The differential effects of extremity and movement side on the scalp distribution of the readiness potential (RP) and the stimulus-preceding negativity (SPN)

Damen, E.J.P.; Freude, G.; Brunia, C.H.M.

Published in:
Electroencephalography and clinical neurophysiology

Publication date:
1996

ELSEVIER

Electroencephalography and clinical Neurophysiology 99 (1996) 508–516

The differential effects of extremity and movement side on the scalp distribution of the readiness potential (RP) and the stimulus-preceding negativity (SPN)

E.J.P. Damen[a], G. Freude[b], C.H.M. Brunia[a,*]

[a]*Physiological Psychology Section, Department of Psychology, Tilburg University, P.O. Box 91053, 5000 LE Tilburg, The Netherlands*
[b]*Central Institute For Occupational Health, Berlin, Germany*

Accepted for publication: 20 June 1996

Abstract

In a time estimation task subjects had to press a button 3 s after the presentation of a warning stimulus. Two seconds after the movement they were informed about their performance by a knowledge of results (KR) stimulus. Preceding the movement a readiness potential (RP) and prior to the presentation of the KR stimulus a stimulus-preceding negativity (SPN) was recorded. Movement side (left/right) and extremity (hand/foot) were varied within subjects to demonstrate that the RP but not the SPN is affected by such manipulations. The scalp distribution of the late part of the RP was affected by movement side and extremity. Yet it exhibited the expected lateral asymmetry only preceding a movement of the left hand or of the right foot. The scalp distribution of the SPN was not affected by extremity. The size of the right hemisphere preponderance of the SPN depended on movement side following a finger flexion, but not following a plantar flexion of the foot. The experimental design was intended to avoid the temporal overlap between movement-related and stimulus-related activity. Yet it is argued that both results of this experiment can best be explained by such an overlap. Copyright © 1996 Elsevier Science Ireland Ltd.

Keywords: Readiness potential (RP); Stimulus-preceding negativity(SPN); Motor preparation; Stimulus anticipation; Post-movement negativity; Knowledge of results (KR)

1. Introduction

The presentation of the warning stimulus (WS) in a reaction time (RT) task with a fixed preparatory interval evokes a dual preparatory process in a subject: an expectant attention for the reaction stimulus (RS) and response preparation. In this type of task both processes are necessarily confounded because of temporal overlap. Therefore, Damen and Brunia (1987) used a task in which this overlap was avoided: subjects had to terminate an estimated time interval by a movement that was followed by the delayed presentation of a stimulus providing knowledge of results (KR) about their time estimation. So, response preparation took place first, to be followed by expectant attention for the KR stimulus. Several studies using this paradigm (Damen and Brunia, 1987; Brunia and Damen, 1988) or a variation of it (Chwilla and Brunia, 1991) demonstrated that the readiness potential (RP) and stimulus-preceding negativity (SPN) have different, but overlapping potential distributions.

Chwilla and Brunia (1991) concluded that the amplitude of the SPN depends on the information value of the KR stimuli. Damen and Brunia (Damen and Brunia, 1990; Damen and Brunia, 1994) concluded that KR stimuli are preceded by a larger negative shift than instruction stimuli and only the SPN preceding KR stimuli exhibited a right hemisphere preponderance. These studies agree on the idea that the SPN reflects an anticipatory attentional process.

In a spatial positioning task with KR, the SPN exhibited a right hemisphere preponderance, irrespective of the hand used in positioning (Grünewald-Zuberbier et al., 1981; Grünewald and Grünewald-Zuberbier, 1983) and

* Corresponding author. Tel.: +31 13 662400.

PII S0921-884X(96)95565-4

EEG 95565

irrespective of the sensory modality of the KR stimulus (Grünewald et al., 1984). But, in a time estimation task with visual KR (Damen and Brunia, 1987; Brunia and Damen, 1988; Damen and Brunia, 1994), this right hemisphere preponderance of the SPN was very large following a left index-finger movement (Damen and Brunia, 1987; Brunia and Damen, 1988; Damen and Brunia, 1994), but almost zero following a right index-finger movement (Chwilla and Brunia, 1991). Such a movement side effect contradicts the idea that the SPN reflects exclusively an anticipatory attentional process. Rather, it suggests a contribution of (reafferent?) sensomotoric activity. For instance, the sources of the late part of the RP may remain active to some degree until the presentation of the KR stimulus and contribute to the SPN. Fortunately, the lateral asymmetry and scalp distribution of the late part of the RP depend on movement side and extremity: preceding finger movements, amplitudes are largest over the hemisphere contralateral to the movement side, whereas preceding foot movements, a paradoxical ipsilateral preponderance is found (Brunia and Vingerhoets, 1981; Boschert et al., 1983a; Boschert et al., 1983b; Brunia and van den Bosch, 1984a; Brunia and van den Bosch, 1984b; Brunia and Haagh, 1986). The central coronal distribution has a pronounced maximum over the vertex preceding foot movements and a more laterally located contralateral maximum preceding finger movements (Kornhuber and Deecke, 1965; Vaughan et al., 1968; Shibasaki et al., 1981; Brunia and Dautzenberg, 1986). So, the scalp distribution reflects the different localisation of the sources of the late part of the RP preceding finger and foot movements. By a systematic variation of movement side (left vs. right) and responding extremity (hand vs. foot), this study tries to evaluate whether or not the SPN is caused mainly by prolonged activity of these sources. Specifically, the following questions were asked: (1) does the scalp distribution of the SPN depend on the extremity used to respond with?; (2) do hand and foot movements have opposite movement side effects on the right hemisphere preponderance of the SPN?

2. Method

2.1. Subjects

Twenty right-handed subjects (range of ages 18–40 years, mean age 24.9 years), 9 females and 11 males, participated in the experiment. Hand dominance was assessed with an abridged version of the Edinburgh Inventory (Oldfield, 1971). Subjects were paid Dfl. 7.50 per hour.

