Clinical Neurophysiology 115 (2004) 131–139

www.elsevier.com/locate/clinph

EEG Biofeedback of low beta band components: frequency-specific effects on variables of attention and event-related brain potentials

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Accepted 8 March 2003

Abstract

Objective: To test a common assumption underlying the clinical use of electroencephalographic (EEG) biofeedback training (neurofeedback), that the modulation of discreet frequency bands is associated with frequency-specific effects. Specifically, the proposal was assessed that enhancement of the low beta components sensorimotor rhythm (SMR: 12–15 Hz) and beta1 (15–18 Hz) affect different aspects of attentional processing.

Methods: Subjects ($n = 25$) were randomly allocated to training with either an SMR or beta1 protocol, or to a non-neurofeedback control group. Subjects were assessed prior and subsequent to the training process on two tests of sustained attention. The neurofeedback participants were also assessed on target P300 event-related potential (ERP) amplitudes in a traditional auditory oddball paradigm.

Results: Protocol-specific effects were obtained in that SMR training was associated with increased perceptual sensitivity 'd prime' (d'), and reduced omission errors and reaction time variability. Beta1 training was associated with faster reaction times and increased target P300 amplitudes, whereas no changes were evident in the control group.

Conclusions: Neurofeedback training of SMR and beta1 band components led to significant and protocol-specific effects in healthy subjects. The data can be interpreted as indicating a general attention-enhancing effect of SMR training, and an arousal-enhancing effect of beta1 training.

Keywords: EEG biofeedback; Neurofeedback; Beta1 activity; Sensorimotor rhythm; Attention; P300

1. Introduction

Various parameters of the human electroencephalogram (EEG) can be brought under operant control by means of a training process involving the real-time display of ongoing changes in the EEG via an EEG biofeedback loop. The principal feasibility of learned self-regulation has been demonstrated for evoked potentials (EPs) (Rosenfeld et al., 1969), event-related potentials (ERPs) (Birbaumer et al., 1981), slow cortical potentials (SCPs) (Birbaumer, 1984; Hardman et al., 1997), and EEG frequency components (Kamiya, 1968), with the latter two being of particular interest due to their reported intrinsic clinical benefits. For instance, the operant modulation of positive and negative SCP shifts has been found to facilitate control over epileptic seizures (Birbaumer et al., 1991; Rockstroh et al., 1993), and has been employed to spectacular effect as a brain-computer communication device for totally paralysed patients (Birbaumer et al., 1999).

Arguably the best established clinical application of AC EEG frequency component training consists of the treatment of epilepsy through learned self-regulation of the 12–15 Hz sensorimotor rhythm (SMR) recorded from central scalp regions over sensorimotor cortex (for review see Sterman, 2000). While the term SMR originally referred exclusively to the occurrence of phasic EEG spindles, the term will here be used to cover both phasic and tonic 12–15 Hz activity over the sensorimotor strip. Trained enhancement of the SMR has been demonstrated to result in increased seizure thresholds in response to exposure to eliptogenic agents in monkeys (Sterman et al., 1978), and to lead to reduced seizure incidence in human epileptics (Sterman and Friar, 1972; Sterman et al., 1974; Sterman and MacDonald, 1978; Sterman and Shouse, 1980). SMR activity over sensorimotor cortex is probably generated

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1388-2457/$30.00
doi:10.1016/S1388-2457(03)00353-5

through thalamocortical interactions during burst firing activity in ventrobasal thalamic relay nuclei (Harper and Sterman, 1972), associated with the suppression of somatosensory afferent gating (Howe and Sterman, 1972). In consideration of its effects on cortical excitability in epilepsy, it has been concluded that SMR neurofeedback training appears to facilitate thalamic inhibitory mechanisms (Sterman, 1996).

From the apparent impact of SMR training on sensorimotor excitation, Lubar and colleagues have extrapolated the application of SMR training to the treatment of hyperactivity disorder (HD) (Lubar and Shouse, 1976; Shouse and Lubar, 1979). Subsequently, the operant enhancement of SMR, trained concurrently with suppression of slower theta (4–8 Hz) components, has often been complemented with or supplanted by training of higher beta band components, such as beta1 (15–18 Hz) in the treatment of attention deficit disorder (ADD) and attention deficit hyperactivity disorder (ADHD) (Lubar and Lubar, 1984; Lubar et al., 1995; Linden et al., 1996). A stated assumption in the use of the SMR and beta1 protocols is that the former addresses problems of hyperactivity and impulse control, while the latter is held to alleviate symptoms of inattentiveness (Lubar and Lubar, 1984; Lubar, 1991; Othmer et al., 1999). However, while recent controlled studies of beta band neurofeedback have produced promising results in the treatment of ADHD (Rossiter and LaVaque, 1995; Fuchs et al., 2003; Monastra et al., 2002), no protocol-specific differential effects between SMR and beta1 training have been demonstrated.

