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REVIEW

Somato-motor inhibitory processing in humans: evidence from neurophysiology and neuroimaging

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Abstract Motor execution processing has been examined using an index of behavioral performance such as reaction times, kinetics, and kinematics. However, difficulties have been associated with the study of motor inhibitory processing because of the absence of actual behavioral performance. Therefore, non-invasive neurophysiological and neuroimaging methods including electroencephalography, magnetoencephalography, transcranial magnetic stimulation, and functional magnetic resonance imaging have been used to investigate neural processes in the central nervous system. We mainly reviewed research on somato-motor inhibitory processing based on data obtained by using these techniques, which can examine 'when', 'where, and 'how' motor inhibition occurs in the brain. Although to date a number of studies have used these techniques separately, few studies have utilized them in a comprehensive manner. In this review, we provide evidence that combining neurophysiological and neuroimaging methods should contribute to our understanding of how executive and inhibitory functions are implemented.

Keywords No-go · Nogo · ERP · MEG · fMRI · TMS

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Introduction

When motor execution is performed in daily life and sports activities, the brain receives information from the surrounding environment, such as visual, auditory, and somatosensory stimuli, and the interior of the body including muscle spindles and visceral sensations. The brain then quickly and accurately judges and evaluates the information inputted, and decisional processing takes place. Voluntary movement involves a complex process with neuronal activities from different brain regions, including motor preparation and motor intention, more or less consciously or unconsciously. In contrast, the brain often makes a decision not to perform these movements. For example, a soccer player occasionally stops to kick a ball when the course of the pass to a team-mate is interrupted by an opponent. This processing is called 'inhibition', and actively suppresses the motor command to accomplish a task. Therefore, the motor control system in the brain includes both motor execution and inhibitory processing.

A number of studies on the motor control system in human brains have so far focused on motor execution processing; however, many issues have yet to be resolved in the study of motor inhibitory processing. The principal reason for this is that motor execution has been studied based on an index of behavioral performance such as the reaction time (RT), electromyography (EMG), and movement kinematics/kinetics, whereas motor inhibition research on a behavior index has been difficult because of the absence of actual behavioral performance. Therefore, other methodologies are needed to clarify the neural basis for motor inhibitory processing. Due to accelerated science technological progress, several non-invasive recording methods have recently been used to measure human brain

activity in motor inhibitory processing. Among these are methods based on neurophysiology, including electroencephalography (EEG), magnetoencephalography (MEG), and transcranial magnetic stimulation (TMS), and those based on neuroimaging, including functional magnetic resonance imaging (fMRI) and positron emission tomography (PET). These methods have also been used when subjects performed Go/No-go paradigms. Go/No-go paradigms include two different stimuli (e.g., green and red signals), which are randomly presented to the subjects. Subjects have to respond by pushing a button with their hand as quickly as possible only after the presentation of a Go stimulus (e.g., green signal). On the other hand, subjects are asked not to respond to a No-go stimulus (e.g., red signal). Thus, brain activity during Go trials reflects motor execution processing, while that during No-go trials involves motor inhibitory processing. By utilizing the neurophysiological and neuroimaging methods, the neural mechanisms responsible for motor inhibitory processing, 'when, where, and how the inhibition occurs in human brain', have been investigated.

In this review, we mainly introduced some recent findings on somato-motor inhibitory processing based on our neurophysiological and neuroimaging methods.

Event-related potentials (ERPs)

Sensory modality and ERPs for inhibitory processing

EEG is indispensable for examining neural activities in the human brain and offers high temporal resolution in the order of milliseconds. EEG technology captures fluctuations in the electrical voltage of the brain through electrodes placed on the scalp in accordance with the standardized guidelines of the International 10–20 system [1]. Because motor execution and inhibitory processing are instantaneously performed within 1 s in the human brain, this technique is necessary to clarify the time course of neuronal activities. Event-related potentials (ERPs) obtained by time-locked averaging EEG have been used to investigate the neural substrates of motor execution and inhibition during Go/No-go paradigms. ERPs are often recorded separately for Go and No-go trials with the same probability to avoid the effect of the stimulus probability and minimize differences in response conflict between event types [2, 3].

Two large components, which show a negative deflection at approximately 140–300 ms (N2) after the stimulus onset and a positive deflection at approximately 300–600 ms (P3), are elicited in No-go trials, and differ from the ERPs recorded in Go trials [4–7]. Moreover, the anteriorization of the No-go-P3 (P3 evoked by No-go stimuli) has often been recorded; No-go-P3 shows a more anterior distribution than Go-P3 (P3 evoked by Go stimuli) [5, 6, 8]. This characteristic has clearly distinguished it from the classical P300 or P3b, which was previously shown to have a parietal distribution in oddball paradigms [9].

ERP components during Go/No-go paradigms have mainly been investigated using visual and auditory stimulations. We recently reported the characteristics of ERPs during somatosensory (tactile) and pain (noxious) Go/No-go paradigms. We assumed that Go/No-go effects would not be dependent on sensory modalities if motor inhibitory processing was truly performed. In our first experiment [10], we stimulated the second (Go) or fifth digit (No-go) of the left hand with ring electrodes, and the probability of the stimulus for the second and fifth digit was even. Subjects were asked to respond by grasping a grip with their right hand (contralateral to the stimulated side) as fast as possible when a Go stimulus was presented. The amplitude of the No-go-N140 component (N140 evoked by No-go stimuli) was found to be more negative than that of the Go-N140 (N140 evoked by Go stimuli) in the frontocentral electrodes. The amplitude of No-go-P300 (P300 evoked by No-go stimuli) was also larger than that of Go-P300 (P300 evoked by Go stimuli) in the frontocentral electrodes (Fig. 1). These characteristics were not observed under resting control conditions, in which subjects were asked to relax and rest quietly with no task. The enhanced No-go-related components, No-go-N140 and No-go-P300, indicated that brain potentials were not dependent on sensory modalities, and reflected common neural activities specific to the inhibitory process.

However, this enhancement may have been specific to the brain response when the fifth digit was stimulated. Therefore, the effects of task conditions on somatosensory N140 and P300 components, such as difficulty, response pattern, and stimulus presentation, as well as stimulus sites, had to be clarified. We used four conditions [11]. In condition 1 (L2 condition), the Go stimulus was delivered to the second digit of the left hand and the No-go stimulus to the fifth digit of the left hand. In condition 2 (L5 condition), the Go and No-go stimuli were reversed in the left hand: the Go and No-go stimuli were delivered to the fifth and second digits, respectively. In condition 3 (R2 condition), the Go stimulus was delivered to the second digit of the right hand and the No-go stimulus was delivered to the fifth digit of the right hand. In condition 4 (R5 condition), the Go and No-go stimuli were reversed in the right hand. Subjects had to respond to a Go stimulus by pushing a button with the thumb contralateral to the stimulated side as quickly as possible under all conditions. The results obtained revealed that enhanced No-go-related components, No-go-N140 and No-go-P300, were confirmed under

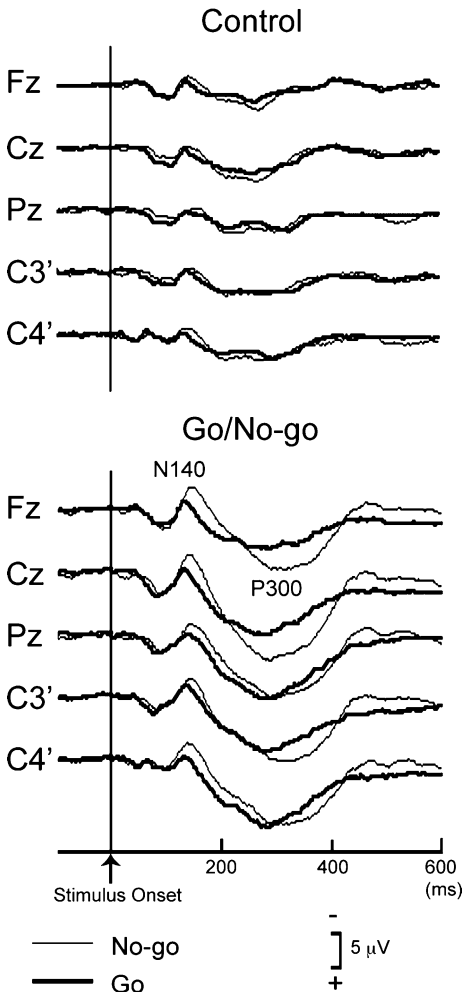

Fig. 1 Grand-averaged ERP waveforms during the resting control condition (*upper*). Grand-averaged ERP waveforms during the Go/No-go paradigm (*lower*)

all conditions (Fig. 2), which indicated that No-go-related components were elicited independent of the response hand or stimulus sites.

We also investigated the characteristics of ERPs using painful (noxious) stimulations during the Go/No-go paradigm [12]. Intraepidermal electrical stimulation, which preferentially and selectively provides Aδ fiber nociceptive stimuli without tactile sensations, was used for the painful stimulation [13, 14]. This intraepidermal method can stimulate the same points on the skin surface, compared with traditional laser stimulation, and does not cause bleeding or burns. Using this stimulation, we recorded ERPs under three conditions: the rest control, Go/No-go paradigm, and choice reaction task (CRT). The N2 and P3 components in No-go trials, which peaked at around 190 and 390 ms after the stimulation, were clearly later than those in our previous studies during standard somatosensory (tactile) stimulation. This delay appeared to reflect the slow conduction velocity through Aδ fibers and the spinothalamic tract to the cerebral cortex, in which pain and temperature sensations are mediated [15, 16]. The amplitudes of No-go-N2 and No-go-P3 were larger than those of Go-N2 and Go-P3 in the frontocentral electrodes in the Go/No-go paradigm, but were not in Control and CRT (Fig. 3). Our results indicated that inhibitory processing was not dependent on the sensory modality used such as visual, auditory, somatosensory, and pain.

