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Central Adaptations to Repetitive Grasping in Healthy Aging

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Abstract

Augmented cortical activity during repetitive grasping mitigates repetition-related decrease in cortical efficiency in young adults. It is unclear if similar processes occur with healthy aging. We recorded movement-related cortical potentials (MRCP) during 150 repetitive handgrip contractions at 70% of maximal voluntary contraction (MVC) in healthy young ($n = 10$) and old ($n = 10$) adults. Repetitions were grouped into two Blocks (Block 1 and 2: repetitions 1–60 and 91–150, respectively) and analyzed separately to assess the effects of aging and block. EMG of the flexor digitorum superficialis and handgrip force were also recorded. No changes in EMG or MVC were observed across blocks for either group. Significant interactions ($P < 0.05$) were observed for MRCPs recorded from mesial (FCz, Cz, CPz) and motor (C1, C3, Cz) electrode sites, with younger adults demonstrating significant increases in MRCP amplitude. Focal MRCP activity in response to repetitive grasping resulted in minimal changes (i.e. Block 1 versus Block 2) in older adults. Central adaptive processes change across the lifespan, showing increasingly less focal activation in older adults during repetitive grasping. Our findings are consistent with previous paradigms demonstrating more diffuse cortical activation during motor tasks in older adults.

Keywords

Movement-related cortical potential; Bereitschaftspotential; Central adaptation; Repetitive voluntary contraction; Aging

Introduction

Age-related reorganization of the central nervous system is evident during simple motor tasks as characterized by electroencephalography (EEG) (Golob et al. 2005; Inuggi et al. 2009; Sailer et al. 2000; Sterr and Dean 2008; Yordanova et al. 2004), functional imaging (fMRI) (Naccarato et al. 2006; Ward 2006; Ward and Frackowiak 2003; Ward et al. 2008), and transcranial magnetic stimulation (TMS) (Peinemann et al. 2001; Talelli et al. 2008a; Talelli et al. 2008b). Some consistent findings have been reported regarding task-related activation whereby cortical activity is more diffuse and less lateralized in advancing age (Cabeza 2001). Presumably, these findings may be explained in terms of adaptive plasticity in the motor system whereby older adults seek to maintain performance despite age-related changes in the brain (Ward and Frackowiak 2003). Such changes include reduction of gray and white matter (Ge et al. 2002; Liu et al. 2003b), degradation of cortical neurons (Dickstein et al. 2007), connectivity changes in cortical motor regions (Rowe et al. 2006), and a reduction in intra-cortical inhibition (Peinemann et al. 2001; Talelli et al. 2008b). Activation of a wider cortical network should not be viewed simply as an effective compensatory mechanism to match performance with a younger counterpart, but also an indication of the inability to selectively activate a given cortical network or region (Inuggi et al. 2009).

Studies of age-related and neurodegenerative changes in the central nervous system show that there are specific task-related effects on central activation, but the majority of these studies are limited to simple motor tasks (e.g. button presses, finger-thumb opposition) performed with minimal effort. This limitation may restrict our understanding of plasticity during repetitive motor actions. The changes during repetitive actions may also reflect in older adults a progressive decline in neural drive during repetition (Hunter et al. 2008), consistent with suboptimal neural output (Gandevia 2001). An exercise-related reduction in motor output is typically measured via motor-evoked potentials using TMS (Gandevia 2001). Utilizing the TMS technique a number of studies have examined cortical excitability during fatiguing contractions (Benwell et al. 2007a; Benwell et al. 2006; Hunter et al. 2008; Maruyama et al. 2006; Sacco et al. 1997; Taylor et al. 2000; Taylor et al. 1996). Taken together, these studies describe an increase in central motor drive through modulation of excitatory (i.e. increase in corticomotor excitability) and inhibitory (i.e. decrease in intra-cortical inhibition) networks (Benwell et al. 2007b). These adaptations are apparently made in order to compensate for diminished central motor drive and thereby to maintain performance of the motor task.

