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Research report

The effects of acute cortical somatosensory deafferentation on grip force control

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ABSTRACT

Grip force control involves mechanisms to adjust to unpredictable and predictable changes in loads during manual manipulation. Somatosensory feedback is critical not just to reactive, feedback control but also to updating the internal representations needed for proactive, feedforward control. The role of primary somatosensory cortex (S1) in these control strategies is not well established. Here we investigated grip force control in a rare case of acute central deafferentation following resection of S1. The subject had complete loss of somatosensation in the right arm without any deficit in muscle strength or reflexes. In the first task, the subject was asked to maintain a constant grip force with and without visual feedback. The subject was able to attain the target force with visual feedback but not maintain that force for more than a few seconds after visual feedback was removed. In the second task, the subject was asked to grip and move an instrumented object. The induced acceleration-dependent loads were countered by adjustments in grip force. Both amplitude and timing of the grip force modulation were not affected by deafferentation. The dissociation of these effects demonstrates the differential contribution of S1 to the mechanisms of grip force control.

1. Introduction

The manual manipulation of objects in our environment is a ubiquitous and largely effortless feature of daily life. However a considerable amount of computation occurs below the level of consciousness to execute these actions. Tasks as seemingly mundane as picking up and moving a glass of water engage complex neural systems to prevent the glass from slipping out of our grasp. When holding an object, we generate a grip force that takes into account the anticipated object weight and surface friction (Westling & Johansson, 1984). When moving

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an object, we anticipate movement-induced variations in the load force and adjust grip force accordingly (Flanagan & Wing, 1993). Anticipation of object properties and loads is a key feature of volitional movement control (Shadmehr, Smith, & Krakauer, 2010).

Anticipatory grip force control is dependent on cutaneous, proprioceptive and visual feedback regarding changes in an object's constitutive properties and motion (Witney, Wing, Thonnard, & Smith, 2004). This process can be described computationally as adaptive feedforward control in which internal models of object properties are updated by incoming sensory information (Wolpert & Ghahramani, 2000). The need for sensory feedback is clear when loading conditions change unpredictably. For example, the ability to appropriately adjust grip force to sudden pulling loads is severely diminished during digital anesthesia (Johansson, Hger, & Backstrom, 1992). The contribution of sensory feedback to compensation of predictable loads is more nuanced. When lifting and moving an object with anesthetized fingers, subjects will elevate grip force to levels inappropriate for the load, but the prediction of when to elevate the grip force relative to the change in load force is maintained (Augurelle, Smith, Lejeune, & Thonnard, 2003; Nowak et al., 2001). Unlike acute digital anesthesia, chronic peripheral deafferentation due to large-fiber sensory neuropathy disrupts the correct timing of grip forces to load forces (Nowak, Glasauer, & Hermsdorfer, 2004). Even simply maintaining a static grip force for several seconds without visual feedback is compromised in these subjects (Rothwell et al., 1982). These results suggest that feedforward grip force control is severely degraded without periodic sensory feedback.

Many different brain areas have been implicated in the adaptive feedforward control of grip force, including those in the cerebellum and cerebral cortex. Lesion studies provide insight into how each area contributes. Cerebellar lesions profoundly impact anticipatory control (Babin-Ratte, Sirigu, Gilles, & Wing, 1999; Muller & Dichgans, 1994; Rost, Nowak, Timmann, & Hermsdorfer, 2005; Serrien & Wiesendanger, 1999). Based on this and other evidence, the cerebellum has been posited to be the site of internal models (Wolpert, Miall, & Kawato, 1998). Sensorimotor cortical circuits may play a role in updating cerebellar internal models by relaying and processing sensory information. In one study, cerebral stroke involving the sensorimotor cortex was found to impair grip force control in a manner similar to that of subjects with acute digital anesthesia (Hermsdorfer, Hagl, Nowak, & Marquardt, 2003). However, middle cerebral artery stroke causes lesions of both sensory and motor circuits, making it difficult to ascribe the deficits specifically to a lack of sensory feedback. Lesions specific to primary somatosensory cortex (S1) in non-human primates result in impaired grip force control (Brochier, Boudreau, Pare, & Smith, 1999). However, to date there has been no comparable lesion study in humans.