However, care is needed when interpreting N2 and P3 components because the generator mechanisms for inhibitory processing may differ among sensory modalities. Previous studies demonstrated that the amplitude of N2 was markedly smaller following auditory than visual stimuli [5, 17, 18]. According to Kiefer and colleagues [18, 19], the No-go-N2 component during auditory paradigms could be recorded more easily in the inferior temporal electrode than in the frontocentral electrodes. Regarding the P3 component, Falkenstein and colleagues reported a larger amplitude in visual paradigms than in auditory paradigms, and the topography was more frontal for auditory stimuli than visual stimuli [17]. In addition, the peak latency of the N2 component was clearly different during the somatosensory modality than during the visual and auditory modalities. For example, the No-go-N140 component during somatosensory paradigms peaked 80–160 ms after the stimulus onset in the frontocentral electrodes (e.g., mean 150 ms in Nakata et al. [10]; mean 150–160 ms in Nakata et al. [3]; 80–160 ms in Hatem et al. [20]; mean 148 ms in Nakata et al. [21]), whereas the N2 component peaked after 180–300 ms in the auditory paradigms (e.g., mean 291 ms in Falkenstein et al. [17]; mean 260 ms in Kiefer et al. [18]; mean 202 ms in Smith et al. [22]; 180–250 ms in Barry et al. [23]) and 200–350 ms in visual paradigms (e.g., mean 306 ms in Falkenstein et al. [17]; mean 280–300 ms in Kato et al. [24]; 200–350 ms in Di Russo et al. [25]). To date, no studies have examined the effects of visual, auditory, and somatosensory stimuli in the same subjects, which is needed to elucidate the common and uncommon effects of sensory modalities on N2 and P3.

The relationship between exogenous factors and ERPs

We manipulated the stimulus conditions, interstimulus interval (ISI), and stimulus probability during somatosensory Go/No-go paradigms to clarify the characteristics of somatosensory ERPs with exogenous factors. The manipulation of ISI more directly reflected 'temporal probability', while that of stimulus probability reflected 'sequential probability'. Temporal probability refers to the occurrence of a particular stimulus within a period of time, whereas sequential probability refers to the probability of a particular stimulus within a number of stimuli [26, 27]. The effects of these manipulations have been investigated to

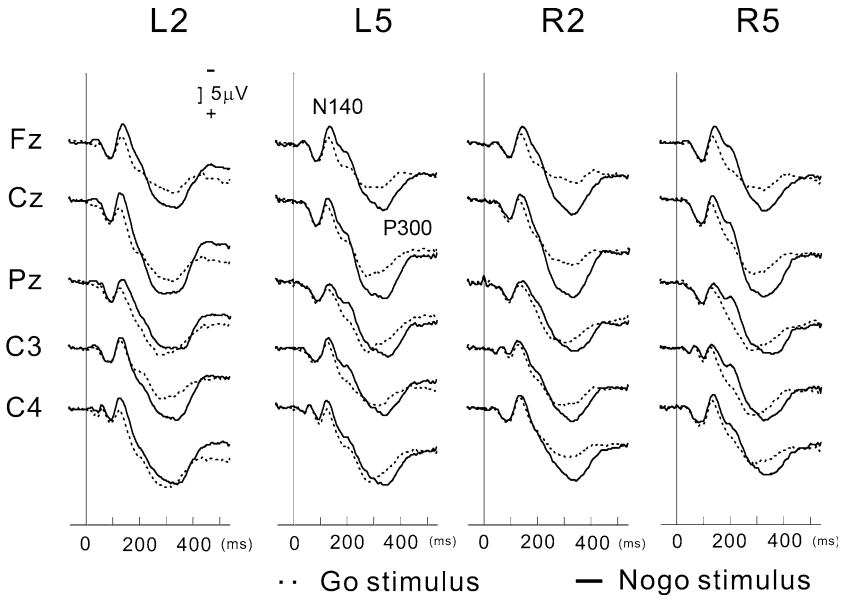

Fig. 2 Grand-averaged ERPs for each Go/No-go paradigm. *L2* the Go stimulus was delivered to the second digit of the left hand. *L5* the Go stimulus was given to the fifth digit of the left hand. *R2* the Go stimulus was delivered to the second digit of the right hand. *R5* the Go stimulus was given to the fifth digit of the right hand

clarify the characteristics of ERP components such as P300 [27–30].

In a study on the effects of ISI, Recio and colleagues [31] reported that the mean amplitudes of N2 and P3 became larger with increasing ISI, and similar effects were found on both Go- and No-go-P3, and Go- and No-go-N2. However, since they analyzed the mean amplitude between 200 and 250 ms for the N2 component, and between 300 and 400 ms for the P3 component, the effects of ISI on the peak amplitude and latency of N2 and P3 remain unclear. Peak latency includes important data relating to the stimulus classification speed or stimulus evaluation time in Go/No-go paradigms [32–34]. Thus, the effects of ISI on the peak amplitude and latency of ERP waveforms need to be analyzed. We established four conditions with different ISI: 1, 2, 4, and 6 s during the somatosensory Go/No-go paradigm. The amplitude of the N140 component increased as the ISI increased. Significant differences were observed in the amplitude of N140 between the No-go and Go trials at 1-s and 2-s ISI, but not at 4-s or 6-s ISI. The amplitude of the P300 component also increased with the ISI. Significant differences were also noted in the amplitude of P300 between the No-go and Go trials at all ISI (Fig. 4) [35].

As examples of the effects of the stimulus probability, Eimer [36] found a more pronounced enhancement of the N2 amplitude by No-go stimuli, with a probability of 25 %, than No-go stimuli, with a probability of 50 %, using visual Go/No-go paradigms. Bruin and Wijers used two visual Go/No-go paradigms, and subjects either responded manually to Go stimuli or counted the occurrence of each Go stimulus silently under different conditions [37]. The stimulus probability for Go and No-go trials varied in both response mode conditions (i.e. Go 25 % and No-go 75 %,

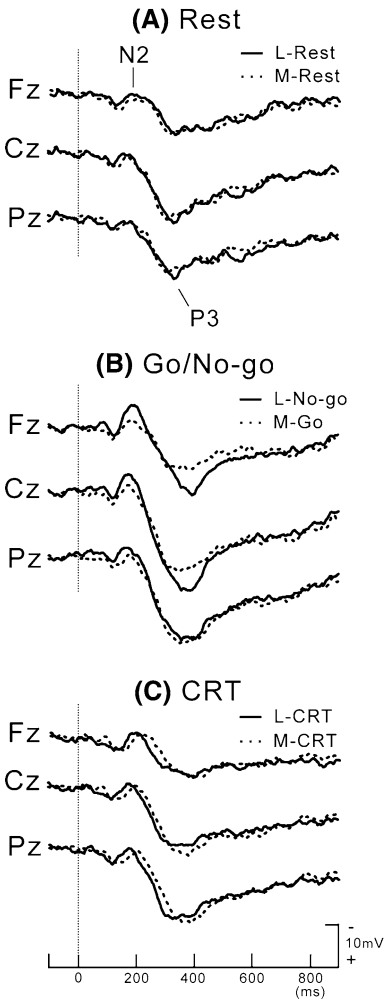

Fig. 3 Grand-averaged ERPs under the three conditions. **a** ERPs in the Rest condition. **b** ERPs in the Go/No-go paradigm. **c** ERPs in the Choice reaction task (CRT)

