The old-but-new theories about human voluntary motor control

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Abstract For more than a century, neuroscientists have debated the problem of how self-generated voluntary motor actions are executed without interference from sensory information elicited by the movements. In the 1950's, two theories were proposed: the corollary discharge theory by Sperry¹⁾ and the efference copy theory by von Holst and Mittelstaedt²⁾. They are essentially identical in proposing the necessity of specific neuronal mechanisms to suppress undesirable percepts or reflexes, both of which might disturb ongoing movements. However, this raises a simple question as to whether the control system always ignores sensory information whenever the intended movements are in progress. Here, we review recent findings and currently debated problems in corollary discharge theory.

Keywords : corollary discharge theory, equilibrium point hypothesis, relativity, brain imaging

Visual stability

Human and other foveate animals execute rapid eye movements (saccades) to fixate a target of interest with the fovea. Despite the presence of a slipping image motion on the retina during saccades, the visual system maintains an accurate and seamless representation of space and time between fixations. This implies that the visual perception of self-generated image displacement on the retina could be 'blocked' during eye movements. The so-called visual stability has commonly been attributed to this ability of the visual system to compensate for retinal shifts evoked by eye movements themselves. This idea was first proposed by von Helmholtz in 1866, who called the adjusting factor the "sense of effort". Thereafter, Nobel laureate Roger Sperry¹⁾ hypothesized that when the brain commands the eyes to move, it also sends a corollary discharge (CD) signal of that command to the visual system. Almost at the same time, von Holst and Mittelstaedt²⁾ suggested that motor commands leave an image of themselves (*efference copy*) somewhere in the central nervous system that is then compared to the afference elicited by the movement (*reafference*). While the concepts of these two theories are slightly different, the central idea common to them is that the oculomotor system sends some information about the motor signal to the visual system when it initiates a movement, and that by this signal the transient shift of the retinal image of the visual world during saccadic periods is blocked from perception. The necessity of this compensatory information in solving the visual stability problem has been repeatedly

claimed, and recent advances in neuroscience have led us to the next stage which confirms the theory as a biological reality.

In animal experiments, multiple roles of the CD signal to achieve visual stability have been suggested; the CD signal is a component required to suppress image motion across the retina caused by the saccade^{3,4)}, to drive a predictive shift of the receptive fields of visually responsive cells prior to a saccade⁵⁻⁷⁾, or to realize both roles leading to the perception of a stable visual world across saccadic periods⁸⁾. Sommer and Wurtz⁶⁾ showed in a 2002 *Science* paper that a pathway from the brainstem oculomotor center to the frontal cortex conveys a CD signal in the brain of monkeys: this pathway might cause visual neurons of the cortex to suddenly shift their receptive field just before a saccade. In their subsequent paper⁷⁾, they completed the possible neuronal correlates for that circuit. They exploited the fact that the signals are relayed via the thalamus. By knocking out the relay neurons, they interrupted the pathway, and found that receptive field shifts were curtailed by more than half. This led them to conclude that the receptive fields in the visual cortex are shifted by the CD signal from the oculomotor center in the brainstem.

In psychophysics, a great number of studies have suggested the existence of the CD signal, some of which are specifically attractive in attempting to replicate a time varying pattern of the CD signal. The most widely accepted current model is based on the experiment by Martin⁹⁾, in which a so-called damped eye position model was used in an attempt to incorporate the empirical finding for the pattern of mislocalization of flashed stimuli presented during saccadic periods. In the model, it was assumed that

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the CD signal encoding an intended eye position between fixations is both anticipatory and damped, leading to an N-shaped timecourse as a whole, beginning to move long before saccade onset but lagging behind the eye movement after the mid portion of the saccadic periods. This model has been supported by subsequent researchers¹⁰⁻¹⁴⁾. However, the pattern of the signal was still hypothetical, since the model was elaborated to replicate the pattern of mislocalization of *stationary* stimuli presented during the course of movements. To date, no evidence has been found to specifically support the existence of a continuously-varying, damped eye position signal^{15,16)}, implying that the empirical data to account for the concrete pattern of the CD signal do not exist.

