



**HAL**  
open science

## Motor Preparation of Step Initiation: Error-related Cortical Oscillations

Arnaud Delval, Aurore Braquet, Nauaman Dirhoussi, Madli Bayot, Philippe Derambure, Luc Defebvre, Celine Tard, Kathy Dujardin

► **To cite this version:**

Arnaud Delval, Aurore Braquet, Nauaman Dirhoussi, Madli Bayot, Philippe Derambure, et al.. Motor Preparation of Step Initiation: Error-related Cortical Oscillations. Neuroscience, 2018, Neuroscience, pp.12-23. 10.1016/j.neuroscience.2018.09.046 . hal-02178934v3

**HAL Id: hal-02178934**

**<https://hal.univ-lille.fr/hal-02178934v3>**

Submitted on 22 Nov 2019 (v3), last revised 26 Nov 2020 (v5)

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

## Motor Preparation of Step Initiation: Error-related Cortical Oscillations

Arnaud Delval,<sup>a,b,\*</sup> Aurore Braquet,<sup>a,b</sup> Nauaman Dirhoussi,<sup>a,b</sup> Madli Bayot,<sup>a,b</sup> Philippe Derambure,<sup>a,b</sup> Luc Defebvre,<sup>a,b</sup> Céline Tard,<sup>a,b</sup> and Kathy Dujardin<sup>a,b</sup>

<sup>a</sup> Univ. Lille, CHU Lille, INSERM, U1171 – Degenerative & Vascular Cognitive Disorders, F-59000 Lille, France

<sup>b</sup> Lille Centre of Excellence for Neurodegenerative Diseases (LiCEND), F-59000 Lille, France

**Abstract**—Gait initiation can vary as a function of the available and engaged attentional resources. Conflict resolution can disrupt movement preparation and lead to “errors” in motor programming. These “errors” are physiologically useful by enabling us to adapt our motor behavior to situations with conflicting information. The objective of the present study was to analyze the patterns of cortical activation associated with motor programming errors and the corresponding error corrections. Incongruent flankers around a target arrow were used to trigger errors in anticipatory postural adjustments (APAs) prior to gait initiation; i.e. perturbed motor programming but normal execution. Thirty healthy adults performed a gait initiation task. The event-related potentials (ERPs) and event-related desynchronization (ERD) after target presentation were analyzed according to the presence or absence of an APA error. The ERP was similar in both conditions, except that the Ne and P300 peak latencies were longer for APA errors. Motor programming errors during gait initiation were characterized by longer, less intense low-beta-band ERD over the sensorimotor cortex and alpha ERS followed by stronger alpha ERD during errors. APA errors were associated with a specific alpha/beta oscillation profile over the sensorimotor cortex; these beta oscillations might be sensitive markers of non-conscious motor error and correction monitoring. © 2018 IBRO. Published by Elsevier Ltd. All rights reserved.

**Key words:** cortical activation, gait initiation, posture, inhibition, attention.

### INTRODUCTION

Gait initiation is a motor program characterized by the transition from a static stable stance to a continuously unstable posture during locomotion. The characteristics of gait initiation can vary as a function of the available attentional resources. Indeed, gait initiation can be modulated when the subject is obliged to deal with conflicting information (Uemura et al., 2012). Gait is initiated in two phases: a motor preparation phase (corresponding to anticipatory postural adjustments, APAs) and then an execution phase (corresponding to the time interval between “toe-off” and “heel strike” for the swing leg). During standard gait initiation (i.e. in the absence of external or internal stimuli requiring modulation of the motor program), healthy subjects display a stereotypical APA pattern. Foot-off of the swing leg is preceded by a shift in body weight that displaces the center of pressure

(CoP) backward and toward the swing leg. Next, the CoP is displaced forward and toward the stance leg. Hence, APAs create the conditions required for progression (Brenière and Do, 1991). Furthermore, APAs along the mediolateral axis are predictive of postural stability (McIlroy and Maki, 1999). However, it is known that self-triggered gait initiation is not always preceded by an APA (Delval et al., 2014; Lu et al., 2017). The lack of a lateral or posterior APA was nevertheless infrequent (in 2% of the trials) in healthy elderly controls during externally triggered rapid stepping (Delval et al., 2014). Conversely, the absence of APAs can be frequently observed in patients with freezing of gait and an increased risk of falls (Delval et al., 2014). The occurrence of APA errors can also perturb the gait initiation program by delaying the onset of movement execution (Cohen et al., 2011). This corresponds to the correction of an APA when the initial direction of postural adjustment is not appropriate (for example, when the CoP moves inappropriately toward the stance leg and is then appropriately moved first toward the swing leg and only then toward the stance leg). This APA error corresponds to a motor program error, which is efficiently corrected and prevents incorrect step initiation from taking place. It is known that APA errors are more frequent in conditions modulated by attention (especially in the presence of incongruent stimuli

\*Correspondence to: A. Delval, Univ. Lille, CHU Lille, INSERM, U1171 – Degenerative & Vascular Cognitive Disorders, F-59000 Lille, France.

E-mail address: [arnaud.delval@chru-lille.fr](mailto:arnaud.delval@chru-lille.fr) (A. Delval).

**Abbreviations:** APAs, anticipatory postural adjustments; CoP, center of pressure; CRN, Correct-Related Negativity; EEG, electroencephalogram; ERD, event-related desynchronization; ERD/S, event-related (de)synchronization; ERN, Error-Related Negativity; ERPs, event-related potentials; Pe, Positive error-related.

(Uemura et al., 2013) or with invalid cues (Tard et al., 2013)) than in conditions with congruent step initiation stimuli. However, it is not known if and how these APA errors are modulated in the cortex.

Cortical areas involved in gait initiation include the sensorimotor cortex, premotor cortex, basal ganglia and brainstem structures. It was initially suggested that the motor programs underlying the elicitation of gait initiation were stored in subcortical structures, and could be elicited by a startling stimulus or a decision for action (Takakusaki, 2008; Queralt et al., 2010; Delval et al., 2012; Watanabe et al., 2016a). However, studies in patients with focal lesions of the supplementary motor area and studies in patients with Parkinson's disease (Viallet et al., 1992; Gantchev et al., 1996) have shown that APAs can be modulated at the supraspinal level, since the supplementary motor area, the basal ganglia and the pontomedullary reticular formation are linked by neural networks. Moreover, inhibitory repetitive transcranial magnetic stimulation over the supplementary motor area shortens the APA duration for a brief period, i.e. for the first stepping trial after stimulation (Jacobs et al., 2009). The output of this pathway is located in the mid-brain locomotor region (which may correspond in part to the cuneiform nucleus and the dorsal part of the pedunculopontine nucleus), which is connected to limbic structures and the basal ganglia (Pahapill and Lozano, 2000).

Attentional control can modulate gait initiation – either directly by involving brainstem structures (for example, the alert process induced by a loud stimulus can product a start-react effect) or indirectly via a cortical loop that includes more complex attentional networks (Delval et al., 2012; Tard et al., 2013). Cortical movement preparation can be measured through electroencephalogram (EEG) features like event-related potentials (ERPs) and event-related (de)synchronization (ERD/S). In the frequency domain, ERD (mainly in the alpha- and beta-bands) is the cortical marker of movement intention. It has been demonstrated that gait initiation is associated with desynchronization of sensorimotor rhythms related to sensorimotor cortex activation (Pfurtscheller and Andrew, 1999). If the EEG is response-locked (i.e. locked to the motor response), a movement-related cortical potential (MRCP) is present before gait initiation or when imagining gait initiation (Vidailhet et al., 1993, 1995). For the gait initiation task, if the EEG is target-locked, the early ERP components are probably influenced by the different physical characteristics of the stimuli (Rektor et al., 2006) and a posterior P300 can be found, higher in case of stimulus driven attention for example (Tard et al., 2013), whereas late components reflect motor preparation (Hamano et al., 1997). More recently, combined ERP and ERD/S recordings via an EEG brain–computer interface were used to detect gait initiation (Jiang et al., 2015; Sburlea et al., 2015).

ERPs are also used to monitor cognitive control of action. During error recognition, a negativity (named the “Error-Related Negativity” (ERN or Ne)) and then a “Positive error-related wave” (Pe) can be observed (for a review, see Wessel and Aron (2017)). The functional significance of ERN was associated with error detection

(Falkenstein et al., 1991). Alternatively, the ERN was proposed to reflect conflict resolution due to a finding of the “Correct-Related-Negativity” (CRN) (Vidal et al., 2000; Meckler et al., 2011). However, errors during gait initiation are mostly non-conscious and the presence of an ERN or Pe during an APA error in healthy subjects remains uncertain. For example, Watanabe found similar frontal ERN and CRN in trials with or without APA errors during gait initiation (Watanabe et al., 2016b). The significance of these potentials remains discussed. Indeed, it has been demonstrated that the ERN occurs also after “partial errors”, i.e., incorrect activities that are not sufficient to produce overt errors (Carbónnell and Falkenstein, 2006), which is observed during spontaneous correction of APA errors. To date, the cortical areas involved in gait initiation errors have not been extensively studied and the focus was only on Fz, FCz, and Cz (Watanabe et al., 2016b). Indeed, the human sensorimotor system needs to be able to rapidly correct for errors in an ongoing motor command brought about by sudden, unexpected changes in the movement environment (such as conflicting information, for example) (Krigolson et al., 2008). The present study was designed to evaluate the cortical changes induced by these adaptive reactions called APA errors. The study's primary objective was to use a combined ERP and time–frequency analysis to evaluate cortical activation during correct gait initiation (i.e. with no APA errors) and during disturbed step initiation (i.e. with APA errors). Our starting hypothesis was that APA errors would be associated with ERP modulations featuring error-related potentials (for example, error-related negativity/positivity (Ne/Pe) (Falkenstein et al., 2000)) and/or changes in beta-band ERS, for example increased beta ERS, as observed in stop-signal paradigms for movements requiring motor inhibition (Duque et al., 2017). Modulations in lower bands (delta–theta) have also been attributed to error monitoring in children, young and elderly adults (Kolev et al., 2001, 2005; Albrecht et al., 2009).