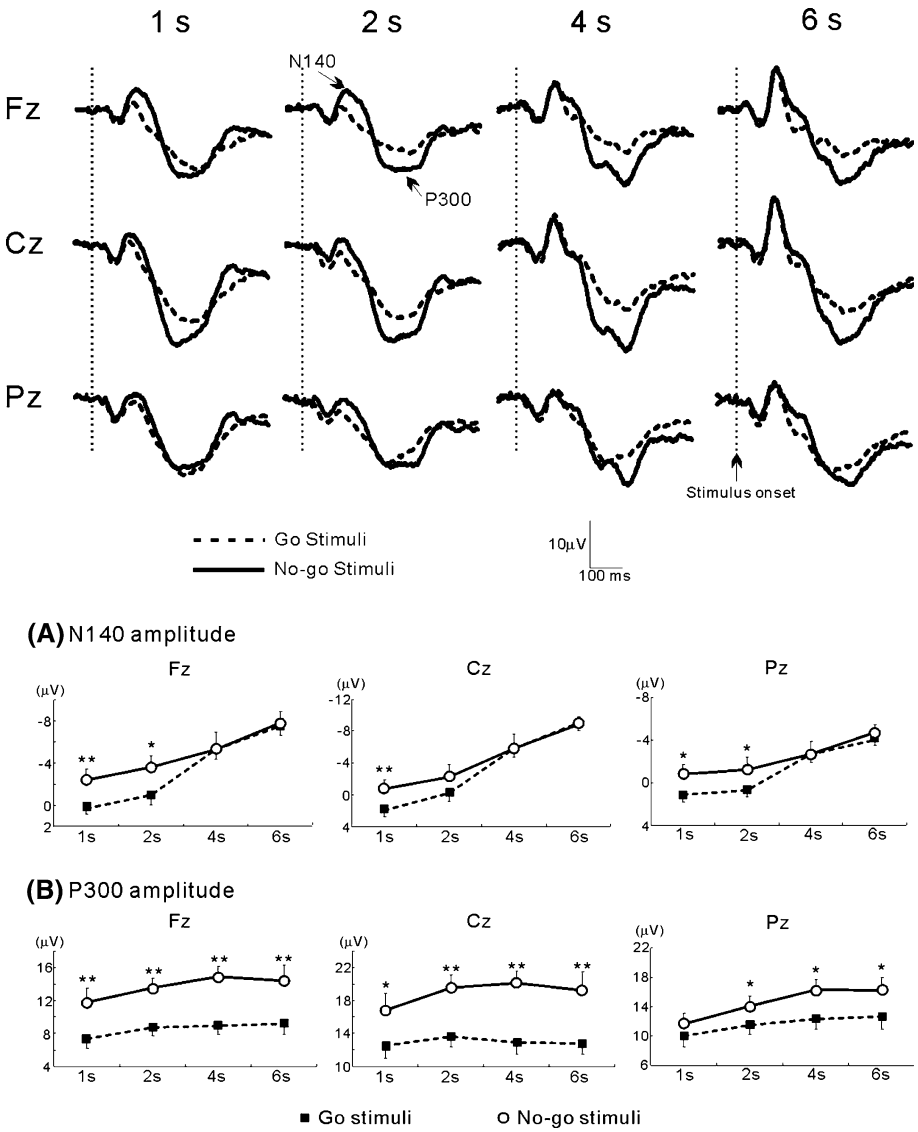

Fig. 4 Grand-averaged ERPs under conditions that were manipulated by an interstimlus interval (ISI) (*upper*). Mean value of the N140 and P300 amplitudes with standard errors (SE) (*lower*). Significant differences between Go and No-go trials: $^{**}p < 0.01$, $^{*}p < 0.05$

Go 50 % and No-go 50 %, and Go 75 % and No-go 25 %). They found similar N2 and P3 Go/No-go effects in both movement and counting tasks, and the amplitude of No-go-N2 and No-go-P3 varied with the No-go stimulus probability. Enriquez-Geppert and colleagues combined Go/No-go and Stop signal paradigms, and investigated the effects of the stimulus probability on visual N200 and P300 components [38]. In their paradigm, the probability of No-go and Stop signals was even (i.e. Go 75 %, No-go 12.5 %, and Stop signal 12.5 % in the 75 % block, and Go 25 %, No-go 37.5 %, and Stop signal 37.5 % in the 25 % block). They found that the amplitudes of P3 during the No-go and Stop trials were significantly larger in the 75 % Go block than in the 25 % Go block. However, the effects of the stimulus probability have not yet been confirmed in somatosensory modalities during Go/No-go paradigms. We set three conditions with three different stimulus probabilities during the somatosensory Go/No-go paradigm: Go = 20 %, No-go = 80 %; Go = 50 %, No-go = 50 %; Go = 80 %, No-go = 20 %. However, this analysis was performed to compare the ERP waveforms between Go 20 % and No-go 20 % trials (i.e. 20 % probability), between Go 50 % and No-go 50 % trials (i.e. 50 % probability), and between Go 80 % and No-go 80 % trials (i.e. 80 % probability). An increase was observed in the peak amplitude of N140 with a decrease in the stimulus probability. In addition, no significant Go/No-go effect was observed on N140 in the 20 %-ERP waveforms including Go 20 % and No-go 20 % trials (Fig. 5). This finding indicated that the effect of the stimulus probability on the amplitude of N140 was larger than the Go/No-go effect during 20 % ERPs. As for P300, the amplitude of both Go- and No-go-P300 increased as the stimulus probability decreased, and the Go/No-go effect was observed under all stimulus probability conditions (Fig. 5). This result indicated that the Go/No-go effect was larger than the stimulus

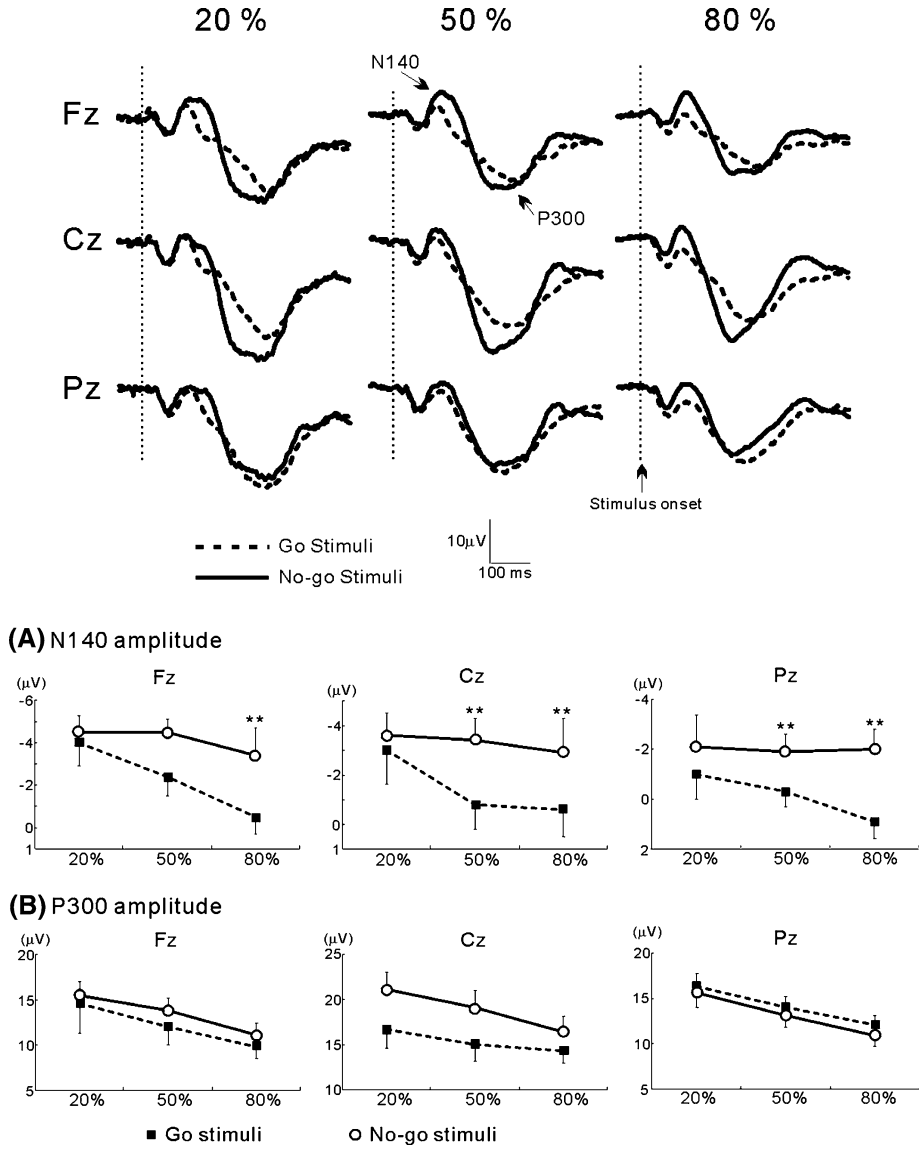

Fig. 5 Grand-averaged ERPs under conditions that were manipulated by the stimulus probability (*upper*). Mean value of the N140 and P300 amplitudes with SE (*uower*). Significant differences between Go and No-go trials: $^{**}p < 0.01$

probability effect on the amplitude of P300, and the effect of the stimulus probability differed between the amplitudes of N140 and P300.

In addition to ISI and the stimulus probability, the effects of other exogenous factors such as the stimulus intensity, stimulus sites (e.g., hand, foot, and face), and response mode (e.g., right foot or left foot) on ERPs have not been investigated during somatosensory Go/No-go paradigms. These factors need to be clarified in future studies.

The relationship between motor execution and inhibition

ERP waveforms are known to be affected by the status of motor preparation. For example, the amplitude of No-go-N2 was shown to be enhanced with an increase in the time pressure for the response speed [32, 39] by the presence of a preceding cue signal [4] and a low error rate [5]. These findings suggested that motor preparation for a faster and more accurate response was related to the larger amplitude of N2 and/or P3 components in Go/No-go paradigms, and that a stronger inhibitory process was needed to suppress motor execution under these conditions than under the opposite conditions. Another factor associated with motor inhibitory processing is the strength of motor execution for Go trials because controlling response force is important for precise motor execution. Several studies have reported a relationship between the response force and movement-related neural activities, and the amplitudes of readiness potential (RP) or Bereitschaftspotential (BP) were enhanced with an increase in muscle force [40, 41]. We previously hypothesized that a stronger inhibitory process may be required to suppress motor execution with a

stronger response force if a higher amplitude of RP for the stronger motor performance reflected an enhanced motor preparation process [42]. We set three conditions with different force levels during somatosensory Go/No-go paradigms: 10, 30, and 50 % of the maximal voluntary contraction (MVC) of each subject. Subjects were asked to adjust their force level to match the target line with the force trajectory line as quickly and accurately as possible only when the Go stimuli were presented. The results obtained revealed that the peak amplitude of No-go-N140 at Fz was significantly enhanced with an increase in the muscle force (Fig. 6), which suggested that a stronger inhibitory process was required to suppress stronger preparatory motor activity in No-go trials.