Here we investigated control of grip force in a rare case of an acute, centrally-deafferented subject with a S1 lesion. We tested static and dynamic grip force control using tasks modeled after those found to reveal key deficits in chronic peripheral deafferentation (Nowak et al., 2004; Rothwell et al., 1982). We hypothesized that acute deprivation of sensory feedback would result in an inability to maintain a constant grip force and to adjust grip force for movement-dependent changes in loading. To the best of our knowledge, this is the first report on the effect of acute central deafferentation on grip force modulation.

2. Methods

2.1. *Subject*

A 39-year old, left-handed patient (C.O.) had a history of simple partial seizures since childhood. At age 4, he was involved in a motor vehicle accident in which he suffered a skull fracture and subsequent scarring on the left side of his brain. He was involved in another motor vehicle accident at age 17 after which he was comatose for 2 weeks. His seizures, which largely affected the right half of his body, began at age 8 and persisted into his 30s despite multiple medications. Seizure semiology included sensory changes in the right upper extremity, followed by progression to tonic-clonic activity in the same region before progressing to facial muscles. He was seizure-free for a period of 5 years but seizures then recurred. Video-electroencephalography localized seizures to the left temporoparietal region, where MRI had revealed a cystic lesion with stable encephalomalacia. He was deemed a suitable candidate for intracranial electrocorticography to further localize the onset zone and map its relationship to sensorimotor cortices. He underwent preoperative high angular resolution diffusion images (HARDI) for tractography. Intracranial recordings documented seizure onset from the anterior margin of the cyst, bounded by the postcentral gyrus. Extra-operative electrical stimulation mapping through the implanted electrodes verified that the involved gyrus was S1 for hand, arm and face. The patient underwent extensive counseling regarding this finding and the potential risks of surgical resection of the seizure onset zone. At the patient's insistence, he was scheduled for surgical resection of the seizure onset zone.

An awake craniotomy was performed with intraoperative motor and sensory mapping, which verified the findings of extra-operative mapping. S1 was removed up to the level of the central sulcus (Fig. 1). Subcortical white matter mapping was undertaken along the cyst wall at 3 different depths as white matter was removed, evoking consistent and reliable motor responses with stimulation. Clinical testing during this dissection noted that the subject was full-strength throughout the procedure, producing robust hand movements and squeezes on command. He was in excellent condition at the conclusion of the procedure and was full strength prior to his departure from the operating room.

C.O. was subsequently seizure free. As expected, he was also left with a complete somatosensory deficit on the right arm, trunk, and face. At a comprehensive neurological exam 23 days after the surgery, he was unable to feel any light touch, pain, temperature changes, or vibration in his right hand and arm. Furthermore, his senses of proprioception, tactile movement, two-point discrimination, graphesthesia, and stereognosis were absent in his right hand and arm. He had a positive Romberg sign, indicating sensory ataxia. However, he

