF, CBF, and cerebral oxygenation. In two studies, after indomethacin administration (a substance that decreases CBF), a decrease in CBF velocity was accompanied by a 0.3–0.5 Hz slowing of PAF and increases in reaction time in a memory test in healthy young adults (Hemler et al., 1990; Kraaier et al., 1992), whereas in another study PAF decreased after hypoxia induced by hemoglobin oxygen (Van der Worp et al., 1991).

1.6. General conclusions about PAF

Summarizing the literature reviewed above, PAF (or IAF) seems to be an index of cognitive capacity (hereafter called *cognitive preparedness*), related to both traits and states of brain function. Regarding its capacity to reflect traits, it has been widely shown that PAF differs between healthy and clinical individuals, with the latter consistently having reduced PAF compared to healthy controls. When tested, clinical individuals are shown to score lower in cognitive performance than matched healthy controls (Passero et al., 1995; Klimesch et al., 1990), suggesting reduced cognitive preparedness. Moreover, PAF has been shown to reflect maturational traits of reading performance in healthy children (Suldo et al., 2001). Although PAF did not reflect general intelligence in two large studies (Anokhin and Vogel, 1996; Posthuma et al., 2001), it has been found to be positively correlated with verbal and non-verbal abilities (Anokhin and Vogel, 1996), as well as with memory and speed of processing in healthy individuals (Klimesch et al., 1990; Li et al., 1996).

Regarding its capacity to reflect states within individuals, PAF (or IAF, or high versus low alpha) has been found to be affected by cognitive tasks (Osaka et al., 1999; Klimesch et al., 1993; Angelakis and Lubar, 2002), mental reproduction of emotional states (Kostyunina and Kulikov, 1995; Kostyunina, 1998), and acute administration of various substances (Knott, 1988; Lindgren et al., 1999; Saletu et al., 1984).

Although there is no evidence that PAF specifically reflects different levels of CBF and/or cerebral oxygenation, there is some data to suggest an at least weak relation between the two. Such a relation, if further supported, would explain both trait and state differences in PAF between and within individuals. In the case of brain pathology, lower PAF may reflect larger degrees of permanent or long-term (i.e. trait) deficits in CBF. In the case of within-individual changes, PAF has been shown to reflect state differences in CBF and/or cerebral oxygenation due to administration of various substances. Likewise, PAF changes within individuals during cognitive manipulation may fit a CBF model, too, suggesting that performance differences are due to changes in brain metabolism. However, until further research supports this hypothesis no conclusive argument can be made on this matter.

1.7. Rationale

Regardless of its physiological substrate (i.e. whether PAF is a direct result or cause of changes in brain metabolism, or just an indirect correlate of it), the present study suggests that PAF measures cognitive preparedness. In the present context, cognitive preparedness refers to a brain trait or state that sets the stage for optimal cognitive performance on a relatively complex task, rather than to a time-locked anticipation of a movement or stimulus as is the case with contingent negative variation, readiness potential, or stimulus-preceding negativity of the event-related potential (Brunia and van Broxtel, 2001). Moreover, cognitive preparedness refers to the brain's capacity for higher-level cognitive functions, and is not a synonym to alertness or arousal that modulate lower-level functions and are inherently autonomic (sympathetic) in nature.

Based on the preceding literature, the following hypotheses were posed. First, PAF is suppressed (in Hz, i.e. slowed) by any *trait* of brain pathology. Second, PAF fluctuates within healthy individuals according to brain *state*, predicting consequent cognitive performance, and being affected by cognitive tasks. In order to test these hypotheses, two experiments were performed. Experiment 1 involved individuals with traumatic brain injury (TBI) in order to investigate the sensitivity of PAF to trait differences, and experiment 2 involved repeated EEG measures from healthy individuals in order to investigate the sensitivity of PAF to state differences.

2. Experiment 1

Most closed TBI patients share attention and concentration deficits, reduced speed of processing, and mental fatigue, which suggest reduced cognitive preparedness. Therefore, we expected patients with closed TBI to exhibit reduced PAF when compared with matched healthy controls. To investigate further the condition under which PAF reflects brain pathology, EEG was recorded and analyzed during 4 different conditions: eyes closed baseline (ECB), eyes-open baseline (EOB), one cognitive task (TASK), and post-task rest (PTR).

2.1. Method

2.1.1. Participants

Ten individuals (5 males and 5 females) with closed TBI due to automobile accidents that caused them acquired attention deficits, and 12 controls matched for age, sex, and education were recruited. The TBI patients were referred either by the Association of Brain Injured patients of Knoxville or from the Disability Service of the University of Tennessee (UT), whereas the controls were recruited from UT's undergraduate population. Ages ranged from 20 to 45. heir accidents occurred from one and a half to 20 years before the study. Participants were not engaged in any therapy nor taking any medication during the period of the study. Written consent was obtained from all participants, according to the Internal Review Board of the University of Tennessee.