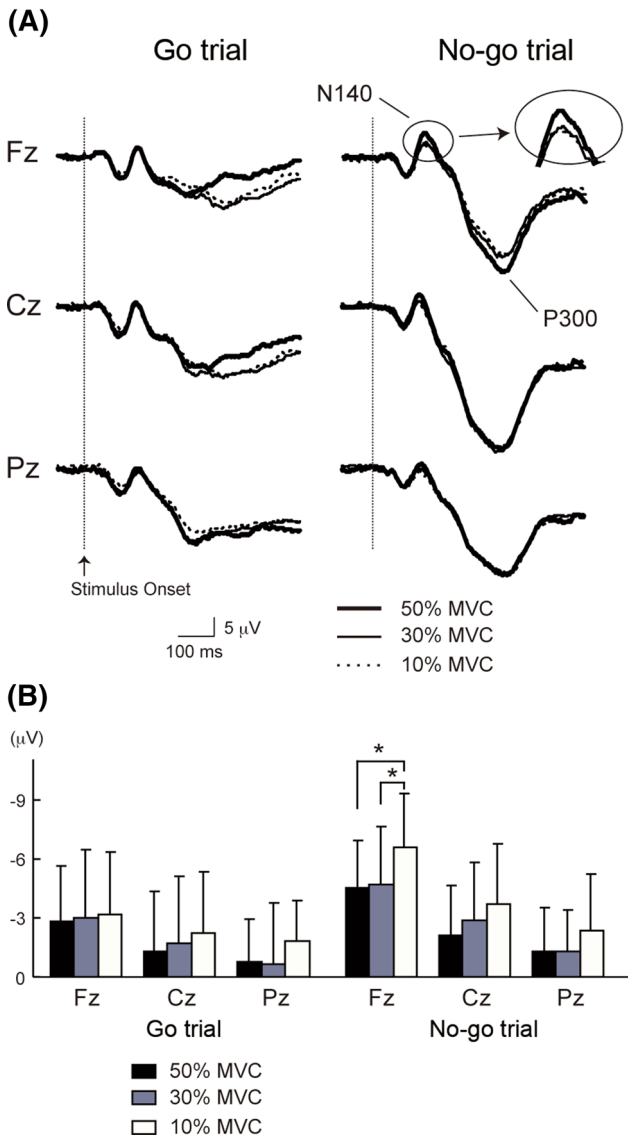

Fig. 6 **a** Grand-averaged ERPs under the three conditions during the Go/No-go paradigm. **b** Mean amplitude with SD for Go- and No-go-N140 at Fz, Cz, and Pz. The amplitude of No-go-N140 was significantly more negative under the 50 % MVC condition than under the 10 and 30 % MVC conditions at Fz. *$p < 0.01$

We also investigated the relationship between the RT, response variability, and somatosensory ERPs during Go/No-go paradigms [21]. In addition to RT, response variability has been shown to be an important factor for evaluating the speed and accuracy of movement. It is often calculated as the standard deviation (SD) of RT [43, 44], which reflects the variability of the time from the stimulus onset to the response, indicating components such as the stimulus evaluation and response selection. A negative correlation was observed between RT and the amplitude of No-go-P300, which indicated that subjects with a shorter RT had a No-go-P300 with a larger amplitude. The latency of Go-P300 correlated with RT (Fig. 7a). The SD of RT correlated with the amplitudes of No-go-P300 and Go-P300, and the latencies of No-go-P300 and Go-P300 (Fig. 7b). This result suggested that the response speed and variability for the Go stimulus in Go/No-go paradigms may have affected neural activity of inhibitory processing for the No-go stimulus.

Taking our ERP studies into consideration, motor inhibitory processing was closely associated with motor execution including strength, speed, and variability, and neural activity relating to the motor inhibition modulated with the required motor execution.

Magnetoencephalography (MEG)

Source modeling analysis

As described in the previous section, evaluating ERPs is a useful method to clarify the neural activities of motor execution and inhibition. However, scalp-recorded EEG cannot provide sufficient resolution to estimate the location of the electrical source in the brain. This has been attributed to the influence of volume currents on scalp-recorded EEG, which severely affect source identification. Furthermore, the large inter-individual variability of intervening tissues, including the scalp, skull, and cerebrospinal fluid, makes it difficult to estimate the dipole location (see reviews [45, 46]).

MEG technology can detect magnetic responses based on neuronal activity in the brain, whereas EEG records the electrical voltage of the brain. Thus, MEG and EEG measure the same neuronal activity with different technologies. However, the critical difference between these techniques is spatial resolution. Because magnetic responses are unaffected by the scalp, skull, or cerebrospinal fluid, MEG has theoretical advantages over EEG. MEG also has high temporal resolution, which permits neural activity to be differentiated on a time scale of milliseconds.

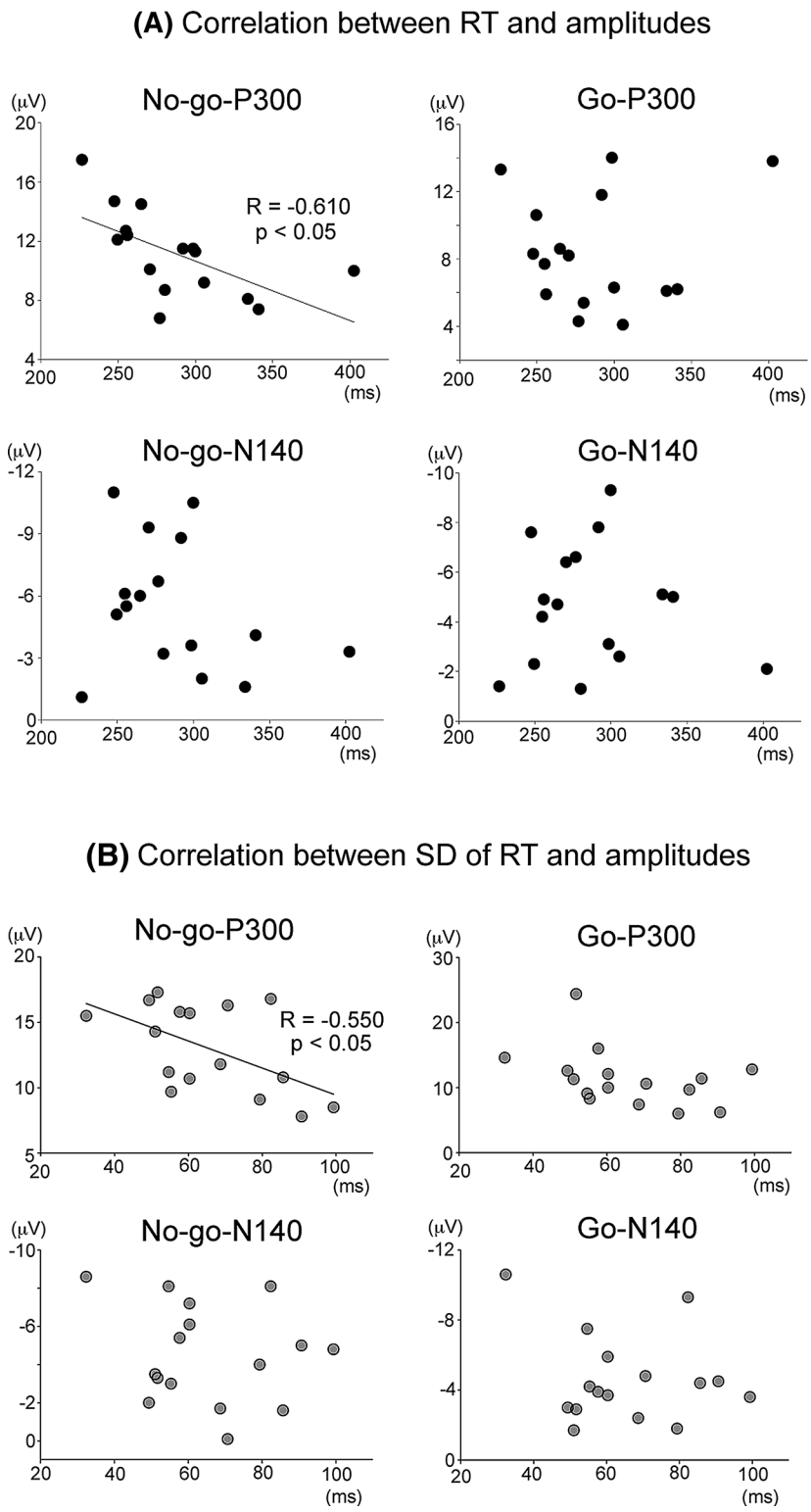

Fig. 7 **a** Correlation between RT and the N140 and P300 amplitudes at Fz, **b** Correlation between the SD of RT and the N140 and P300 amplitudes at C3

We used MEG and attempted to identify the regions involved in inhibitory processing during the somatosensory Go/No-go paradigm [3]. We used three conditions. Condition 1 was the resting control with no specific tasks. In condition 2, the Go stimulus was delivered to the second digit of the left hand, while the No-go stimulus was delivered to the fifth digit of the left hand. In condition 3, the Go and No-go stimuli were reversed in the left hand, i.e. the Go and No-go stimuli were delivered to the fifth and second digits, respectively. The result obtained showed that