## EXPERIMENTAL PROCEDURES

### Participants

Thirty healthy adult volunteers (16 females, 14 males; 29 right-handed; mean  $\pm$  standard deviation (SD) age: 39.4  $\pm$  14.2 years) participated in the study after providing written, informed consent. None of the participants had a history of medication use (neuroleptics, benzodiazepines, etc.) or disease (neurological, orthopedic or psychiatric) that could have interfered with gait. The mean  $\pm$  SD Montreal Cognitive Assessment score (Nasreddine et al., 2005) was 28.5  $\pm$  2. Participants with a score of less than 26 out of 30 were excluded. The study was approved by the local independent ethics committee (CPP Nord-Ouest, Lille, France; reference: 2015-A00013-46).

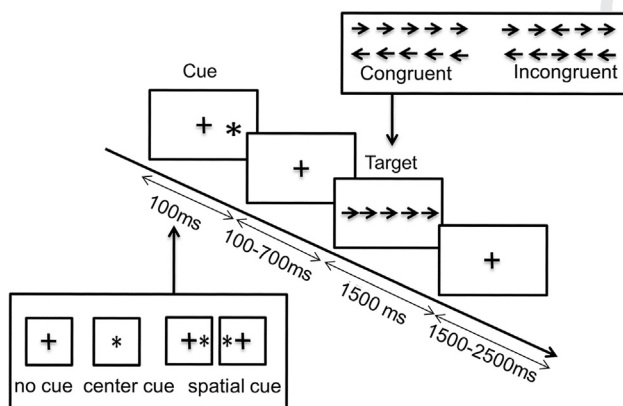
### The experimental setting

The participant was told to stand in a stable, comfortable, natural posture on a force platform, with his/her feet

171 parallel and with a gap of a few centimeters between the feet. A computer display screen was placed at head  
172 height 1 meter in front of the participant. The attentional task was an adaptation of the attentional network test  
173 (Fan et al., 2002) (Fig. 1). The participant was instructed to initiate a forward step after presentation of the visual  
174 target (an arrow pointing to the right or to the left, which was visible for 1500 ms). If the arrow pointed to the left,  
175 the participant had to initiate gait with the left foot; conversely, if the arrow pointed to the right, the participant  
176 had to initiate gait with the right foot. The balance weight shift between the 2 feet was controlled online (position of  
177 the CoP between the 2 feet visualized by the Nexus software). The present study only assessed the condition with  
178 incongruent flankers (i.e. flankers pointing in the opposite direction to the target arrow), in which the frequency of  
179 APA errors is reportedly higher (Uemura et al., 2013). Indeed, differences in ERP amplitudes have been  
180 reported between congruent and incongruent conditions in a similar study design in seated condition (Neuhaus  
181 et al., 2010). A total of 144 incongruent trials (out of a total of 300) were available for each participant.

193 **Motion analysis**

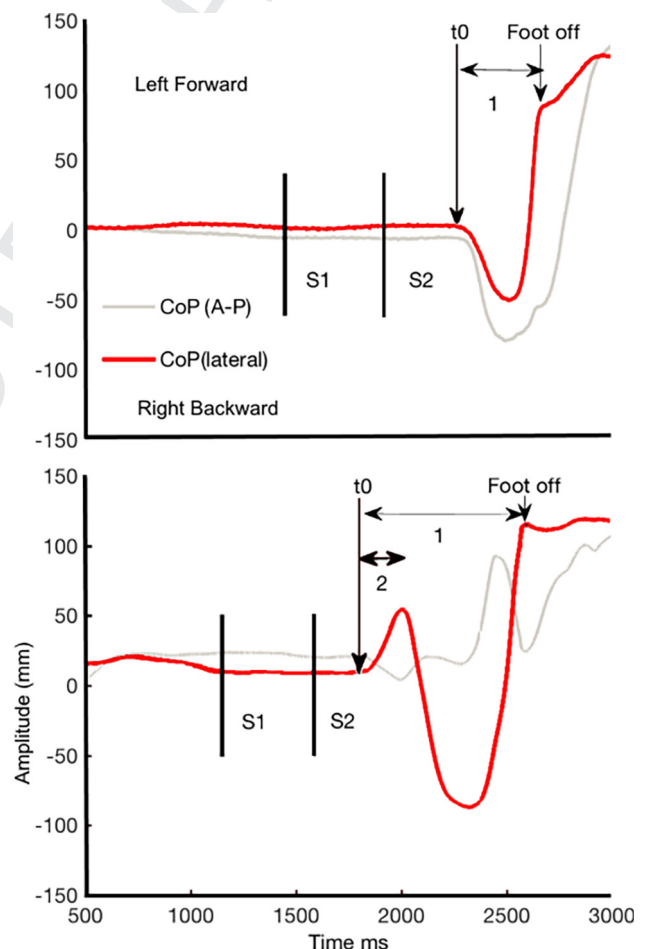
194 Data were collected with a three-dimensional motion analysis system (VICON 370®, Oxford Biometrics,  
195 Oxford, UK), using eight infrared cameras and a sampling frequency of 100 Hz. The CoP was measured  
196 with two force platforms (the ORG-5 model from AMTI®, Watertown, MA, USA) at a sampling frequency  
197 of 1000 Hz. Reflective markers were placed on precise, reproducible, anatomic landmarks on each foot: the toe



198 **Fig. 1.** Attentional Network test. A schematic diagram of the  
199 attentional task. S1: cue; S2: target. The target appeared for  
200 1500 ms and was surrounded by flankers (four arrows: two on each  
201 side) that were either congruent or incongruent. The targets could  
be preceded by valid or invalid spatial cues, i.e. asterisks indicating  
(or not) the direction of the arrow. Four blocks of 75 trials were  
administered. The blocks were separated by short breaks of variable  
duration. The cue and target conditions were presented in a pseudo-  
random order: 156 congruent trials, 144 incongruent trials, 49 trials  
with no cue, 61 trials with a central cue, and 190 trials with a spatial  
cue (142 valid and 48 invalid cues). Only incongruent trials were  
analyzed in the present study. The proportion of no cue trials was  
16.3%, the proportion of neutral cue trials was 20.3%, the proportion  
of valid cue trials was 47.3%, and the proportion of invalid cue trials  
was 16%.

202 (the head of the second metatarsal), the lateral  
203 malleolus, and the heel. The data were then computed  
204 by the same operator using an in-house MATLAB®  
205 routine (The MathWorks, Natick, MA, USA).

206 The direction of the APA was considered to be normal  
207 if the CoP moved backward and sideways toward the  
208 swing foot. Conversely, the direction of the APA was  
209 considered to be abnormal (i.e. an APA error) if the CoP  
210 moved first toward the stance foot and only then toward  
211 the swing foot (see Fig. 2). The reaction time (RT) was  
212 defined as the time interval between the appearance of  
213 the target (S2) and the beginning of the APA or  $T_0$ . An  
214 RT < 100 ms was classified as a false start and was  
215 excluded from further analyses. Incorrect starts (i.e.  
216 starts with the wrong foot) were also excluded. An in-  
217 house MATLAB® routine detected changes in CoP  
218 velocity > mean + 3 SD of the baseline period (-1500  
219 to 1000 ms before target stimulus), the experimenter  
220 then chose the start of the APAs according to the  
221 curves in X and Y axis. Toe-off was detected visually



222 **Fig. 2.** Normal Anticipatory Postural Adjustments (APA) and APA  
223 error. Step initiation with the right foot by a study participant. Top  
224 panel: a normal APA, where the center of pressure (CoP) shifts to the  
225 swing leg (right) and then to the stance leg (left) (lateral CoP: red  
226 line). Bottom panel: an APA error: the CoP shifts toward the stance  
227 leg (left) but the trajectory is corrected. (For interpretation of the  
228 references to color in this figure legend, the reader is referred to the  
229 web version of this article.)

(from the toe marker trajectory in the sagittal plane) and then reported on the CoP curve. It corresponded to the time the CoP shifted forward. The APA duration was assessed by subtracting  $T_0$  from the toe-off time. For APA errors, the correction time was defined as the time interval between  $T_0$  and the sideways corrective shift (i.e. the beginning of the APA in the correct direction).

#### 229 Acquisition of electroencephalographic data

230 The electroencephalogram (EEG) was recorded with an  
231 Ag/AgCl 128-scalp-electrode cap (Waveguard®, ANT  
232 Neuro, Enschede, The Netherlands), positioned  
233 according to the 10/05 international system (Oostenveld  
234 and Praamstra, 2001). Data were acquired with ASA™  
235 software (ANT Neuro), using a 0.01- to 100-Hz band-  
236 pass filter, a sampling frequency of 512 Hz, and impe-  
237 dances below 20 kΩ. The data were pre-processed with  
238 ASA™ software in order to reject ocular artifacts and apply  
239 a 50-Hz notch filter to the recordings. Next, interpolation  
240 was performed for artifact-affected electrodes, with a  
241 maximum interpolation rate of 10% ( $n = 13$ ) for the whole  
242 set of 128 scalp electrodes.

243 The EEG data were then analyzed with Brain Vision  
244 Analyzer 2.0 software (BrainProducts). Muscle artifacts  
245 were manually removed from the EEG layout;  
246 thereafter, we segmented the EEG data into 2500-ms  
247 epochs that were time-locked with respect to target  
248 onset (1500 ms before and 1000 ms afterward). The  
249 median (min–max) number of epochs selected (after  
250 artifact rejection) per participant was 49 (20–98) for APA  
251 errors and 69 (38–107) for normal APAs.

252 *ERP analyses.* ERP were analyzed with the EEGLAB  
253 toolbox (Delorme and Makeig, 2004), using a baseline  
254 from 1500 ms before the target onset to 1000 ms for  
255 target-locked ERP and from 1500 ms before the APA  
256 onset to 650 ms for response-locked ERP. The time win-  
257 dows analysis was from S2 to 1000 ms after target onset.  
258 We first analyzed ERP scalp distribution maps. Next,  
259 the ERP wave's characteristics were assessed and col-  
260 lected by grand-averaging over the main central elec-  
261 trodes (Fz, Cz, Pz).