The mirror paradigm

When we look into a mirror and move our eyes, we cannot catch our own eyes in motion. Since its first documentation in Erdmann and Dodge's report in 1898, this 'mirror paradigm' has been widely cited to illustrate the saccadic suppression phenomenon¹⁷⁻¹⁹⁾. Many researchers support the notion that our inability to see our own eye movements during saccades is of central origin; viz., from factors arising from a CD of the eye movement command, which instructs the visual system to suppress its sensitivity^{3,4)}; others suggest that such a central process is unnecessary, and that saccadic suppression can be attributed to peripheral (retinal) processes²⁰⁾. Besides such divergent accounts for the same phenomenon, the 'mirror paradigm' poses a practical difficulty from two perspectives: 1. eye displacements which are small relative to the size of the saccades are least likely to be detected by the gazing observer²¹⁾, and 2. even in larger saccades the actual gaze during saccadic periods is not directed to either of the two eyes but to the open space between them, simply suggesting a limitation of space and time available for an intra-saccadic percept of our own eye(s) in motion.

To circumvent these negative effects, Suzuki and Yamazaki²²⁾ examined an experimental situation in which horizontal saccadic eye movements were fed back to gazing observers using an oscilloscope screen (Fig. 1A). The subjects conducted rightward horizontal saccades over the oscilloscope screen, where eye movements represented by electro-oculographic (EOG) signals were displayed on-line as horizontal motion of a light spot. The scale of the light spot was carefully adjusted such that its horizontal moving distance between start and ending positions was equal to the corresponding change of gaze direction between separate fixations. During the saccadic period, three different stimuli with 1 millisecond (ms) durations were presented simultaneously: a vertical pulse stimulus (Sp) superimposed on the motion of the light spot and two *stationary* stimuli, S1 and S2, which were flashed near the initial fixation point (T1) and near target position (T2), respectively [Panel A(1)]. All these stimuli were triggered

simultaneously when the EOG signal passed several different amplitude levels between T1 and T2 [Panel A(3)]. Note that in physical space, the Sp changed its position along with the light spot, while S1 and S2 remained stationary near T1 and T2, respectively.

The subjects conceived that the motion of a light spot between separate fixations was too rapid to track, and thus invisible. If this were true, the Sp would be similarly invisible. However, the same subjects reported that Sp was continually apparent near the target (T2) position irrespective of the triggered time (or amplitude) over nearly all the saccadic periods (Fig. 1B). The physical gaze was always directed to a light spot and thus rotated in the same direction with the same velocity as the motion of the light spot. Nevertheless, the apparent positions of the Sp disagreed with the physical positions; rather, it was predictive as if the gaze was regulated to be directed to the target position across the saccadic periods (Fig. 2A).

Fig. 1 Procedure of a single trial. **A(1)** Preparatory period. A small square shows the spatial position of a light spot. Two-colored (red and green) LEDs for S1 and S2 were used to provide positions of the T1 and T2 targets, respectively. Each trial begins by fixating T1 (a red LED). The short vertical bar is not real, but shows how the trigger levels of stimuli varied in the saccadic path. **A(2)** Presenting a trigger stimulus. After variable delay periods, T1 is turned off and the T2 (a red LED) on the right is simultaneously turned on. The subjects saccaded to T2 as rapidly as possible. **A(3)** An image of probing stimuli. Three stimuli are presented simultaneously when the light spot passes 3° from the T1 position. They are a vertical pulse stimulus (Sp) superimposed on the horizontal sweep of an oscilloscope and two stationary stimuli flashed at S1 and S2 for the left and right green LEDs, respectively. **B** An image of perceived positions of the three stimuli. The spatial position of Sp is greatly mislocalized near T2. Both S1 and S2 are mislocalized toward the right, but S2 shows a lesser extent of mislocalization than S1. Therefore, the spatial separation of two stationary stimuli seems to be compressed relative to their physical separation.