2.1.2. Materials

Six cognitive tasks during which EEG was recorded were taken from the Captain's Log software program (Braintrain, Richmond, VA) that measures and trains different types of attention. Each task lasts for 3 min, and all together lasted for approximately 20 min. In the first task the participant listened to two patterns of rhythm and had to chose whether they were the same or different by clicking on the respective ('same' or 'different') box. In the second task the person chose between two patterns of melody. In the third task the person had to click the mouse each time two of 3boxes (the center one with each of the lateral ones) matched in color. In the fourth task the person clicked the mouse each time the box matched in color with the rectangular border line. In the fifth task the person saw a series of numbers/letters appearing in sequence and had to click the mouse each time he/she saw the number or letter designated as the target at the beginning. In the sixth task the person clicked the mouse each time he/she saw a box of particular size, but not when boxes of other sizes appeared.

2.1.3. Apparatus

EEG was recorded at 128 samples per second with a Lexicor Neurosearch 24 EEG recorder using an electrode cap with 19 electrodes placed according to the 10/20 system. The recording montage was referential to physically linked ears, and the amplifiers were bandpass filtered at 0.5–32 Hz The Captain's Log tasks were presented on a computer screen 50 cm from the participants' eyes. The participants responded using the computer's mouse. Speakers with adjustable volume were also used.

2.1.4. Procedure

All data were collected in a quiet windowless laboratory room with fluorescent lighting and no other persons present except for the participant and the experimenter. Participants were fitted with the electrode cap, and impedance at all channels was reduced to below 5 kOhms. EEG was recorded during an eyes-open (EOB) and an eyes-closed (ECB) resting baseline, during the 6 Captain's Log cognitive tasks, and during a post-task eyes-open resting (PTR) condition. During all recordings, participants were seated on an armchair. During the resting conditions, participants were asked to stay still and relaxed, and during the eyes-open recordings they were asked to group and minimize their eyeblinks as much as possible. During the task recordings, participants were instructed to perform to their best ability.

2.1.5. Data analysis

All raw EEG data were visually inspected and all epochs with artifacts due to muscle movement (including eye and eyelid movements) or tension were removed from further analysis. Epochs with slow-wave activity (below 4 Hz) seen primarily in FP1 and FP2 were rejected, being interpreted as vertical eye or eyelid movements; and epochs with converging slow potentials (below 1 Hz) between F7 and F8 were rejected, being interpreted as lateral eye movements. Moreover, epochs with intense activity above 40 Hz primarily in peripheral channels were rejected, being interpreted as muscle tension. Visual inspection of epoch-by-epoch spectra revealed clear peaks within the alpha band for almost all participants, conditions, and electrodes. Average PAF for each recording was calculated and reported using the EEG Workstation 2.0 software (NovatechEEG, Inc., Knoxville, TN). PAF was defined as the discrete frequency that has the highest magnitude within the range of 8–13 Hz in each recording for each of the 19 channels. All EEG recordings

were down-sampled to 64 samples per second (by averaging every two samples) with an epoch length of 256 samples, to yield frequency resolution of 0.25 Hz (Congedo et al., 2002). Although such a high resolution may risk missing information from close by frequencies, it was selected to maximize frequency accuracy. Depending on artifact rejection, the final files averaged ranged from 30 to 100 s in length. Participants with TBI tended to have more artifacts, especially during tasks, so their final files ended up shorter (closer to 30 s for the TASK condition).

Since most data were not found to be normally distributed, non-parametric Mann–Whitney *U* tests were used to compare PAF between TBI individuals and healthy controls during ECB, EOB, TASK, and PTR. Because of the large number of statistical analyzes, the alpha-level for statistical significance was adjusted to avoid type-I error. Since each hypothesis involved more than one condition, the alpha-level was adjusted using a sequential Bonnferoni adjustment for multiple comparisons for the number of conditions involved. This technique increases the power of the standard Bonnferoni adjustment, reducing the probability of type-II error (Rice, 1988; Miller, 1981; Holm, 1979). It should be noted that this method for correcting the alpha-level for multiple comparisons is quite conservative.

2.2. Results and discussion

Significant results are reported in Table 1 and illustrated in Fig. 1. Although statistically significant *P*-values corrected for multiple comparisons are shown in bold-typed numbers, all reported *P*-values are non-adjusted, for comparison. Average PAF across 19 electrodes was significantly lower in individuals with TBI as compared to matched healthy controls during PTR only (TBI: 8.83, controls: 9.81; Mann–Whitney z: 2.24, $P < .01$).