However, changes in excitability as assessed using TMS may not be analogous with changes in voluntary drive (Schillings et al. 2006). Several studies (Freude and Ullsperger 1987; Johnston et al. 2001; Liu et al. 2005a; Schillings et al. 2006) have investigated central adaptations to repetitive contractions by analyzing movement-related cortical potentials (MRCP), EEG-derived cortical activity occurring around the time of movement. [For review of MRCP see: (Shibasaki and Hallett 2006)] Contrary to motor-evoked potentials, recording of the EEG does not introduce artificial input into the system and indexes activity at the level of the cortex, and measurements are performed during, rather than following, repetitive contractions (Schillings et al. 2006). Further, as noted by Johnston and colleagues (Johnston et al. 2001), MRCPs have the advantage of assessing global changes occurring simultaneously across the cortex, and they disclose distinct periods of motor preparation, initiation, and execution. To this end, several MRCP studies (Freude and Ullsperger 1987; Johnston et al. 2001; Liu et al. 2005a; Schillings et al. 2006) had subjects perform 120–200 repetitive hand grip contractions at a high intensity, and recorded MRCP over the duration of the protocol. Over time, the amplitude of MRCP (i.e. electrocortical activation) significantly increased over SMA and contralateral sensorimotor areas. This increase in

MRCP amplitude was interpreted as a central adaptation to counteract suboptimal motor output (Schillings et al. 2006). Thus, regardless of methodology (i.e. TMS or EEG), there appear some consistency amongst findings concerning how the central nervous system may adapt in order to maintain performance during a repetitive motor task. However, these results are limited primarily to young healthy adults. The existing data do not provide an understanding of whether this adaptation is present with advancing age.

Utilizing the MRCP repetitive grasping paradigm cited above (Freude and Ullsperger 1987; Johnston et al. 2001; Liu et al. 2005a; Schillings et al. 2006), we examined the influence of aging on central adaptations. In light of recent work describing age-related EEG differences for baseline motor performance (Inuggi et al. 2009), we chose to explore the possibility of an interaction between age and MRCP amplitude change. We hypothesize that older adults maintain motor task performance dissimilarly from younger adults whereby they require mobilization of additional neural resources.

Methods

Subjects

Twenty subjects volunteered to participate and were subdivided into ‘Young’ (age mean $\pm$ SD, 24.1 ± 1.0 ; age range: 22–25) and ‘Old’ (68.8 ± 4.6 ; 59–78) groups. Each group consisted of 10 subjects (8 men and 2 women). All subjects were right-handed according to the Edinburgh handedness inventory (Oldfield 1971). None had history of musculoskeletal injury or neurological events, nor were any depressed (as confirmed by the Beck Depression Inventory) or taking any psychoactive medication for at least six months prior to entry into the study. All subjects gave their written informed consent to the experimental procedures, which conformed to the Declaration of Helsinki and was approved by the local Human Research Protection Office.

Experimental Procedures

Subjects were comfortably seated in an armchair with their dominant hand and forearm stabilized in a semi-supinated position by a vacuum positioning pillow (see Fig. 1). A strain gauge grip dynamometer (Biopac SS25; Goleta, CA) was placed in their dominant hand from which isometric handgrip force was recorded. This dynamometer did not allow for individual adjustments, but subjects were given instruction and practice on how to squeeze the device using a power grip configuration. Thereafter, a maximum voluntary contraction (MVC) was recorded as the largest force produced in a 3–5 s attempt. Subjects were given three attempts and the average was used for subsequent analysis. Standardized instructions were provided to all subjects.