262 For target-locked ERP, we focused on P300 over Pz.  
263 For response-locked ERP, we analyzed ERN/CRN over  
264 Fz and P300/Pe over Pz. The amplitude of potentials  
265 was measured as the difference between the maximum  
266 peak of the ERP waveform and the mean baseline  
267 voltage (which occurs prior to the stimulus). Latency  
268 was defined as the interval between target presentation  
269 and the point of highest positive amplitude in the time  
270 window of the potential. For target-locked ERP, time  
271 window of P300 ranged from 250 to 500 ms after target  
272 presentation. For response-locked ERP, the time  
273 window of the ERN/CRN ranged from –50 to 200 ms  
274 after APA start and from 0 to 400 ms for the posterior  
275 component.

276 *ERD/ERS analyses.* Time–frequency analysis  
277 requires computing the power spectrum over a sliding  
278 latency window. ERD data were analyzed using  
279 EEGLAB software (Delorme and Makeig, 2004) with a

500-ms baseline (between 1500 and 1000 ms before 280  
the target's appearance, target-locked and response- 281  
locked). To characterize event-related EEG oscillations 282  
like ERD and ERS, we applied a time–frequency analysis 283  
based on a continuous wavelet transform. We used a ver- 284  
sion of sinusoidal wavelets in which the number of cycles 285  
increases slowly with frequency (e.g. 1.5 cycles at 4 Hz, 286  
and 5.6 cycles at 30 Hz) with a window width of 213 sam- 287  
ples (416 ms). This procedure has been described in 288  
(Delorme and Makeig, 2004), and similar approaches for 289  
time windows of around 2500 ms have been described 290  
in (Fan et al., 2007). Time–frequency analyses were per- 291  
formed between 4–7 Hz (the theta-band), 8–12 Hz (the 292  
alpha-band), and 13–30 Hz (the beta-band, divided in a 293  
low-beta-band (13–20 Hz) and a high-beta-band (20– 294  
30 Hz)). 295

*Cortical sources.* A realistic head model was built by 296  
segmenting a template MRI data with Freesurfer 297  
software (Dale et al., 1999). The lead field matrix was 298  
then computed for a cortical mesh with 15,000 vertices, 299  
using Brainstorm software (Tadel et al., 2011) and Open- 300  
MEEG software (Gramfort et al., 2010). The weighted 301  
minimum-norm estimate was then used to reconstruct 302  
the cortical sources (using Brainstorm toolbox (Tadel 303  
et al., 2011)) in the time window corresponding to motor 304  
preparation. 305

#### 306 Statistical analyses

307 Characteristics of APAs were compared using a one-way 308  
ANOVA after checking normality of the distributions. To 309  
evaluate differences in cortical activation (ERPs, ERD/S 310  
and source localization) in trials with an APA error vs. 311  
trials with a normal APA, we used a non-parametric 312  
permutation (randomization) test to obtain the p-value 313  
for each electrode and each time point (for ERP and 314  
ERD/S) and for each source reconstruction. The false 315  
discovery rate (FDR) method was used to correct for 316  
multiple comparisons (Genovese et al., 2002), and 317  
enabled us to determine which electrodes differed 318  
between the two conditions at the different time points 319  
(scalp maps) and to compare ERD/ERS maps between 320  
both conditions. These analyses were performed with 321  
the EEGLAB toolbox (Delorme and Makeig, 2004), which 322  
includes MATLAB statistical routines at this purpose. For 323  
comparisons between source localizations, we used the 324  
scripts included in the Brainstorm toolbox (Tadel et al., 325  
2011)). Peak amplitudes and latencies (for P300 on Pz, 326  
for example) were compared in a t-test (in SPSS 17 for 327  
Windows) after checking the normality of distribution in 328  
a Kolmogorov–Smirnov test. The threshold for statistical 329  
significance was set to  $p < 0.05$  for all analyses.

## 330 RESULTS

### 331 Behavioral data

In trials with incongruent flankers, the APA error rate was 332  
41.0%. 333

*The false start rate.* The false start rate (i.e. 334  
RTs < 100 ms) was 11.9%. These trials were excluded 335

336 from further analysis, since they did not correspond to  
337 APA errors.

338 *The error step rate.* A start with the wrong foot was  
339 rare, since it occurred in only 0.56% of the trials. These  
340 trials were excluded from the analysis because they did  
341 not correspond to correct error monitoring. The low  
342 number of these events prevented us from analyzing  
343 them separately.

344 The mean  $\pm$  SD RT was  $0.27 \pm 0.08$  s for normal  
345 APAs and  $0.23 \pm 0.06$  s for APA errors ( $p < 0.001$ ).  
346 The mean  $\pm$  SD APA duration was longer for APA  
347 errors ( $0.64 \pm 0.13$  s) than for normal APAs ( $0.47$   
348  $\pm 0.10$  s) ( $p < 0.001$ ). The mean  $\pm$  SD correction time  
349 for APA errors was  $0.20 \pm 0.07$  s (i.e.  $0.43 \pm 0.07$  s  
350 after target presentation, on average).

### 351 Erp

352 *Target-locked ERP:* as shown in Fig. 3, the ERP scalp  
353 distribution maps revealed an early anterior component  
354 (N2, see discussion), a late central negative component  
355 (corresponding to preparation of movement) and a  
356 posterior positive component (at the same scalp sites as  
357 the P300). There were no differences in the ERP maps  
358 between the “APA error” and “normal APA” conditions  
359 except for P300 component (see Figs. 3 and 4).

360 The mean  $\pm$  SD P300 peak latency (Pz electrode)  
361 was longer for the APA error condition than the normal  
362 APA condition ( $0.50 \pm 0.08$  s vs.  $0.47 \pm 0.08$  s,  
363 respectively;  $p < 0.01$ ). No differences in P300 peak  
364 amplitude were observed.

365 *Response-locked:* in both APA conditions, early  
366 negativity (in the time window of ERN or CRN)  
367 occurred, later in case of APA error. Late positivity  
368 (P300 or Pe, see discussion) also occurred in both  
369 conditions, later in case of APA error on posterior  
370 regions. The mean  $\pm$  SD ERN/CRN peak latency (Fz  
371 electrode) was longer for the APA error condition than  
372 the normal APA condition ( $0.12 \pm 0.05$  s vs.  $0.08$   
373  $\pm 0.05$  s, respectively;  $p < 0.001$ ). No differences in  
374 peak amplitude were observed. The mean  $\pm$  SD Pe/  
375 P300 peak latency (Pz electrode) was longer for the  
376 APA error condition than the normal APA condition  
377 ( $0.26 \pm 0.07$  s vs.  $0.19 \pm 0.06$  s, respectively;  
378  $p < 0.001$ ). No differences in peak amplitude were  
379 observed.

### 380 ERD data

381 As shown in Figs. 5 and 6, we observed similar theta-  
382 band ERS (between 200 and 600 ms, target-locked;  
383 starting at T0, response-locked) in both conditions.

384 Alpha ERS was significantly more pronounced in trials  
385 with an APA error (starting around 300 ms after S2,  
386 target-locked; during APA, response-locked) and was  
387 followed by a stronger alpha ERD (response-locked).

388 Central beta ERD was observed, starting 200 ms after  
389 S2 (target-locked); or just before T0 (response-locked).  
390 This feature lasted significantly longer over Cz in trials  
391 with an APA error (Fig. 6). Moreover, beta ERD over  
392 the sensorimotor cortex was more attenuated in the low-  
393 beta-band (i.e. 13–20 Hz) than in the high-beta-band

(20–30 Hz) in trials with an APA error (relative to trials  
with a normal APA) in target-locked analysis (Figs. 5  
and 6). We can observe that this beta ERD was present  
during both normal APAs and APA errors but was more  
prolonged (response-locked and target-locked) in case  
of error.

### Cortical sources of changes in the EEG signal during motor programming

Cortical sources in the 0- to 600-ms time interval (target-  
locked) are shown in Fig. 7. Occipital and temporoparietal  
regions were activated at 200 ms, and then the  
sensorimotor cortex and the frontal dorsolateral cortex  
were activated during both normal APAs and APA errors.