The results of this experiment are consistent with previous research showing a stable direction of PAF difference between normal brain function and pathology. As expected, individuals with TBI had lower PAF than healthy controls, as is the case with other neurological or psychiatric syndromes, including stroke, dementia, and schizophrenia (see Section 1). Although PTR was the only condition to show statistically significant differences between TBI and control participants after correcting for multiple comparisons, the other 3 conditions (ECB, EOB, and TASK) were marginally significant (i.e. at the 0.1 level). Interestingly, these differences between individuals with TBI and non-clinical controls were most prominent during the post-task resting condition, analogous to other physiological measures that assess pathology during post-stress rest (e.g. heart rate, blood pressure).

3. Experiment 2

Although experiment 1 extended previous findings (see Section 1) supporting the hypothesis that PAF reflects a trait of cognitive preparedness, it did not directly test whether

Table 1
PAF for TBI vs. controls

	ECB		EOB		TASK		PTR	
	M-W z	*P*-value	M-W z	*P*-value	M-W z	*P*-value	M-W z	P-value
FP1	−0.96	ns	−1.53	ns	−1.2	ns	−1.57	ns
FP2	−2.29	0.01	−1.81	0.04	−0.1	ns	−1.51	ns
F7	−0.46	ns	−1.29	ns	−0.57	ns	−2.49	**0.006**
F3	−1.59	ns	−1.28	ns	−1.04	ns	−1.93	**0.03**
FZ	−2.2	0.015	−1.33	ns	−1.66	ns	−1.81	**0.04**
F4	−1.83	0.035	−0.67	ns	−0.8	ns	−1.32	ns
F8	−2.4	0.008	−0.53	ns	−0.27	ns	−1.24	ns
T3	−1.23	ns	−1.09	ns	−0.13	ns	−1.86	**0.035**
C3	−1.65	ns	−1.53	ns	−1.87	0.035	−2.66	**0.004**
CZ	−1.69	0.047	−0.27	ns	−1.73	0.047	−3.07	**0.001**
C4	−1.69	0.047	−0.47	ns	−1.75	0.04	−2.8	**0.002**
T4	−0.66	ns	−0.1	ns	−0.46	ns	−1.07	ns
T5	−1.5	ns	−2.19	0.015	−2.52	**0.006**	−1.33	ns
P3	−1.93	0.03	−2.32	0.01	−1.29	ns	−2.36	**0.008**
PZ	−2.75	**0.002**	−2.49	0.006	−2.55	**0.004**	−2.92	**0.001**
P4	−1.73	0.047	−2	0.025	−2.08	0.018	−2.43	**0.007**
T6	−1.39	ns	−0.13	ns	−0.4	ns	−1.56	ns
O1	−0.8	ns	−0.93	ns	−0.87	ns	−0.93	ns
O2	−1.33	ns	−1.52	ns	−2.18	0.015	−2.19	**0.015**
MEAN	−1.58	0.061	−1.52	0.07	−1.35	0.09	−2.24	**0.0102**

Z-scores (Mann–Whitney) and *P*-values (1-tailed) for the differences in peak alpha frequency (PAF) between participants with traumatic brain injury (TBI) ($n = 10$) and non-clinical controls ($n = 12$) during eyes-closed baseline (ECB), eyes-open baseline (EOB), cognitive task (TASK), and post-task eyes-open rest (PTR). Bold typed *P*-values are significant after correcting for multiple comparisons. In all significant differences, TBI participants had lower PAF than controls. Note that PTR shows the greatest effect.