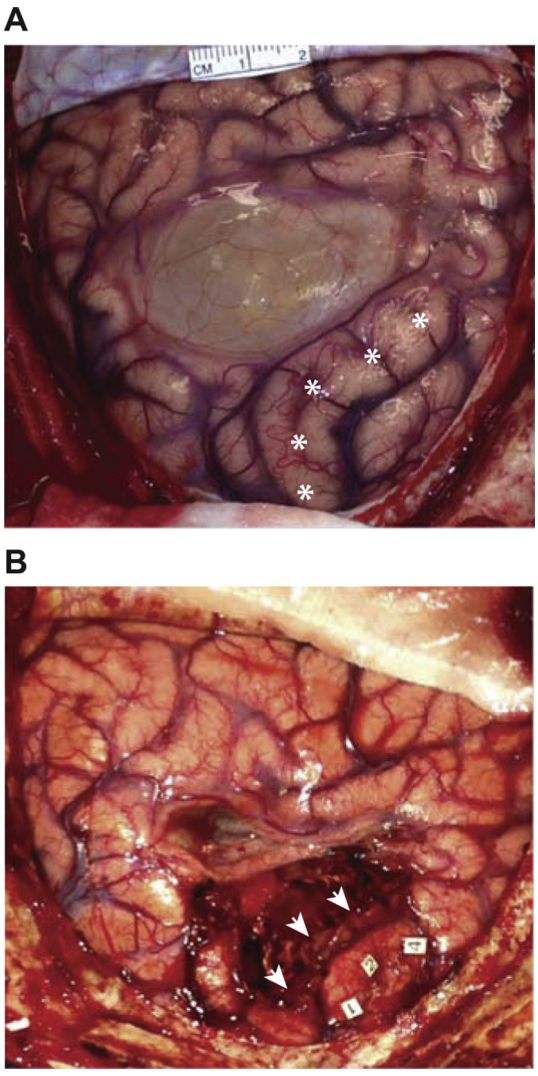

Fig. 1 – Operative findings and surgical resection of somatosensory cortex. A, Pre-resection findings. Cystic lesion abutting the posterior extent of somatosensory cortex (*). B, Following resection of cystic lesion and seizure onset zone within somatosensory cortex. The intact hand motor cortex is illustrated (sterile number tickets) in relation to the fundus of the skeletonized central sulcus (arrow heads). Subcortical white matter mapping during awake resection verified integrity of descending corticospinal fibers anterior to the resection cavity.

was full strength in all extremities, with normal tone, bulk and power in all muscle groups. Notably, the maximum power grip force with his right (dominant) and left hand was 403 N and 375 N, respectively, which are within normal range. Reflexes were normal. Visually-guided reaching with the right arm appeared qualitatively normal and performed at a similar pace as with the left arm. There was no sensory deficit in the left hand and arm. Identical findings were noted at his follow-up exams 70 and 126 days after the surgery. Informed consent was obtained from C.O. for the following two behavioral experiments.

2.2. *Static grip force experiment*

We first studied how maintenance of a steady grip force was affected by resection of S1. C.O. was asked to squeeze a compression load cell using a power grip to achieve a target, steady force. Output of the load cell (LMD500, Futek Advanced Sensor Technology Inc) was passed through an instrumentation amplifier and digitized on a RZ2 BioAmp Processor (Tucker–Davis Technologies). Digital signal processors of the RZ2 were configured to save the force data to file at 305 samples/s and control the presentation of auditory and visual cues that defined the behavioral task.

The task was composed of 10–15 trials with each hand per session. Each trial lasted 7 sec and began with the onset of an audible tone (500 Hz) and appearance of visual feedback regarding the current grip force. Real-time visual feedback of grip force was achieved by mapping force to the angular position of an arrow on a dial displayed on a computer monitor using OpenController software (Tucker–Davis Technologies). The target force range (22.5–27.5 N) was represented visually by a red region on the dial. The subject was instructed to squeeze the load cell to move the arrow to the red region. The arrow was only visible for the first 2 sec of the trial regardless of whether the target was reached. The subject was instructed to maintain the target grip force without visual feedback until the audible tone ended (5 sec after the arrow disappeared), marking the end of the trial. The subject was instructed to relax his grip between trials.

Data analysis was performed in Matlab (MathWorks). Trials in which the subject was inattentive, as indicated by latent grip force onset, or released his grip too early were excluded. Two performance measures were computed for each trial. The *attained force* was defined as the average force from 1.5 to 2.5 sec after trial onset, at the transition from presence to absence of visual feedback. The *maintained force* was defined as the average force in the last 1 sec of the trial. The central tendency of each measure pooled across trials was reported as mean ± 95% student-t confidence intervals.

2.3. *Dynamic grip force experiment*

The second behavioral task examined dynamic regulation of grip force in the deafferented subject. Specifically, we quantified grip force – load force coupling in the healthy and deafferented hands of C.O. using a wireless, instrumented object (Nowak et al., 2001). The object was a 8.5 × 5.5 × 4.0 cm plastic box weighing 250 g that housed a 3-axis accelerometer (MMA7361LC, Freescale Semiconductor), a force-sensing resistor (FSR 402, Interlink Electronics), custom electronics on a printed circuit board (PCB), and a battery (UBP002, Ultralife Corp). The PCB contained analog-to-digital converters and a radiofrequency transceiver to acquire and transmit the 4 channels of sensor data wirelessly to a computer, which stored the data at 250 samples/s/channel. The electronics were based on the sensor node of the PennBMBI system developed recently (Liu et al., 2015).