2.2. Apparatus

Subjects were seated in a comfortable chair in a dimly lit, sound-attenuated, and electrically shielded chamber. A display was attached to the wall at eye-level 1.5 m in front of the subject. It contained five translucent, congruent squares (area 2.25 cm^2) arranged in the shape of a plus sign. Each square could be illuminated independently by a red LED mounted behind it. The central square was illuminated during the whole session and served as a fixation stimulus. The visual stimuli (duration 300 ms), a plus sign, a minus sign, or a vertical line, were produced by illuminating the peripheral squares. The acoustic stimuli were pure tones of 1000 Hz (intensity 70 dB(A), duration 100 ms) and 4400 Hz (intensity 80 dB(A), duration 300 ms) presented by a speaker located right behind the subject at a distance of 1 m. On the tips of both arms of the chair, tubes with a push-button on one side were mounted. Subjects held the tube between thumb and index-finger in a pincers-grasp, the index-finger resting on the push-button. Closure of the microswitch (force 9.8 N) under the push-button was used to detect the movement. Each foot rested on a footplate, with a microswitch placed under it, which could only be closed by moving the footplate down by means of a plantar flexion of the foot (force 19.6 N). The footplate was kept in place by a set of springs. The electromyogram (EMG) of the dorsal interosseus I muscle of each hand, a synergist in the flexion of the index-finger was recorded bipolarly by two non-polarising Beckman 2.1 mm Ag-AgCl electrodes placed over the belly of this muscle (heart-to-heart distance 1 cm). The EMG of both calf muscles, agonists in the plantar flexion of the foot, was recorded bipolarly by two Ag-AgCl surface electrodes placed 4 cm apart over the distal part of each soleus muscle. The EMGs were fed to differential amplifiers (–3 dB bandwidth 3.8 Hz (roll-off 31 dB/octave) to 520 Hz (roll-off 13.5 dB/octave)) and subsequently full-wave rectified and low-pass filtered (–3 dB cut-off 50 Hz, roll-off 29 dB/octave).

To record the electroencephalogram (EEG), non-polarising Beckman 8 mm Ag-AgCl electrodes were affixed to the scalp with an adhesive and conductive paste (Grass EC2) at F3, F4, P3, P4 and Cz of the international 10/20 system and at C1′, C2′, C3′, C4′, T3′, T4′. C3′ and C4′ were located 1 cm anterior to C3 and C4, respectively. C1′ and C2′ were located halfway between C3′ and Cz and C4′ and Cz, respectively. T3′ and T4′ were located halfway between C3′ and T3 and C4′ and T4, respectively. Linked mastoids served as a reference. The electro-oculogram (EOG) was recorded bipolarly with two 2.1 mm Beckman Ag-AgCl electrodes, one located directly above and the other laterally below the right eye. EEG and EOG signals were fed to modified Nihon Kohden amplifiers (–3 dB bandwidth 0.005–35 Hz (roll-off 6 dB/octave)). Inter-electrode impedance was less than 3 kΩ. Preceding and following each block of trials, trains of square waves (100 μV peak-to-peak) were recorded for calibration purposes. The full-wave rectified and low-pass filtered EMGs of both dorsal interosseus I muscles were digitised only during trial blocks with hand movements, whereas those of both

calf muscles were digitised only during trial blocks with foot movements. A DEC SBC 11/21$^+$ computer and 10 programmable timers controlled stimulus presentation and acquired performance data. The full-wave rectified and low-pass filtered EMGs, EOG, EEG signals and pulses corresponding to experimental events were written out on paper by a 14 channel Nihon Kohden polygraph and digitised online in 9 s epochs from 1 s before the start of a trial at a rate of 200 Hz by a 12-bit A/D converter connected to a VAX 730 computer.

2.3. Procedure

Subjects participated in one session in which the same time estimation task was presented in 4 blocks of trials. Each trial started with the presentation of an acoustic stimulus (1000 Hz, 70 dB(A), 100 ms), which signalled the subject to start timing a 3 s interval. As soon as this interval was judged to have elapsed, the subject closed a microswitch and 2 s later one of 3 visual stimuli providing KR about the correctness of the produced interval was presented. The produced interval was defined to be correct if it deviated not more than 5% from the required 3 s interval in either direction. A minus sign indicated produced intervals smaller than 2850 ms, whereas a plus sign signalled intervals greater than 3150 ms. A vertical line indicated correct performance, i.e. the produced interval was at least 2850 and at most 3150 ms. If no response was emitted within 5 s after the start stimulus, the trial was terminated by a loud high-pitched tone (4400 Hz, 80 dB(A), 300 ms). The intertrial interval (ITI) was varied pseudo-randomly from 6 to 10 s (rectangular distribution with a mean of 8 s and a step size of 0.5 s) and was timed from the moment a trial ended, either by presentation of visual KR or by presentation of the acoustic time-out stimulus, until the presentation of the acoustic start stimulus of the next trial. Each block consisted of 80 trials that were not ended by the presentation of the time-out stimulus.

There were 4 different responses: (1) flexion of the left index-finger; (2) flexion of the right index-finger; (3) plantar flexion of the left foot; and (4) plantar flexion of the right foot. The responses were varied between blocks according to 4 different orders to each of which 5 subjects were assigned randomly. Each response occupied a position only once across all orders.

To reduce variability in strategies employed in time estimation, subjects were requested to count silently and to refrain from rhythmical activity during timing (e.g. not to tap their feet, hands, fingers, not to blink, not to move their tongue, lips etc.). They also had to remove their watches. It was stressed to keep the duration of the responses as short as possible. They were instructed to make no eye movements and to refrain from blinking from at least 1 s before the presentation of the start stimulus until at least 0.5 s following presentation of the KR stimulus. They were encouraged to produce as many correct intervals as possible. At the end of each block the experimenter informed them about the number of correctly timed intervals and told them whether their performance was improving or not.