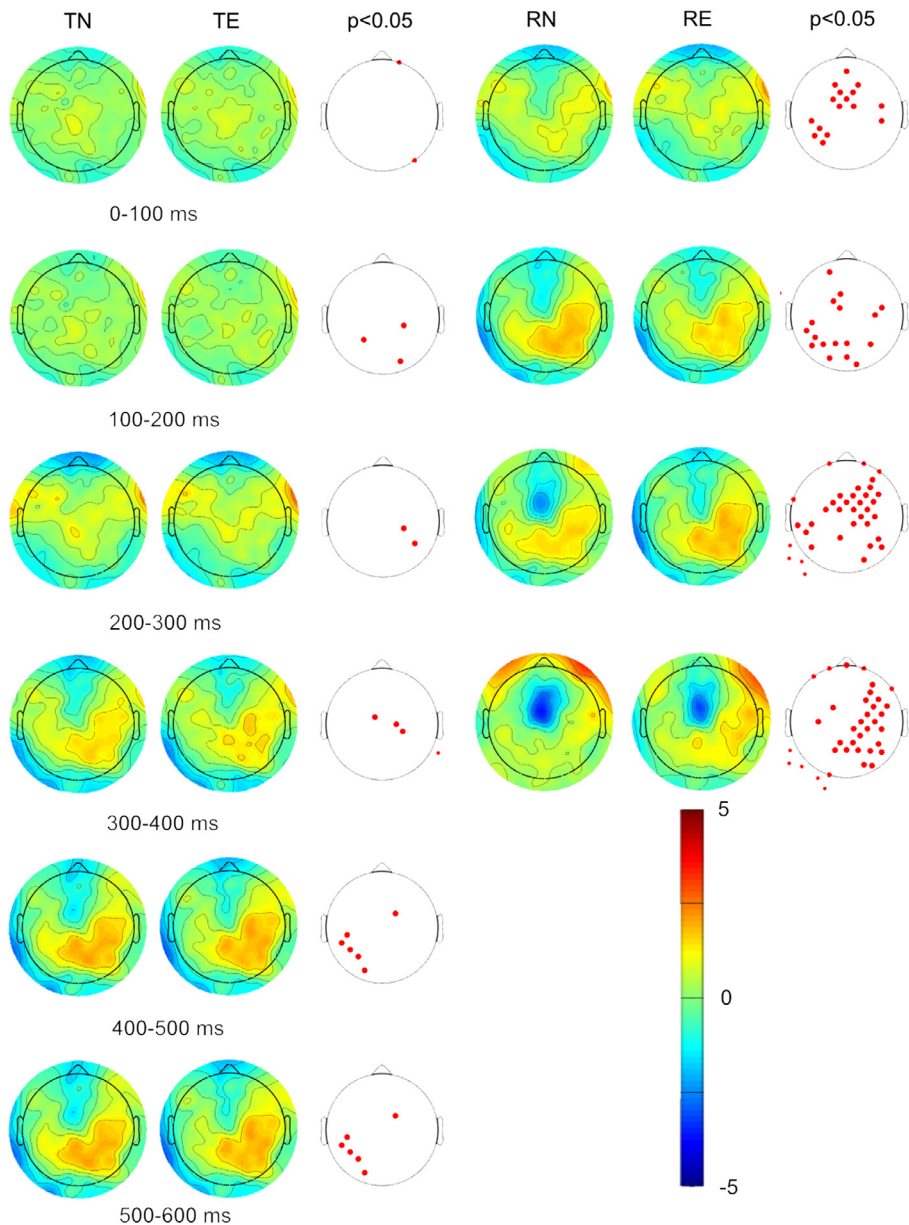
## DISCUSSION

Our present results showed that an error in motor  
programming during gait initiation in healthy subjects  
was not associated with obvious differences in ERPs.  
We only observed a longer P300 peak latency in trials  
with an APA error. However, we observed extended  
beta ERD over the sensorimotor cortex, and more  
pronounced alpha ERS followed by an ERD in trials with  
an APA error.

### Are APA errors low-level errors?

It is known that the motor program can be adjusted during  
APAs. This process might involve rapid, direct  
sensorimotor loops via visual afferences for stimulus  
detection and proprioceptive afferences for the ongoing  
APA. Hence, healthy subjects are able to adjust the  
motor program after it has started by delaying foot lift  
until the correct motor program has been selected.  
Response inhibition – the ability to rapidly cancel an  
action – is a critical component of executive function. In  
gait initiation, response inhibition quickly corrected APAs  
initiated in the wrong direction (around 200 ms after the  
start of the APA error (Tard et al., 2015)). This means that  
subjects can react to the perception of conflicting informa-  
tion and quickly reorient ongoing actions. Many research-  
ers have investigated the neural substrates of behavioral  
inhibition by applying laboratory tasks based on the stop-  
signal paradigm and that require a planned action to be  
stopped (Duque et al., 2017); however, these tasks  
require complete inhibition of the motor program, rather  
than just correction (as in APA errors).

Moreover, the different types of errors described in the  
literature appear to have different neural bases: Hill and  
Raab (Hill and Raab, 2005) first distinguished the correc-  
tion of errors induced externally and internal errors gener-  
ated by the subject itself. Another distinction has been  
made between low-level errors (i.e. non-conscious,  
quickly corrected errors) involving posterior regions of  
the brain, and high-level errors (i.e. conscious errors that  
are not always corrected) involving the medial frontal lobe  
(Krigolson and Holroyd, 2007). The errors in our study  
would be classified as internal, low-level, since the partici-  
pants were not aware of them; although some partici-



**Fig. 3.** Event-related potentials in normal Anticipatory Postural Adjustments (APA) and APA error. Top view of topographic voltage maps for each condition (i.e. a normal APA-N- or an APA error-E-). 0 ms corresponds to either the target presentation (S2), target-locked (T), or to the start of the APAs (response-locked:R). Cold and hot colors correspond to negative and positive ERPs, respectively. Red dots correspond to electrodes with differences between conditions (whatever the direction), as indicated by permutation tests with FDR correction. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

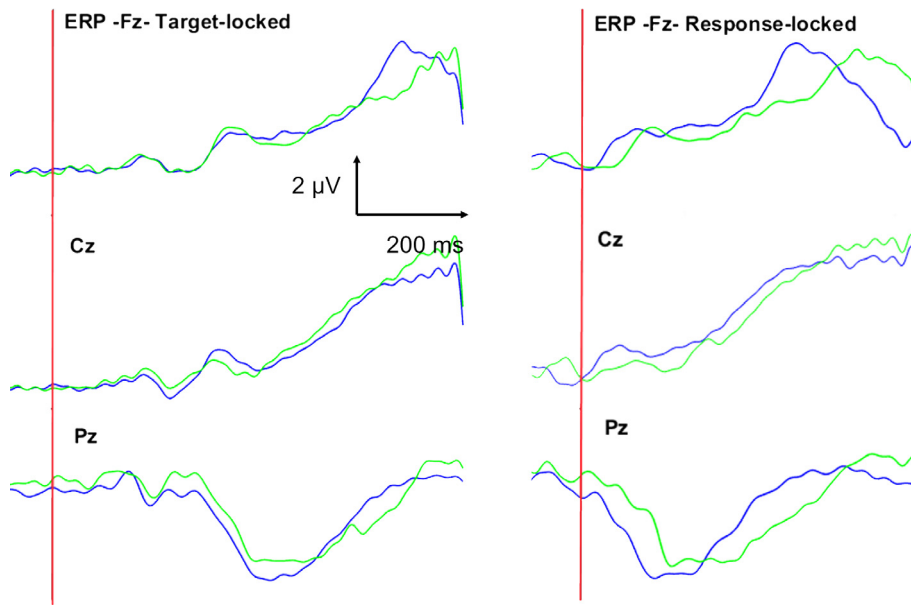
449 pants described “start hesitation” in a few trials, most  
450 were unaware of the quickly corrected motor program.

451 Cortical activations during unexpected events have  
452 recently been reviewed (Wessel and Aron, 2017). Most  
453 of the relevant studies were based on the use of stop-  
454 signal paradigms and fMRI to identify the underlying  
455 motor inhibition system. The right inferior frontal cortex,  
456 pre-supplementary motor area and subthalamic nucleus  
457 (STN) of the basal ganglia are all involved, with down-  
458 stream effects on the pallidum, thalamus, and primary  
459 motor cortex. Indeed, the STN’s role has been empha-

460 sized by several fMRI studies (Aron and Poldrack, 2006; Li  
461 et al., 2008); activity in the STN is greater during both stop successes  
462 and stop errors than in “go” trials,  
463 and greater for stop errors than  
464 for stop successes (Li et al.,  
465 2008). These findings suggest that  
466 the STN has a role in suppressing  
467 thalamocortical output, which  
468 thereby blocks motor response  
469 execution via a hyperdirect path-  
470 way (Aron and Poldrack, 2006).  
471 The mechanisms in our paradigm  
472 were less clear, since error correc-  
473 tion re-oriented a movement rather  
474 than stopping it completely as in  
475 stop-signal paradigms. Subjects  
476 were able to shift their weight  
477 toward the swing leg to correct  
478 the error. This could correspond  
479 to “partial errors”, i.e., incorrect  
480 activities which are not sufficient  
481 to produce overt errors  
482 (Carbonell and Falkenstein,  
483 2006). These latter could produce  
484 both ERN and CRN. However, we  
485 would have expected more ample  
486 negativity over frontal regions in  
487 case of APA errors.  
488  
489

### Cortical markers of error monitoring

490 Evaluating the precise timing of  
491 cortical activations requires  
492 electrophysiological recordings  
493 based on local field potentials or  
494 EEG. It is generally thought that  
495 an anterior component (ERN/Ne)  
496 reflects error inhibition (Kopp  
497 et al., 1996), conflict detection  
498 (Carter et al., 1998) or the  
499 comparison (response checking)  
500 of the neural representation of the  
501 actual (erroneous) response and  
502 the representation of the required  
503 (i.e. correct) response. In our  
504 paradigm, however, successful  
505 error inhibition was followed by a  
506 motor programming correction  
507 and then appropriate movement execution. With regard  
508 to response checking, the participant had to recognize  
509 the engaged motor program (the left or right foot) –  
510 usually a non-conscious process – and determine  
511 whether or not it corresponded to the appropriate  
512 response. This process is much the same in APA errors  
513 and normal APAs.  
514 We did not observe any differences in the ERN/CRN  
515 amplitudes. The amplitudes of CRN and ERN were also  
516 similar for the stepping task in (Watanabe et al., 2016b)  
517 that used a Simon task to elicit APA errors. They pro-  
518  
519



**Fig. 4.** Event-related potentials in normal Anticipatory Postural Adjustments (APA) and APA error in midline derivations. ERP for Fz, Cz and Pz sites in two conditions: blue: grand average of normal APA vs. green, grand average of APA error. 0 ms corresponds to either the target presentation (S2), target-locked, or to the start of the APAs (response-locked). Target-locked: P300 occurred later in case of APA error. Response-locked: negative components (?Ne and CRN) were observed, later in case of APA error. P300 or Pe occurred later in case of APA error. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

520 posed different interpretations: the more convincing one  
 521 in our opinion is that APA errors are brief, covert, and  
 522 are likely corrected by initiating a step with the appropriate  
 523 leg. As a consequence, ERN amplitude may have  
 524 become smaller because APA errors were not recognized  
 525 as definite errors. Once again, this error is not conscious.  
 526 The late, posterior, positive ERP component observed  
 527 after presentation of the target is more difficult to interpret.  
 528 Considering target-locked responses, the ERP peaked in  
 529 the parietal cortex at about 400 ms (target-locked) and  
 530 100–200 ms (response-locked). It might therefore be a  
 531 P300 component. It is very similar to the response-  
 532 locked posterior component. The latency (but not the  
 533 amplitude) differed according to the presence or not of  
 534 an APA error. The absence of a difference in amplitude  
 535 was not very surprising because P300's amplitude is  
 536 primarily modulated by the stimulus's rarity (as in the oddball  
 537 paradigm). Here, only incongruent targets were considered.  
 538 They elicited a large P300, regardless of the forth-  
 539 coming motor preparation (Neuhaus et al., 2010; Deiber  
 540 et al., 2013). Alternatively, the ERP component might cor-  
 541 respond to Pe. The latter is thought to reflect (i) error cor-  
 542 rection, (ii) a delayed parietal P300 (since it is present in  
 543 correct trials) or (iii) additional error processing or post-  
 544 error processing (for a review, see Falkenstein, 2010).  
 545 Here, the distribution is posterior and not anterior. More-  
 546 over, in our paradigm, there were no amplitude differ-  
 547 ences between trials with and without APA errors. It  
 548 must be borne in mind that the variability in Pe depends  
 549 on error detectability: the larger the difference between  
 550 the representations (i.e. the easier the error is to detect),  
 551 the larger and/or earlier the Pe. There are several possi-

552 ble explanations for the lack of differ-  
 553 ence in the amplitude of Pe. The  
 554 participant was not given any infor-  
 555 mation about APA error monitor-  
 556 ing. Indeed, both Ne and Pe are  
 557 closely related to conscious per-  
 558 ception of the error (Nieuwenhuis  
 559 et al., 2001; Charles et al., 2013).  
 560 In fact, in the work by Charles  
 561 et al., the ERN was absent only  
 562 when subjects reported that they  
 563 did not see the target. That was  
 564 not the case in our study since sub-  
 565 jects well identified the target (no  
 566 error of step side).