The controlled assessment of specific cognitive and electrocortical effects from training self-regulation of these frequency bands would be of great value in order to provide an empirical rationale for their clinical application to specific symptoms, and for furthering an understanding of their etiology. In a recent study, Egner and Gruzelier (2001) have supplied the first systematic evidence for protocol-specific effects of beta band protocols. They showed that, in a group of healthy volunteers who were trained on both the SMR and beta1 protocols, significant changes in attention performance and event-related potentials (ERPs) could be predicted on the basis of the subjects' individual neurofeedback learning rates, and that learning rates on the two protocols showed differential relations with dependent measure changes. Specifically, it was found that SMR learning was associated with commission error reduction, while beta1 learning displayed the opposite association. Learning on both protocols was positively correlated with increased target P300 ERPs in an auditory oddball task, which indexes integration of task-relevant stimuli in working memory (Donchin and Coles, 1988). These findings could be interpreted as supporting SMR's role in improving impulse control, and beta1 training as increasing impulsive response tendencies, while both protocols may be associated with improved integration of relevant environmental stimuli.

However, conclusions drawn from this study were limited by the fact that subjects were trained on both protocols, resulting in correlational analyses for distinguishing between the impact of the two protocols. Furthermore, possible practice effects on the attention task were not controlled for, and the attention test employed did not provoke a substantial amount of omission errors, preventing the authors drawing any conclusions regarding the beta band protocols' effects on inattentiveness. The current experiment was devised to test for protocol-specific effects of SMR and beta1 neurofeedback by directly contrasting attention performance and target P300 ERPs between the protocols in an independent-groups design. On the basis of the previous findings (Egner and Gruzelier, 2001), it was hypothesized that SMR training would result in reduced commission errors and increased d' scores and P300 amplitudes, while beta1 training would result in increased commission error incidence and P300 amplitudes.

In order to control for possible practice and motivational factors affecting the attention measures, a control group engaging in a training regime of equal duration and experimenter-contact was included in the study. As the investigation was carried out as part of a large-scale project at a music conservatoire (Royal College of Music, London), the control group was involved in an 'Alexander technique' training program. The Alexander technique refers to a system of kinaesthetic education aimed at avoiding excessive postural tension, and constitutes the most widely practised behavioural training in professional orchestral musicians (Watson and Valentine, 1987). This intervention was not expected to affect attention performance.

2. Methods

2.1. Subjects

Participants were 25 students (7 males, 18 females; mean age 21.7 years, SD 2.24) from the Royal College of Music (London). The subjects volunteered for participation, gave their informed consent, and the investigation received ethical approval from the Riverside Research Ethics Committee (ref. RREC 2224). The subjects did not receive monetary reward for participation. Participants were screened with a health-related questionnaire in order to exclude subjects with a history of mental or neurological illness, or currently receiving psychoactive medication. No volunteers had to be excluded.

2.2. Design

The subjects were randomly allocated to one of 3 training groups: beta1 neurofeedback ($n = 8$), SMR neurofeedback ($n = 9$), or a group that engaged in Alexander technique training ($n = 8$). For the purpose of testing hypotheses concerning protocol-specific effects on target P300 ERPs,

the SMR, and beta1 groups underwent EEG recordings before and after their NFT regime. In order to test the hypotheses concerning the protocol-specific effects on behavioural attention measures, the neurofeedback groups and the Alexander technique training group were assessed on attention tests before and after their respective training regimes.

2.3. Sustained attention measures

Sustained attention was assessed on two continuous performance tests within two weeks before and after the training interventions. The Test of Variables of Attention (TOVA; Universal Attention Disorders Inc.), a widely used diagnostic tool in the clinical assessment of ADD/ADHD, was employed. The TOVA is a computerized visual go/no-go task that consists of presentation of two types of stimuli, 'targets' which require the subject to respond as quickly as possible by pushing a switch button, and 'non-targets,' which require the subject to refrain from responding. The stimuli are non-language based, consisting of a white rectangle on a black background, which has a smaller black rectangle inserted either above (targets) or below (non-targets) its geometrical centre. Stimuli are presented for 100 ms with an inter-stimulus interval (ISI) of 2 s, for a duration of 21.6 min. The first half of the test contains a low ratio of targets to non-targets (1:3.5; 'infrequent target condition') and purports to measure inattentiveness/distractibility as reflected by errors of omission. The second half of the test is characterized by a high target to non-target ratio (3.5:1; 'frequent target condition') and is aimed at assessing impulsivity, as indicated by errors of commission. Also recorded were reaction time (RT) and reaction rime variability (RTV). A further measure that takes into account both of these error types is perceptual sensitivity or 'd prime' (d'), which expresses a ratio of hit rate to false alarm rate, derived from signal detection theory (Green and Swets, 1966). Customarily, for calculating d', the hit rate is defined as ['H' = ((number of targets − number of omissions) + 0.5)/(number of targets + 1)], and the false alarm rate as ['F' = (number of commissions + 0.5)/(number of non-targets + 1)]. From this, d' is calculated as [$d' = H\ (1 - F)/F(1 - H)$].