C.O. was instructed to hold the object with a precision grip and avoid contact with the palm. The tip of the thumb was positioned on the FSR, which was secured to a vertical face of the outside of the plastic box. The other fingertips were

positioned on the opposing vertical face of the box, yielding a grip aperture of 4 cm. With this hand posture, the FSR could measure the precision grip force. However, FSRs are pressure sensors, not force sensors. To make the FSR insensitive to the contact area, an epoxy dome approximately 3 mm high in the center and spanning the entire 12.7-mm sensing area was applied to the surface of the sensor (Jensen, Radwin, & Webster, 1991). Furthermore, the FSR was conditioned and calibrated according to published guidelines to ensure it was a reliable sensor of dynamic, compressive forces (Hall, Desmoulin, & Milner, 2008).

Upon correctly gripping the object, C.O. was instructed to make swift upward and downward arm movements, primarily through flexion and extension of the shoulder, to move the object over a vertical distance of about 40 cm. He was instructed to keep the object orientation as consistent as possible over the course of these movements as if "painting a wall", which he did using minor wrist and elbow angle adjustments. The goal was to minimize angular accelerations of the object such that the dominant load forces were due to the gravitational and translational accelerations measured with the onboard accelerometer. Load force was then calculated as the product of the object mass and the L^2 norm of the measured acceleration vector.

Grip force and load force data were collected for both upward and downward movements. However, C.O. found it very difficult to maintain the correct hand posture during downward movements with his deafferented arm. Despite adjusting his hand before each movement, the palm of the hand or proximal fingers routinely came in contact with non-force sensing parts of the object over the course of the movement, which resulted in incorrect grip force measurements. This was not because the load forces were greater during downward movements and causing the thumb to slip off the FSR. The peak load forces during upward and downward movement of the deafferented arm were not significantly different ($t_{39} = .30$, $p = .765$). Furthermore, the grip force measurement errors occurred early in the downward movements, on average 213 msec before the peak force was encountered during deceleration of the load towards the end of the movement. The difficult in maintaining the hand posture at the beginning of the movement may have been related to the fact that the downward movement started in a relatively uncomfortable position with the shoulder flexed beyond 90°, possibly affecting his coordination or attention to his hand posture. Therefore, the data from downward movements of both the healthy and deafferented hand were omitted from the analysis.

Although visual feedback is not particularly relevant to grip force-load force coupling, the subject was instructed not to look at his hand but rather look straight ahead at the experimenter who paced the movements. In the experimental session, trials with the healthy arm were made first, followed by trials with the deafferented arm.

To quantify the coupling between grip force and load force modulations, two measures were computed for each trial. The *peak amplitude ratio* was defined as the ratio between the grip amplitude and load amplitude, where both amplitudes were calculated by subtracting the static force value (i.e., gravitational force prior to movement) from the peak value. The *relative peak timing* was defined as the difference between the time of maximum grip force and the time of maximum load force. As with the prior analysis, the central tendency of each measure pooled across trials was reported as mean ± 95% student-t confidence intervals. In addition, a cross-correlation analysis between the load and grip force trajectories was performed over a −.5-sec to .75-sec window relative to the peak load force of each trial.

3. Results

3.1. Static grip force control

We quantified the ability of C.O. to maintain a steady grip force in three post-resection sessions (Fig. 2). The results were qualitatively similar across sessions and were pooled for statistical analysis. With 2 sec of visual feedback on his current grip force, the subject was able to attain a target grip force (22.5–27.5 N) with both his healthy left hand and deafferented right hand. The attained force on each trial was defined as the average force in the [1.5, 2.5]-s interval after trial onset. 95% student-t confidence intervals for the mean attained force with the healthy and deafferented hand was 25.4 ± .4 N and 23.7 ± .3 N, respectively. Although the mean attained forces were both within the target force range, the mean attained force with the deafferented hand was significantly lower than that of the healthy hand ($t_{49} = 5.76$, $p < .001$). Also, the grip force trajectory from rest to target was mostly monotonically increasing with the healthy hand but oscillatory with the deafferented hand (Fig. 2).