On the same timescale, by contrast, the apparent positions of S1, once specified near the target (T2) position at lower trigger amplitudes, shifted in the direction opposite to that of the saccades, and then matched their veridical positions near saccade termination when the trigger amplitude was set near T2. The characteristics of mislocalization of S1 were equivalent to those reported elsewhere^{23,24}, while those of Sp are new. Interestingly, if the pattern of mislocalization of Sp reflects the spatial pattern of the referent position signal Θ_r , its relation to the other two variables replicates the predictions of the CD theory: an error in stationary stimuli S1 (Θ_e) originating from a spatiotemporal misalignment of Θ_r with the physical eye position Θ_p .

$$\Theta_e = \Theta_r - \Theta_p \quad 1)$$

The phenomenon of shifts of visual frame of reference, or so-called receptive field shifts, of visually responsive cells may be a key element in this finding. If the predictive shift of receptive fields in many visual areas (visual frame of reference) preceding physical foveation of the target is part of the process that stabilizes the visual world during a saccade, we may also see a similar shift in the perceived direction of gaze during saccadic periods when it is properly fed back to a gazing observer. Equation 1) may be the first attempt to rationalize an experimentally confirmed variable Θ_r as an eye position signal in a framework of the CD theory.

Recently, Hunt and Cavanagh²⁵ have shown that when observers saccaded to a clock with a fast-moving hand, they saw a time that predated their eye movements: 39 ms earlier than the time when eye movement ended in 8° saccades. Consistent with their finding, the variable Θ_r could serve as more direct evidence to show that gaze during saccades is not tied to what is actually falling on the fovea, but instead is tied to the target position, where the fovea will be placed once a saccade is completed. Besides the saccadic suppression mechanism(s) proposed previously^{3,4,20,24}, this scheme may account for our inability to see our own eyes in the ‘mirror paradigm’, since our gaze during saccades is mostly tied to a target falling on the peripheral retina, where spatial resolution decreases rapidly relative to the fovea.

Relativity in visual spacetime

When an observer moves at higher speeds in space, that observer underestimates both the spatial and temporal separation of events occurring in that space. The compression of spacetime is a major prediction of Einstein’s special theory of relativity²⁶. As an analogy to the physical system, such compression may be postulated in an inner representation of the visual world during saccades²⁷⁻²⁹. Morrone et al.³⁰ demonstrated that space compression accompanies an underestimation of the perceived (subjective) time interval between temporally separated stimuli.

Since these two illusory effects occurred in nearly the same time range, they suggest the possibility of a single unifying mechanism of both space and time compressions. Moreover, they speculated that the spacetime compression originates from anticipatory repositioning of visual receptive fields^{8,25,31,32}, leading to an immediate relativistic consequence in perceptual space and time²⁹. But this attractive hypothesis awaits further empirical support.

To approach the mechanisms of spacetime compression, one could refer to Fig. 2B. As described earlier, a gazing observer sees Sp remaining stationary near the target (T2) across the saccadic periods, while S1, which was once mislocalized near the target, moves away from that target as the trigger amplitude increases. This means that the events (Sp) internal to the moving (gazing) observer are not moving, while those fixed in an external world are moving away relative to the observer. Now, imagine the view of a second observer (e.g., experimenter) who is seeing the same two stimuli (Sp and S1) without eye movement. This (second) observer can see the two stimuli in physical space, such that the Sp is shifted toward the target (T2) as the trigger amplitude increases, while S1 remains stationary near T1, regardless of the trigger amplitude. This contrasting view of the same stimuli by two observers is simple, but profound when we claim that either view of the two observers is an absolute stationary frame of reference. The two observers’ perspectives seem to be an analogy of a moving frame of reference in relativity theory.

Of more importance, in Fig. 2B, is the comparison of the perceived positions of the two stationary stimuli S1 and S2. Both stimuli were once mislocalized in the same direction as saccades, but recovered their veridical positions in such a way that both space and time were compressed relative to their physical separations. To illustrate this, let us define the positions and times when each of the two mislocalization curves reach their physical positions, which are marked as (x_1, t_1') and (x_2, t_2') for S1 and S2, respectively, in Panel B. The pattern of mislocalization of these two stimuli may be explained using the framework of a moving (observational) frame of reference in the special theory of relativity; that is, the events (Sp) internal to the moving (gazing) observer remain stationary near the target T2, while events S1 and S2, both occurring in external space relative to the observer, exhibit a linked compression of space and time as follows.