In addition to the TOVA, a further attention test was devised in order to provoke a more substantial amount of omission errors in this healthy sample. This auditory task will be referred to as a *divided attention task* and consisted of a sequence of monaurally presented sound stimuli (pure sinusoidal tones of 90 dB intensity and 40 ms duration with instantaneous rise and fall times) that differed in pitch (1000 Hz versus 1100 Hz) and ear channel of presentation (left versus right). These 4 stimulus categories had equal probability of occurrence ($P = 0.25$) and were presented in a random sequence of 240 trials with an ISI of 1 s that was randomly varied by 0.2 s. Subjects had to attend to both ear channels and were instructed to respond to high pitch tones presented to the left ear by pressing a response key with their left hand, and to respond to the low pitch sound in the right ear by pressing another response key with their right hand. Responses had to be withheld to low pitch stimuli presented to the left ear and high pitch stimuli presented to the right ear. The attention measures extracted from the divided attention task corresponded with those constituting the TOVA variables, i.e. errors of omissions, errors of commission, reaction time, reaction time variability (here defined as the standard deviation), and d'.

2.4. EEG recordings

EEG was recorded within 14 days prior and subsequent to the neurofeedback training regime. Recordings were taken via an ECI Electro-Cap from 28 locations placed according to the standard 10–20 system (Jasper, 1958) during an auditory oddball task. Electrodes were referenced off-line to linked earlobes, and the ground electrode was placed 1.5 cm anterior to the central frontal (FZ) electrode. Impedances were kept below 5 kΩ. EEG data were digitized at a sampling rate of 500 Hz and passed through a 0–100 Hz bandpass filter (24 dB/octave roll-off). Recording, digitization and subsequent off-line data processing were carried out with a SynAmps amplifier and Neuroscan system (version 4.1; Neuroscan Labs, Sterling, VA). The electro-oculogram (EOG) was recorded with tin cup electrodes placed on the orbis ocularis muscle above and below the left eye in order to detect eye-blinks, and on the left and right outer canti, approximately 1 cm lateral to either eye, for detecting lateral and horizontal eye movements. An EOG artefact correction method developed by Croft and Barry (2000) was applied to the EEG data off-line. Pre- and post-training recordings for each subject were conducted at approximately the same time of day (± 2 h).

2.5. Oddball task

The oddball task consisted of two sub-tasks, which differed only in terms of attentional instructions. In the first sub-task, subjects had to attend and respond to stimuli in their left ear channel, whereas in the second sub-task, subjects had to attend and respond to stimuli presented to the right ear. In each task, 350 stimuli were presented monaurally in a pseudo-random sequence via headphones, using a Neuroscan Stim interface system (Neuroscan Labs). The mean ISI of 1 s was randomly varied by 100 ms. Sound stimuli were generated by a Neurosoft Sound program system (Neuroscan Labs) and consisted of pure sinusoidal tones of 90 dB intensity and 40 ms duration with instantaneous rise and fall times. The stimuli differed in pitch (low pitch = 1000 Hz vs. high pitch = 1100 Hz), task relevance (attended vs. unattended), and frequency of occurrence (frequent/standard vs. rare/deviant). In the first sub-task, participants were instructed to attend only to the left ear channel (task-relevant) and discriminate between

low-pitch standard tones ($P = 0.4$) and high-pitch deviant target tones ($P = 0.1$) by pressing a response button to the target stimuli, whilst ignoring the concurrent presentation of irrelevant standards ($P = 0.4$) and irrelevant deviants ($P = 0.1$) in the right ear channel. In the second sub-task these instructions were reversed between the two ear channels.

2.6. P300 ERP

EOG-corrected data were epoched into periods of 612 ms, starting 100 ms prior to the onset of each stimulus and lasting until 512 ms post-stimulus. These epochs were baseline-corrected for the 100 ms pre-stimulus interval, and any epochs containing EEG fluctuation exceeding $\pm 100\ \mu V$ were rejected as artefact-contaminated. The remaining epochs were filtered (high-pass 0.5 Hz, low-pass 30 Hz) and averaged into the 4 categories of attended standard (AS), unattended standard (UAS), attended deviant (AD), and unattended deviant (UAD) stimuli. For the AD epochs, only those that had been correctly responded to were selected for averaging. The AD data resulting from the two oddball tasks (right ear versus left ear attendance) were averaged together. The target P300 component peak amplitude was defined for each subject individually as the highest positive deflection in the averaged ERP within a post-stimulus time interval of 250–400 ms for attended deviant stimuli. As in the previous study (Egner and Gruzelier, 2001), statistical testing was carried out for target P300 amplitude averaged over central frontal (F3, Fz, F4), central (C3, Cz, C4), and parietal (P3, Pz, P4) electrodes, as well as at the single electrode site level.