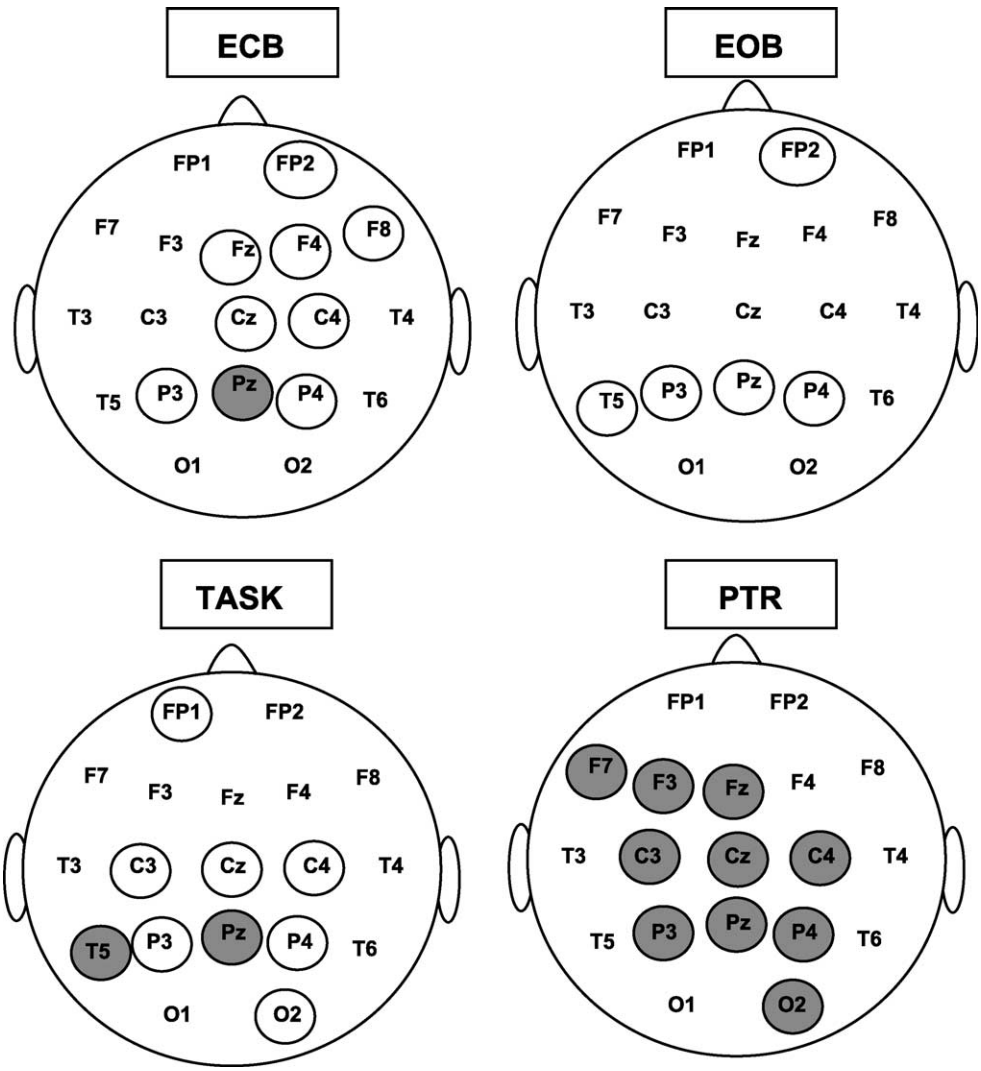

Fig. 1. Peak alpha frequency (PAF, in Hz) differences between individuals with traumatic brain injury (TBI) and non-clinical controls for eyes-closed baseline (ECB), eyes-open baseline (EOB), task (TASK), and post-task eyes-open baseline (PTR). Gray circles: significantly higher PAF for non-clinical controls corrected for multiple comparisons. White circles: significance not corrected. PTR shows the greatest difference between the two groups.

PAF also reflects a *state* of cognitive preparedness within individuals. To test the latter, EEG was recorded from a group of 19 non-clinical individuals before and after a working memory task. The procedure was repeated on two different days to make within-individual comparisons possible. Three hypotheses were tested. First, pre-task PAF was expected to correlate higher with working memory performance of the same day than of a different day, showing that, within individuals, day-to-day variations in PAF reflect variations in cognitive preparedness. Second, individuals who had lower PAF before the task were expected to raise their PAF as a result of performing the task, thereby correcting their initial unpreparedness. Individuals who had higher PAF before the task were supposed to be already prepared and, therefore, were expected to raise their PAF less, if not at all. Finally, participants were expected to have higher pre-task PAF at day 2 compared to day 1 in order to prepare for the task they previously performed on day 1, thereby allowing them to perform better on the task.

3.1. Method

3.1.1. Participants

Nineteen undergraduate psychology college students participated, 6 male and 13 female, ranging in age from 19 to 23. ll volunteered for extra course credit. Written consent was obtained from all participants, according to the Internal Review Board of the University of Tennessee.

3.1.2. Apparatus and materials

EEG was recorded at 128 samples per second with a Lexicor Neurosearch 24 EEG recorder, using an electrode cap with electrodes placed according to the 10/20 system. The recording montage was referential to physically linked ears, and the amplifiers were bandpass filtered at 0.5–32 Hz.

The Digit Span subtest of the WAIS-R was administered as a working memory task. In this subtest, the experimenter spoke sets of digits of increasing size (range: 2–9) at a pace of one digit per second, and the participants had to repeat them verbally in the correct order. In a second phase, participants had to repeat other number sets in the reverse order. PAF was calculated and reported using the Workstation software (NovatechEEG, Inc., Knoxville, TN).

3.1.3. Procedure

Participation involved two sessions on different days, during which identical procedures took place. First, an electrode cap was placed on each participant and impedances were adjusted to below 5 kOhms. EEG was recorded during an eyes-closed baseline (ECB) immediately after which the Digit Span test was administered. EEG was then immediately recorded again (i.e. less than 1 min after the Digit Span completion) during a post-task eyes-closed resting condition (PTR).

3.1.4. Data analysis

Data analyzes were identical to that of experiment 1. Visual inspection of epoch-by-epoch spectra revealed clear peaks within the alpha band for almost all participants, conditions, and electrodes. Correlations between PAF before and after Digit Span of each day with Digit Span performance of each day were tested for significance using Spearman's non-parametric test. To test for PAF differences before and after Digit Span, participants were divided into two groups (independently for each day), one whose PAF at initial resting baseline (ECB) was higher than the group's median, and one whose PAF was lower. Then, differences between PAF before and after Digit Span were tested, using Mann–Whitney *U* tests. Finally, baseline PAF and Digit Span differences between days 1 and 2 were tested, using Mann–Whitney *U* tests.