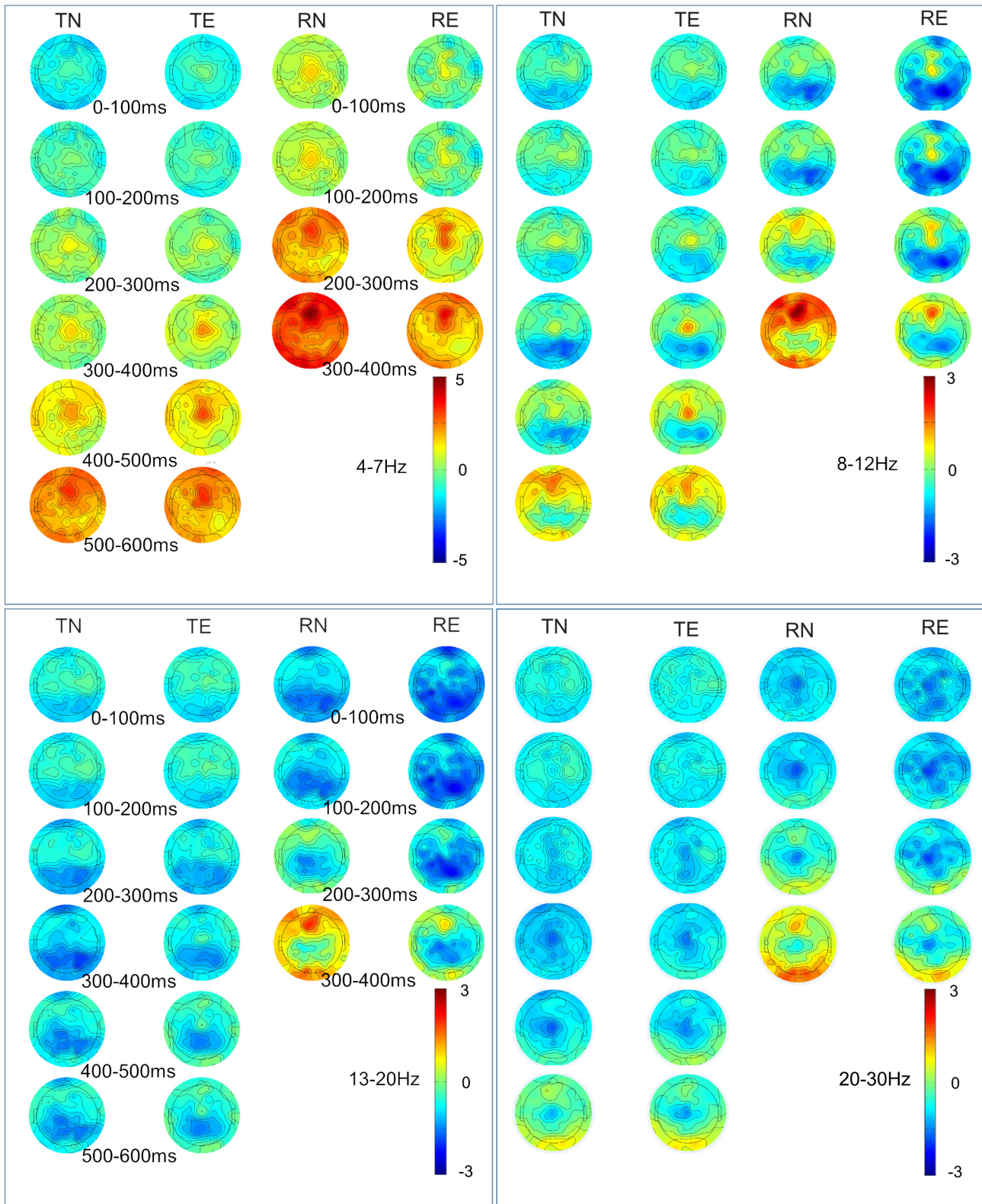
567 Instructing the participant to  
 568 pay attention (or not) to the error  
 569 stimulus (Ramautar et al., 2006)  
 570 can also amplify the ERP. In  
 571 Ramautar et al.'s study, Pe was  
 572 much more pronounced for per-  
 573 ceived errors than for unperceived  
 574 errors. We suggest that these  
 575 ERPs reflect cognitive processing  
 576 of the stimulus (i.e. incongruent  
 577 flankers surrounding the arrow)  
 578 more than perception of the APA  
 579 error. These scalp ERPs did not  
 580 seem to be relevant for studying  
 581 the non-conscious monitoring of

582 an ongoing action, when the error was corrected online  
 583 before the possible erroneous outcome (i.e. initiation with  
 584 the wrong foot). According to Krigolson and Holroyd, the  
 585 P300 component has a role in the online control process  
 586 for low-level errors (Krigolson and Holroyd, 2007). In a  
 587 corrective limb adjustment task using a joystick (in which  
 588 the target's location changed unexpectedly following  
 589 movement onset, in order to elicit errors), the researchers  
 590 concluded that if P300 arises after behavioral changes  
 591 associated with the online control of movement, then it  
 592 cannot be involved in the evaluation of target errors  
 593 (Krigolson et al., 2008). Indeed, the P300 started after  
 594 the participants had begun to adjust their motor output  
 595 to accommodate the target perturbation. Moreover,  
 596 Krigolson and Holroyd did not observe a difference in  
 597 amplitude according to the presence or absence of cor-  
 598 rection. As suggested by Krigolson et al. (Krigolson  
 599 et al., 2008), we hypothesize that P300 reflects the updat-  
 600 ing of an internal model of the movement environment –  
 601 processing of flankers, for example (Donchin and Coles,  
 602 1988).

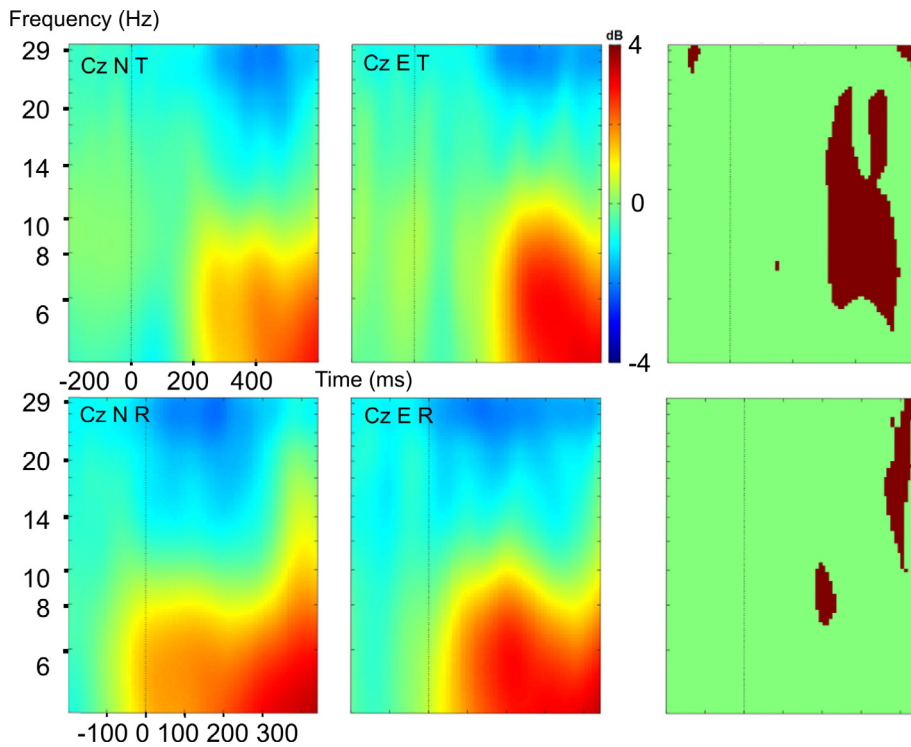
#### Time–frequency analysis and motor programming

603  
 604 Non-phase-locked (induced) changes can be studied in a  
 605 time–frequency analysis, which highlights the cortical  
 606 oscillations related to an external or internal event  
 607 (Rektor et al., 2006). Indeed, motor-related cortical oscil-  
 608 lations are generally assessed by quantifying increases or  
 609 suppressions in spectral power. For example, increases  
 610 in amplitude of the cortical oscillations in the delta-band  
 611 (2–4 Hz) and the gamma (bands 60–200 Hz) are





**Fig. 5.** Event-related desynchronization/synchronization (ERD/ERS) in normal Anticipatory Postural Adjustments (APA) and APA error. Top view of spectral maps (in dB) in the 4–7 Hz, 8–12 Hz, 13–20 Hz and 20–30 Hz bands for normal APAs (N) and APA errors (E). Target-locked (T) and response-locked (R) are shown. 0 ms correspond either to the target presentation or to T0 (start of the APAs). The color at each image pixel indicates the power (in dB) of a given frequency band. Hot colors correspond to an increase in power relative to the baseline, and cold colors correspond to a decrease in power. Theta, alpha ERS were observed in both conditions after target presentation. Beta ERD was observed in both conditions but was more prolonged for APA errors. The low-beta ERD was less intense for APA errors (target-locked only). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)