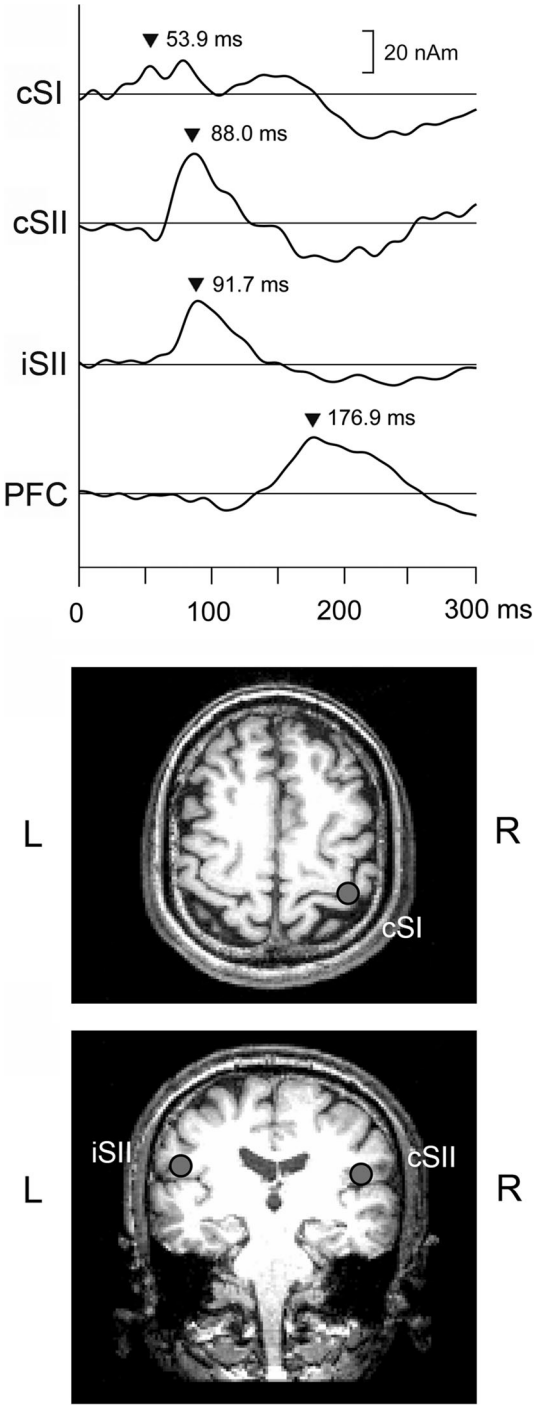

Fig. 8 The time-course of the ECD strengths of cSI, cSII, iSII, and prefrontal activities in No-go trials for the left second digit of a representative subject (*upper*). Location of the main response estimated in a representative subject superimposed on MRI scans (*lower*). *cSI* primary somatosensory cortex contralateral to the stimulation, *cSII* secondary somatosensory cortex contralateral to the stimulation, *iSII* secondary somatosensory cortex ipsilateral to the stimulation

the first major MEG signals peaked at approximately 50 ms in the hemisphere contralateral to the stimulated side, and the generator was estimated by equivalent current dipole (ECD) analysis in the postcentral wall, the primary somatosensory cortex (SI) (Fig. 8). The second signals peaked at 80–120 ms bilaterally, and were presumably generated in the upper bank of the Sylvian fissure, which corresponded to the secondary somatosensory cortex (SII), in both hemispheres (Fig. 8). These MEG signals were recorded under all conditions, independent of the sites stimulated (the second and fifth digits). The long-latency signal recorded only in No-go trials, No-go-M170, was then estimated around the posterior part of the inferior frontal sulci in the prefrontal cortex (PFC) (Fig. 9).

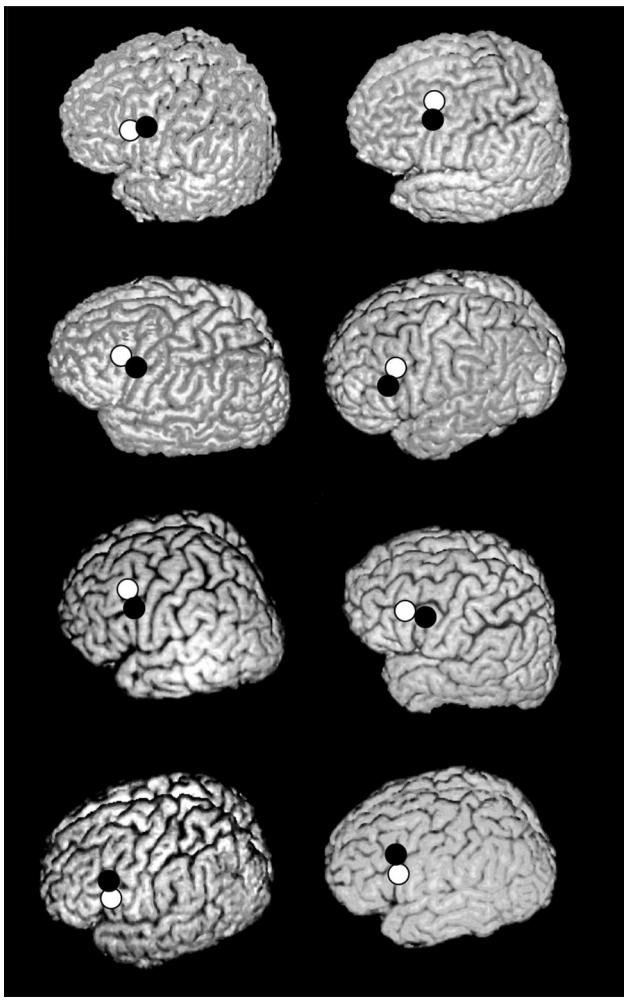

Fig. 9 No-go-dominant activity in a three-dimensional image of all eight subjects. Activity was estimated around the posterior part of the inferior frontal sulci in the prefrontal cortex. *Black and white points* indicate activities for the left second and fifth digits, respectively

Some previous studies attempted to identify No-go dominant activity during the visual Go/No-go paradigm by utilizing MEG [47–50]. Sasaki and colleagues [47] reported specific activity in No-go trials from the bilateral dorsolateral prefrontal cortex approximately 135 ms after the stimulus onset. In addition, several previous ERP studies have investigated the characteristics of the difference in ERP

waveforms between Go and No-go trials by subtracting the waveforms [5, 6, 33, 51–54]. Thorpe and colleagues [33] reported the significant differences between the Go and No-go stimuli from 150 ms onwards after the stimulus onset at the frontal electrodes. Falkenstein [6] showed that the N2d component, which was defined as the most negative peak at electrode Fz in the 100–300 ms window, peaked 172 ms after auditory stimuli and 238 ms after visual stimuli. As described above, the peak latency of N2 (N140) was clearly different among the 3 sensory modalities; visual, auditory, and somatosensory. However, the difference between the Go and No-go trials may be detectable after 150 ms onwards of the stimulus onset. During the somatosensory Go/No-go paradigms, the peak latency of the subtraction waveforms was approximately 180 ms after the stimulus onset [54], which was clearly later than that of N140 component. These MEG findings and our results using ERPs and MEG suggest that neural activity for motor inhibition started at least 130–200 ms after the No-go stimulus onset from the PFC. We also assumed that the starting time of this activity depended on the required tasks.

Cortical rhythmic activity

Several studies on EEG spectral power have examined the characteristics of cortical oscillations in No-go trials during Go/No-go paradigms [55–62]. A common finding was that the power of the theta, alpha, and beta frequency bands decreased or increased 300–900 ms after the onset of a No-go stimulus. For example, Leocani and colleagues [58] reported that the spectral power at 10 and 18–22 Hz decreased 300–600 ms after the stimulus onset, and the power at 10 and 18–22 Hz increased after 900–1,200 and 600–900 ms, respectively. Harmony and colleagues [62] reported a complex spatiotemporal pattern of spectral power decreases and increases under Go- and No-go conditions. These power changes may be due to a decrease or increase in the synchrony of underlying neuronal populations. The former case is called event-related desynchronization (ERD) (i.e. suppression), and the latter, event-related synchronization (ERS) (i.e. rebound) [63]. The role of cortical oscillatory activity in sensory, motor, and cognitive processing has been attracting interest as a key factor in binding mechanisms [64, 65]. These oscillations have been suggested to reflect an idling cortex generated by a large area of highly synchronous neuronal firing in the absence of inputs, or, alternatively, changes in coherent activity resulting from synchronous inputs from other brain regions [66].

We previously investigated the characteristics of cortical rhythmic activity in inhibitory processing during somatosensory Go/No-go paradigms by MEG [67]. Recordings were conducted under three conditions. In condition 1, the Go stimulus was delivered to the second digit, and the No-go stimulus to the fifth digit. Participants responded by pushing a button with their right thumb for the Go stimulus. In condition 2, the Go and No-go stimuli were reversed. Condition 3 was the resting control. The results obtained showed that a rebound in the amplitude was recorded in the No-go trials for theta, alpha, and beta activities, peaking after 600–900 ms. A suppression in the amplitude was recorded in the Go and No-go trials for alpha activity, peaking after 300–600 ms, and in Go and No-go trials for beta activity, peaking after 200–300 ms (Fig. 10). These findings indicated that cortical rhythmic activity clearly has several dissociated components related to different motor and cognitive functions, including response inhibition, execution, and decision-making. Moreover, we have to consider why the peak latency of the oscillatory responses was longer than those of the N2 and P3 components of ERPs, and how they corresponded to ERPs. We speculated that rebounds for theta, alpha, and beta activities in No-go trials reflected specific neural activities in motor inhibitory processing. We described the findings of TMS studies in the next section, but the amplitude of motor-evoked potentials (MEPs) was reduced after the onset of No-go stimuli. According to Waldvogel and colleagues [68], the inhibition of MEP amplitudes lasted for 500 ms after the No-go stimuli. No