3.2. Results and discussion

Average PAF across 19 electrodes recorded during ECB at day 1 was significantly correlated with Digit Span performance on the same day ($r = 0.48$, $P < 0.05$) but not with Digit Span performance on day 2 ($r = 0.32$, $P = 0.10$). Likewise, average PAF recorded during ECB at day 2 was significantly correlated with Digit Span performance of the same day ($r = 0.42$, $P < 0.05$) but not with Digit Span performance of day 1 ($r = 0.27$, $P = 0.14$) (see Table 2 and Fig. 2). Although PAF recorded before Digit Span (ECB) was correlated with Digit Span performance, PAF recorded after Digit Span (PTR) was not correlated with Digit Span performance.

PAF across 19 electrodes was significantly increased after Digit Span (i.e. in the PTR) for those participants who had it below the group median before the task on both days (day 1: 8.47/9.55 Hz, $P < 0.01$; day 2: 8.85/9.29 Hz, $P < 0.01$; days 1 and 2 collapsed: 8.66/9.42, $P < 0.001$), whereas it did not increase significantly for those who had it above the group median (day 1: 9.95/10.30 Hz, $P = 0.107$; day 2: 10.33/10.40 Hz, $P = 0.107$; days 1 and 2 collapsed: 10.14/10.35, $P = 0.054$) (see Table 3 and Fig. 3).

Table 2
Correlations of PAF with Digit Span

	PAF day 1				PAF day 2			
	DS day 1		DS day 2		DS day 1		DS day 2	
	Rho	*P*-value	Rho	*P*-value	Rho	*P*-value	Rho	*P*-value
FP1	0.31	ns	0.10	ns	0.22	ns	0.43	**0.036**
FP2	0.43	**0.038**	0.23	ns	0.28	ns	0.65	**0.002**
F7	0.48	**0.023**	0.15	ns	0.36	ns	0.28	ns
F3	0.46	**0.028**	0.15	ns	0.35	ns	0.49	**0.020**
FZ	0.41	**0.047**	0.21	ns	0.32	ns	0.44	**0.035**
F4	0.42	**0.040**	0.20	ns	0.21	ns	0.43	**0.038**
F8	0.33	ns	0.30	ns	0.20	ns	0.28	ns
T3	0.58	**0.006**	0.42	0.041	0.24	ns	0.28	ns
C3	0.36	ns	0.12	ns	0.28	ns	0.37	ns
CZ	0.22	ns	0.08	ns	0.27	ns	0.37	ns
C4	0.23	ns	0.02	ns	0.34	ns	0.31	ns
T4	0.42	**0.043**	0.51	0.015	0.21	ns	0.34	ns
T5	0.33	ns	0.31	ns	0.32	ns	0.31	ns
P3	0.25	ns	0.26	ns	0.30	ns	0.43	**0.038**
PZ	0.13	ns	0.00	ns	0.32	ns	0.39	ns
P4	0.36	ns	0.14	ns	0.20	ns	0.52	**0.014**
T6	0.37	ns	0.15	ns	0.33	ns	0.44	**0.034**
O1	0.12	ns	0.13	ns	0.31	ns	0.29	ns
O2	0.22	ns	0.15	ns	0.27	ns	0.26	ns
MEAN	0.48	**0.021**	0.32	0.096	0.27	0.136	0.42	**0.041**

Spearman's correlation coefficients (rho) and *P*-values for the association between peak alpha frequency (PAF) at days 1 and 2 with Digit Span (DS) at days 1 and 2. Bold faced *P*-values were significant after correcting for multiple comparisons. Note that significant correlations are only those between the same day PAF and DS.

Baseline (ECB) PAF (averaged across 19 electrodes) on day 2 was significantly higher by 0.4 Hz than on day 1 (day 1: 9.2, day 2: 9.6; $P < 0.05$), and Digit Span performance was higher on day 2 than on day 1 (day 1: 16.4, day 2: 18.3; $P < 0.05$) (see Fig. 4).