After 2 sec, the visual feedback was extinguished and the subject was instructed to maintain the grip force for another 5 sec until an auditory tone signaled the end of the trial. In the absence of feedback, the grip force was qualitatively steady for the healthy hand and unsteady for the deafferented hand (Fig. 2). Maintained force was defined as the average force in the last second of the trial. The mean maintained force with the left and right hand was 27.3 ± .6 N and 19.9 ± 2.0 N, respectively. The maintained force with the deafferented hand had both lower mean ($t_{34} = 7.29$, $p < .001$) and higher variance ($F_{24,28} = .095$, $p < .001$) than the maintained force with the healthy hand. Thus the lack of sensation degraded the subject's ability to maintain a steady grip force with his deafferented hand.

3.2. Dynamic grip force control

In the final post-resection session, 126 days after surgery, C.O. performed a task to assess his dynamic grip force control (Fig. 3). With a precision grip, he held an object that measured his grip force and acceleration-dependent load forces. When the object was held steady, a static load due to gravitational acceleration was present (2.5 N). To prevent the object from slipping he had to generate a sufficient frictional force, which was proportional to his grip force against the vertical walls of the object. In this steady state, the mean grip force with his healthy and deafferented hand was 5.4 ± 1.6 N and 6.6 ± 1.2 N, respectively. The mean static grip force with the deafferented hand was higher than that with his healthy hand, suggesting

