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Motor Preparation of Step Initiation: Error-related Cortical Oscillations

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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

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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).

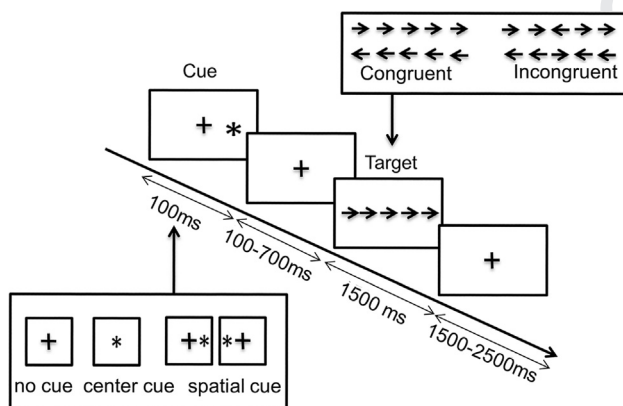
The experimental setting

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**

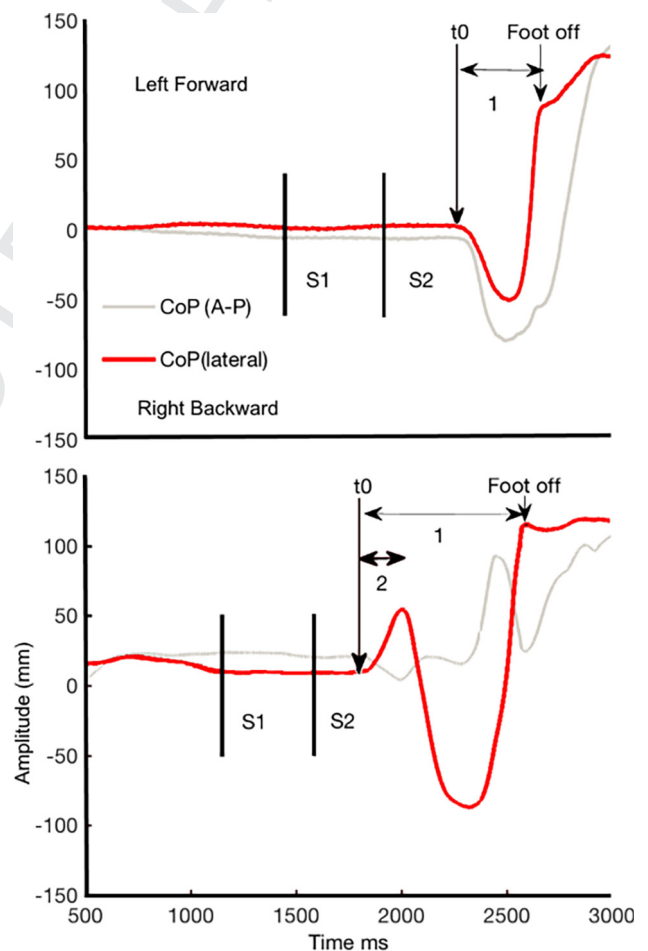
194 Data were collected with a three-dimensional motion analysis system (VICON 370®, Oxford Biometrics,
195 Oxford, UK), using eight infrared cameras and a sampling frequency of 100 Hz. The CoP was measured
196 with two force platforms (the ORG-5 model from AMTI®, Watertown, MA, USA) at a sampling frequency
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 ± 0.08 s for normal
345 APAs and 0.23 ± 0.06 s for APA errors ($p < 0.001$).
346 The mean \pm SD APA duration was longer for APA
347 errors (0.64 ± 0.13 s) than for normal APAs (0.47
348 ± 0.10 s) ($p < 0.001$). The mean \pm SD correction time
349 for APA errors was 0.20 ± 0.07 s (i.e. 0.43 ± 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 ± 0.08 s vs. 0.47 ± 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 ± 0.05 s vs. 0.08
373 ± 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 ± 0.07 s vs. 0.19 ± 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-