Each subject then performed 150 intermittent handgrip contractions at 70% of their MVC. Contractions were performed in sets of 30 contractions each with a brief pause (<2 min) separating sets. These sets were segmented for analysis purposes into two Blocks of trials: one early (trials 1–60) and one late (trials 91–150). Repetitive grasping was self-initiated and subjects were instructed to contract once every 5–8 s. In order to maintain the appropriate contraction intensity, real-time visual force feedback was provided to the subject by means of a custom LabVIEW program (v8.0, National Instruments). A computer screen positioned at eye-level approximately 0.5 m in front of the subject displayed a large sphere that would illuminate once the subject reached the target force of 70% MVC. Once reached, subjects were instructed to relax immediately (Schillings et al. 2006). MVC was re-assessed following the completion of the fifth set of 30 trials.

All subjects completed the fatigue severity scale (Krupp et al. 1989) prior to any testing. They were also asked to subjectively rate their perceived exertion (RPE; Borg 1982), at rest and following completion of the repetitive contractions.

Electrophysiological Recording

Continuous EEG was recorded using an appropriately sized 61-channel elastic nylon Quick-cap, and data were acquired using the 70-channel NeuroScan Synamps2 amplifier system using the Scan 4.3 software (Compumedics; Charlotte, NC). Vertical and horizontal electro-oculograms (EOG) were recorded using Ag/AgCl electrodes placed above and below the right eye and the left and right outer canthi, respectively. Surface EMG was measured bipolarly using two miniature Ag/AgCl electrodes placed 20 mm apart over the flexor digitorum superficialis (FDS) muscle belly of the dominant arm as identified through palpation. Prior to EOG and EMG electrode application, the skin was cleaned and vigorously abraded. EEG, EOG, EMG, and force from the grip dynamometer were all continuously and synchronously recorded through the Synamps 2 amplifier and Scan 4.3 software. This system has a common mode rejection ratio of 100 dB, 24-bit A/D resolution, and input impedance of 10 M Ω . Data were recorded with a bandwidth of DC-100 Hz and sampled at 1 kHz. Impedances were kept below 5 k Ω for all electrodes.

EEG Analysis

Offline analysis was performed utilizing custom MATLAB programs (v7.3.0; Math Works, Inc.; Natick, MA). Raw EEG data were inspected visually to identify and remove signal artifacts. Data were high-pass filtered at 0.01 Hz (90 dB) to eliminate the baseline shift associated with DC recording and were referred to a common average of the signals from all electrode sites. For each trial (i.e. repetition), the onset of force was used to synchronize a 4 s epoch, 3 s before the onset and 1 s after. Force onset was defined as the point when the signal from the grip dynamometer exceeded a threshold of two standard deviations above the activity level at the beginning of the epoch and subsequently remained above that level for at least 500 ms. Acceptable epochs were averaged together forming an average MRCP for each participant. Averaging was performed separately on trials 1–60 (Block 1) and 91–150 (Block 2), and therefore allowed us to analyze main effects associated with Block.

MRCPs were decomposed into three distinct components that have been described and analyzed previously (Chiang et al. 2004; do Nascimento et al. 2005; Falvo et al. 2010; Kristeva et al. 1990; Slobounov et al. 2004; Yoshida et al. 2008): (1) mean amplitude between –600 and –500 ms prior to movement onset, *Bereitschaftspotential* (BP), (2) mean amplitude between –100 ms and movement onset, *motor potential* (MP), and (3) mean amplitude from onset to +100 ms, *movement-monitoring potential* (MMP). These measurement windows were used to characterize the time course of the MRCP and are illustrated in Fig. 2. Amplitudes were computed with reference to a baseline of –3000 to –2500 ms prior to movement onset. Onset of negativity (i.e. MRCP onset) was identified as the point when the baseline signal deviated from a 95% confidence interval and subsequently remained above that level for at least 500 ms from the period of –2500 to –2000 ms preceding movement. Our electrode sites of interest were consistent with prior MRCP studies utilizing this paradigm (Freude and Ullsperger 1987; Johnston et al. 2001; Liu et al. 2005a; Schillings et al. 2006) and included: Cz, FCz, CPz, C1, and C3. These electrodes were then grouped into two regions of interest, e.g. left motor region (LM = Cz, C1, C3), and central mesial (CM = FCz, Cz, CPz).