**Fig. 6.** Event-related desynchronization/synchronization (ERD/ERS) in normal Anticipatory Postural Adjustments (APA N) and APA error (E) over Cz. ERD/ERS in the different frequency bands over Cz in trials with a normal APA (N top row) or an APA error (E second row). The line indicates the target presentation time (target-locked: T) or T0 (start of the APAs, response-locked: R). ERD is shown in blue, and ERS is shown in red in dB. We observed alpha ERS during APAs more intense for APA errors, beta ERD during the normal APA and during the APA error. This ERD was longer (target-locked, response-locked) and less intense (target-locked) in the low-beta-band for APA errors. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

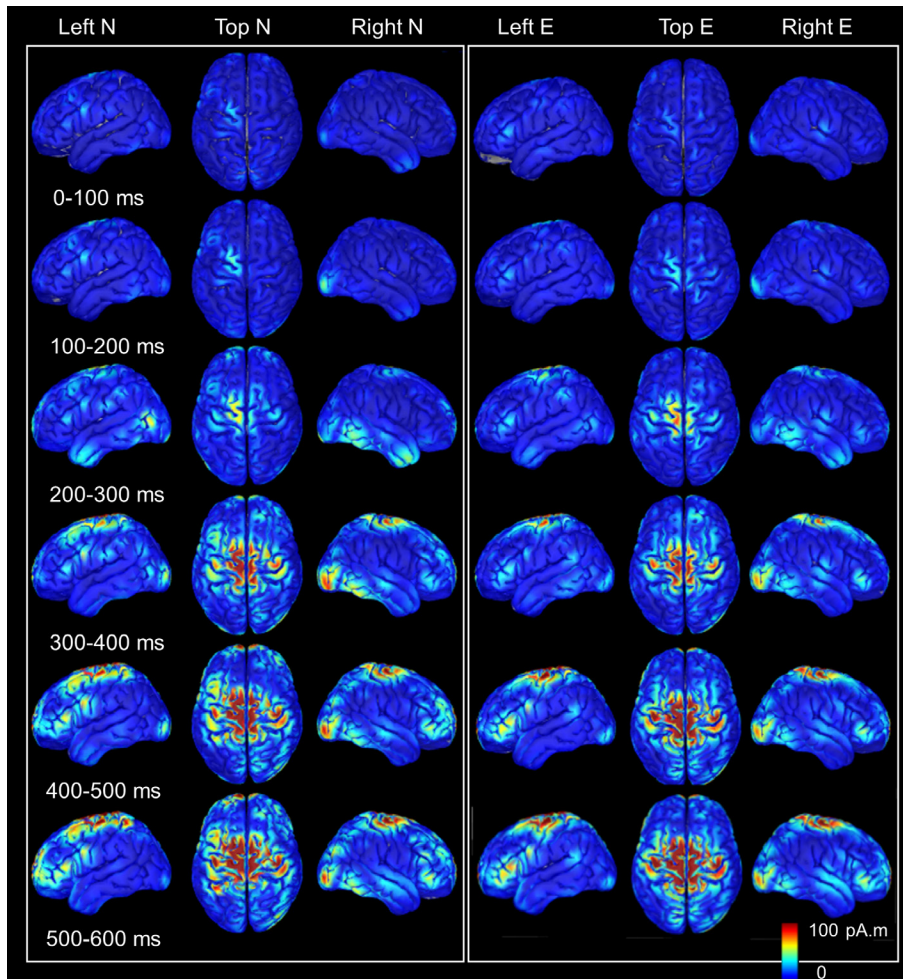
612 observed during both the planning and execution of  
613 movement (Combrisson et al., 2017). The initiation of vol-  
614 untary movements has also been linked to desynchro-  
615 nization of cortical activity in the alpha-band (8–12 Hz)  
616 and the beta-band (13–30 Hz) in electrocorticography  
617 and scalp EEG recordings (and then decrease of ampli-  
618 tude of oscillations in the corresponding frequency band)  
619 over the motor and premotor cortex (Pfurtscheller, 1981;  
620 Neuper et al., 2006).

621 Firstly, we observed theta synchronization and a more  
622 pronounced alpha synchronization in case of error.  
623 Secondly, beta ERD was observed over sensorimotor  
624 cortex.

625 Theta-band ERS is linked to an alert effect (Luu et al.,  
626 2004) and to stimuli monitoring and discrimination (Wang  
627 et al., 2005) engaged in attentional processes (Luu et al.,  
628 2004; Fan et al., 2007; Song et al., 2014). Alpha ERS  
629 (coupled with theta ERS) at the start of APAs was fol-  
630 lowed by stronger posterior alpha ERD in case of APA  
631 error. This particular pattern has been previously  
632 described during different variants of the Simon task dur-  
633 ing errors in an upper limb task (van Driel et al., 2012) and  
634 seems consistent. These patterns according to the vari-  
635 ants of the attentional task (in terms of amplitude, loca-  
636 tion, coupling between different cortical regions) were

637 influenced by performance monitoring (theta ERS), error speed  
638 processing (posterior alpha ERD) (van Driel et al., 2012). Increasing  
639 alpha ERS is supposed to facilitate the goal-directed behavior  
640 (Dockree et al., 2007) by reflecting active mechanisms of sensory  
641 suppression for irrelevant-task stimulus (Foxe and Snyder, 2011)  
642 which could be particularly useful for proper motor execution in case  
643 of APA errors triggered by incongruent stimuli. Then, alpha ERD  
644 (response-locked) is strongly associated to visual perception and  
645 involved in task anticipation to modulate the excitability in human  
646 parieto-occipital cortex (Capotosto et al., 2017). Alpha ERD is there-  
647 fore stronger in trials with APA errors in order to facilitate the mod-  
648 ulation of the sensorimotor cortex with stimulus' perception. It signs  
649 the further orientation and maintenance of visual attention (posterior  
650 predominant, engagement of parieto-occipital areas (Foxe  
651 et al., 1998)).

652 Beta ERD is thought to reflect the activation of regions engaged  
653 in visuospatial attention or motor execution (Pfurtscheller and  
654 Lopes da Silva, 1999). During tasks requiring attention, signifi-  
655 cant increases in the delta-, theta- and gamma-bands have been  
656 reported during the planning phase and especially during  
657 execution. In contrast, alpha, beta and low-gamma power  
658 falls after an execution cue (Combrisson et al., 2017). In  
659 our paradigm, the occurrence of beta ERD following the  
660 appearance of the target was consistent with this pattern.  
661 Oscillations in the beta- and gamma-bands during motor  
662 preparation have been studied in paradigms that compare  
663 successful stops with unsuccessful stops (Swann et al.,  
664 2009, 2012). Overall, brief beta ERS is followed by longer  
665 beta ERD. The latter is more pronounced in successful  
666 stop trials. These findings provide insight into our results  
667 – even though our study did not feature successful vs.  
668 unsuccessful stops. In trials with APA errors, we observed  
669 prolonged beta ERD over the sensorimotor cortex; this  
670 probably reflected the fact that movement preparation  
671 was longer when an APA error occurred (Cohen et al.,  
672 2011). We also observed less intense low-beta ERD dur-  
673 ing APA errors. In a study evaluating a shifting cognitive  
674 task during gait (Wagner et al., 2016), two different beta  
675 oscillations were noted: beta ERD (expressing motor exe-  
676 cution and motor readiness related to gait movements)  
677 and a frontal beta ERS (related to cognitive top-down con-  
678 trol on gait). The less intense ERD in the APA error con-  
679 dition might be due to the summation of concomitant ERD  
680



**Fig. 7.** Source localization of cortical activity in the 0- to 600-ms time window N: normal APAs; E: APA errors. The source localizations were the same in both conditions.

698 (for movement preparation) and ERS (for the cognitive  
699 load, with a complicated process for conflict resolution  
700 and correction of the engaged motor program). Further-  
701 more, beta frequency oscillations are associated with  
702 the maintenance of the “status quo”, such as holding a  
703 fixed position. In the event of an APA error, the status  
704 quo is disrupted. For example, stabilized gait on treadmill  
705 is characterized by beta-band desynchronization that  
706 lasts for at least 15 steps (Wagner et al., 2016). Here,  
707 the presence of differences in low-beta ERD during an  
708 APA error suggested that the status quo had been dis-  
709 rupted (Engel and Fries, 2010). However, low-beta mod-  
710 ulations have been studied more frequently in cognitive  
711 tasks (including memory tasks) than in motor tasks. Other  
712 spectral properties (such as phase and phase-amplitude  
713 coupling) have also been shown to carry information with  
714 regard to the oscillatory dynamics underlying motor pro-  
715 cesses, and an analysis of these variables might be of  
716 value in studying network dynamics during low-level  
717 errors (Combrisson et al., 2017). There was a contradic-  
718 tion between the findings of ERN and theta/alpha ERD/  
719 S. Contrary to ERP, alpha synchronization appeared in  
720 this study to be more sensitive, probably because of its

time resolution and the relative  
specificity of the different fre-  
quency bands to monitor brief cov-  
ert errors such as APA errors.  
Indeed, previous studies (Kolev  
et al., 2005, 2005; Albrecht et al.,  
2009) found modulations in low-fre-  
quency band (delta, theta) and  
ERN in different paradigms (choice  
reaction task, flankers...) provok-  
ing overt errors in different popula-  
tions but once again, APA errors  
are of different nature.

**Limitations**

There is a strong relationship  
between the ERP and ERD/ERS.  
However, ERD and ERS are not  
systematically time-locked to the  
target ERP. Theta- and delta-  
band ERSs correspond most  
closely to N200 and P300,  
respectively (Huster et al., 2013).  
In fact, an increase in the ampli-  
tude of P300 is invariably associ-  
ated with an increase in power in  
the low-frequency bands – as  
observed in the present study.  
Removing or not the ERP signal  
of the time–frequency analysis is  
still subject to debate. In a non-  
presented analysis, we removed  
the ERP signal from the EEG  
before performing event-related  
spectral analysis in the beta-band;  
this enabled us to study the  
induced response alone and not  
the evoked response: no clear dif-

ferences (topographical or statistical maps) were  
observed after the removal.