2.7. Neurofeedback training apparatus

Neurofeedback training was carried out employing two different commercially available hardware/software packages. Some of the training was carried out with a Neurocybernetics (Encino, CA) EEG Biofeedback System and ProComp (Thought Technology Ltd, Montreal, QC) differential amplifier. Signal was acquired at 160 Hz, A/D converted and band-filtered to extract beta1 (15–18 Hz), SMR (12–15 Hz), theta (4–7 Hz), and 'high beta' (22–30 Hz) components with a smoothing time constant of 0.5 s. Amplitude measures in the filter-bands were transformed online into audio-visual feedback representations, geometrical shapes that continuously changed size according to the amplitude in a given filter-band, and displayed via a 16in monitor to the trainees. Operant contingencies were such that rewards ('points' and auditory 'beeps') were gained whenever the trainee enhanced either beta1 (in the beta1 protocol) or SMR (in the SMR protocol) activity without concurrent rises in theta and high beta activity, relative to a 2 min pre-feedback baseline measure.

Also employed was the WaveRider Pro (Mindpeak, Sebastopol, CA) amplifier and software package. The WaveRider Pro acquired EEG (high-pass filter: 0.5 Hz; low-pass filter: 40 Hz; 76 dB roll-off) at a sampling rate of 128 Hz, and extracted frequency bands through fast Fourier transformation with a frequency resolution of 1 Hz. The feedback interface of this apparatus consisted of a number of line graphs, which expressed continuous representations of changes in (a) the absolute target frequency amplitude, (b) the target frequency-to-theta (4–7 Hz) ratio, and (c) the target frequency-to-broadband (0.5–30 Hz) ratio activity. The visual feedback of (a) and (c) was furthermore linked to auditory feedback sounds that would rise and fall in pitch according to changes in the given EEG parameters. These auditory feedback sounds were individually tailored for each subject to represent their preferred instruments and scales. All neurofeedback EEG was recorded from electrode Cz, referenced and grounded to earlobes, with impedances kept below approximately 10 kΩ.

2.8. Neurofeedback training procedure

Participants took part in 10 once-weekly training sessions consisting of either SMR or beta1 training (depending on group-membership) of 15 min duration. Six subjects in the SMR group and 6 in the beta1 group were trained with the WaveRider Pro equipment, and the remainder were trained with NeuroCybernetics equipment. For sessions trained with NeuroCybernetics equipment, these consisted of 5 feedback periods of 170 s with 10 s breaks in between them, while for training sessions with WaveRider equipment, sessions were continuous. The workings of the feedback loop were explained to the participants, and they were instructed to let the feedback process guide them into learning how to affect the feedback representations in the desired direction. Participants were seated in a comfortable chair approximately 1.5 m from the feedback monitor, and feedback was initiated after a 2 min baseline period.

3. Results

3.1. TOVA

The hypothesized commission error changes in the beta1 and SMR groups, and d′ increase in the SMR group, were assessed by planned comparisons (paired t tests) at one-tailed $P < 0.05$ levels. In order to determine differential effects of the neurofeedback protocols on variables for which no a priori hypotheses existed, 3×2 (group × time) mixed-subjects analyses of variance (ANOVAs) were applied, followed by post hoc comparisons assessing within-group changes for each group (paired t tests), and between-group differences in change scores (independent t tests). Descriptive statistics of all TOVA measures are presented in Table 1.

Table 1
Means and standard deviations on all TOVA measures in each group before and after training

Group	Variables	Time 1		Time 2	
		Mean	SD	Mean	SD
SMR	Omission errors	1.00	3.00	0.11	0.33
	Commission errors	5.67	3.04	4.67	3.91
	d′	6.18	0.90	6.55	0.89
	RT (ms)	320	37	319	51
	RTV (ms)	84	20	70	27
Beta1	Omission errors	0.00	0.00	0.00	0.00
	Commission errors	3.38	3.54	4.25	5.95
	d′	6.86	0.73	6.85	0.78
	RT (ms)	366	33	329	29
	RTV (ms)	75	17	68	12
Alexander	Omission errors	0.25	0.46	0.63	1.41
	Commission errors	4.25	3.69	2.38	1.60
	d′	6.40	1.23	6.49	1.21
	RT (ms)	343	58	341	54
	RTV (ms)	74	14	76	17

RT, reaction time; RTV, reaction time variability.