Two-dimensional (2D) topographical maps were created to reflect spatial features of the MRCP considering the entire 61-electrode montage. Separate 2D maps were created for each of the three distinct MRCP components by Block, using group mean data. Group mean

data rather than single-subject data were used to better represent true cortical activity preceding movement in terms of overall patterns in the data (Fang et al. 2004).

EMG and Force Analysis

EMG recorded during repetitive grasping and MVC attempts was band pass filtered in the 1–100 Hz frequency range and rectified, and maximum amplitudes were calculated. For repetitive grasping, maximum EMG amplitudes were calculated over a 2500 ms epoch (e.g. 1000 ms prior to onset to 1500 ms after onset), and then averaged across trials similar to the MRCP described above. For MVC attempts, maximum amplitudes were computed over the duration of the MVC attempt.

The force signal from the dynamometer was digitally high-pass filtered using a fourth order, zero-lag Butter-worth filter (5 Hz). Force–time histories were analyzed for peak and mean force during grasping and MVC attempts, respectively. Peak rate of force development (RFD) was also computed as the highest values of the slope coefficients of the tangent computed during a sliding 5 ms window (Viitasalo et al. 1980).

Statistics

EMG, and force data were analyzed using a mixed ANOVA design with Group and Block (Block 1, Block 2) as between- and within-subject factors, respectively. MRCP data were analyzed similarly along with the inclusion of baseline grip strength as a covariate (mixed ANCOVA). To account for multiple repeated-measures analyses, *P* values were based on the Huynh–Feldt corrected degrees of freedom. Post-hoc analyses, with correction, were performed with independent or paired *t*-tests where appropriate. All data are presented as mean $\pm$ SD and were analyzed using SPSS (v17; SPSS Inc., Chicago, IL). For all comparisons, a probability of less than or equal to 0.05% was considered to be statistically significant.

In addition to these primary analyses, we also performed statistical cluster plots as an exploratory analysis to utilize the high-density recording montage and assist with interpretation of scalp topography. These methods have been described in detail elsewhere (Murray et al. 2002; Wylie et al. 2003). In brief, cluster plots were generated by calculating pointwise, two-tailed *t*-tests of MRCP responses between groups (e.g. young versus old). Results were then displayed as an intensity plot (Fig. 6). The recording epoch, electrode location, and *t* test result (color value) at each data point are represented by *x*, *y*, and *z* axes, respectively. Electrode locations were as follows: Occipital (Oz, O1, O2); Occipito-Parietal (POz, PO1, PO2, PO7, PO8); Parietal (PZ, P1, P2, P3, P4, P5, P6, P7, P8); Parieto-Central (CPz, CP1, CP2, CP3, CP4, CP5, CP6, TP7, TP8); Central (Cz, C1, C2, C3, C4, C5, C6); Fronto-Central (FCz, FC1, FC2, FC3, FC4, FC5, FC6, FT7, FT8); Frontal (Fz, F1, F2, F3, F4, F5, F6, F7, F8); and Pre-Frontal (FPz, FP1, FP2, AFz, AF1, AF2, AF7, AF8).

Results

Young and Old subjects scored similarly and low on the fatigue severity scale (average score = 2.1). Their RPE following completion of the protocol was also similar ($P = 0.75$) and all were able to successfully complete all 150 contractions. Two-way ANOVA found no significant interaction or Block main effect for any force or EMG variable during MVC testing. Similarly, no differences were found in force or EMG variables during the repetitive grasping. In addition, the absence of behavioral change between Blocks suggests subjects performed the task similarly across Blocks. This is supported by similar ($P > 0.05$) inter-trial intervals during Blocks 1 and 2 for Young (Block 1 = 8.6 ± 2.3 s; Block 2 = 8.5 ± 2.2 s) and Old (5.9 ± 1.6 s; 6.4 ± 1.9) groups. Inter-trial intervals are defined as the time period from

force offset of one contraction to force onset of the ensuing contraction (i.e. inter-trial pacing).