2.4. Data analysis

The paper record with EEG (10 μV/mm) and EOG (20 μV/mm) signals of each subject was examined for artifacts and for excessive change in EOG amplitude during the epoch from 1 s preceding the start of the trial until 0.5 s following presentation of the KR stimulus. Only trials in which the change in EOG amplitude within the critical epoch was less than 60 μV and no other artifacts (dc-drift, head movement artifacts, electrostatic electrode artifacts) were present in the EEG were accepted for averaging. Trials terminated by the time-out stimulus and trials on which the produced interval was less than 1500 ms were rejected too. The data of a subject were used for statistical analysis only if for each block at least 30 trials were accepted for averaging. Only 16 subjects passed this criterion.

All electrophysiological signals were averaged time-locked to the movement from 2.5 s preceding the button press until 0.5 s following presentation of KR. All potential shifts were baseline-corrected with respect to the mean amplitude of the samples in the 500 ms epoch from 2500 to 2000 ms preceding the button press.

Performance was measured as the percentage intervals that were correct, too short, or too long. These percentages were subjected to a repeated measures multivariate analysis of variance (MANOVA procedure from the SPSS-X package) with the factors Extremity (hand vs. foot), Movement Side (left vs. right) and KR (correct, too short, too long).

Grand averages of the slow cortical potentials time-locked to the button press were computed for each block of trials separately. All peak measures were computed with respect to the baseline. The amplitude of the RP was defined as the mean amplitude in the last 200 ms prior to switch-closure, the SPN amplitude as the mean amplitude in the last 200 ms prior to the presentation of the KR stimulus. Each amplitude measure, separately, was subjected to one repeated measures MANOVA across the lateral electrode positions with the factors Extremity (E: hand vs. foot), Movement Side (M: left vs. right), Position (P: F3/4, C1/2′, C3/4′, T3/4′, P3/4) and Hemisphere (H: left vs. right) and another across the central coronal electrode positions with the factors Extremity (E), Movement Side (M) and Position (P: T3′, C3′, C1′, Cz, C2′, C4′, T4′). To test whether interactions with the factor Position reflect true differences in topography between conditions, the data were normalised by dividing by the square root of their sum of squares (McCarthy and Wood, 1985) before MANOVAs were computed.

Only statistical tests with an outcome probability of less

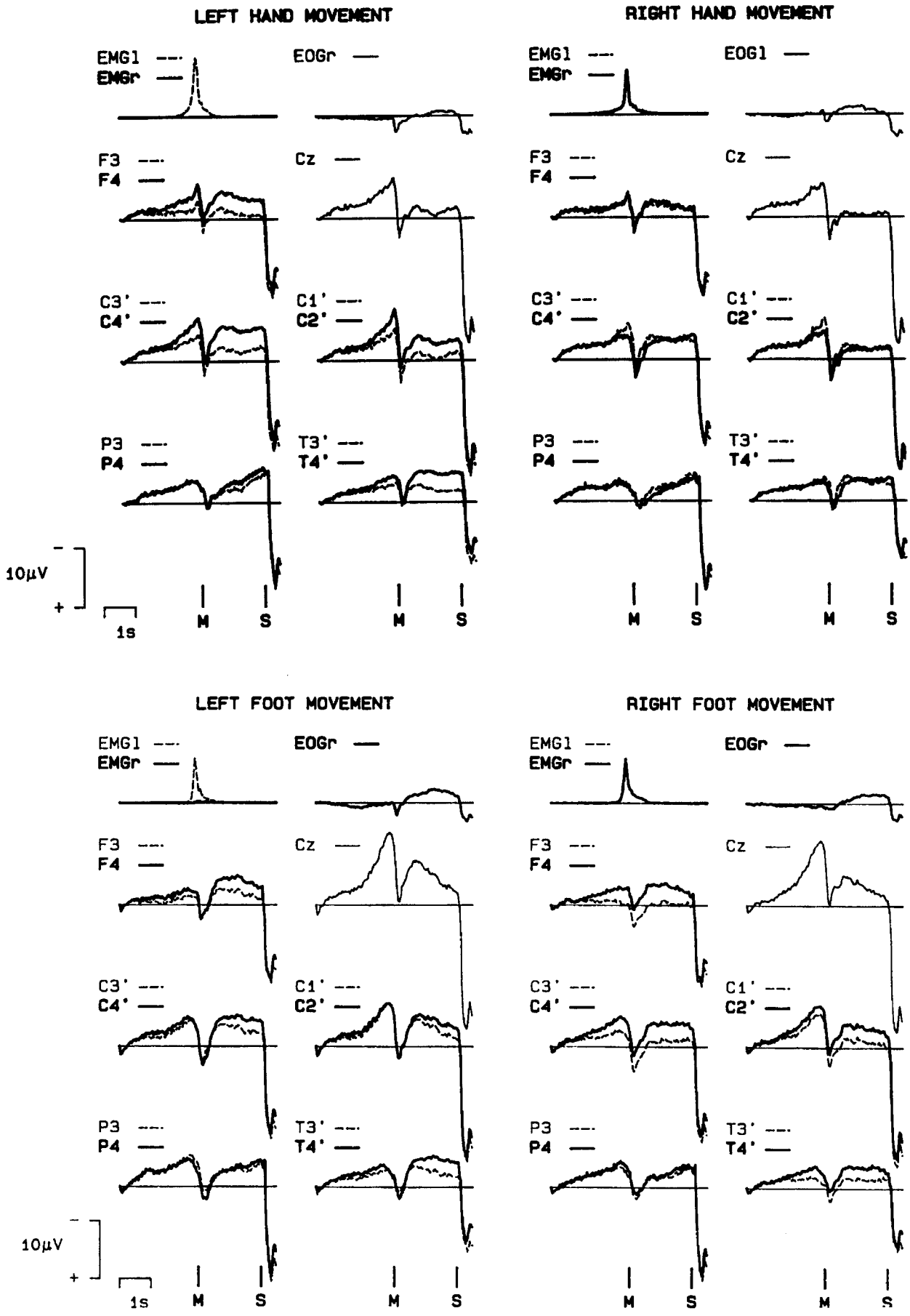
LEFT HAND MOVEMENT
RIGHT HAND MOVEMENT
LEFT FOOT MOVEMENT
RIGHT FOOT MOVEMENT
EMG1
EMGr
EOGr
EOG1
F3
F4
Cz
C3'
C4'
C1'
C2'
P3
P4
T3'
T4'
10μV
1s
M
S