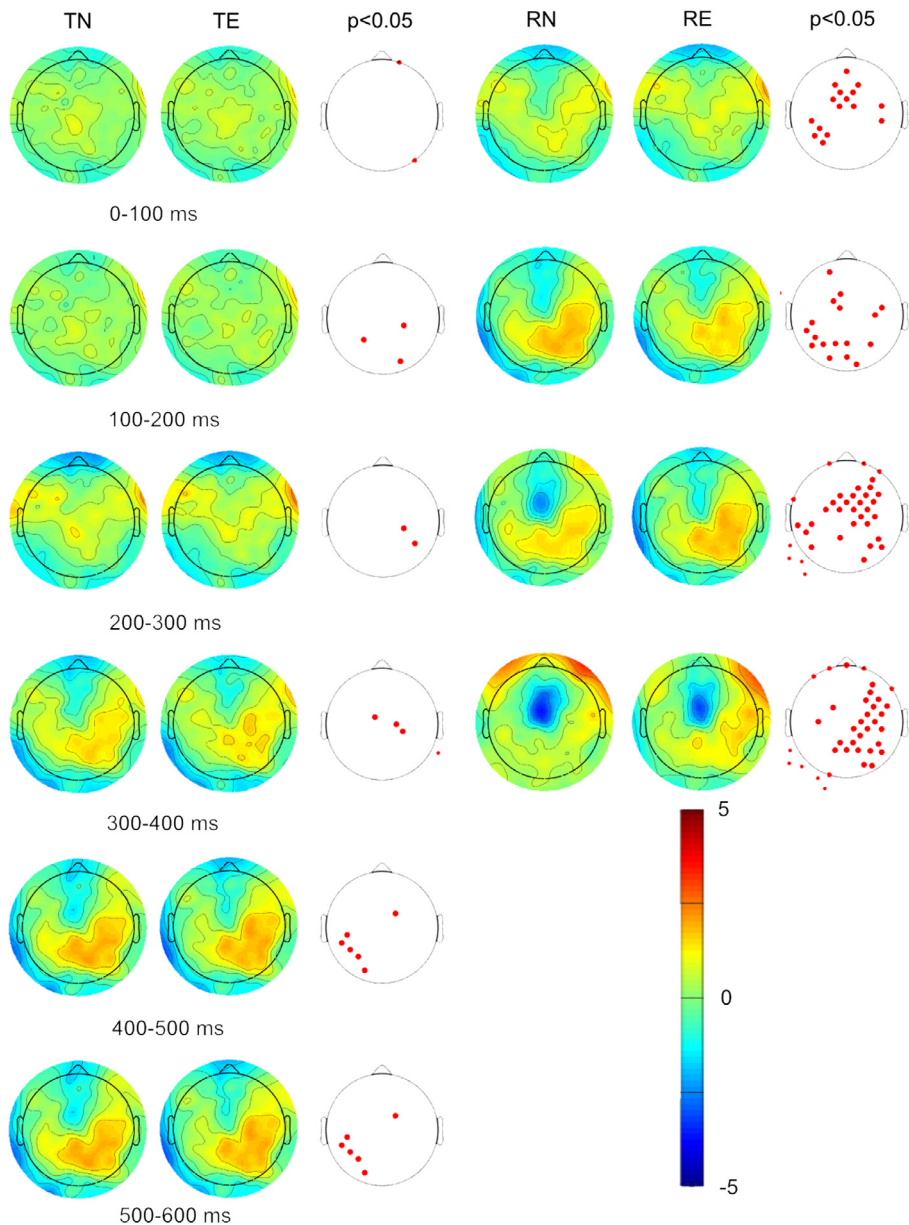


Fig. 3. Event-related potentials in normal Anticipatory Postural Adjustments (APA) and APA error. Top view of topographic voltage maps for each condition (i.e. a normal APA-N- or an APA error-E-). 0 ms corresponds to either the target presentation (S2), target-locked (T), or to the start of the APAs (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-
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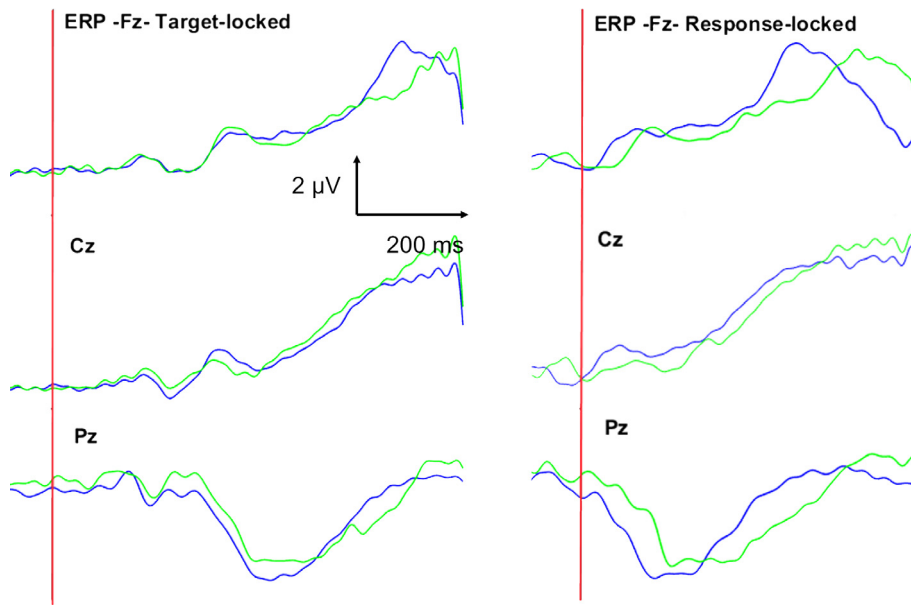


Fig. 4. Event-related potentials in normal Anticipatory Postural Adjustments (APA) and APA error in midline derivations. ERP for Fz, Cz and Pz sites in two conditions: blue: grand average of normal APA vs. green, grand average of APA error. 0 ms corresponds to either the target presentation (S2), target-locked, 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 dif-
 553 ference in the amplitude of Pe. The
 554 participant was not given any infor-
 555 mation about APA error monitoring.
 556 Indeed, both Ne and Pe are
 557 closely related to conscious percep-
 558 tion 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