Using a mixed ANCOVA, we observed significant interactions for each MRCP component in both the CM ($F_{(1,17)} = 5.16\text{--}6.55$, $P = 0.02\text{--}0.03$) and LM regions ($F_{(1,17)} = 4.64\text{--}5.26$, $P = 0.03\text{--}0.05$). Paired one-way t -tests with Bonferroni correction ($\alpha/6 = 0.008$), indicated significant increases in amplitude from Block 1 to Block 2 in Young subjects for CM (BP, $P = 0.005$; MP, $P = 0.004$; and MMP, $P = 0.005$) and LM (BP, $P = 0.002$; MP, $P = 0.001$; and MMP, $P = 0.001$) electrode clusters. However, no significant Block differences were observed in the Old group. Onset times demonstrated a main effect for Block for CM ($F_{(1,18)} = 9.07$, $P = 0.01$) and LM ($F_{(1,18)} = 4.17$, $P = 0.05$), but only LM showed a significant interaction ($F_{(1,18)} = 5.30$, $P = 0.03$). Paired one-way t -tests ($\alpha/2 = 0.02$) indicated onsets began earlier for Young subjects at CM ($P = 0.01$) and LM ($P = 0.01$), but only at CM ($P = 0.04$) for the Old group. Mean data are presented in Fig. 3. Waveforms in Fig. 4 are representative of group mean data for CM and LM electrode clusters. 2D spatial plots (Fig. 5) are also comprised of group mean data, and activity was plotted for time intervals defining the three MRCP components.

Discussion

In the present study, electrocortical activation was investigated during repetitive grasping to study the effects of age on central adaptation. The major finding of this study underscores age-related differences in electrocortical activation. Specifically, focal activity is significantly increased in younger adults, but such an increase is minimal in older adults as observed by MRCP activity.

Effects of Aging

Similar to prior MRCP studies in healthy young adults (Freude and Ullsperger 1987; Johnston et al. 2001; Liu et al. 2005a; Schillings et al. 2006), we observed a significant increase in activity over both central mesial and contralateral motor areas upon repeated movements (Fig. 4). However, this adaptation was minimal (non-significant) in older adults, indicating an age-related difference in central adaptation during repetitive grasping in agreement with our hypothesis. To our knowledge, no studies have examined age-related differences on brain activation patterns during such a paradigm, although several have focused in healthy young adults (Benwell et al. 2007a; Benwell et al. 2007b; Benwell et al. 2006; Freude and Ullsperger 1987; Johnston et al. 2001; Liu et al. 2003a; Liu et al. 2005a; Liu et al. 2005b; Maruyama et al. 2006; Schillings et al. 2006). Evidence from these prior studies has suggested that young adults demonstrate an increase in central motor drive (e.g. premotor, motor, sensorimotor) to counteract suboptimal voluntary drive. Our results in young adults confirm these findings in that cortical activity increased progressively for each MRCP component (BP, MP, MMP) over CM electrodes and for most components (MP, MMP) over LM electrode sites. In addition, MRCP onset began earlier over sensorimotor regions further reflecting supraspinal adaptation responsible for movement preparation and execution. However, similar findings were not found in older adults where we found no evidence for adaptation, i.e. no differences observed between Blocks 1 and 2.

In addition to adaptation within cortical motor centers, another possible countermeasure would be the involvement of additional brain regions to maintain sufficient neural drive, which has been interpreted as an increase in central effort (Dobkin 2008). There is some evidence for this additional recruitment in the present experiment (Figs. 5, 6), particularly in the period prior to movement onset. In a previous study, fMRI data recorded during intermittent handgrip contractions demonstrated a progressive increase in activity in ipsilateral sensorimotor cortex, prefrontal cortex, cingulate gyrus, and cerebellum (Liu et al.