One-way ANOVAs on the initial scores of all TOVA variables indicated no differences between groups prior to training. As can be seen in Table 1, the hypothesized trends for commission errors in the beta band groups are reflected in the average scores, but neither the error decrease in the SMR group (one-tailed $P = 0.19$) nor the increase in the beta1 group (one-tailed $P = 0.21$) were statistically significant. A group × time ANOVA of commission error incident disclosed no significant effects either. While no omnibus group × time effects were detected, the predicted changes in the d′ measure in the SMR group were confirmed due to marginally significant d′ increments in the SMR group only (t[d.f. = 8] = −1.75, $P = 0.058$), as is depicted in Fig. 1. No significant changes were found for omission error incidence, and Table 1 reveals that, similar to previous studies applying the TOVA to healthy subjects (Egner and Gruzelier, 2001), only a negligible amount of omission errors were incurred.

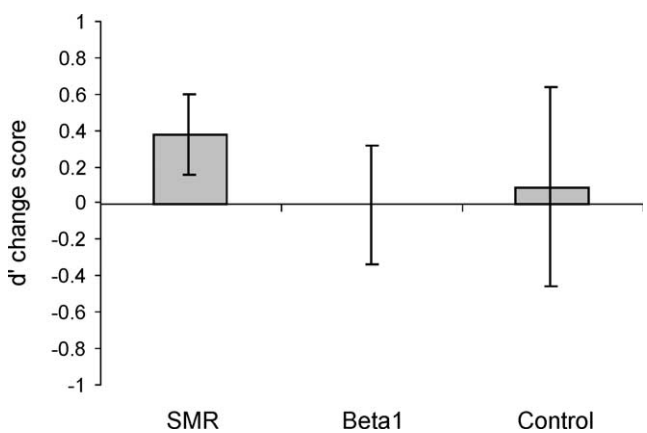

Fig. 1. Post-training changes on mean d′ scores (± SEM) on the TOVA task for the SMR, beta1, and control groups.

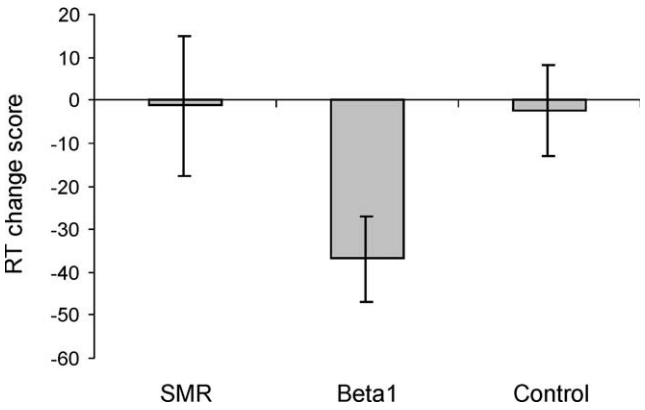

Fig. 2. Post-training changes (in ms) on mean reaction times (± SEM) on the TOVA task for the SMR, beta1, and control groups.

With no significant omnibus group × time effects found for RT, a significant RT reduction was evident in the beta1 group only (t[d.f. = 7] = 3.65, $P < 0.01$), as shown in Fig. 2. This reaction time reduction in the beta1 group was significantly different from RT changes in the control group (t[d.f. = 14] = −2.33, $P < 0.05$). Furthermore, a marginally significant main effect of time was found for RTV ($F[1, 22] = 3.59$, $P < 0.05$), due to trends for reduced RTV in the SMR and beta1 groups (see Table 1).

3.2. Divided attention task

Descriptive statistics for the variables extracted from the divided attention task are presented in Table 2, where it can be seen that the task succeeded in provoking a substantial

Table 2
Means and standard deviations on the divided attention task measures in each group before and after training

Group	Variables	Time 1		Time 2	
		Mean	SD	Mean	SD
SMR	Omission errors	14.00	3.33	7.75	1.88
	Commission errors	4.00	0.85	3.25	0.70
	d′	5.69	0.51	6.56	0.58
	RT (ms)	503	25	489	25
	RTV (ms)	147	11	128	12
Beta1	Omission errors	10.11	3.34	11.33	3.67
	Commission errors	5.67	1.64	4.11	1.29
	d′	5.86	0.52	6.35	0.71
	RT (ms)	526	14	502	23
	RTV (ms)	146	11	141	9
Alexander	Omission errors	8.38	3.01	8.50	3.55
	Commission errors	3.63	1.02	3.88	0.81
	d′	6.50	0.49	6.44	0.64
	RT (ms)	494	15	483	14
	RTV (ms)	118	6	115	69

RT, reaction time; RTV reaction time variability.