Table 1

Results of the MANOVAs on the peak-amplitudes of the RP and the SPN recorded from the lateral electrodes

	RP			SPN		
	d.f.	*F*-ratio	*P*	d.f.	*F*-ratio	*P*
Extremity (E)	1,15	0.04	0.838	1,15	0.00	0.958
Movement Side (M)	1,15	4.16	0.059	1,15	3.02	0.103
Electrode Position (P)	*4,12*	*13.81*	*0.000*	*4,12*	*5.87*	*0.007*
Hemisphere (H)	*1,15*	*5.78*	*0.030*	*1,15*	*8.72*	*0.010*
E × M	1,15	0.26	0.619	1,15	0.00	0.995
E × P	*4,12*	*10.46*	*0.001*	4,12	1.92	0.171
E × H	1,15	1.49	0.241	1,15	3.33	0.088
M × P	4,12	1.28	0.333	4,12	0.45	0.768
M × H	1,15	2.48	0.136	*1,15*	*9.58*	*0.007*
P × H	4,12	1.54	0.252	4,12	1.06	0.417
E × M × P	4,12	0.68	0.618	4,12	1.39	0.297
E × M × H	*1,15*	*74.54*	*0.000*	*1,15*	*5.64*	*0.031*
E × P × H	4,12	2.96	0.065	4,12	1.09	0.405
P × M × H	*4,12*	*14.46*	*0.000*	4,12	1.05	0.422
E × P × M × H	*4,12*	*14.97*	*0.000*	4,12	1.77	0.199

Interactions in which the Electrode Position factor was involved were computed after normalisation of the data as recommended by McCarthy and Wood (1985). Results printed in italics are significant at the 5% level.

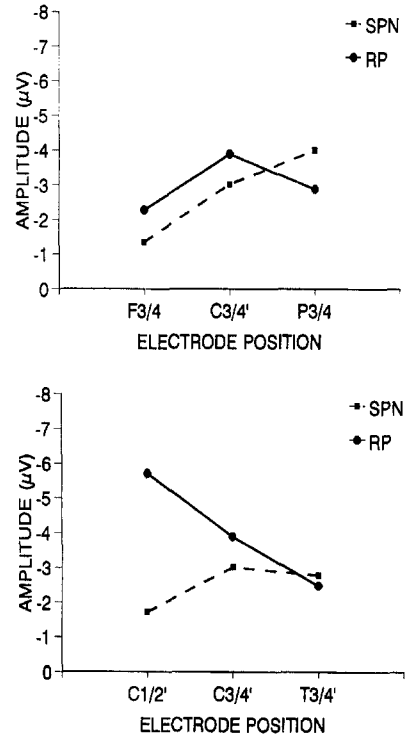

Fig. 2. Average RP (solid line) and SPN (dashed line) peak-amplitude scalp distributions across the factors Extremity, Movement side and Hemisphere. The left-hand panel displays the antero-posterior gradients, the right-hand panel displays the central coronal gradients of the RP and the SPN.

than 5% will be reported as significant effects. Post-hoc tests were computed according to the Scheffé method described by O'Brien and Kaiser (1985), in which univariate *F*-statistics are compared to the critical Scheffé value *Fs* for the particular contrast tested. Interactions were broken down into simple effects to clarify their interpretation and the overall α was held constant at 5% by dividing α by the number of possible simple effects that could be computed. Although the significance of interactions with the factor Position was based on the normalised data, corresponding post-hoc tests and simple effects were computed on the original non-normalised data.

3. Results

3.1. Performance

The repeated measures MANOVA on the performance scores did not result in significant effects. Across conditions subjects produced on average 27.9% too short intervals, 41.5% correct intervals and 30.1% too long intervals. Only 0.5% of trials was terminated by a time-out stimulus. So, performance did not differ significantly between conditions.

3.2. Slow cortical potentials

Fig. 1 shows that the movement was preceded by a clear RP which was followed by a second slow cortical potential shift of negative polarity, the SPN. The EMG traces show a clear unilateral activation of the intended muscles.

3.3. Readiness potential

The results of the repeated measures MANOVA on the RP recorded from the lateral electrode positions are summarised in Table 1. The Position effect indicated that RP amplitudes differed between recording sites (see Fig. 2). The lateral antero-posterior gradient of the RP (left-hand panel of Fig. 2) had a slight central maximum. None of the post-hoc Scheffé comparisons was significant. The lateral coronal distribution (right-hand panel of Fig. 2) decreased

Fig. 1. Grand averages of the RP and SPN. The top half displays both potentials for hand movements. EMGl and EMGr stand for the EMGs recorded from the left and right m. dorsal interosseus I, respectively. The bottom half displays both potentials for foot movements. EMGl and EMGr stand for the EMGs recorded from the left and right calf muscles, respectively. EOGr indicates the obliquely recorded eye-movement potentials from the right eye. Negative polarity up. For EEG and EOG signals the amplitude calibration marker corresponds to 10 μV, for the EMGs it corresponds to 150 μV. Marker 'M' stands for movement and marker 'S' for the presentation of the KR stimulus.