Experiment 2 showed PAF to be sensitive to brain states within individuals on different days. PAF significantly predicted cognitive performance on a working memory task that was performed immediately after EEG recording, whereas it did not predict performance within a few days (although it showed a non-significant trend). This finding is further validated by the similarity of correlations between the two days (same day correlations: day 1/1, $r = 0.48$; day 2/2, $r = 0.42$; different day correlations: day1/2, $r = 0.32$; day 2/1, $r = 0.27$). These results show that, in non-clinical individuals, pre-task PAF can be sensitive to a cognitive state as well as a trait in that it may reflect day-to-day, or even moment-to-moment, changes in cognitive preparedness within individuals. The fact that post-task resting PAF did not correlate significantly with cognitive performance supports the idea that PAF reflects preparedness. PAF at

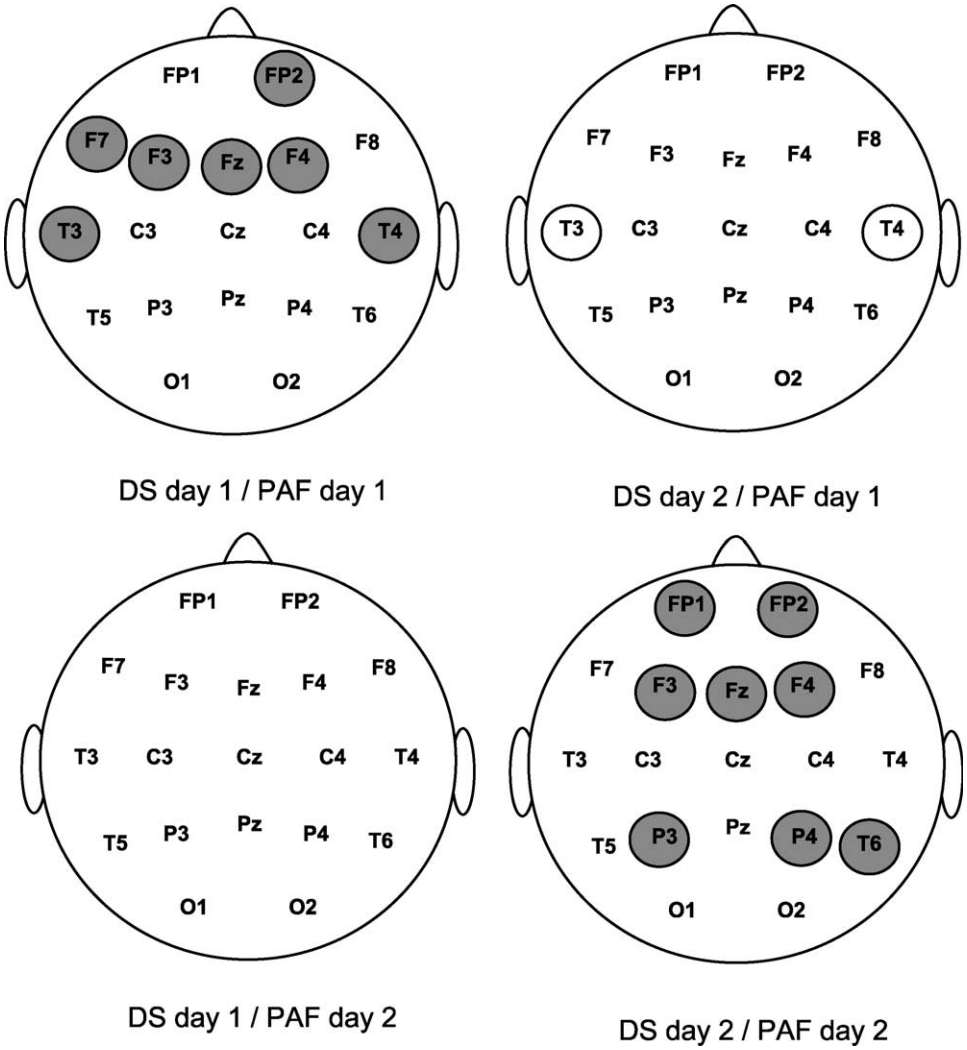

Fig. 2. Spearman's correlation coefficients between WAIS-Digit Span (DS) and peak alpha frequency (PAF) on two different days. Gray circles: significance corrected for multiple comparisons. White circles: significant not corrected. Only same day DS and PAF are significantly correlated. Notice frontal topography common at both days.

initial baseline indicated how prepared individuals were to perform the upcoming task, whereas PAF at post-task rest did not indicate how well individuals performed in a preceding task.

Furthermore, of particular interest is the topography of this preparedness (see Fig. 2), which in both days is observed mostly at frontal electrodes (i.e. over a cortical area that is known to specialize in working memory functions). This observation extends Osaka (1984) findings of lateralized PAF increases according to task type (see Section 1). Although the used EEG montage of physically-linked ears reference may misrepresent the actual topography of WM preparedness, the reliability of topographical distribution between the two days makes it of interest in it self. Future research may use alternative, bias-free, EEG montage to more validly localize such phenomena.

The fact that only (initially) low-PAF participants had significantly higher PAF after engaging in the task showed not only that PAF before a cognitive task predicted performance on the task but also that performing a cognitive task affected PAF. It was shown that individuals who were not fully prepared for the task, as indicated by their initial PAF, were forced by the task to correct their unpreparedness by increasing their PAF. This observation extends the findings of Osaka (1984) and Osaka et al. (1999) in which PAF increases were observed during performance of a task, and supports the hypothesized relationship between PAF and cognitive performance.