In the first half of the saccadic periods (Fig. 2B), S2 initiated mislocalization curves with negative slopes being parallel with S1 in the corresponding periods. It is noticed that these two slopes are uniformly tilted, but the space separation between them averages 4.22°, nearly a space compression factor of two relative to their physical separation (9.0°). This is interpreted as a superposition of the shift and compression components of mislocalization^{33,34}, such that the former reflects uniformly tilted paths while the latter reflects the shortening of spatial separation be-

tween these two paths. Also, the latter corresponds to the compression of time as well. Because, the first recovery times when the perceived positions of two stimuli match their physical locations, are shortened as their physical distance from the initial fixation point to stimuli increases. By this effect, the *line of simultaneity* passing through the recovery times of two stimuli (t_1' and t_2') near their physical positions x_1 and x_2 , respectively, is tilted leftward, and the definition of a new space axis x' becomes possible. From the viewpoint (x, t) of the gazing observer, therefore, the recovery time t_2' is estimated as 18 ms after the saccadic onset, nearly a time compression factor of two relative to the movement time of 39 ms for t_1' of S1. This means that the gazing observer perceived the target (T2) near its veridical position 21 ms before physically foveating it.

The receptive fields of visual neurons in several retinotopically organized areas begin to shift just before the

instant of a saccade^{5-7,15,32}. Owing to this predictive nature of remapping that shifts the visual frame of reference, the perceived direction of gaze may be adjusted to the predicted location of the fovea, where post-saccadic visual input (i.e., target) will be placed²⁵. The spatial constancy of apparent positions of pulse stimuli (Sp) near the target, which precedes physical foveation of the target (Fig. 2), may reflect this predictive adjustment of the visual frame of reference. If so, the other problem for the system may be to rearrange accurate spatial representation with reference to this new frame on-line, to complete spatiotopic specificity. In this process, the retinotopic inputs which were once remapped near the target position may be continually shifted in the opposite direction to the saccade with an amount to counteract the retinal shift. This may be seen in the highly negative relationship between the perceived S1 position and eye orientation (Fig. 2A). But the magnitude and timecourse of the mislocalization

Fig. 2 **A)** Relationship among eye orientations, apparent positions of pulse (Sp) and of two stationary stimuli S1 and S2 during saccadic periods. They are represented by closed circles, open circles and open triangles, respectively. A line segment with an arrow head represents the direction of eye movement. Data were obtained from a single subject. Note that the perceived positions of the Sp triggered at different amplitudes are gathered near target T2. The perceived positions of S1 are at first mislocalized near the target, followed by a shift in the opposite direction to the saccade across the saccadic period. The S2, which is mislocalized in a similar direction to S1, recovers its physical location earlier than S1. Note that in the former half of the saccadic period, the tilt of mislocalized curves of two stationary stimuli (S1, S2) seems to be nearly homogeneous. **B)** Schematic illustration of spacetime compression based on the results in panel A). First, we define the *Newtonian* space-time coordinates (x, t), where the tilted time axis t_1' is to specify the corresponding position of S1. If the geometry of space is *Euclidian* and time is universal across space, two events are specified as simultaneous when they occur on the line parallel to the space axis x . However, this does not hold true due to the earlier recovery time of S2 near its physical location than that of S1. This corresponds to the rightward tilt of the *line of simultaneity*, or the tilt in the space axis from vertical (x) to leftward (x'), leading to the definition of other spacetime coordinates (x', t'). The superimposed representation of the two coordinates, (x, t) and (x', t'), and their interrelationship are analogues of the *Minkowski* spacetime diagram in the special relativity theory. From the viewpoint of our gazing observer (x, t), therefore, two relativistic-like effects, space and time compressions, can be seen. Our observer sees earlier recovery of S2 ($=t_2'$) than that of S1 ($=t_1'$), implying that the time is compressed relative to their physical separation. At the same time, the spatial distance of two simultaneous events appears to contract in the direction of motion (space compression). Taken together, what is novel here is to present a single unifying mechanism of space and time compression which could be ascribed to the tilt of the space axis from x to x' . Plots in both panels are based on a different, unpublished data set from those depicted in the published paper²².

curves of S1 are not representative of the other stimulus S2 (Fig. 2B). During the times when each of the two stationary stimuli took a negative slope in mislocalization curves, their spatial separation was compressed relative to their physical separation, leading to the definition of a tilted space axis (x'): the more distal stimulus from an initial fixation target shows an earlier recovery time near its physical location. This compression may help to mediate an earlier perception of the target in a spatiotopically appropriate position in advance of its physical foveation²⁵. Although the neural substrates underlying spacetime compression are still unclear, the key element may be in the visual remapping process that is neither instantaneous nor uniform over the retina, leading to transient deformations in spatio-temporal receptive field organization during remapping³¹.