2003a). The investigators reasoned that similar to motor neuron pools in the spinal cord, the brain may also recruit more cells (Liu et al. 2003b). Enhanced brain activation may also be a function of the increase in sensory feedback during repetitive grasping (Liu et al. 2005b). Alternatively, Liu and colleagues have proposed the “shifting of activation center” hypothesis to explain the recruitment of additional brain regions (Liu et al. 2007). They hypothesize that the brain has multiple motor control centers with parallel projections to motor neuron pools; therefore, if neurons in one location become unable to maintain adequate output, the brain may shift activation to another group of neurons. Through EEG source reconstruction, these authors demonstrated that during 200 unilateral maximal handgrip contractions, the center of brain activation shifted in the direction of anterior, inferior, and ipsilateral locations (Liu et al. 2007). This concept has recently been supported via EEG-EMG coherence maps recorded during sustained fatiguing elbow flexion which demonstrated augmented ipsilateral activation (Yang et al. 2009). As described in simplified form by Yang et al., under normal circumstances, contralateral M1 controls voluntary muscle contractions directly via monosynaptic corticospinal pathways which is underscored by strong beta (15–30 Hz) band coherence. Presumably, as the brain shifts to more indirect polysynaptic pathways to muscle, i.e. shifting the activation center, a weakened coupling would be expected (Yang et al. 2009).

There appears to be converging and consistent evidence of central adaptive processes during repetitive grasping, but adaptation appears significantly blunted in the CM and LM studied here for older adults. Several possible mechanisms may explain these findings. In response to age-related deterioration of the nervous system (e.g. shrinkage of M1, loss of neurons), older adults recruit larger neuronal populations (Inuggi et al. 2009; Ward 2006) to produce an intended movement. Greater activation may also be the result of the normal aging process in which integration of sensorimotor processing becomes less efficient. In other words, greater computational effort is required on the part of older adults to perform a task at the same absolute level as a younger adult and this increased effort is reflected at the systems level (Ward and Frackowiak 2003). This would seem plausible as information processing related to anticipation and preparation of a motor response changes with age. These changes are related to alterations in cognitive processes that engage pre- and supplementary motor areas, areas subject to structural changes with aging (Sterr and Dean 2008). Our statistical cluster plots (Fig. 6) and 2D spatial plots (Fig. 5) appear to confirm these previous findings, particularly in parietal (Block 1) and frontal regions (Block 2).

Studies have also observed the absence of lateralized activity (Sterr and Dean 2008) or a shift towards ipsilateral M1 activation with increasing age (Naccarato et al. 2006; Talelli et al. 2008a; Ward et al. 2008), likely due to reduction in interhemispheric inhibition (Peinemann et al. 2001; Talelli et al. 2008b). Ipsilateral deactivation of M1 is believed to occur through transcallosal inhibition, which may be impaired or reduced in older subjects (Ward and Frackowiak 2003; Ward et al. 2008). Along with activation of a wider cortical network, this shift in activation may also be in agreement with Liu et al.’s shifting of activation center hypothesis. All of these studies fit into a similar physiological concept, namely that the aging brain would need to mobilize additional primary and non-primary motor resources to accomplish successful task performance (Sailer et al. 2000). We speculate that given the finite resources available in central sensorimotor and premotor regions, older adults may need to recruit additional networks that subserve motor function (Fig. 6).

Paradigm Considerations

A detailed assessment of multiple EMG sites is lacking in the present investigation as a result of practical limitations with our recording system, i.e. limited available channels for detailed assessment of EMG activity at sites other than the principal effector muscle.