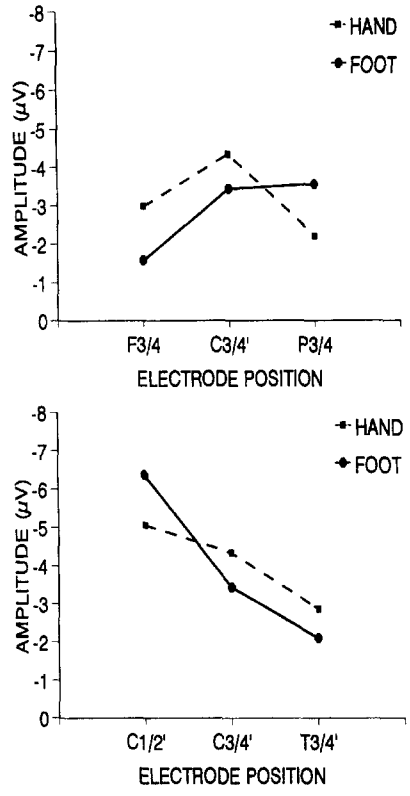

Fig. 3. RP peak-amplitude scalp distributions averaged across the factors Movement Side and Hemisphere. The left-hand panel displays the antero-posterior gradients of the RP. The right-hand panel displays the central coronal gradients of the RP. RP amplitudes preceding hand movements are indicated by dashed lines and those preceding foot movements by solid lines.

from a near-midline (C1/2′) maximum to a temporal minimum as the post-hoc comparisons (C1/2′-C3/4′, $F(1,15) = 42.68$; C1/2′-T3/4′, $F(1,15) = 51.12$; C3/4′-T3/4′, $F(1,15) = 28.65$; $Fs = 16.30$) confirmed. The scalp distribution of the RP also depended on the responding extremity (E × P): it had a central maximum preceding finger movements but a more parietal maximum preceding foot movements (left-hand panel of Fig. 3), as the post-hoc contrast of the RP amplitude difference between C3/4′ and P3/4 as a function of extremity ($F(1,15) = 18.99 > Fs = 16.30$) confirmed. RP amplitudes decreased more steeply from a near-midline maximum (right-hand panel of Fig. 3) in lateral directions preceding foot movements than preceding finger movements, as the significant post-hoc contrast (C1/2′-C3/4′, $F(1,15) = 42.17 > Fs = 16.30$) confirmed. Furthermore, the lateral asymmetry of the RP differed as a function of Extremity and Movement Side (E × M × H). Preceding a left-hand movement, the average RP-amplitude was more negative over the contralateral than over the ipsilateral hemisphere (Hemisphere (left hand): $F(1,15) = 16.86$, $P < 0.0125$), but not preceding a right hand movement (Hemisphere (right hand): $F(1,15) = 7.28$, $P > 0.0125$). Prior to a right foot response the mean RP amplitude was largest over the ipsilateral hemisphere (Hemisphere (right foot): $F(1,15) = 20.05$, $P < 0.001$), but not preceding a left foot response (Hemisphere (left foot): $F(1,15) = 0.06$, $P > 0.8$). From Fig. 1, and the significant P × M × H and E × P × M × H effects, it can be inferred that this movement side-dependent lateral asymmetry was not present at each recording site. Dissection of this interaction in simple hemisphere effects for each factorial combination of levels of the remaining factors revealed the sites at which RP amplitudes differed between hemispheres (see Table 2).

The results of the MANOVA on RP amplitudes recorded from all coronal electrodes are displayed in Table 3. The significant effects reflect the way movement side and extremity affected the coronal distribution of the RP. The coronal distribution had a near-midline maximum contralateral to the responding hand and a maximum over the vertex preceding foot movements, irrespective of movement side (Fig. 4).

3.4. Stimulus preceding negativity

The results of the MANOVA on the SPN recorded from lateral electrode positions are summarised in Table 1. The effect of Position indicates that the SPN amplitude differed between recording sites. The parasagittal distribution of the SPN (left-hand panel of Fig. 2) tended to a parietal maximum, although none of the 3 pairwise post-hoc Scheffé contrasts was significant (F3/4-C3/4′, $F(1,15) = 7.45$; C3/4′-P3/4, $F(1,15) = 2.63$; F3/4-P3/4, $F(1,15) = 7.29$; $Fs = 16.30$). The SPN (right-hand panel of Fig. 2) amplitude increased from a near midline (C1/2′) minimum to a lateral centro-temporal maximum, although none of the pairwise post-hoc contrasts (C1/2′-C3/4′, $F(1,15) = 16.09 < Fs = 16.30$; C3/4′-T3/4′, $F(1,15) = 0.40 < Fs = 16.30$) was statistically significant. The Hemisphere effect confirmed that more negativity was recorded from the right than from the left hemisphere sites (Fig. 1). But, the size of this right hemisphere preponderance was dependent on Movement Side and Extremity (E × M × H): in the case of finger movements (see Fig.

Table 2

Dissection of the Extremity × Movement side × Hemisphere interaction from the MANOVA on the RP recorded from lateral electrodes in simple Hemisphere effects for each factorial combination of levels of the factors Position, Extremity and Movement side

Electrode Position	Left hand	Right hand	Left foot	Right foot
F3/4	17.75*	0.24	2.78	36.83*
C1/2′	38.88*	22.16*	0.02	6.74
C3/4′	20.75*	11.85	0.04	15.51*
T3/4′	2.70	1.92	0.28	8.23
P3/4	0.06	0.00	9.70	6.85

The d.f. for all F-tests were 1 by 15. The 0.05 significance level was Bonferroni-corrected by dividing α by the number of tests (20), resulting in $\alpha = 0.0025$ for individual tests. Asterisks indicate significant effects.