Finally, the results indicate that improved performance on Digit Span during the second trial (day 2) was accompanied by an analogous increase in pre-task PAF. These results suggest that part of the practice effect for improved performance during replication of a specific task is due to cognitive preparedness.

Table 3
Post-task PAF increases for low-PAF and high-PAF participants

	Day 1 high-PAF		Day 1 low-PAF		Day 2 high-PAF		Day 2 low-PAF	
	M-W z	*P*-value	M-W z	*P*-value	M-W z	*P*-value	M-W z	*P*-value
FP1	−0.42	ns	−2.39	**0.009**	−0.42	ns	−2.21	**0.014**
FP2	−0.35	ns	−2.16	**0.016**	−1.47	ns	−2.21	**0.014**
F7	−0.85	ns	−2.24	**0.012**	−0.83	ns	−2.39	**0.008**
F3	−0.84	ns	−1.72	**0.043**	−0.74	ns	−1.60	ns
FZ	−0.56	ns	−2.44	**0.007**	−0.74	ns	−1.60	ns
F4	−0.42	ns	−2.21	**0.014**	−1.19	ns	−1.83	**0.034**
F8	−0.56	ns	−2.32	**0.010**	−0.76	ns	−2.21	**0.014**
T3	−0.56	ns	−1.96	**0.025**	−0.41	ns	−1.20	ns
C3	−0.21	ns	−1.84	**0.033**	−1.13	ns	−1.29	ns
CZ	−0.77	ns	−2.25	**0.012**	−0.65	ns	−1.58	ns
C4	−0.59	ns	−1.60	ns	−1.38	ns	−0.74	ns
T4	−0.17	ns	−1.84	**0.033**	−1.37	ns	−0.74	ns
T5	−2.31	0.010	−2.67	**0.004**	−0.37	ns	−1.24	ns
P3	−2.50	0.006	−2.37	**0.009**	−0.27	ns	−1.84	**0.033**
PZ	−1.84	0.033	−2.26	**0.012**	−0.56	ns	−2.03	**0.021**
P4	−1.90	0.029	−2.20	**0.014**	−1.63	ns	−0.96	ns
T6	−1.84	0.033	−2.43	**0.008**	−0.96	ns	−0.32	ns
O1	−2.67	0.004	−2.27	**0.012**	−0.60	ns	−2.12	**0.017**
O2	−1.98	0.024	−2.55	**0.005**	−1.16	ns	−2.20	**0.014**
MEAN	−1.24	0.107	−2.55	**0.005**	−1.24	0.107	−2.43	**0.008**

Mann–Whitney z scores (M-W z) and *P*-values for peak alpha frequency (PAF) increases after Digit Span as compared with pre-task baseline, for participants with PAF higher than the group's median (high-PAF) and with lower than the group's median (low-PAF), for days 1 and 2. Bold-typed *P*-values are corrected for multiple comparisons. Note that participants with lower initial PAF showed the greatest effect.

4. General discussion

The present study supported the hypothesis that PAF can reflect traits and states of cognitive preparedness. In the case of individuals with TBI whose cognitive preparedness is chronically suppressed, depressed PAF reflects a trait. In non-clinical individuals whose cognitive preparedness varies over time, PAF also reflects their state. In experiment 2, PAF was shown to be affected by a preceding cognitive task. This finding may explain further other results from experiments 1 and 2. At initial rest, healthy individuals may show a random distribution of their PAF due to an undetermined state of cognitive preparedness, therefore not

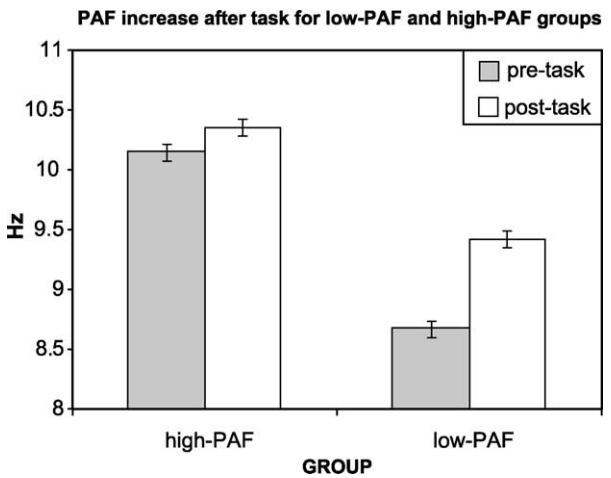

Fig. 3. Peak alpha frequency (PAF, in Hz) increases at post-task eyes-closed rest (PTR), as compared with pre-task eyes-closed baseline (ECB) for individuals whose PAF was higher than the sample median at ECB (high-PAF) and for individuals whose PAF was lower than the sample median at ECB (low-PAF). Both days are collapsed. Gray bars: ECB. White bars: PTR. Error bars show two standard errors of the mean.