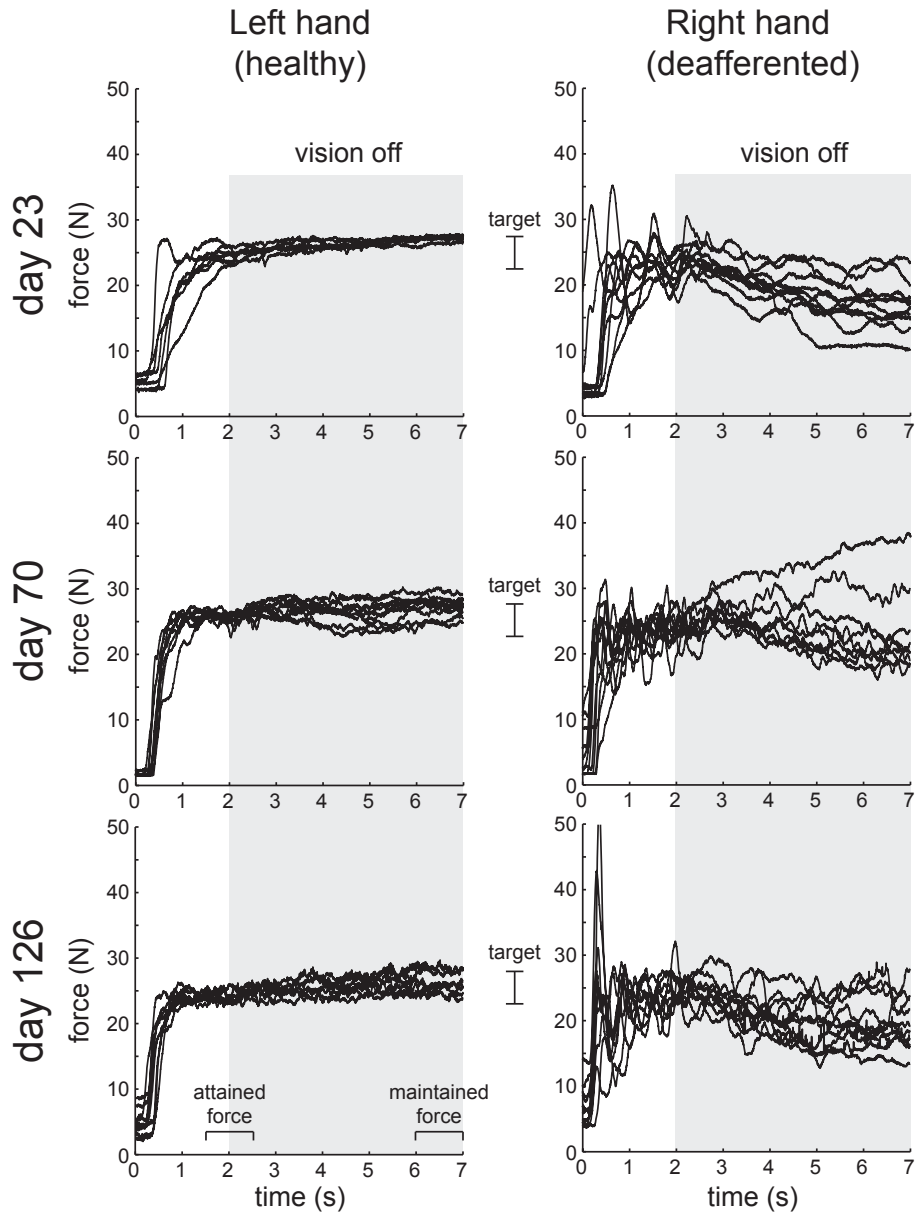

Fig. 2 – Results of the static grip force experiment conducted in each of three post-resection sessions. Guided by visual feedback during the first 2 sec of the trial, the subject reliably attained the target force of 25 ± 2.5 N in both hands. In the subsequent 5-sec hold period without visual feedback (gray region), the grip force was held steady in the healthy hand but drifted from the target in the deafferented hand. Time intervals defining the attained and maintained force performance metrics are indicated.

the use of a higher safety margin against slip without touch sensation. However, the difference in means was not significant ($t_{46} = -1.72$, $p = .099$).

To test dynamic grip force control, C.O. was instructed to move the object upward over a ~40 cm distance using primarily shoulder flexion. Acceleration of the object upward produced a transient inertial load downward, which added with the downward gravitational force resulted in a higher total load force (Fig. 3A). Deceleration of the object to stop the upward movement caused a transient inertial load upward, which subtracted from the gravitation force resulted in a lower total load force. Finally, the load force returned to the static, gravitational load when the hand stopped at the end of the movement. The mean peak load forces produced by movement of the healthy and deafferented arms were comparable, 6.0 ± .5 N and 5.1 ± .3 N, although significantly different ($t_{46} = 2.16$, $p = .036$).

During the upward movement of the object, the grip force used by C.O. was transiently modulated (Fig. 3B). Qualitatively, the grip force increase mirrored the amplitude and timing of

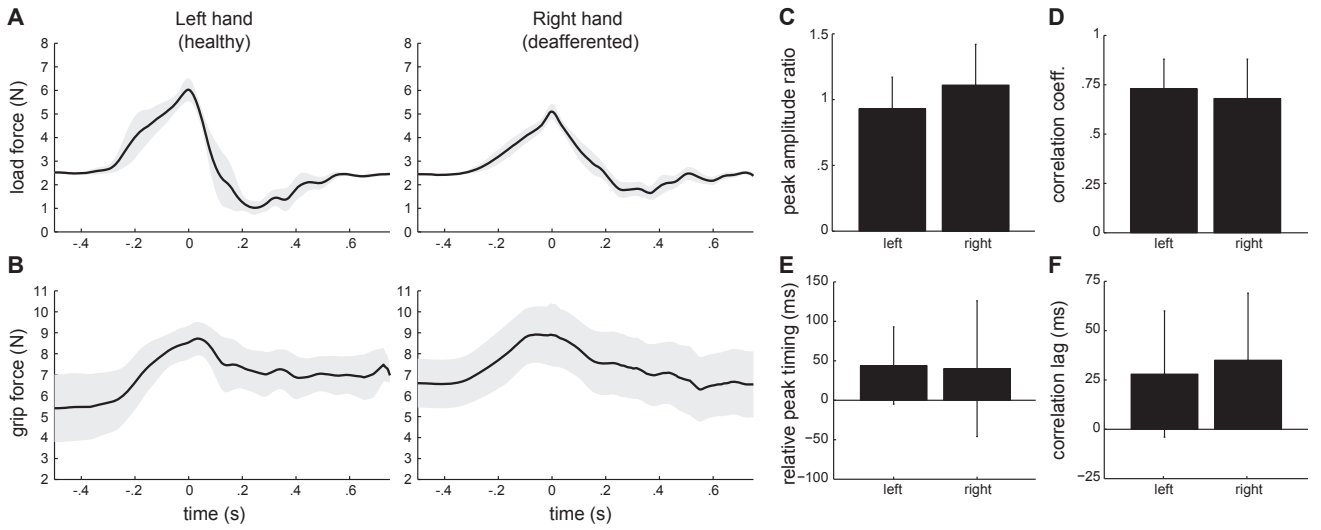

Fig. 3 – Results of the dynamic grip force experiment conducted 126 days after resection. A, Steady-state (gravitational) and transient (inertial) load forces were experienced as the subject rapidly moved an instrumented object upwards over ~40 cm. Load and grip force trajectories for each trial were aligned on the peak load force. The mean force across trials (black lines) and 95% confidence intervals on the mean (gray regions) are shown. B, A transient increase in grip force that was in phase with the peak load force was observed in both the healthy and deafferented hand. C–F, Four performance metrics comparing grip force-load force coupling. There were no significant performance differences between the left and right hand.