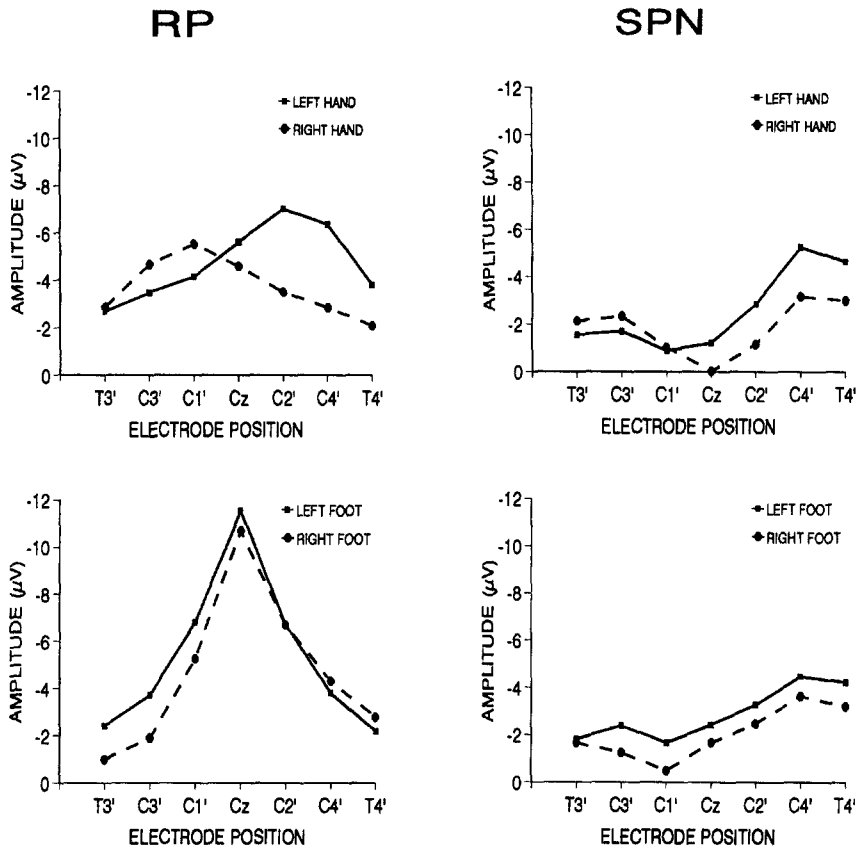

Fig. 4. The coronal scalp distributions of the RP and SPN peak-amplitudes for each type of movement separately. The left-hand column displays the central coronal RP distributions preceding left and right hand movements (top) and preceding left and right foot movements (bottom). The right-hand column displays the central coronal SPN distributions following left and right hand movements (top) and following left and right foot movements (bottom). Solid lines correspond to a left-sided movement, dashed lines to a right-sided movement.

1), a right hemisphere preponderance was found ($F(1,15) = 13.56$, $P < 0.01$) following a left hand, but not following a right hand movement ($F(1,15) = 0.16$, $P > 0.69$). In case of foot movements (see Fig. 1), the right hemisphere preponderance was independent of movement side (Hemisphere (left foot): $F(1,15) = 12.52$, $P < 0.01$; Hemisphere (right foot): $F(1,15) = 9.76$, $P < 0.01$).

The MANOVA on the SPN recorded from electrodes placed on the central coronal line (Table 3) revealed only a significant effect of Position. In all conditions the coronal distribution of the SPN had a minimum near Cz or C1′ and amplitudes increased in lateral directions(right hand panels of Fig. 4), but more so over the right than over the left hemisphere. The lack of a significant E × M × P effect indicates that the coronal distribution of the SPN was not significantly affected by movement side or extremity. This does not point to activation of differently localised electrocortical sources in different conditions.

4. Discussion

The lack of difference in performance between conditions makes an explanation of effects on the slow cortical potentials in terms of differences at the behavioural level highly unlikely.

The coronal potential gradient of the RP reflected the more medial localisation of the electrocortical sources preceding foot than preceding finger flexions (e.g. Vaughan et

Table 3

Results of the MANOVAs on the peak amplitudes of the RP and the SPN recorded from the electrodes placed over the central coronal line

	RP			SPN		
	d.f.	*F*-ratio	*P*	d.f.	*F*-ratio	*P*
Extremity (E)	1,15	2.52	0.133	1,15	0.24	0.628
Movement Side (M)	*1,15*	*3.80*	*0.007*	1,15	2.73	0.119
Electrode Position (P)	*6,10*	*9.50*	*0.001*	*6,10*	*4.00*	*0.026*
E × M	1,15	.14	0.713	1,15	0.01	0.933
E × P	*6,10*	*6.74*	*0.004*	6,10	1.48	0.279
M × P	*6,10*	*5.89*	*0.007*	6,10	1.52	0.267
E × M × P	*6,10*	*21.18*	*0.000*	6,10	2.20	0.130

Interactions in which the Electrode Position factor was involved were computed after normalisation of the data as recommended by McCarthy and Wood (1985). Results printed in italics are significant at the 5% level.

al., 1968; Shibasaki et al., 1981; Boschert et al., 1983a; Boschert et al., 1983b). Prior to foot movements the maximum of the antero-posterior gradient was located more centro-parietally than centrally, as was also noted by Brunia (Brunia and van den Bosch, 1984b; Brunia et al., 1985). The lateral parts of the precentral gyrus are more anteriorly located than the medial parts of the precentral gyrus (Vaughan et al., 1968). So, the more posterior maximum of the antero-posterior RP gradient with foot movements in the present experiment presumably reflects the more posterior localisation of the activated medial part of the precentral gyrus.