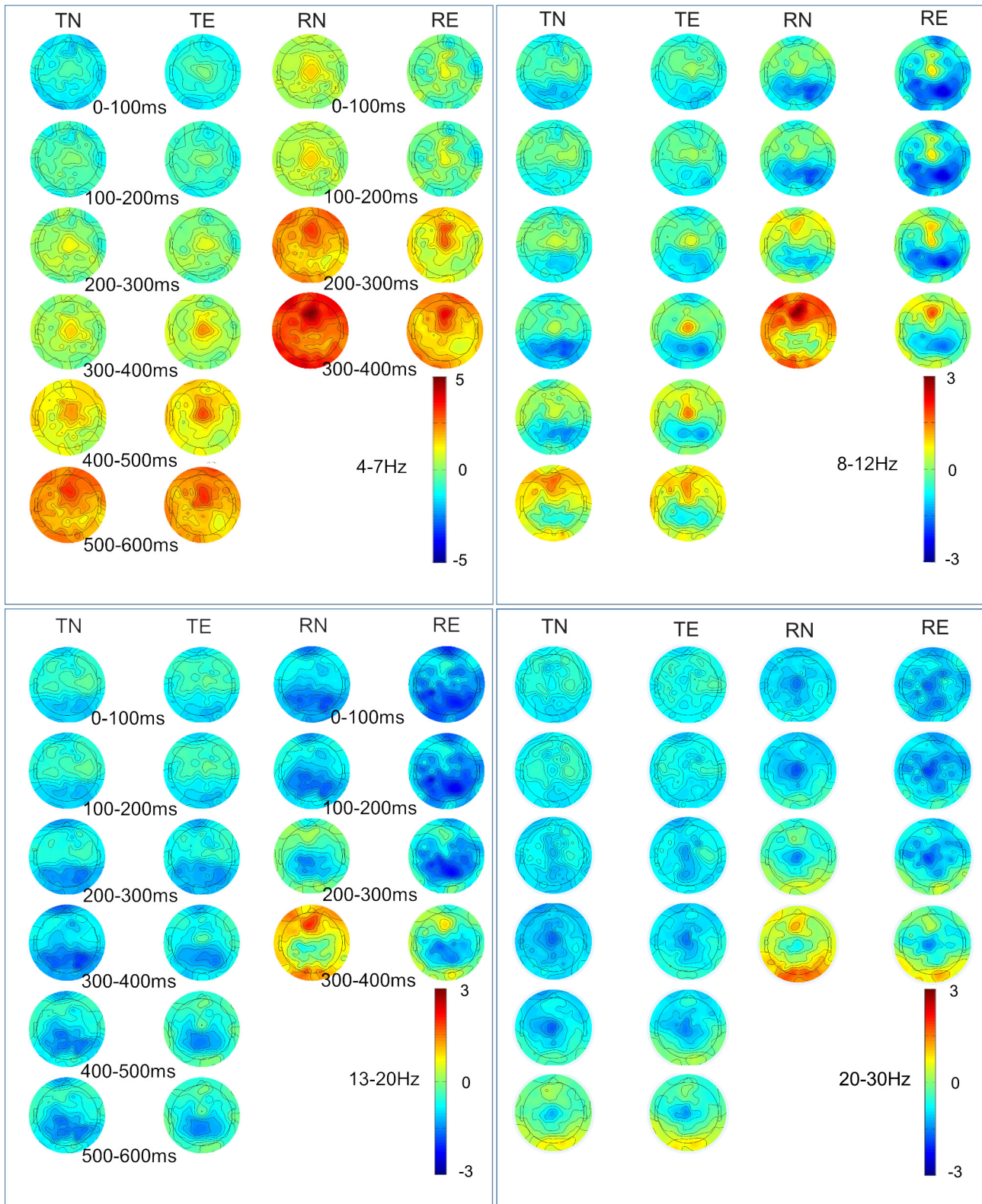


Fig. 5. Event-related desynchronization/synchronization (ERD/ERS) in normal Anticipatory Postural Adjustments (APA) and APA error. Top view of spectral maps (in dB) in the 4–7 Hz, 8–12 Hz, 13–20 Hz and 20–30 Hz bands for normal APAs (N) and APA errors (E). Target-locked (T) and response-locked (R) are 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.)

