Available online at www.sciencedirect.com

ScienceDirect

Journal homepage: www.elsevier.com/locate/cortex

Research report

The effects of acute cortical somatosensory deafferentation on grip force control

Andrew G. Richardson [a,d,*], *Mark A. Attiah* [a], *Jeffrey I. Berman* [b], *H. Isaac Chen* [a], *Xilin Liu* [c], *Milin Zhang* [c], *Jan Van der Spiegel* [c] *and Timothy H. Lucas* [a,d]

[a] *Department of Neurosurgery, University of Pennsylvania, Philadelphia, PA, USA*
[b] *Department of Radiology, University of Pennsylvania, Philadelphia, PA, USA*
[c] *Department of Electrical and Systems Engineering, University of Pennsylvania, Philadelphia, PA, USA*
[d] *Center for Neuroengineering and Therapeutics, University of Pennsylvania, Philadelphia, PA, USA*

ARTICLE INFO

Article history:
Received 11 December 2014
Reviewed 1 May 2015
Revised 16 September 2015
Accepted 13 October 2015
Action editor David Carey
Published online 30 October 2015

Keywords:
Somatosensory
Motor control
Deafferentation
Lesion
Internal model

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

* *Corresponding author*. University of Pennsylvania, 502C Stemmler Hall, 3450 Hamilton Walk, Philadelphia, PA 19104, USA.
E-mail address: andrew.richardson@uphs.upenn.edu (A.G. Richardson).
http://dx.doi.org/10.1016/j.cortex.2015.10.007

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