The lateral asymmetry of the late part of the RP was not fully in agreement with the literature (e.g. Vaughan et al., 1968; Brunia and Vingerhoets, 1981; Shibasaki et al., 1981; Boschert et al., 1983a; Boschert et al., 1983b; Brunia and van den Bosch, 1984a; Brunia and van den Bosch, 1984b; Brunia et al., 1985). The lateral asymmetries were not perfect mirror images (see Table 2). Preceding a movement of the left index finger the frontal and central positions exhibited a contralateral preponderance of negativity, whereas preceding a movement of the right index finger only the near-midline central site exhibited a contralateral preponderance of negativity. Prior to a movement of the left foot the RP was bilaterally symmetrically distributed, whereas preceding a movement of the right foot, the frontal and lateral central positions exhibited an ipsilateral preponderance of negativity. So, only prior to a movement of the left index finger or of the right foot the late part of the RP was clearly laterally asymmetric.

The answer to the first question we asked is that the scalp distribution of the SPN seems not to depend on the responding extremity. The central coronal scalp distribution of the SPN was the opposite of that of the RP: amplitudes decreased from a centro-temporal maximum to a near-midline minimum. This agrees with an electrocortical source near the temporal areas suggested by the dipole modelling study of Böcker et al. (1994). In antero-posterior direction the SPN had a frontal minimum and a centro-parietal maximum. Since both potential gradients were unaffected by the factor Extremity, it is not likely that the SPN contains a contribution from the electrocortical generators of the late part of the RP.

Although the effect of movement side on the right hemisphere preponderance of the SPN differed between hand and foot movements, no reversal was found. The SPN exhibited a right hemisphere preponderance, which, as in previous studies (Damen and Brunia, 1987; Brunia and Damen, 1988; Damen and Brunia, 1994), was large following a left-hand and small following a right-hand movement (e.g. Chwilla and Brunia, 1991). However, the right hemisphere preponderance of the SPN did not differ between right and left foot movements.

This differential effect of finger and foot movements on the right hemisphere preponderance of the SPN and the lack of lateral asymmetry of the RP preceding left foot and right hand movements may have a common explanation: a temporal overlap of cortical negative potential shifts. Damen and Brunia (1994) not only recorded the RP and SPN in a time estimation task, but also the RP in a self-paced movement task. In the 2.5 s epoch following a self-paced movement the recorded dc-potentials did not fully return to baseline. Over precentral, postcentral and temporal sites these post-movement shifts were similarly lateralised as the late part of the RP. Brunia (Brunia and van den Bosch, 1984b; Brunia et al., 1985) reported a similar post-movement negativity showing a contralateral preponderance following hand movements and an ipsilateral preponderance following foot movements during a 1 s post-movement epoch. By subtracting the potentials recorded in the self-paced movement task from those recorded in the time estimation task, Damen and Brunia (1994) obtained a pure SPN exhibiting a right hemisphere preponderance that was independent of movement side and starting before movement onset at the lateral frontal, precentral and post-central sites. Böcker et al. (1994) also suggested that the SPN starts before movement onset. So, in the time estimation tasks the RP amplitude may be contaminated by a laterally asymmetrical pre-movement part of the SPN and the SPN may be contaminated by laterally asymmetrical post-movement negativity.

In related studies the RP was possibly also contaminated by a pre-movement part of a SPN. McAdam and Seales (1969) and Hink et al. (1982) recorded an enhanced RP in conditions in which the presentation of a feedback stimulus followed the movement. Although they suggested that the RP itself increased in amplitude due to motivation, it is equally well possible that the RP was superimposed on a negative potential shift related to the anticipation of the feedback stimulus. However, because the stimulus followed the response immediately, McAdam and Seales (1969) could not detect a separate SPN prior to feedback. Hink et al. (1982) probably filtered out such a SPN in the 1.4 s delay between movement and stimulus presentation by their time constant of 1.33 s.

If the RP is superimposed on the pre-movement part of the pure SPN, then prior to left hand or right foot movements the right hemisphere preponderances of both RP and pure SPN amplify each other. The left hemisphere preponderance of the RP prior to right hand or left foot movements would be reduced by the right hemisphere preponderance of the pure SPN. This would nicely fit the present RP data, especially at the frontal, lateral central and temporal sites (see Fig. 1).

If a pure SPN is superimposed on post-movement negativity, then after a left hand movement, the right hemisphere preponderances of both the pure SPN and post-movement negativity would add to a clear right hemisphere preponderance of the measured SPN. After a right hand movement the right hemisphere preponderance of the pure SPN would be counteracted by a left hemisphere

preponderance of the post-movement negativity and a bilaterally symmetrical SPN would be recorded. For hand movements the interaction between SPN and post-movement negativity presumably is strong because both are maximally lateralised at lateral central and temporal sites (Damen and Brunia, 1994). But what happens after a foot movement? Because, to our knowledge, only recordings of post-movement negativity up to 1 s after foot movements exist, its scalp distribution at 2 s post-movement is unknown. If one could assume that its lateral asymmetry is maximal near the midline and decreases in lateral directions, its interaction with the more laterally located SPN would be less than following finger movements. This would explain the right hemisphere preponderance following a movement of either foot. This interpretation needs further verification by recordings of movement-related potentials from multiple recording sites in a 2 s epoch following the movement. Finally, it should be noted that an experimental design aimed at avoiding temporal overlap of different cortical potential shifts is no guarantee for their actual temporal separation.

Acknowledgements

This research was made possible by a grant from the Dutch Organisation for Scientific Research (NWO) to C.H.M.B. (grant no. NWO 560-265-30).