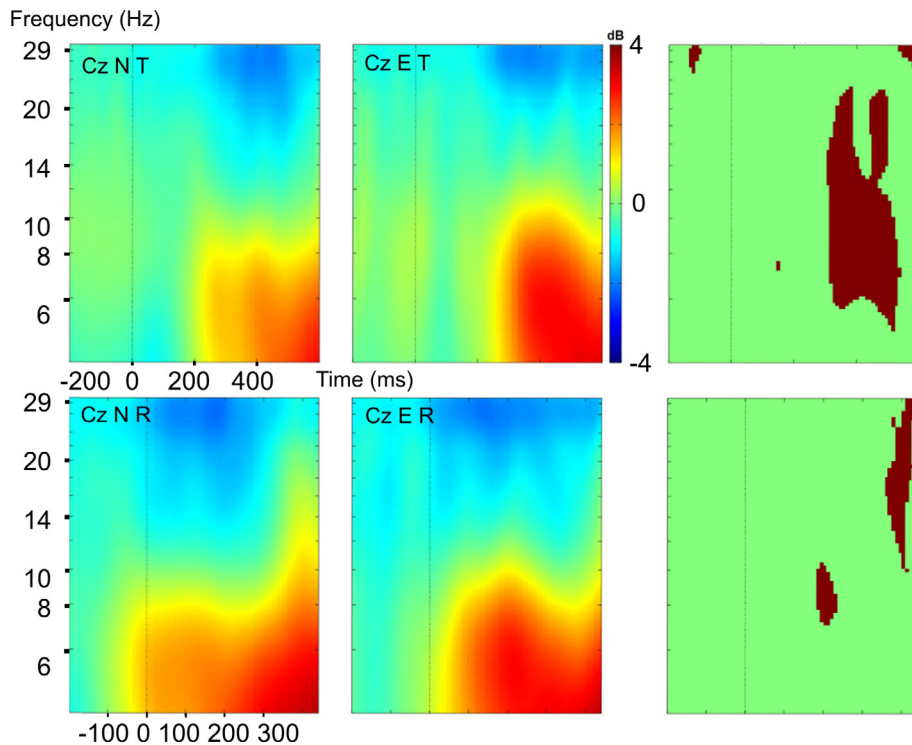


Fig. 6. Event-related desynchronization/synchronization (ERD/ERS) in normal Anticipatory Postural Adjustments (APA N) and APA error (E) over Cz. ERD/ERS in the different frequency bands over Cz in trials with a normal APA (N top row) or an APA error (E second row). The line indicates the target presentation 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.)