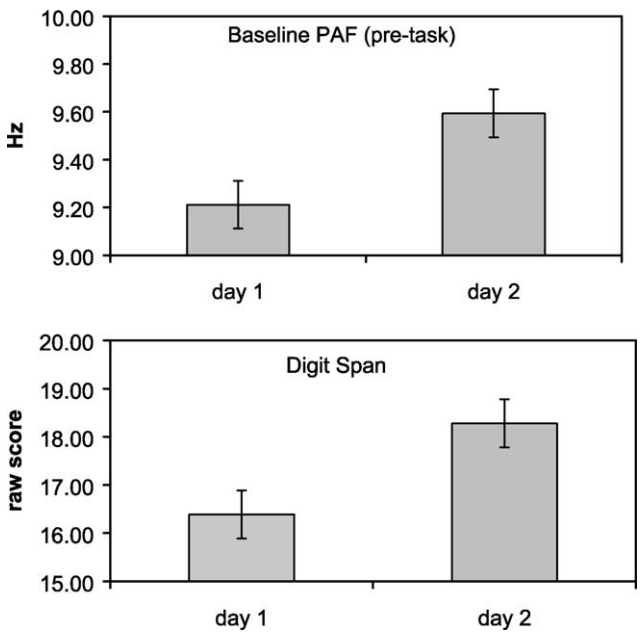

Fig. 4. Peak alpha frequency (PAF, in Hz) at pre-task eyes-closed baseline (ECB) and Digit Span (DS) for days 1 and 2. Both PAF and DS are significantly higher at day 2. Error bars show two standard errors of the mean.

showing a significant group difference relative to individuals with TBI. However, when a task forces them to correct their unpreparedness, then healthy individuals show their PAF potential, which clinical individuals are not able to attain. Related findings were reported by Klimesch (1997), who showed that groups of bad and good memory performers had greater IAF differences during a memory task than during an initial resting baseline.

We believe that PAF reflects cognitive preparedness, i.e. a capacity for higher-level cognitive functions, rather than lower-level functions such as arousal, for several reasons. Kostyunina and Kulikov (1995) (Kostyunina, 1998) found PAF increases during anger but PAF decreases during fear, although both states should induce high arousal due to sympathetic activation. Therefore, arousal itself is not enough to explain a positive correlation between PAF and task performance. Moreover, general arousal does not fit the maturational increases of PAF from childhood to adulthood, unless one is ready to accept that adults are more aroused than children. Similarly, the maturational model for PAF proposed by Suldo et al. (2001) regarding reading performance in children suggests a relationship of PAF with higher cognition. If younger children read as well as older ones and have similar PAF levels to them as opposed to same age children who read less well and have lower PAF, it seems more realistic to attribute PAF and reading performance to better development and function of cortical areas responsible for higher cognitive functions, rather than to brain-stem induced arousal.

One implication of this understanding of PAF and cognitive preparedness is that it can be used to improve existing neurodiagnostic tools. The results of this study and those of other studies (e.g. Angelakis and Lubar, 2002) show that the alpha frequency band is particularly responsive to a cognitive task as reflected in recordings during rest immediately after the task. One possibility is that future versions of qEEG normative databases (Thatcher, 1999) include PAF and post-task resting conditions. The results of experiment 2 would seem to challenge this suggestion, since PAF is shown here to vary within individuals according to temporary states, thereby losing its validity as a reliable trait measure. However, the finding of variable PAF was based on an initial resting baseline rather than on a post-task resting condition, as was the case in experiment 1. Instead, PAF during a post-task resting condition can be used to assess traits of brain function, suggesting that the electroencephalogram resembles other indices like heart rate and blood pressure that best reflect traits of the underlying physiology during post-stress resting conditions. Furthermore, the frontal topographical distribution of correlational results from experiment 2 suggests that further research might investigate whether PAF over different cortical areas can predict cognitive potential for functions subserved by these areas.

The finding that PAF can be increased by performing a cognitive task provides neurophysiological evidence that cognitive exercise itself may be an alternative to substance administration for boosting cognitive preparedness. Alternatively, individuals could be trained to voluntarily control their PAF – and cognitive preparedness – with the help of EEG biofeedback (*neurofeedback*), a technique that has been used successfully to self-regulate EEG rhythms (Lubar and Lubar, 1999; Sterman, 2000).

In conclusion, the present study supported the sensitivity of PAF to detect both trait and state differences in cognitive preparedness across groups and within individuals. Trait differences were found to be better detected during post-task resting conditions whereas state differences were more prominent during an initial resting baseline. In addition to predicting cognitive preparedness, PAF was found to be affected by cognitive tasks, showing a two-way relationship between PAF and cognitive preparedness.

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