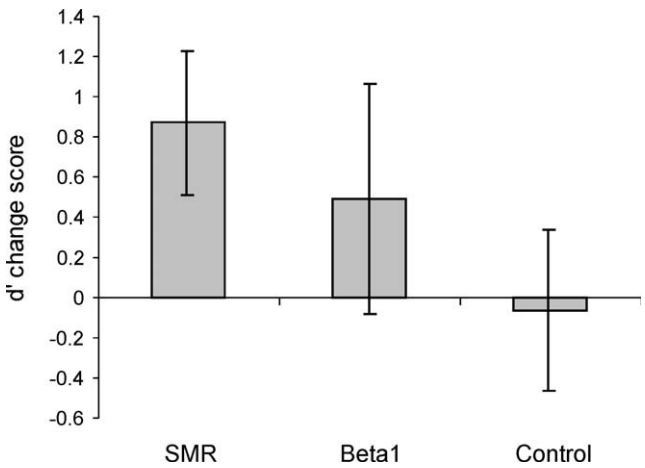

Fig. 3. Post-training changes on mean d′ scores (±SEM) on the divided attention task for the SMR, beta1, and control groups.

number of omission errors in all 3 groups. One-way ANOVAs on the initial scores of all TOVA variables disclosed no differences between groups prior to training. There was neither an omnibus effect, nor the predicted directional within-group differences found on the variable of commission error incidence. However, similar to the TOVA data, the SMR group was found to exhibit the hypothesized d′ increments (t[d.f. = 7] = −2.42, $P < 0.05$; see Fig. 3).

A marginally significant time × omission error interaction effect ($F[2, 22] = 2.83$, $P = 0.08$) was due to a significant reduction in omission errors found in the SMR group only (t[d.f. = 7] = 2.70, $P < 0.05$), displayed in Fig. 4. This omission error reduction in the SMR group differed significantly from omission change in the control group (t[d.f. = 14] = 2.48, $P < 0.05$). A main effect of time on RT ($F[1, 22] = 6.36$, $P < 0.05$) was found, due to a general trend for faster post-training reactions across groups (see Table 2). Furthermore, a significant main effect of time on RTV was obtained ($F[1, 22] = 6.54$, $P < 0.05$) due to mean decreased RTV in the SMR and beta1 groups, with significant within-group reduction of RTV evident in the SMR group only (t[d.f. = 7] = 3.53, $P < 0.05$), which was furthermore significantly different from the control group change score (t[d.f. = 14] = 2.18, $P < 0.05$).

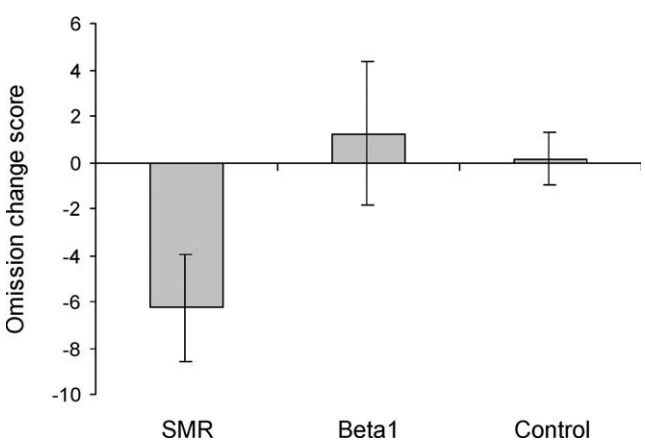

Fig. 4. Post-training changes on mean omission errors (±SEM) on the divided attention task for the SMR, beta1, and control groups.

3.3. Target P300

On this relatively simple task, mean numbers of errors were negligible both before (omission errors: mean = 0.87, SD = 2.25; commission errors: mean = 0.73, SD = 1.12; correct responses: 99.68%) and after training (omission errors: mean = 0.94, SD = 1.68; commission errors: mean = 0.38, SD = 0.70; correct responses: 99.74%). No significant changes in error detection were found. For P3b amplitude averaged over frontal, central, and parietal sites, the hypothesized P3b increase was found in the beta1 group (t[d.f. = 7] = −2.16, $P < 0.05$), but no changes between pre-training and post-training measures were detected in the SMR group (see Fig. 5). At the single electrode level, the beta1 group displayed significantly increased target P300 amplitudes at C3 (t[d.f. = 7] = −2.31, $P < 0.05$), Cz (t[d.f. = 7] = −2.73, $P < 0.05$), and Pz (t[d.f. = 7] = −2.05, $P < 0.05$). Again, no significant changes were detected in the SMR group.