References

Böcker, K.B.E., Brunia, C.H.M. and van den Berg-Lenssen, M.M.C. A spatiotemporal dipole model of the Stimulus Preceding Negativity (SPN) prior to feedback stimuli. Brain Topogr., 1994, 7: 71–88.

Boschert, J., Hink, R.F. and Deecke, L. Finger movement versus toe movement-related potentials: further evidence for Supplementary Motor Area (SMA) participation prior to voluntary action. Exp. Brain Res., 1983a, 52: 73–80.

Boschert, J., Brickett, P., Weinberg, H, and Deecke, L. Movement-related potentials preceding toe plantarflexion and dorsiflexion. Hum. Neurobiol., 1983b, 2: 87–90.

Brunia, C.H.M. and Damen, E.J.P. Distribution of slow potentials related to motor preparation and stimulus anticipation in a time estimation task. Electroenceph. clin. Neurophysiol., 1988, 69: 234–243.

Brunia, C.H.M. and Dautzenberg, J.E.M.W. Cortical potentials in man preceding a plantar flexion and dorsiflexion of the foot. In: W.C. McCallum, R. Zappoli and F. Denoth (Eds.), Cerebral Psychophysiology: Studies in Event-Related Potentials. Elsevier, Amsterdam, 1986, pp. 238–241.

Brunia, C.H.M. and Haagh, S.A.V.M. Preparation for action: slow potentials and EMG. In: H. Heuer and A. Sanders (Eds.), Perspectives on Perception and Action. Springer, Berlin, 1986, pp. 28–40.

Brunia, C.H.M. and van den Bosch, W.E.J. The influence of response side on the readiness potential prior to finger and foot movements. In: R. Karrer and P. Tueting (Eds.), Brain and Information: Event-Related Potentials. New York Academy of Sciences, New York, 1984a, pp. 434–437.

Brunia, C.H.M. and van den Bosch, W.E.J. Movement-related slow potentials, I: a contrast between finger and foot movements in right-handed subjects. Electroenceph. clin. Neurophysiol., 1984b, 57: 515–527.

Brunia, C.H.M. and Vingerhoets, A.J.J.M. Opposite hemisphere differences in movement-related potentials preceding foot and finger flexions. Biol. Psychol., 1981, 13: 261–269.

Brunia, C.H.M., Voorn, F.J. and Berger, M.P.F. Movement-related slow potentials, II: a contrast between finger and foot movements in left-handed subjects. Electroenceph. clin. Neurophysiol., 1985, 60: 135–145.

Chwilla, D.J. and Brunia, C.H.M. Event-related potentials to different feedback stimuli. Psychophysiology, 1991, 28: 123–132.

Damen, E.J.P. and Brunia, C.H.M. Changes in heart rate and slow brain potentials related to motor preparation and stimulus anticipation in a time estimation task. Psychophysiology, 1987, 24: 700–713.

Damen, E.J.P. and Brunia, C.H.M. Is the occurrence of the SPN restricted to feedback stimuli? In: C.H.M. Brunia, A.W.K. Gaillard and A. Kok (Eds.), Psychophysiological Brain Research, Vol. 1. Tilburg University Press, Tilburg, 1990, pp. 187–191.

Damen, E.J.P. and Brunia, C.H.M. Is a stimulus conveying task-relevant information a sufficient condition to elicit a Stimulus-Preceding Negativity (SPN)? Psychophysiology, 1994, 31: 129–139.

Grünewald, G. and Grünewald-Zuberbier, E. Cerebral potentials during voluntary ramp movements in aiming tasks. In: A.W.K. Gaillard and W. Ritter (Eds.), Tutorials in ERP Research: Endogenous Components. Elsevier, Amsterdam, 1983, pp. 311–327.

Grünewald, G., Grünewald-Zuberbier, E., Hömberg, V. and Schuhmacher, H. Hemispheric asymmetry of feedback-related slow negative potential shifts in a positioning movement task. In: R. Karrer and P. Tueting (Eds.), Brain and Information: Event-Related Potentials. New York Academy of Sciences, New York, 1984, pp. 470–476.

Grünewald-Zuberbier, E., Grünewald, G., Runge, H., Netz, J. and Hömberg, V. Cerebral potentials during skilled slow positioning movements. Biol. Psychol., 1981, 13: 71–87.

Hink, R.F., Kohler, H., Deecke, L. and Kornhuber, H.H. Risk-taking and the human Bereitschaftspotential. Electroenceph. clin. Neurophysiol., 1982, 53: 361–373.

Kornhuber, H.H. and Deecke, L. Hirnpotentialänderungen bei Willkürbewegungen und passiven Bewegungen des Menschen: Bereitschaftspotential und reafferente Potentiale. Pflügers Arch., 1965, 284: 1–17.

McAdam, D.W. and Seales, D.M. Bereitschaftspotential enhancement with increased level of motivation. Electroenceph. clin. Neurophysiol., 1969, 27: 73–75.

McCarthy, G. and Wood, C.C. Scalp distributions of event-related potentials: an ambiguity associated with analysis of variance models. Electroenceph. clin. Neurophysiol., 1985, 62: 203–208.

O'Brien, R.G. and Kaiser, M.K. MANOVA method for analyzing repeated measures designs: an extensive primer. Psychol. Bull., 1985, 97: 316–333.

Oldfield, R.C. The assessment and analysis of handedness: the Edinburgh Inventory. Neuropsychology, 1971, 9: 97–113.

Shibasaki, H., Barrett, G., Halliday, E. and Halliday, A.M. Cortical potentials associated with foot movement in man. Electroenceph. clin. Neurophysiol., 1981, 49: 213–226.

Vaughan, Jr., H.G., Costa, L.D. and Ritter, W. Topography of the human motor potential. Electroenceph. clin. Neurophysiol., 1968, 25: 1–10.