The manifold concept in modern physics may help our understanding of the compression of perceptual spacetime during saccades. During fixation, the retinal input could be of a continuous spatial manifold within which visible objects are embedded^{35,36}. However, if this spatial manifold is tilted intrasaccadically (Fig. 2B), the space from the viewpoint of gazing (moving) observers is not defined (and thus not perceived) as a continuum of a single spatial manifold, such that the mapping from physical space to perceived space does not appear to be one-to-one, but rather as one-to-many. Correspondingly, the size of the spatial gap (vertical distance) between two adjacent, co-aligned stimuli is seen as less than their physical distance (space compression). Similarly, the size of the temporal gap (horizontal distance) between two adjacent, co-aligned stimuli is seen as less than their physical separation (time compression). It is as if both space and time are linked to be compressed relative to their physical separations. These perspectives share aspects with *Minkowski* spacetime to describe physical systems in special relativity theory²⁶, that is, space and time are not separable but are combined into a single continuum and thus both metrics contract dependent on the state of the gazing observer.

Posture-movement paradox

In a seminal paper by von Holst and Mittelstaedt², they emphasized that voluntary movements to move the body segments away from initially stabilized postures do not evoke resistance arising due to sensory feedback information, whereas if the same initial posture is disturbed by an external force and then forced into the same movement trajectory, posture stabilizing mechanisms actively operate to oppose such disturbance. To explain this so-called 'posture-movement paradox', they assumed that when initiating intentional motions, the nervous system simultaneously sends a copy of motor commands to muscles to suppress motion-evoked afferent feedback, leading to the cancellation of the afferent influences that would

cause resistance to motion. However, this proposal has been confronted with the long-held criticism that afferent feedback is effectively utilized and functional throughout intentional movements³⁷⁻⁴⁰, which would in some cases cause resistance strong enough to drive the segments back to their initial positions⁴¹. To complete an intended final position, therefore, the system must prepare some effective mechanisms to reset the posture stabilizing mechanisms that are suitable for the upcoming, intended final posture. This scenario is a core concept of the equilibrium point hypothesis proposed by Anatol Feldman.

Feldman and Orlovsky⁴² found that descending signals adjust a specific neurophysiological variable, the *threshold* limb position at which relevant muscles are ready to be recruited when stretched. By shifting or simply resetting this threshold position, the brain may create the spatial frame of reference in which muscles are constrained to work without pre-determining how they should work, or to work without resolving inverse problems^{43,44}. During posture, the posture-stabilizing mechanisms operate only if the limb has deviated from a current threshold limb position. Similarly, the central task in the generation of intentional movements may be to reset the posture-stabilizing mechanisms to a new posture. By shifting the threshold position, the nervous system converts movement-resisting into movement-producing forces, which solves the posture-movement paradox^{41,43-45}. According to this scheme, the dynamics is regarded as a recovery process of a segment's posture toward its new equilibrium state.

In a similar vein, Feldman⁴⁴ verified that threshold position resetting would be a necessary element of visual stability during saccadic eye movements. According to him, a saccade is assumed to result from a monotonic shift in the referent position Θ_r' of the eye to bring the projection of the target to the fovea. The resulting activity of eye muscles (Θ_e) is not explicitly programmed but emerges depending on the difference between the physical eye position Θ_p and its referent position Θ_r' .

$$\Theta_e = \Theta_r' - \Theta_p \quad (2)$$

The eyes rotate until Θ_p matches Θ_r' , or until the saccadic target becomes projected to the fovea. This form makes sense when the visual system uses a common frame of reference to designate action and perception, implying that shifts in the referent eye position underlie both oculomotor action and visual perception. This form is essentially similar to equation 1), but note that the term Θ_r in equation 1) is constant, while the term Θ_r' in equation 2) is variable, and thus resettable from glance to glance over spatiotopic coordinates. Resetting of the posture-stabilizing mechanisms and the resulting eye movements, as well as visual space constancy, can thus rely on central shifts in the threshold (referent) eye position (Θ_r') rather than on preprogramming of motor action through the computation of inverse dynamics.