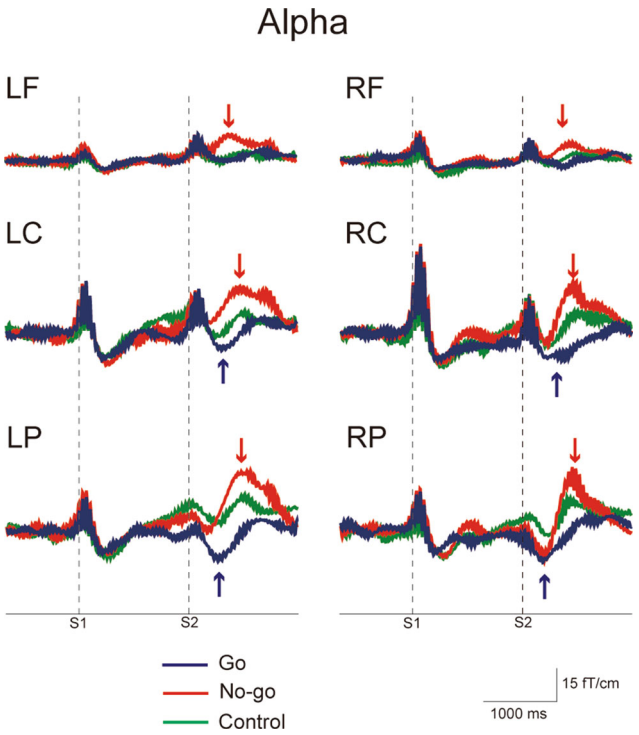

Fig. 10 Grand-averaged waveforms for alpha bands in each region. *Blue*, *red*, and *green lines* indicate waveforms for Go, No-go, and Control, respectively. *Red arrows* show the peak of the rebound, and *blue arrows* indicate the peak of the suppression. *LF* left frontal, *LC* left central, *LP* left parietal, *RF* right frontal, *RC* right central, *RP* right parietal

study has yet determined how long inhibitory processing lasts in the corticospinal tract; however, findings based on the cortical rhythmic activities of EEG and MEG suggest the existence of specific inhibitory functions, which differ from the neural activities of No-go-N2 and No-g-P3.

Functional magnetic resonance imaging (fMRI)

Findings in previous studies

fMRI measures blood oxygenation level-dependent (BOLD) signals, and those reflect magnetic differences between oxyhemoglobin and deoxyhemoglobin. Changes in BOLD signals have been correlated with neuronal activity [69], and fMRI is useful for clarifying neural networks with millimeter-order spatial resolution. Using this technique, many studies have attempted to clarify the neural networks involved in inhibitory processing during Go/No-go paradigms in the human brain, which include the dorsolateral (DLPFC) and ventrolateral (VLPFC) prefrontal cortices, supplementary motor area (SMA), anterior cingulate cortex (ACC), temporal and parietal lobes, and cerebellum [70–89]. Konishi and colleagues [72, 73], as well as Garavan and colleagues [74, 75], reported right-hemisphere dominance in response inhibition, which included the DLPFC, VLPFC, insula, and inferior parietal lobule (IPL), and right VLPFC in particular plays a key role in the neuronal networks involved in response inhibition in both the oculomotor and manual response modalities [88]. However, previous studies also described the activities of the bilateral PFC [70, 79, 81]. We considered that the difference in activation between the bilateral and right-lateralized PFC may be associated with various factors to perform the required Go/No-go paradigms, such as the task difficulty, presenting stimulation, response mode, and the strategy in each subject. Casey and colleagues [71] also showed that the DLPFC and VLPFC responded differently to change in target probabilities. They observed an inverse relationship such that, when activity in the DLPFC increased, activity in the VLPFC decreased, and vice versa. Mostofsky and colleagues [84], who used visual Movement and Count Go/No-go paradigms, reported that the pre-SMA was activated in No-go trials during both Go/No-go paradigms, which suggested that the pre-SMA is a common region in the neural networks involved in inhibitory processing independent of the required response mode.

Regions responsible for somato-motor inhibitory processing

We investigated the regions responsible for inhibitory processing during somatosensory Go/No-go paradigms by even-related fMRI [90]. Subjects preformed two different types of Go/No-go paradigms: (1) Movement and (2) Count. Our results showed that the response inhibition network involved the DLPFC, VLPFC, SMA, ACC, IPL, insula, and temporoparietal junction (TPJ), which were consistent with previous finding obtained using visual Go/No-go paradigms. These activities were confirmed in both Movement and Count No-go trials (Fig. 11). Therefore, these results indicated that the network for inhibitory processing was not dependent on sensory modalities, and reflected common neural activities.

We also analyzed brain activity involved in motor execution processing in Go trials [91]. We compared executive functions with different motor outputs during somatosensory Go/No-go paradigms: (1) Movement and (2) Count. We observed a common network for Movement and Count Go trials in several regions of the brain including the DLPFC, VLPFC, SMA, posterior parietal cortex (PPC), IPL, Insula, and superior temporal gyrus (STG) (Fig. 12). Direct comparisons revealed that the primary sensorimotor area (SMI), premotor area (PM), and ACC were activated more during Movement than Count Go trials. In contrast, the VLPFC was activated more during Count than Movement Go trials. These results suggested that there were two neural networks for the supramodal executive function, common and uncommon, depending on the required response mode.

We also evaluated the Negative BOLD effect on inhibitory processing during Go/No-go paradigms [92]. Recent fMRI studies have shown not only an increase but also a decrease in BOLD signals during tasks. This decrease is often referred to as 'Negative BOLD' or deactivation. The physiological basis for Negative BOLD has recently been investigated in detail, and non-human studies have described a coupling between Negative BOLD signals and decreases in neural activity [93, 94]. We observed a common Negative activation during Movement No-go and Count No-go trials in the right superior frontal gyrus (SFG), which corresponded to BA 8 (Fig. 13). These findings suggested that the right SFG region was responsible for the Negative BOLD effect on inhibitory processing, which was independent of the required response mode.

The neural basis for motor execution and inhibitory processing during somatosensory Go/No-go paradigms has been clarified based on the fMRI findings. However, neural mechanisms still need to be clarified in this study field. First, the temporal dynamics of brain regions involved in inhibitory processing need to be elucidated because fMRI has the limited temporal resolution associated with hemodynamic imaging. Previous ERP studies reported the time course of the brain regions using multi-dipole brain electric source analysis (BESA) [18, 95, 96] and low-resolution electromagnetic tomography (LORETA) [38, 97, 98]. For

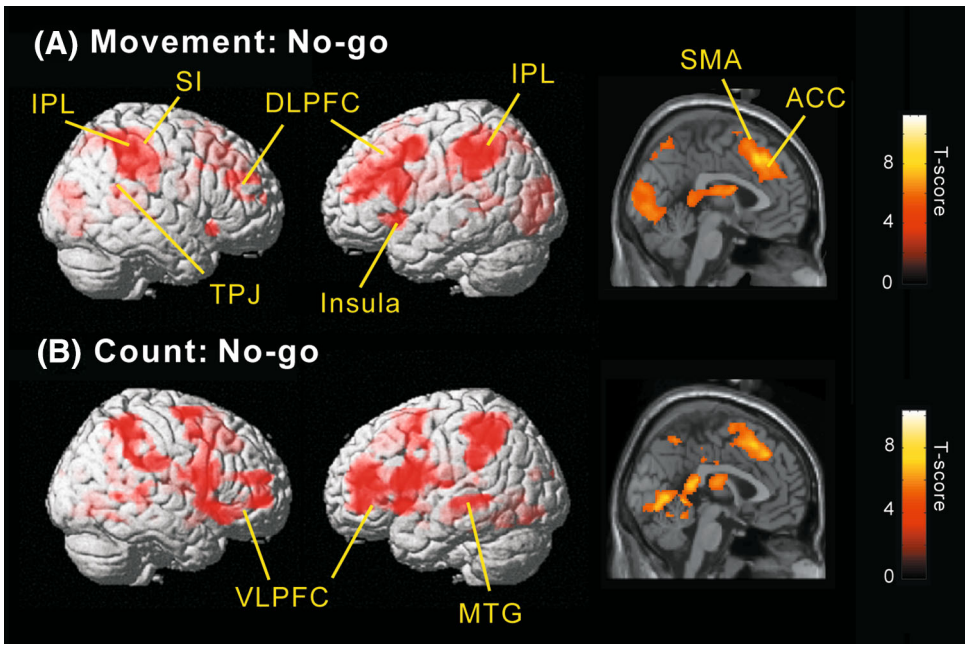

Fig. 11 Group activation maps showing activated brain regions in Movement No-go and Count No-go trials. *DLPFC* dorsolateral prefrontal cortex, *IPL* inferior parietal lobule, *SI* primary somatosensory area, *TPJ* temporoparietal junction, *SMA* supplementary motor area, *ACC* anterior cingulate cortex, *VLPFC* ventrolateral prefrontal cortex, *MTG* middle temporal gyrus