The attention network task enabled the study of  
different components of attention (Fan et al., 2002). How-  
ever, trials with cues might influence the RTs or the pro-  
portion of pre-APAs (i.e. APAs occurring between the  
cue and the target presentation but not followed by a step,  
with a return to the baseline posture at the moment of tar-  
get presentation (Tard et al., 2015)); however, the propor-  
tions of valid, invalid cued and uncued trials did not differ  
after removal of trials with artifacts. The proportion of no  
cue trials was 17.8% (normal APA) vs. 16.7% (APA error)  
vs 16.3% (before removal of trials with artifacts), the pro-  
portion of neutral cue trials was 19.3% (normal APA) vs.  
18.2% (APA error) vs. 20.3% (before removal of trials with  
artifacts), the proportion of valid cue trials was 47.5%  
(normal APA) vs. 47.4% (APA error) vs. 47.3% (before  
removal of trials with artifacts), the proportion of invalid  
cue trials was 15.4% (normal APA) vs 17.6% (APA error)  
vs. 16% (before removal of trials with artifacts). Further-  
more, we excluded false start trials (i.e. those with an  
RT < 100 ms). The median number of trials by subject /  
condition after removal of artifacts was 49 (20–98) for

781 APA errors and 69 (38–107) for normal APAs. One sub-  
782 ject has a very low number of trials taken into account  
783 but the ERP was clearly identifiable in this particular case.  
784 The subject was thus not excluded.

785 Our present results highlighted a cortical marker of  
786 gait initiation APA errors in healthy subjects. Differences  
787 in sensorimotor activation (reflected by differing alpha/  
788 beta-band ERS/ERD patterns) were observed during  
789 APA errors. It remains to be seen how these cortical  
790 oscillations are influenced by cortical-subcortical loops.  
791 Future research should consider the role of the basal  
792 ganglia (and specifically the STN) in movement  
793 inhibition. In contrast to ERP analysis, time–frequency  
794 methods are useful for monitoring non-conscious errors.  
795 These methods could also be used to monitor motor  
796 programming errors (in patients with dysexecutive  
797 syndrome, for example), and could be implemented in  
798 brain–computer interface algorithms.

### 799 UNCITED REFERENCE

800 [Capotosto et al. \(1991\).](#)

### 801 REFERENCES

- 802 Albrecht B, Heinrich H, Brandeis D, Uebel H, Yordanova J, Kolev V,  
803 Rothenberger A, Banaschewski T (2009) Flanker-task in children:  
804 time-frequency analyses of response monitoring. *J Psychophysiol*  
805 23:183–190.
- 806 Aron AR, Poldrack RA (2006) Cortical and subcortical contributions to  
807 stop signal response inhibition: role of the subthalamic nucleus. *J*  
808 *Neurosci* 26:2424–2433.
- 809 Brenière Y, Do MC (1991) Control of gait initiation. *J Mot Behav*  
810 23:235–240.
- 811 Capotosto P, Baldassarre A, Sestieri C, Spadone S, Romani GL,  
812 Corbetta M (1991) Task and regions specific top-down modulation  
813 of alpha rhythms in parietal cortex. *Cereb Cortex N Y N* 1991  
814 27:4815–4822.
- 815 Carbonell L, Falkenstein M (2006) Does the error negativity reflect  
816 the degree of response conflict? *Brain Res* 1095:124–130.
- 817 Charles L, Van Opstal F, Marti S, Dehaene S (2013) Distinct brain  
818 mechanisms for conscious versus subliminal error detection.  
819 *NeuroImage* 73:80–94.
- 820 Cohen RG, Nutt JG, Horak FB (2011) Errors in postural preparation  
821 lead to increased choice reaction times for step initiation in older  
822 adults. *J Gerontol A Biol Sci Med Sci*. Available at: [http://www.  
823 ncbi.nlm.nih.gov/pubmed/21498431](http://www.ncbi.nlm.nih.gov/pubmed/21498431) [Accessed June 10, 2011].
- 824 Combrisson E, Perrone-Bertolotti M, Soto JL, Alamian G, Kahane P,  
825 Lachaux J-P, Guillot A, Jerbi K (2017) From intentions to actions:  
826 Neural oscillations encode motor processes through phase,  
827 amplitude and phase-amplitude coupling. *NeuroImage*  
828 147:473–487.
- 829 Dale AM, Fischl B, Sereno MI (1999) Cortical surface-based analysis.  
830 I. Segmentation and surface reconstruction. *NeuroImage*  
831 9:179–194.
- 832 Deiber M-P, Ibañez V, Missonnier P, Rodriguez C, Giannakopoulos P  
833 (2013) Age-associated modulations of cerebral oscillatory  
834 patterns related to attention control. *NeuroImage* 82:531–546.
- 835 Delorme A, Makeig S (2004) EEGLAB: an open source toolbox for  
836 analysis of single-trial EEG dynamics including independent  
837 component analysis. *J Neurosci Methods* 134:9–21.
- 838 Delval A, Dujardin K, Tard C, Devanne H, Willart S, Bourriez J-L,  
839 Derambure P, Defebvre L (2012) Anticipatory postural  
840 adjustments during step initiation: elicitation by auditory  
841 stimulation of differing intensities. *Neuroscience* 219:166–174.
- 842 Delval A, Moreau C, Bleuse S, Tard C, Ryckewaert G, Devos D,  
843 Defebvre L (2014) Auditory cueing of gait initiation in Parkinson's  
844 disease patients with freezing of gait. *Clin Neurophysiol Off J Int*  
845 *Fed Clin Neurophysiol* 125:1675–1681.
- 846 Dockree PM, Kelly SP, Foxe JJ, Reilly RB, Robertson IH (2007)  
847 Optimal sustained attention is linked to the spectral content of  
848 background EEG activity: greater ongoing tonic alpha  
849 (approximately 10 Hz) power supports successful phasic goal  
850 activation. *Eur J Neurosci* 25:900–907.
- 851 Donchin E, Coles MGH (1988) Is the P300 component a  
852 manifestation of context updating? *Behav Brain Sci* 11:357–374.
- 853 Duque J, Greenhouse I, Labruna L, Ivry RB (2017) Physiological  
854 markers of motor inhibition during human behavior. *Trends*  
855 *Neurosci* 40:219–236.
- 856 Engel AK, Fries P (2010) Beta-band oscillations—signalling the status  
857 quo? *Curr Opin Neurobiol* 20:156–165.
- 858 Falkenstein M, Hohnsbein J, Hoormann J, Blanke L (1991) Effects of  
859 crossmodal divided attention on late ERP components. II. Error  
860 processing in choice reaction tasks. *Electroencephalogr Clin*  
861 *Neurophysiol* 78:447–455.
- 862 Falkenstein M, Hoormann J, Christ S, Hohnsbein J (2000) ERP  
863 components on reaction errors and their functional significance: a  
864 tutorial. *Biol Psychol* 51:87–107.
- 865 Fan J, Byrne J, Worden MS, Guise KG, McCandliss BD, Fossella J,  
866 Posner MI (2007) The relation of brain oscillations to attentional  
867 networks. *J Neurosci Off J Soc Neurosci* 27:6197–6206.
- 868 Fan J, McCandliss BD, Sommer T, Raz A, Posner MI (2002) Testing  
869 the efficiency and independence of attentional networks. *J Cogn*  
870 *Neurosci* 14:340–347.
- 871 Foxe JJ, Simpson GV, Ahlfors SP (1998) Parieto-occipital  
872 approximately 10 Hz activity reflects anticipatory state of visual  
873 attention mechanisms. *Neuroreport* 9:3929–3933.
- 874 Foxe JJ, Snyder AC (2011) The role of alpha-band brain oscillations  
875 as a sensory suppression mechanism during selective attention.  
876 *Front Psychol* 2:154.
- 877 Gantchev N, Viallet F, Aurenry R, Massion J (1996) Impairment of  
878 posturo-kinetic co-ordination during initiation of forward oriented  
879 stepping movements in parkinsonian patients.  
880 *Electroencephalogr Clin Neurophysiol* 101:110–120.
- 881 Genovese CR, Lazar NA, Nichols T (2002) Thresholding of statistical  
882 maps in functional neuroimaging using the false discovery rate.  
883 *NeuroImage* 15:870–878.
- 884 Gramfort A, Papadopoulos T, Olivi E, Clerc M (2010) OpenMEEG:  
885 opensource software for quasistatic bioelectromagnetics. *Biomed*  
886 *Eng Online* 9:45.
- 887 Hamano T, Lüders HO, Ikeda A, Collura TF, Comair YG, Shibasaki H  
888 (1997) The cortical generators of the contingent negative variation  
889 in humans: a study with subdural electrodes. *Electroencephalogr*  
890 *Clin Neurophysiol* 104:257–268.
- 891 Hill H, Raab M (2005) Analyzing a complex visuomotor tracking task  
892 with brain-electrical event related potentials. *Hum Mov Sci*  
893 24:1–30.
- 894 Huster RJ, Enriquez-Geppert S, Lavalée CF, Falkenstein M,  
895 Herrmann CS (2013) Electroencephalography of response  
896 inhibition tasks: functional networks and cognitive contributions.  
897 *Int J Psychophysiol Off J Int Organ Psychophysiol* 87:217–233.
- 898 Jacobs JV, Lou JS, Kraakevik JA, Horak FB (2009) The  
899 supplementary motor area contributes to the timing of the  
900 anticipatory postural adjustment during step initiation in  
901 participants with and without Parkinson's disease. *Neuroscience*  
902 164:877–885.
- 903 Jiang N, Gizzi L, Mrachacz-Kersting N, Dremstrup K, Farina D (2015)  
904 A brain–computer interface for single-trial detection of gait  
905 initiation from movement related cortical potentials. *Clin*  
906 *Neurophysiol* 126:154–159.
- 907 Kolev V, Falkenstein M, Yordanova J (2005) Aging and error  
908 processing: time-frequency analysis of error-related potentials. *J*  
909 *Psychophysiol* 19:289–297.
- 910 Kolev V, Yordanova J, Schürmann M, Başar E (2001) Increased  
911 frontal phase-locking of event-related alpha oscillations during  
912 task processing. *Int J Psychophysiol Off J Int Organ*  
913 *Psychophysiol* 39:159–165.