4. Discussion

Of the hypothesized cognitive-behavioural effects of the SMR and beta1 neurofeedback protocols, the expected commission error decrease in the SMR group and commission error increase in the beta1 group were not confirmed by the current data. The expected d′ improvements in the SMR group on the other hand were confirmed in both the TOVA and the divided attention task. The expected target P300

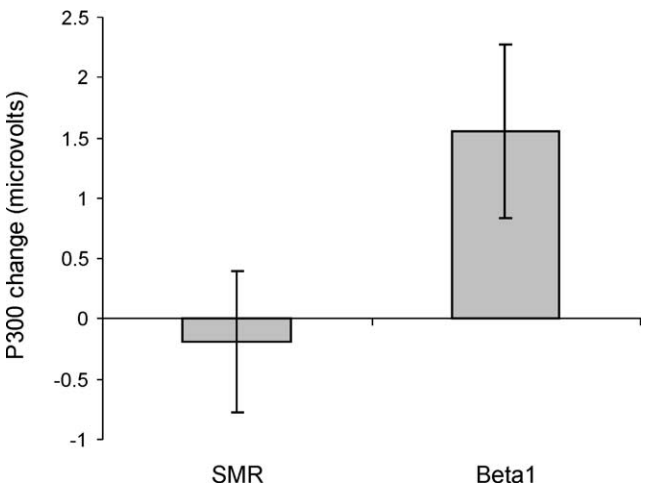

Fig. 5. Post-training changes on mean oddball target P300 amplitudes (±SEM) averaged for frontal, central, and parietal electrodes for the SMR and beta1 groups.

increments subsequent to beta band training were found exclusively in the beta1 group, with no changes evident in the SMR group. These significant P3b increments were found for amplitudes averaged over frontal, central, and parietal sites, as well as at C3, Cz, and Pz at the single electrode level. In addition to these findings concerning hypothesized protocol-specific effects, it was found that SMR training was associated with reduced omission errors and RTV on the divided attention task, and beta1 training resulted in reduced RTs on the TOVA. While of these data only the omission error reduction in the SMR group was associated with a significant overall interaction effect, it is noteworthy that both the RTV reduction in the SMR group and the RT reduction in the beta1 group were significantly different from change scores in the Alexander technique control group. Furthermore, there were no initial differences between groups on any of the dependent measures, and the control group did not display significant within-group changes on any of the variables assessed.

These data would seem to preclude the attribution of training effects to practice or motivational factors. Furthermore, the fact that effects differed between SMR and beta1 protocols discounts the interpretation of effects being due to some generic aspect involved in the nature of the neurofeedback training procedure, as the only factor that differentiated between the two protocols was the reinforced frequency. These results support the generic conclusion that SMR and beta1 neurofeedback protocols have significant and protocol-specific effects on cognitive-behavioural and electrocortical measures of attentional processing. In the following, the results obtained from the experimental investigations are interpreted in the light of the previous empirical and theoretical literature in an attempt to develop a coherent account of the effects and workings of learned beta band self-regulation.

4.1. SMR training

The trained enhancement of SMR activity has been conceptualized as a method for reducing impulsiveness/hyperactivity (e.g. Lubar and Shouse, 1976), as well as for enhancing attention processing more generally (Sterman, 1996). However, an unequivocal demonstration of the protocol-specific impact of SMR training on different aspects of attention and electrophysiology in clinical as well as non-clinical samples has remained elusive. The attribution of protocol-specific effects to the SMR protocol has previously been impeded by either the lack of a control group (Shouse and Lubar, 1979; Tansey, 1984, 1985), or by a failure to separate the effects of SMR and beta1 protocols when trained in the same group of subjects (Lubar and Lubar, 1984; Rossiter and LaVaque, 1995; Fuchs et al., 2003). In contrast to previous data showing a positive association between SMR feedback learning and commission error reduction (Egner and Gruzelier, 2001), the current results supply evidence for effects of SMR training on inattentive features of attention performance. These results can be interpreted as supportive of the proposal that the training results in decreased somatosensory and motor interference in attentive cognitive processing (Sterman, 1996).

The failure to replicate the association with reduced commission errors may lead to the conclusion that SMR training does not reliably reduce impulsive responses in healthy subjects. For a theoretical integration of these data, however, it appears most instructive to consider that improvements on the d' measure, which takes into account both omission and commission error incidence, have been reliably associated with SMR training across the previous and the current study on all the attention tests (visual and auditory) included. On the basis of this finding, it could be argued that the SMR training has led to reliable overall improvements of attention, but with varying relative contributions of impulsive versus inattentive error reduction. These in turn may be dependent on the initial error profile and the possible scope for improvement. For instance, while the d' improvement in the previous study (Egner and Gruzelier, 2001) was primarily a function of reduced commission errors, the SMR training group in the current study displayed a d' improvement on the divided attention task, which was primarily affected by a significant reduction in omission errors. The improvement on d' in the current TOVA data on the other hand, was a function of slight (non-significant) reductions in both commission and omission errors.