Therefore, it may be questioned whether cortical changes are manifested by recruitment of non-prime movers of the involved arm as well as activation of contralateral musculature (i.e. mirror movements). We do not consider these significant problems for two reasons. First, all subjects were provided a thorough familiarization that specifically focused on activating the involved hand for gripping and were continuously monitored. Also, if unwanted movements were made during performance of a trial this would likely interject movement artifacts within the EEG recording which is screened for and eliminated during data analysis. Second, larger EMG montages including assessment of contralateral musculature (e.g. finger, arm, shoulder sites) has been performed in three separate studies employing 100–200 intermittent handgrip MVCs (Liu et al. 2007; Liu et al. 2005a; Liu et al. 2005b). EMG activity of the prime and non-prime movers of the contralateral limb remained low and similar to pre-exercise values, and activity of the non-prime movers of the involved limb did not significantly change over the protocol and remained low (Liu et al. 2007; Liu et al. 2005a; Liu et al. 2005b). As the present study utilized the same intermittent handgrip design, albeit at a 30% lower intensity, we expect minimal contribution from muscles outside of the prime movers of the involved hand.

Despite substantial increases in RPE from baseline (Young: +47%; Old: +45), we observed no significant changes in MVC or EMG. This differs from a previous study in young adults where following 120 handgrip contractions at 70% MVC, post-exercise MVC was 58% of its baseline value (Johnston et al. 2001). However, subjects of this study were asked to maintain 70% MVC contraction level for four seconds after reaching the target whereas in our study subjects immediately relaxed after reaching the target. Despite this, our observed increases in MRCP are consistent with and support previous findings (Johnston et al. 2001), albeit their MRCP amplitudes on average were higher particularly during the MP phase of the MRCP waveform. Our protocol more closely replicates that performed by Schillings and colleagues (Schillings et al. 2006) in which subjects were also instructed to relax after reaching the target 70% MVC. Although their protocol included 50 more contractions than the present study, they also were unable to demonstrate a significant decrease in MVC or increase in EMG. The authors suggested their observed increase in MRCP amplitude reflects diminished efficiency of the motor cortex.

Conclusions and Future Directions

Successful performance of repetitive grasping reflects the adaptable and plastic nature of cortical networks governing motor function. From our data, it appears that cortical regions relied upon for dominant hand grasping (premotor, contralateral motor) may sufficiently augment activity to maintain an appropriate level of motor output. During the gripping protocol studied here, however, it appears this central adaptation is attenuated with aging. As a result, older adults may rely upon mobilization of additional brain regions, in support of a shifting of activation hypothesis (Liu et al. 2007). This adaptation is likely due in part to a greater central effort at baseline. Future research is needed to clarify these mechanisms in individuals with and without pathology, considering amplitude as well as spatial-depth changes, as amplitude alone may not be sensitive in discriminating effects.

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Fig. 1. Illustration of experimental protocol *Note:* Subjects were seated in a semi-recumbent position and computer monitor was placed at eye-level at a distance of approximately 0.5 m

Fig. 2. Measurement windows of the movement-related cortical potential (MRCP): Bereitschaftspotential (BP), motor potential (MP), and movement-monitoring potential (MMP). *Note:* The time of '0' on the X-axis indicates movement onset

Fig. 3. Grand averaged BP, MP, and MMP amplitude for central mesial (CM) and left motor (LM) electrode sites across the top and bottom panels, respectively. *Error bars* are in standard error units

Fig. 4. Grand average MRCP waveform at CM (*top*) and LM (*bottom*) electrode sites for Experiment 1. *Note:* These are used for clarification and are not exact

Fig. 5. 2D topographical maps of component amplitudes. *Note:* Each map is oriented such that the anterior-posterior axis is arranged vertically with nasion located at the top

Fig. 6. Statistical cluster plots. *Color values/dashes* represent the result of pointwise *t* tests evaluating the young versus old subject responses during Block 1 (**a**) and Block 2 (**b**) across our 4 s epoch ('0 s' = movement onset). Electrode positions (Y-axis) represent the entire recording montage. Only *P* values < 0.05 are plotted according to the intensity bar (Color figure online)