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

Beta ERD is thought to reflect the activation of regions engaged in visuospatial attention or motor execution (Pfurtscheller and Lopes da Silva, 1999). During tasks requiring attention, significant increases in the delta-, theta- and gamma-bands have been reported during the planning phase and especially during execution. In contrast, alpha, beta and low-gamma power falls after an execution cue (Combrisson et al., 2017). In our paradigm, the occurrence of beta ERD following the appearance of the target was consistent with this pattern. Oscillations in the beta- and gamma-bands during motor preparation have been studied in paradigms that compare successful stops with unsuccessful stops (Swann et al., 2009, 2012). Overall, brief beta ERS is followed by longer beta ERD. The latter is more pronounced in successful stop trials. These findings provide insight into our results – even though our study did not feature successful vs. unsuccessful stops. In trials with APA errors, we observed prolonged beta ERD over the sensorimotor cortex; this probably reflected the fact that movement preparation was longer when an APA error occurred (Cohen et al., 2011). We also observed less intense low-beta ERD during APA errors. In a study evaluating a shifting cognitive task during gait (Wagner et al., 2016), two different beta oscillations were noted: beta ERD (expressing motor execution and motor readiness related to gait movements) and a frontal beta ERS (related to cognitive top-down control on gait). The less intense ERD in the APA error condition might be due to the summation of concomitant ERD

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

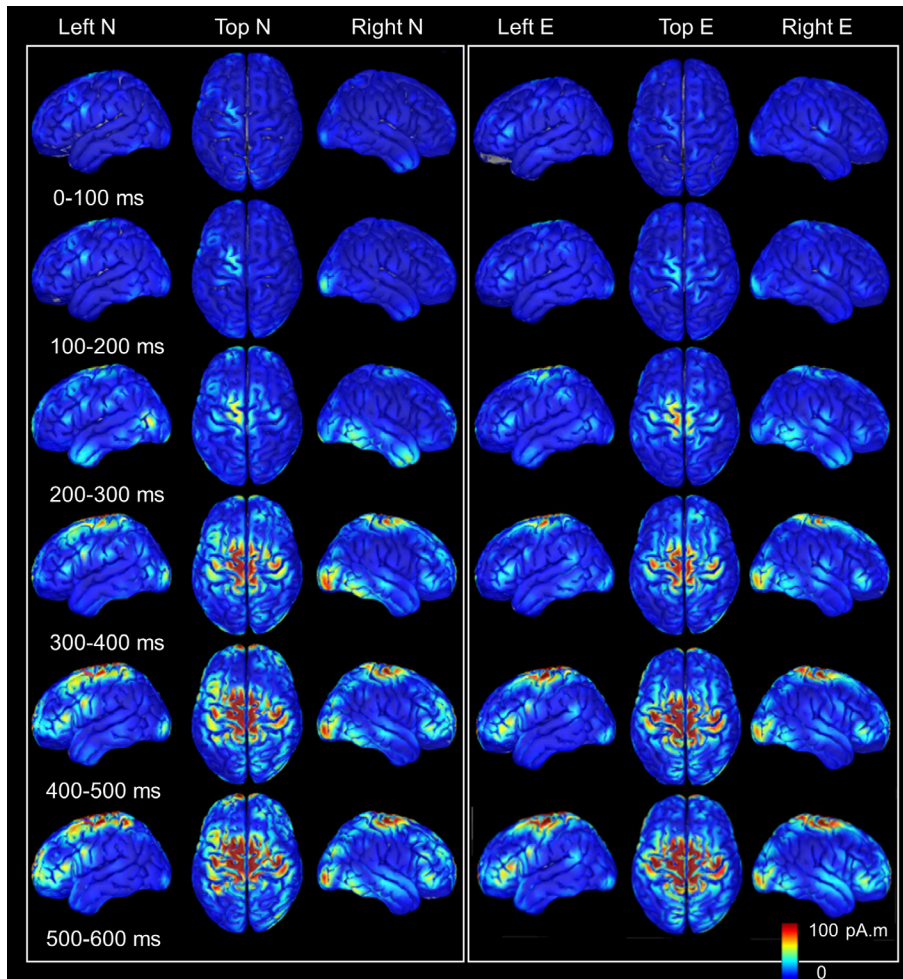


Fig. 7. Source localization of cortical activity in the 0- to 600-ms time window N: normal APAs; E: APA errors. The source localizations were the same in both conditions.

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.

UNCITED REFERENCE

799
800 [Capotosto et al. \(1991\).](#)

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