the load force increase in both healthy and deafferented arm movements. The grip force increased presumably to prevent slip due to the increased load force at peak acceleration, although he was not consciously aware of this grip force modulation. To quantify the coupling between the transient grip and load forces, we computed two measures, the peak amplitude ratio and relative peak timing (see Methods), and performed a cross-correlation analysis. The mean peak amplitude ratio for healthy and deafferented arm movements was .93 ± .24 and 1.11 ± .31, respectively (Fig. 3C). These ratios were not significantly different from each other ($t_{46} = .42$, $p = .679$). The mean coefficient of correlation between the grip and load force trajectories for healthy and deafferented arm movements was .73 ± .15 and .68 ± .20, respectively (Fig. 3D). The correlations were not significantly different from each other ($t_{46} = .79$, $p = .437$). Thus the amplitude of the grip force increase was statistically the same as the amplitude of the load force increase and the coupling was not affected by deafferentation.

The mean peak timing was 44 ± 49 msec and 40 ± 86 msec for healthy and deafferented arm movements, respectively, where a positive value indicates the peak grip force lagged the peak load force (Fig. 3E). These lags were not significantly different from zero ($t_{24} = .11$, $p = .916$; $t_{22} = 1.06$, $p = .301$) or significantly different from each other ($t_{46} = -.94$, $p = .354$). In the cross-correlation analysis, the lag at maximum correlation was 28 ± 32 msec for healthy arm movements and 35 ± 34 msec for deafferented arm movements (Fig. 3F). These lags were not significantly different from each other ($t_{46} = -.55$, $p = .585$), although the lags for the deafferented arm movements were significantly different from zero ($t_{22} = 2.44$, $p = .023$). Overall, the data suggests that, as with amplitude, the timing of the grip force modulation was not affected by deafferentation.

4. Discussion

Here we examined the motor effects of acute central deafferentation in a patient who underwent removal of a lesion in S1. This case presents a unique contribution to the literature characterizing the somatosensory effects on grip force modulation. Prior work has analyzed effects of central lesions including acute stroke (Nowak, Hermsdorfer, & Topka, 2003), chronic stroke (Hermsdorfer et al., 2003), and demyelinating conditions (Thonnard, Detrembleur, & Van den Bergh, 1997). Unlike vascular or demyelinating lesions that disrupt broad networks (Nowak, Hermsdorfer, & Topka, 2003), the present case was associated with pure sensory loss without accompanying paresis.

We tested the hypotheses that central deafferentation disrupts (a) the maintenance of isometric power grip forces in the absence of visual feedback and (b) anticipatory modulation of precision grip forces during dynamic loading. The data confirmed the former hypothesis but rejected the latter. These results inform our understanding of the neural substrate for feedback and feedforward mechanisms of grip force modulation.

Damage to primary afferents results in dense sensory deficits. Deficits in the ability to maintain grip force are profound (Rothwell et al., 1982) and lasting (Nowak, Hermsdorfer, Marquardt, et al., 2003). In the static task, it was unclear *a priori* whether selective S1 deafferentation would result in similar grip force disruption. S1 is at least two synapses downstream

of the termination of primary afferents in dorsal column nuclei. Secondary afferents arising from the brainstem activate diverse networks in cerebellum, thalamus and association cortices, in addition to S1. The profound inability to maintain constant grip force following S1 lesion suggests that S1 networks are required to maintain and correct internal models of motor programs.

Under normal circumstances, unexpected sensory feedback from errant force execution updates relevant forward models (Nowak, Glasauer, & Hermsdorfer, 2013). In the absence of sensory feedback, forward models maintain a default program based on memory of the task. In agreement with Rothwell et al. the subject did not appear to have insight from efference copy that the motor program had become errant. The unstable forces suggest that the memory trace of the default program is used to establish an initial set point, but is not sufficient to drive a constant force for more than one or two seconds. Stable internal representations appear to require at least intermittent feedback to establish and update predictive models of grasping force control (Nowak & Hermsdorfer, 2006).