In summary, the current results can be interpreted as supporting the notion that SMR training's effects on sensorimotor control have benefits that extend beyond the direct impact on impulsive aspects of attention. Currently, the most parsimonious way of accounting for improved perceptual sensitivity and attentiveness is to propose that the presumed improved regulatory control in the somatosensory/sensorimotor pathways leads to more efficient higher order attention processing, i.e. the cognitive integration of task-relevant stimuli, by means of reduced processing interference. In support of this interpretation, Vernon et al. (2003) have recently found SMR training to be associated with improved semantic memory performance.

4.2. Beta1 training

The use of beta1 neurofeedback in the application to attentional disorders has stemmed from clinical observations of neurofeedback practitioners (e.g. Lubar and Lubar, 1984), and has more recently been justified in theoretical terms on the basis of abnormally low beta activity levels in some sub-groups of ADD/ADHD children (e.g. Dykman et al., 1982; Mann et al., 1991). In contrast to the physiological basis of the SMR as recorded over sensorimotor cortex, the generation of beta activity at any cortical site is not very well understood apart from the traditional notion of reflecting generic cortical activation

due to desynchronisation of alpha activity. In terms of its purported effects, beta1 training has been described as alleviating specifically the inattentive aspects of attentional disorders (Lubar and Lubar, 1984; Othmer et al., 1999). Similar to the attention-enhancing effects that have been attributed to SMR training however, the specific impact of beta1 training on inattentiveness has not been unequivocally demonstrated.

In healthy subjects it has been reported that beta1 feedback learning was negatively correlated with commission error reduction, and positively associated with increments in target P300 amplitudes, and these combined data were interpreted as the possible relation between beta1 training and increased activation in an attentional alertness/vigilance network (Egner and Gruzelier, 2001). In the current study, beta1 training resulted in decreased reaction times on one of the attention tests, but was not found to produce any effects on either impulsive or inattentive errors in test performance. The training's enhancing effect on target P300 amplitudes, however, was replicated. These data could be summarized by stating that beta1 training appears to reliably enhance target P300 amplitudes, and that these effects were accompanied by behavioural effects reflective of impulsive response tendencies, comprised by associations with increased commission errors (Egner and Gruzelier, 2001), and reduced reaction times (in the current study). Thus, while the precise behavioural effects did not replicate between the two studies, the effects obtained may still be reasonably conjectured to be reflective of one particular underlying response tendency. This tendency for fast but not necessarily accurate responses in turn has been associated with increased arousal in a noradrenergic alertness/vigilance attention network (Posner and Peterson, 1990; Posner and Raichle, 1994).

This theoretical conjecture assumes that the P300 increments associated with beta1 training in these studies are reflective of a higher general cortical background excitation (possibly mediated by noradrenergic neuromodulation), rather than of an enhancement of the specific neural processes associated with the cognitive correlates of the P300, i.e. central resources concerned with stimulus evaluation. It is well documented that P300 amplitudes are modulated not only by cognitive variables, but also by biological determinants of arousal states (for review see Polich and Kok, 1995), in that higher arousal is related to higher P300 amplitudes. The fact that the training was not associated with improved attention performance in terms of error incidence suggests that in healthy subjects, such enhancement of cortical excitation may lead to arousal levels beyond those required for optimal task performance.

In view of this interpretation of the results, the data are compatible with the general proposal that beta1 training may serve to raise cortical excitation in under-aroused ADD/ADHD samples (Lubar, 1991; Lubar and Lubar, 1999). The results expand this conceptualization by showing that the behavioural and electrocortical effects in healthy subjects indicate an impact on cortical arousal as maintained by an alertness/vigilance system, but not necessarily improving overall cognitive processing. This theoretical elaboration on the efficacy of beta1 neurofeedback evokes a number of hypotheses to guide future investigations. For instance, activation of the alerting network of attention can be assessed independently from other attentional systems (the orienting and executive networks) by means of continuous performance tasks which contrast reaction times to target stimuli between conditions where a pre-stimulus alerting signal is present or absent (Fan et al., 2002).

In conclusion, the current study has replicated associations between learned enhancement of SMR activity and improved perceptual sensitivity in sustained attention tasks, as well as between learned beta1 enhancement and oddball target P300 amplitudes in healthy subjects. In addition, SMR training was found to lead to omission error reduction and reaction time variability reduction, and beta1 training to faster reaction times. These data were interpreted as supporting the notion that SMR training can lead to general improvements in attention performance not limited to impulsive response tendencies, and that these effects may be accounted for in terms of reduced sensorimotor processing interference with higher cognitive function. Beta1 training effects were interpreted as reflecting a tendency towards fast but not necessarily accurate responses due to general arousal increments possibly mediated by increased activation in a noradrenergic alertness/vigilance network of attention.

Acknowledgements

This research was funded by the Leverhulme Trust and generously supported by the Royal College of Music.

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