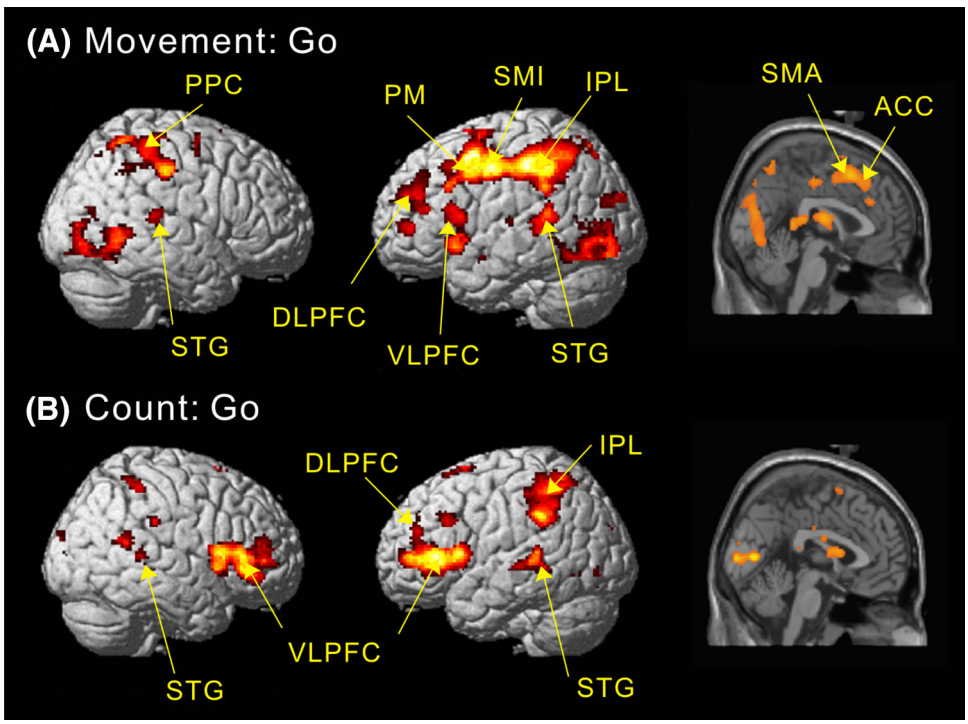

Fig. 12 Group activation maps showing activated brain regions in the Movement Go and Count Go trials. *PM* premotor area, *SMI* primary sensorimotor area, *STG* superior temporal gyrus, *PPC* posterior parietal cortex

example, Kiefer and colleagues [18] used auditory Go/No-go paradigms and reported that bilateral sources in the inferior prefrontal areas peaked at approximately 260 ms, and sources then peaked in the left precentral area at 500 ms and the frontal midline at 590 ms. Bokura and colleagues [97] used visual Go/No-go paradigms and showed that the source of the N1 component, which peaked at approximately 170 ms, was in the bilateral inferior temporal lobes, and the source of the No-go-N2 component, which peaked at approximately 265 ms, was located in the right lateral orbitofrontal and cingulate cortex, with weaker source activity being observed in the left thalamus. The source of No-go-P3 peaking at approximately 365 ms was in the left lateral orbitofrontal cortex. These studies demonstrated the temporal dynamics of No-go-related brain regions. However, the detailed mechanisms for each region are not fully understood because of the limitations of EEG recording and analysis methods.

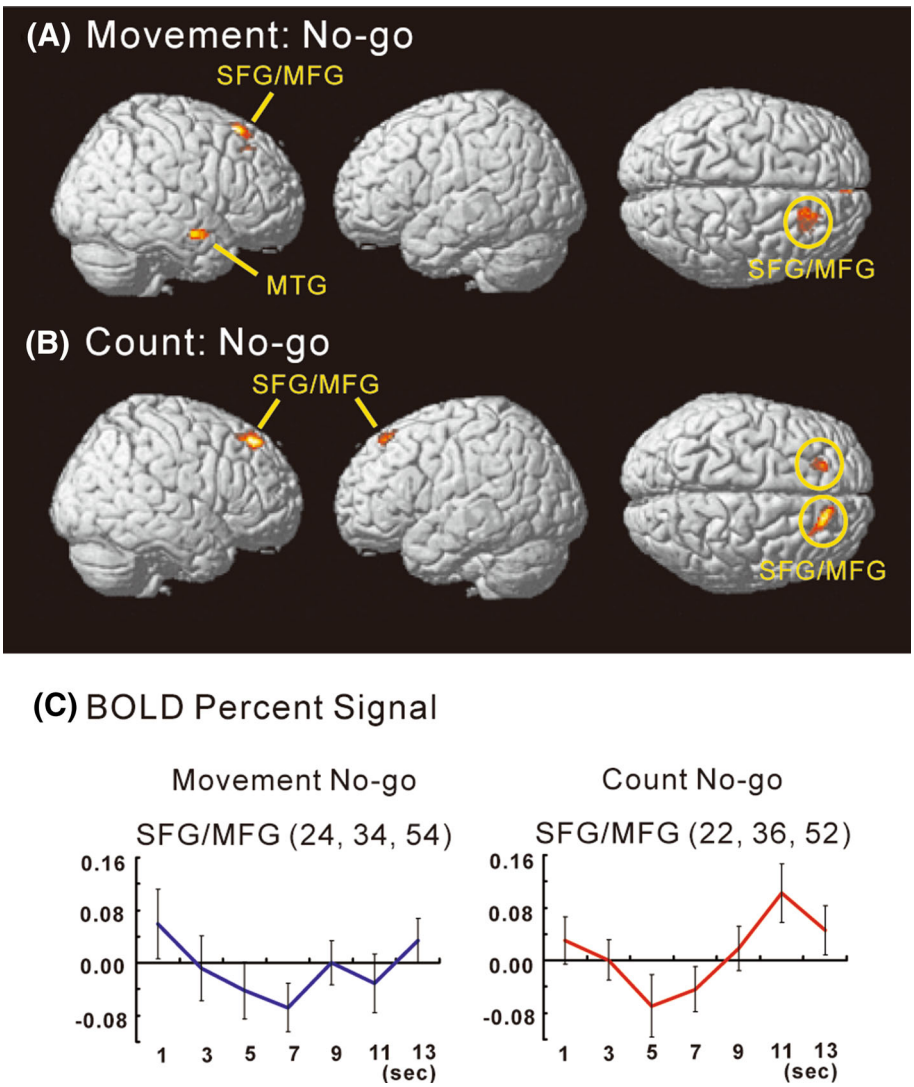

Fig. 13 Group activation maps showing deactivated brain regions in the **a** Movement No-go and **b** Count No-go trials. **c** Time course of hemodynamic responses in Movement No-go and Count No-go. *MFG* middle frontal gyrus, *MTG* middle temporal gyrus

Second, the role of each region in the neural networks involved in motor inhibition needs to be clarified. As described, numerous studies have reported the function of the PFC in motor inhibition, while the role of other regions has remained unclear. For example, activation in the PPC including IPL and superior parietal lobule (SPL) has been reported during many kinds of Go/No-go paradigms [74–79, 81–83, 85–90], whereas its role in motor inhibition remains controversial. Kalaska and Crammond [99] found that the decision not to move was clearly reflected in cell activity in the dorsal PM, but not in parietal cortex area 5, in the monkey brain, which suggested that PPC was not directly responsible for motor inhibition. The activities of the STG and middle temporal gyrus (MTG) were also observed during No-go trials in previous fMRI studies [75, 79, 83, 87]; however, the function of this area in the neural network involved in inhibitory processing remains unclear. Regarding functional connectivity in the neural networks involved in inhibitory processing, a recent study reported that the pre-SMA and MI have significant connectivity with the caudate head and subthalamic nucleus (STN), and also that the VLPFC and pre-SMA are reciprocally connected [100]. According to Sharp and colleagues [101], the VLPFC was associated with the attentional capture of an unexpected stimulus, whereas the pre-SMA was related to the inhibition of ongoing action. However, further studies are needed to address the role of each region in the neural networks involved in motor inhibition.

Transcranial magnetic stimulation (TMS)

Approximately 25 years ago, Sasaki and colleagues investigated the timing of cortical inhibition during visual Go/No-go paradigms in the monkey [102]. A stimulation was delivered directly to the PFC at different latencies after the onset of the Go stimulus, and suppressive or delayed

actions in the Go response were detected, especially at 100–150 ms. This is one of the most direct studies investigating the role of cortical inhibition in the PFC.

However, it is impossible to use the same methods in a human study to examine cortical inhibitory processing. Therefore, TMS has been used to investigate both excitatory and inhibitory effects on the corticospinal tract when subjects performed Go/No-go paradigms [53, 68, 103–106]. TMS of the primary motor cortex (MI) can produce relatively synchronous muscle responses, MEPs. If MEP amplitudes were larger during a task than during the resting control condition, the state was evaluated as an increase in cortico-spinal excitability. On the other hand, if MEP amplitudes were smaller during a task than during the resting control condition, it was considered as a decrease in cortico-spinal excitability. The common findings of these studies in Go/No-go paradigms were a decrease in MEP amplitudes 100–200 ms after No-go stimuli, and an increase after Go stimuli. These studies also reported the inhibition of both agonist and antagonist muscles [104] and the contralateral homologus muscle [105].

Our previous study utilizing TMS used the same somatosensory Go/No-go paradigms to produce different force levels during Go trials with ERPs [42]. We set three conditions with different force levels during somatosensory Go/No-go paradigms: 10, 30, and 50 % of the MVC of each subject, and each condition was performed separately. TMS was set over the left MI, and MEPs were recorded from the first dorsal interosseous muscle (FDI). TMS was triggered 150 ms after Go and No-go trials, and the MEP amplitudes in each condition were compared with those of the resting control. The results obtained revealed that the MEP amplitude for No-go trials was significantly smaller under the 50 % MVC condition than those under either the 10 or 30 % MVC conditions, and that the MEP amplitude for Go trials was significantly larger under the 50 % MVC condition than under the 10 % MVC condition (Fig. 14). As shown in our ERP study, the peak amplitude of No-go-N140 at Fz was significantly enhanced with an increase in the muscle force following No-go stimuli (Fig. 6a, b). Moreover, the MEP amplitudes in No-go trials became significantly smaller with an increase in the muscle force. While No-go-N140 components may be associated with neural activity in the PFC, the decrease in the MEP amplitudes from FDI reflects a decrease in cortico-spinal excitability. These results suggest that the strength of the inhibitory process is modulated by output changes in muscle strength.