Implications from imaging studies

CD and Sensorimotor Interaction. Movement is the only way of interacting with the world. Indeed, all communication, including speech, gestures and writing, is mediated via the motor system. To control our action, the human brain uses sensory signals to determine future actions. In this chapter, we would like to introduce the research topics of sensorimotor integration using brain imaging in humans. Human studies have provided some insights into the functional role of CD in two operations: resolving ambiguity in the origin of sensory information and enabling proper motor performance. The CD mechanism plays an important role in the vocalization and auditory system, and also plays a part in coordinating the skeletomotor and somatosensory systems⁴⁶. CD is produced only if the motor commands interact with unpredicted sensory inputs and inhibits the neural response to self-generated sensory signals. Recently, an interesting study was reported on the somatosensory system. Everyone experiences the phenomenon that we cannot tickle ourselves. However, if someone externally touches the side of our abdomen, we suddenly feel tickled. The neural mechanism of this phenomenon was reported using brain image techniques. Comparing the self-produced and externally produced stimuli, more activity in somatosensory areas was found when an unpredicted stimulus was externally delivered^{47,48}. When a movement is self-produced, its sensory consequences can be accurately predicted, and this prediction can be used to attenuate the sensory effects of the movement. The sensory prediction is made by an internal forward model of the motor system⁴⁹. By comparing the predicted with actual sensory feedback, it is possible to distinguish the sensory consequences of our movements from sensory signals due to changes in the outside world. Monitoring self-generated actions allows us to recognize them as our own.

Sensorimotor Interaction during the Preparation of Voluntary Movement. For precise movement, a motor program requires sensory signals from the target body area to be moved, and the appropriate integration of sensory and motor signals is essential. When we execute voluntary movement, somatosensory information processing, during its course from the peripheral to the somatosensory areas, is influenced by top-down modulation (centrifugal mechanism) mediated by a network comprising brain structures, motor and higher-order brain systems. This phenomenon has been reported as modulation in the short- and long-latency components of somatosensory evoked potentials (SEPs) and magnetic fields (SEFs). With regard to modulation in the somatosensory cortices produced by voluntary movement, changes of the SEP/SEF amplitude were found not only during movement⁵⁰⁻⁵³, but also just prior to it (preparatory period)⁵⁴⁻⁵⁶. It is assumed that these elec-

trophysiological changes are associated with a decrease in tactile sensitivity commonly observed before the onset of movement of the limb that received the sensory stimulation⁵⁷.

We tried to elucidate the neural mechanism of sensorimotor interaction in somatosensory areas, especially centrifugal modulation in the primary somatosensory area (SI) and the secondary somatosensory area (SII), during the preparatory period of self-initiated voluntary movement. We hypothesized that if motor commands interact with sensory inputs in the central nervous system, neurons in the motor and sensory areas should show changes of activity when these commands are issued. The results showed that, in the period of 500 ms before the onset of movement, an attenuation of activation in the SI and enhancement in the SII were found^{58,59}. In this time period, the activity in the primary motor area and supplementary motor area is enhanced for the preparation of upcoming muscle contraction. The opposite effects of movement on SI and SII cortices indicated that motor and higher-order brain systems regulate sensory information at several processing stages by the centrifugal process. Motor commands can facilitate or suppress sensory responsiveness and, thus, probably perception, depending on temporal and behavioral constraints.