- 914 Krigolson OE, Holroyd CB (2007) Hierarchical error processing: 980  
915 different errors, different systems. *Brain Res* 1155:70–80. 981
- 916 Krigolson OE, Holroyd CB, Gyn GV, Heath M (2008) 982  
917 Electroencephalographic correlates of target and outcome 983  
918 errors. *Exp Brain Res* 190:401–411. 984
- 919 Li C-SR, Yan P, Sinha R, Lee T-W (2008) Subcortical processes of 985  
920 motor response inhibition during a stop signal task. *NeuroImage* 986  
921 41:1352–1363. 987
- 922 Lu C, Amundsen Huffmaster SL, Harvey JC, MacKinnon CD (2017) 988  
923 Anticipatory postural adjustment patterns during gait initiation 989  
924 across the adult lifespan. *Gait Posture* 57:182–187. 990
- 925 Luu P, Tucker DM, Makeig S (2004) Frontal midline theta and the 991  
926 error-related negativity: neurophysiological mechanisms of action 992  
927 regulation. *Clin Neurophysiol Off J Int Fed Clin Neurophysiol* 993  
928 115:1821–1835. 994
- 929 McIlroy WE, Maki BE (1999) The control of lateral stability during 995  
930 rapid stepping reactions evoked by antero-posterior perturbation: 996  
931 does anticipatory control play a role? *Gait Posture* 9:190–198. 997
- 932 Meckler C, Allain S, Carbone L, Hasbroucq T, Burle B, Vidal F 998  
933 (2011) Executive control and response expectancy: a Laplacian 999  
934 ERP study. *Psychophysiology* 48:303–311. 1000
- 935 Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, Whitehead 1001  
936 V, Collin I, Cummings JL, Chertkow H (2005) The Montreal 1002  
937 Cognitive Assessment, MoCA: a brief screening tool for mild 1003  
938 cognitive impairment. *J Am Geriatr Soc* 53:695–699. 1004
- 939 Neuhaus AH, Urbanek C, Oppen-Rhein C, Hahn E, Ta TMT, Koehler 1005  
940 S, Gross M, Dettling M (2010) Event-related potentials associated 1006  
941 with Attention Network Test. *Int J Psychophysiol Off J Int Organ* 1007  
942 *Psychophysiol* 76:72–79. 1008
- 943 Neuper C, Wörtz M, Pfurtscheller G (2006) ERD/ERS patterns 1009  
944 reflecting sensorimotor activation and deactivation. *Prog Brain* 1010  
945 *Res* 159:211–222. 1011
- 946 Nieuwenhuis S, Ridderinkhof KR, Blom J, Band GP, Kok A (2001) 1012  
947 Error-related brain potentials are differentially related to 1013  
948 awareness of response errors: evidence from an antisaccade 1014  
949 task. *Psychophysiology* 38:752–760. 1015
- 950 Oostenveld R, Praamstra P (2001) The five percent electrode system 1016  
951 for high-resolution EEG and ERP measurements. *Clin* 1017  
952 *Neurophysiol Off J Int Fed Clin Neurophysiol* 112:713–719. 1018
- 953 Pahapill PA, Lozano AM (2000) The pedunculopontine nucleus and 1019  
954 Parkinson's disease. *Brain J Neurol* 123(Pt 9):1767–1783. 1020
- 955 Pfurtscheller G (1981) Central beta rhythm during sensorimotor 1021  
956 activities in man. *Electroencephalogr Clin Neurophysiol* 1022  
957 51:253–264. 1023
- 958 Pfurtscheller G, Andrew C (1999) Event-Related changes of band 1024  
959 power and coherence: methodology and interpretation. *J Clin* 1025  
960 *Neurophysiol Off Publ Am Electroencephalogr Soc* 16:512–519. 1026
- 961 Pfurtscheller G, Lopes da Silva FH (1999) Event-related EEG/MEG 1027  
962 synchronization and desynchronization: basic principles. *Clin* 1028  
963 *Neurophysiol Off J Int Fed Clin Neurophysiol* 110:1842–1857. 1029
- 964 Queralt A, Valls-Solé J, Castellote JM (2010) Speeding up gait 1030  
965 initiation and gait-pattern with a startling stimulus. *Gait Posture* 1031  
966 31:185–190. 1032
- 967 Ramautar JR, Kok A, Ridderinkhof KR (2006) Effects of stop-signal 1033  
968 modality on the N2/P3 complex elicited in the stop-signal 1034  
969 paradigm. *Biol Psychol* 72:96–109. 1035
- 970 Rektor I, Sochůrková D, Bocková M (2006) Intracerebral ERD/ERS in 1036  
971 voluntary movement and in cognitive visuomotor task. *Prog Brain* 1037  
972 *Res* 159:311–330. 1038
- 973 Sburlea AI, Montesano L, de la Cuerda RC, Alguacil Diego IM, 1039  
974 Miangolarra-Page JC, Minguez J (2015) Detecting intention to 1040  
975 walk in stroke patients from pre-movement EEG correlates. *J* 1041  
976 *NeuroEngineering Rehabil* 12:113. 1042
- 977 Song K, Meng M, Chen L, Zhou K, Luo H (2014) Behavioral 1043  
978 oscillations in attention: rhythmic  $\alpha$  pulses mediated through  $\theta$  1044  
979 band. *J Neurosci Off J Soc Neurosci* 34:4837–4844. 1045
- Swann N, Tandon N, Canolty R, Ellmore TM, McEvoy LK, Dreyer S, 980  
DiSano M, Aron AR (2009) Intracranial EEG reveals a time- and 981  
frequency-specific role for the right inferior frontal gyrus and 982  
primary motor cortex in stopping initiated responses. *J Neurosci* 983  
29:12675–12685. 984
- Swann NC, Cai W, Conner CR, Pieters TA, Claffey MP, George JS, 985  
Aron AR, Tandon N (2012) Roles for the pre-supplementary motor 986  
area and the right inferior frontal gyrus in stopping action: 987  
electrophysiological responses and functional and structural 988  
connectivity. *NeuroImage* 59:2860–2870. 989
- Tadel F, Baillet S, Mosher JC, Pantazis D, Leahy RM (2011) 990  
Brainstorm: a user-friendly application for MEG/EEG analysis. 991  
*Comput Intell Neurosci* 2011 879716. 992
- Takakusaki K (2008) Forebrain control of locomotor behaviors. *Brain* 993  
*Res Rev* 57:192–198. 994
- Tard C, Dujardin K, Bourriez J-L, Derambure P, Defebvre L, Delval A 995  
(2013) Stimulus-driven attention modulates the release of 996  
anticipatory postural adjustments during step initiation. 997  
*Neuroscience* 247C:25–34. 998
- Tard C, Dujardin K, Girard A, Debaughrien M, Derambure P, 999  
Defebvre L, Delval A (2015) How does visuospatial attention 1000  
modulate motor preparation during gait initiation? *Exp Brain Res.* 1001
- Uemura K, Oya T, Uchiyama Y (2013) Effects of visual interference 1002  
on initial motor program errors and execution times in the choice 1003  
step reaction. *Gait Posture* 38:68–72. 1004
- Uemura K, Yamada M, Nagai K, Shinya M, Ichihashi N (2012) Effect 1005  
of dual-tasking on the center of pressure trajectory at gait initiation 1006  
in elderly fallers and non-fallers. *Aging Clin Exp Res* 24:152–156. 1007
- van Driel J, Ridderinkhof KR, Cohen MX (2012) Not all errors are 1008  
alike: theta and alpha EEG dynamics relate to differences in error- 1009  
processing dynamics. *J Neurosci Off J Soc Neurosci* 32:16795–16806. 1010
- Viallet F, Massion J, Massarino R, Khalil R (1992) Coordination 1011  
between posture and movement in a bimanual load lifting task: 1012  
putative role of a medial frontal region including the 1013  
supplementary motor area. *Exp Brain Res Exp Hirnforsch* 1014  
*Expérimentation Cérébrale* 88:674–684. 1015
- Vidailhet M, Atchison PR, Stocchi F, Thompson PD, Rothwell JC, 1016  
Marsden CD (1995) The Bereitschaftspotential preceding 1017  
stepping in patients with isolated gait ignition failure. *Mov Disord* 1018  
*Off J Mov Disord Soc* 10:18–21. 1019
- Vidailhet M, Stocchi F, Rothwell JC, Thompson PD, Day BL, Brooks 1020  
DJ, Marsden CD (1993) The Bereitschaftspotential preceding 1021  
simple foot movement and initiation of gait in Parkinson's disease. 1022  
*Neurology* 43:1784–1788. 1023
- Vidal F, Hasbroucq T, Grapperon J, Bonnet M (2000) Is the "error 1024  
negativity" specific to errors? *Biol Psychol* 51:109–128. 1025
- Wagner J, Makeig S, Gola M, Neuper C, Müller-Putz G (2016) 1026  
Distinct  $\beta$  band oscillatory networks subserving motor and 1027  
cognitive control during gait adaptation. *J Neurosci* 1028  
36:2212–2226. 1029
- Wang C, Ulbert I, Schomer DL, Marinkovic K, Halgren E (2005) 1030  
Responses of human anterior cingulate cortex microdomains to 1031  
error detection, conflict monitoring, stimulus-response mapping, 1032  
familiarity, and orienting. *J Neurosci Off J Soc Neurosci* 25:604–613. 1033
- Watanabe T, Ishida K, Tanabe S, Nojima I (2016a) Preparatory state 1034  
and postural adjustment strategies for choice reaction step 1035  
initiation. *Neuroscience* 332:140–148. 1036
- Watanabe T, Tsutou K, Saito K, Ishida K, Tanabe S, Nojima I (2016b) 1037  
Performance monitoring and response conflict resolution 1038  
associated with choice stepping reaction tasks. *Exp Brain Res* 1039  
234:3355–3365. 1040
- Wessel JR, Aron AR (2017) On the globality of motor suppression: 1041  
unexpected events and their influence on behavior and cognition. 1042  
*Neuron* 93:259–280. 1043

(Received 5 December 2017, Accepted 29 September 2018)  
(Available online xxx)