Attempts to rapidly achieve the desired target force with visual feedback revealed highly variable forces. Occasionally he overshot target forces by as many as 25 N. This finding persisted for over 120 days despite the fact that the task was over-learned by the subject. The persistent variability in attaining target force at the onset of the static task is an interesting finding. Initiation is thought to be an entirely feedforward mechanism. On an over-learned task, one might hypothesize that forward models would provide consistent estimates of the desired grip force. Instead, our subject was dependent on visual feedback to correct grip force errors to achieve the target level. Such variability is common when the prediction of the required force is inaccurate (Nowak & Hermsdorfer, 2003b; Quaney, Rotella, Peterson, & Cole, 2003; Thonnard et al., 1997). When the required force (or joint position) is well-known, subjects can achieve accurate movements of deafferented fingers (Rothwell et al., 1982). It is possible, therefore, that an accurate memory trace of the target force was never retained by the affected hemisphere due to S1 disruption.

In the dynamic task, we examined the hypothesis that acute central deafferentation would disrupt feedforward models of precision grip force control. The task induced predictable loads that could be compensated by appropriate grip force scaling. Unlike patients with chronic disruption of primary afferents, acute central deafferentation was associated with preserved scaling of grip force level. Both intact and affected limbs demonstrated economical scaling. Peak grip force slightly preceded peak load force, ensuring that the object was not dropped at the point of maximal load force at the inflection point of the down cycle. Temporal coupling between grip forces and time-varying loads also remained intact. This preserved force-load coupling suggests that the internal models of dynamic grip force control do not depend upon intact S1.

Though there is considerable evidence supporting the existence of feedforward internal models responsible for anticipatory grip force adjustments, there is debate as to their anatomical substrate. The localization of lesions which do not impair feedforward control mechanisms include primary afferents (Augurelle et al., 2003; Nowak & Hermsdorfer, 2003a; Nowak et al., 2001), basal ganglia (Hermsdorfer et al., 2003; Nowak & Hermsdorfer, 2006), internal capsule (Hermsdorfer et al., 2003), combined motor and sensory cortices (Hermsdorfer et al., 2003), and now, primary sensory cortex in isolation.

Of remaining networks, the cerebellum appears to be the most compelling candidate to support forward models of grip force control (Kawato, 1999; Wolpert et al., 1998). Individuals with cerebellar lesions typically lack anticipatory grip control (Babin-Ratte et al., 1999; Muller & Dichgans, 1994; Rost et al., 2005; Serrien & Wiesendanger, 1999). Similar deficits in precision grip were noted when the lesion specifically involved the dentate nucleus (Fellows, Ernst, Schwarz, Topper, & Noth, 2001). In normal subjects, increased cerebellar activation is seen when unexpected weight changes are introduced in a grip and lift task, although other networks including the inferior parietal lobule and inferior frontal cortex may also be involved (Jenmalm, Schmitz, Forssberg, & Ehrsson, 2006).

It is important to note that the static and dynamic tasks used power and precision grips, respectively, and involved different force output ranges (20–30 N *vs* 5–10 N). An imaging study found that different cortical networks are involved in power and precision grip, with the former being confined to the contralateral hemisphere and the latter involving cortical areas in both hemispheres (Ehrsson et al., 2000). Thus one could attribute the performance dissociation between the two tasks to the difference in cortical networks controlling power versus precision grip rather than the difference in networks controlling static versus dynamic grip. In particular, the involvement of undamaged ipsilateral sensorimotor areas in the dynamic task may have enabled appropriate scaling of grip force with load force. Subsequent imaging studies found almost no difference in the networks controlling power and precision grip (Kuhtz-Buschbeck et al., 2008; Takasawa et al., 2003). However, due to the use of two different grips in the two tasks, the observed performance dissociation between the tasks does not necessarily support the hypothesis that static and dynamic grips are separately controlled sensorimotor processes.

In conclusion, S1 injury impaired feedback mechanisms required to maintain power grip force and impaired accurate initiation of grip forces on well-learned tasks. Feedforward mechanisms implicated in anticipatory precision grip modulation, meanwhile, remained unaffected by S1 lesion.

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