Our sensory systems are constantly bombarded by a large amount of sensory stimuli, from which we must extract the few stimuli that are important to control our movement. From this viewpoint, we are able to recognize that attenuation of SI activation is involved in filtering the information. Although much attention has been given to sensorimotor interaction in SI, there is little evidence of such a phenomenon in the SII and the role of SII in motor execution has not been fully elucidated in humans. Compared with the SI, the SII is speculated to serve a higher level of cognitive function in somatosensory information processing, such as attention, decision-making, object recognition, and the integration of nociceptive and non-nociceptive inputs⁶⁰⁻⁶⁴. Our results show that these cortical areas play different roles in the function of sensorimotor integration. When we are moving, the sensory threshold is attenuated. By contrast, exploration using the fingertips is sensible during movement execution (active touch). This neural mechanism can be explained by an enhancement of SII activation. To clarify the function of the activation of SII concerning sensorimotor integration, we conducted further research.

Sensorimotor Interaction during the Execution of Voluntary Movement. Recognizing one's own movement is essential to the control of voluntary movement. Movement causes changes to sensory inflow, as well as changes in the positions of body parts. The movement of one's body parts is perceived not only by visual information but also by somatosensory feedback from muscles, skins and tendons which provide us with information on the status of

each part being moved in a moment. Under normal conditions, the visual estimation of limb position is congruent with the somatosensory estimate and motor command, and movement is usually achieved automatically without awareness of the component processes. By contrast, in novel motor tasks or situations that produce conflict or incongruence between intentions and sensorimotor consequences, a mismatch between the actual sensory feedback and predicted movement of a given body part disrupts motor execution. The crucial question is that of the brain mechanism that integrates the sensory information and motor commands for motor control.

We investigated whether activation in somatosensory areas was affected by a discordance between an intended and an executed action⁶⁵. We provided unintended visual feedback of body movement using a mirror box, and measured the brain activities during movement execution using magnetoencephalography (Fig. 3A). In the Non-veridical condition, subjects inserted their hands into a mirror box with the forearm supine. The position of the right hand was adjusted so that the mirror image precisely overlapped the view of the masked left hand. A mirror image of the right hand was therefore presented instead of the left hand. Since the actual visual information of the left hand was masked by the mirror, a mirror image of the right hand was provided. The motor task was self-paced thumb movement of the left hand. Electrical stimulation for the recording of somatosensory responses was delivered to the median nerve at the left wrist. Subjects watched the stationary image of this left hand while performing self-paced movement of the left thumb. This condition typically produces the feeling that the movement is not controlled by themselves or the moving body part does not belong to them. In the Normal condition, the mirror box was removed.

The results showed activity in the SI to be inhibited and activity in the SII to be enhanced with voluntary movement, and neural responses in the SII and parietal cortex were strongly affected by the unexpected visual feedback (Fig. 3B). In the Normal and Non-veridical conditions, subjects experienced appropriate somatosensory feedback, but in the Non-veridical condition, what they watched was incongruent with the expected visual feedback and produced a state of cognitive conflict. These results provide evidence that the SII and parietal cortex had cross-modal functions in somatosensory and visual modality during motor execution, and that the visual information plays a crucial role in activation of somatosensory areas during motor execution. We assume that this modulation during conflicting visual feedback might be involved in computing motor errors by comparing the actual hand location to the estimated location for controlling movement. Further research may be useful to elucidate the functional role of somatosensory areas for motor control using somatosensory and visual information.

Fig. 3 **A)** The schema of the experimental paradigm. Subjects performed self-paced movement of the left thumb with normal visual feedback (Normal condition) and incongruent non-veridical visual feedback (Non-veridical condition). Since the actual visual information for the left hand was masked by the mirror, a mirror image of the right hand was provided. In the Non-veridical condition, subjects watched a stationary image of the hand while they performed self-paced movement of the left thumb. Electrical stimulation for the recording of somatosensory responses was delivered to the median nerve at the left wrist. **B)** The location of equivalent current dipoles (ECDs) superimposed on 3D images. The early responses were estimated around the posterior bank of the central sulcus, corresponding to the SI. The temporal components were located in the upper wall of the sylvian fissure in both hemispheres, corresponding to the SII. The source of the parietal area was estimated to be in the posterior wall of the postcentral sulcus. The amplitude of components in the SII was significantly larger in the Non-veridical than in the Normal condition. By contrast, the PC was significantly smaller in the Normal condition than in the Non-veridical condition. ** $P < 0.01$, * $P < 0.05$: between two conditions.

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