In addition to previous studies using TMS over MI, one study directly applied TMS over PFC. Chambers and colleagues [107] investigated the critical role of the right PFC, MFG, and parietal cortex in inhibitory processing using repetitive TMS. They showed that temporary deactivation of the VLPFC in the right hemisphere impaired inhibitory control of the left and right hands, and TMS over the MFG and parietal cortex did not significantly alter inhibitory performance.

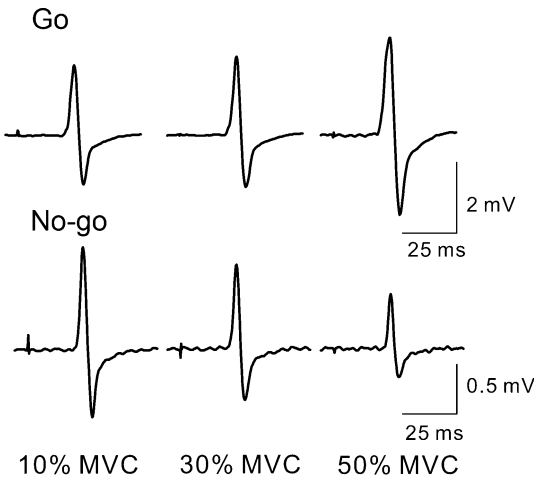

Fig. 14 MEPs for the Go and No-go trials under each condition from one representative subject. The MEP amplitude decreased gradually with an increase in the muscle force in the No-go trial, but increased in the Go trial

Muscle relaxation and Stop-signal

Neural mechanisms for muscle relaxation

In addition to motor inhibitory processing using Go/No-go paradigms, several studies have focused on muscle relaxation. While motor execution processing involving muscle contractions have often been emphasized, voluntary muscle relaxation should also play an important role in motor control. Previous studies have shown that unskilled novices co-contracted muscles in the upper and lower limbs (i.e. unclear reciprocal contraction of agonistic and antagonistic muscles) while playing the drums [108], piano [109], or badminton [110]. Dysfunctions associated with clinical diseases such as Parkinsonism [111] and focal dystonia [112] are also related to improper relaxation, due to both cortical and subcortical impairments. Similar to research on motor inhibitory processing, neurophysiological and neuroimaging studies using EEG [112–118], MEG [119], TMS [120, 121], and fMRI [122–124] have been used to elucidate the neural mechanisms underlying muscle relaxation. Common findings in these studies were that muscle relaxation was an active process that required cortical activation rather than merely ending muscle contraction. For example, previous fMRI studies demonstrated that muscle relaxation evoked the activation of some brain regions, including the MI, SMA, and pre-SMA [122, 124]. Spraker and colleagues found that the neural mechanisms

underlying slow and precisely controlled force reductions involved the DLPFC and ACC [124]. Based on the activated brain regions, there appeared to be differences in the neural networks involved in muscle relaxation and motor inhibition. Muscle relaxation may be related more to motor-related brain regions involving the MI, SMA, and PM, while motor inhibition during Go/No-go paradigms may be associated more with broad brain regions such as the DLPFC, VLPFC, SMA, ACC, IPL, insula, and TPJ, which were described in the previous section. This was supported by EEG studies that focused on muscle relaxation. Several studies reported movement-related cortical potentials (MRCPs) preceding voluntary muscle relaxation of the hand muscles, and waveforms were similar between muscle relaxation and contraction [112, 113, 115]. MRCPs are generally recorded before self-initiated voluntary movement, and reflect movement preparation processing (reviewed in Shibasaki and Hallett [125]). These potentials begin with a slow rising negativity, called the Bereitschaftspotential (BP), and progress to a steeper, later negativity starting approximately 500 ms before movement onset, called the negativity slope (NS′). The generation of MRCPs mainly involves movement-related regions. BP and NS′ are generated from the pre-SMA, SMA, PM, MI, SI, ACC, and subcortical structures including the basal ganglia and thalamus, as shown by intracranial recordings [126–131], dipole modeling [132, 133], MEG [134, 135], and studies with monkeys [136, 137]. Previous studies recording MRCPs during muscle relaxation suggested that a certain preparatory activity including muscle relaxation and contraction may be based on similar neural substrates, especially in the movement-related brain regions. In TMS studies, Buccolieri and colleagues [120] measured MEP amplitudes following a single or pair of TMS pulses, which were plotted relative to the onset of muscle relaxation. They demonstrated that the enhancement observed in cortical activation that was related to inhibition played a role in suppressing corticospinal excitability during muscle relaxation. Begum and colleagues [121] used single-pulse TMS and double-pulse TMS, and also recorded MEPs and the silent period (SP). MEP amplitudes decreased prior to the onset of muscle relaxation (−21 to −70 ms), while the SP remained unchanged. Intra-cortical inhibition was smaller prior to muscle relaxation. They suggested that multiple inhibitory mechanisms acted in diverse ways to achieve motor inhibition.

Neural mechanisms for inhibitory processing during Stop signal paradigms

Many studies have focused on inhibitory processing in Stop signal paradigms. As described above, Go/No-go paradigms require the high-level cognitive functions of decision-making, response selection, motor execution, and motor inhibition. No-go signals are delivered before any action is undertaken. In contrast, the Stop signal paradigm needs motor responses to be withheld such that the cancellation of the planned motor command is needed as a response to Stop cue. Stop signals are delivered after a Go command has been issued. They contain a higher load on response inhibition processes than the Go/No-go paradigms. To date, many fMRI studies have investigated the inhibitory processing involved in Stop signal paradigms [80, 138–142]. Rubia and colleagues [80] found commonly activated regions between the Go/No-go and Stop signal paradigms, including the left and right VLPFC, right DLPFC, ACC, pre-SMA, right IPL, and left middle temporal cortex. They also reported the stronger activation of regions in Go/No-go paradigms than in Stop signal paradigms, including the left PM, left IPL, and left SMA, which were in greater demand during Go/No-go paradigms than during the Stop signal paradigm. Aron and colleagues also [138] recorded BOLD signals in patients with lesions in the right PFC and compared them to those in control healthy subjects. They found a correlation between the stop signal reaction time (SSRT) and volume of damage to the right VLPFC, and no correlations in other regions. Leung and Cai [140] used Stop signal paradigms, and found significant activation during the inhibition of both manual and saccadic responses in the bilateral VLPFC, bilateral insula, SFG, right DLPFC, and right IPL. Tabu and colleagues [142] also reported common activation during both hand and foot Stop signal paradigms in the bilateral VLPFC, bilateral insula, bilateral DLPFC, pre-SMA, and parietal cortex. Badry and colleagues [143] used a TMS during Stop signal paradigms, and reported the significant suppression of MEP amplitudes recorded from bilateral hand and foot muscles in successful Stop trials, but not in failed Stop trials. Their findings suggested that the cortical activation of inhibitory processing affected not only the cortico-motoneuronal excitability of the task-performing hand but also the entire motor system. The findings of these studies indicated that the neural substrates are basically similar between Go/No-go and Stop signal paradigms, with the activation pattern depending on the required tasks.

Neuroplasticity of inhibitory function

Athletes participate in long-term training and practice, often starting very early in childhood, throughout their entire careers. Therefore, ‘the athlete’s brain’ offers a good opportunity for studying neuroplasticity. Motor-skill learning relates to the acquisition of a motor ability as a result of repetition or long-term training [144]. Previous studies have examined the neuroplasticity of inhibitory function during the Go/No-go paradigm among athletes.

Kida and colleagues [145] assessed the effects of baseball experience or skill levels on simple RTs and Go/No-go RTs in 82 university students (22 baseball players, 22 tennis players, and 38 non-athletes) and 17 professional baseball players. They observed no significant differences in simple RT either for sports experience or for skill levels, whereas the Go/No-go RT for baseball players was significantly shorter than that of tennis players and non-athletes. The Go/No-go RT of higher-skill baseball players was significantly shorter than that of lower-skill players, while that of professional baseball players was the shortest. These findings suggested that intensive physical training in sports, including Go/No-go decisional processing, improves the Go/No-go RT, but not simple RT.

Nakamoto and Mori [146] recorded ERPs during visual Go/No-go paradigms, and found that the amplitudes of P300 in No-go trials were larger in baseball players than in control subjects at the frontal (Fz) electrodes when the stimulus–response mapping used was similar to that in baseball batting. They suggested that the brains of baseball players have stronger inhibitory functions that stop movements. Similar findings using Go/No-go paradigms were also reported in fencers [147].

The findings of these studies indicated that reinforced neural networks and plastic changes are induced by the acquisition and execution of compound motor skills during extensive daily physical training requiring quick decision-making and specific attention. However, to precisely identify the brain regions in which plastic changes occur, different non-invasive recording methods, such as fMRI and MEG, should be performed in future studies.

In addition to plasticity, a 'deficit' in inhibitory function is an important topic for understanding the underlying mechanisms of motor inhibitory processing. This was reviewed in a recent article [148].

Conclusions

The present review mainly described spatial and temporal neural networks in somato-motor inhibitory processing. An investigation of the neural substrates in the human brain that cleverly and skilfully manipulate the body is warranted, and these neural networks should be related to motor execution and inhibition processing. In addition, human society is influenced by our brains, whether consciously or not, in order to control self-expression and self-restraint. Thus, we assume that the research theme, 'inhibitory processing', is more directly accessible to approach the final goal of human sciences, 'What it is to be human?'. The combination of neurophysiological and neuroimaging methods, such as ERPs, MEG, fMRI, and TMS, should contribute to our understanding of how executive and inhibitory functions are implemented.

Conflict of interest None.

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