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Coordination deficits in ideomotor apraxia during visually targeted reaching reflect impaired visuomotor transformations

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ABSTRACT

Ideomotor limb apraxia, commonly defined as a disorder of skilled, purposeful movement, is characterized by spatiotemporal deficits during a variety of actions. These deficits have been attributed to damage to, or impaired retrieval of, stored representations of learned actions, especially object-related movements. However, such deficits might also arise from impaired visuomotor transformation mechanisms that operate in parallel to or downstream from mechanisms for storage of action representations. These transformation processes convert extrinsic visual information into intrinsic neural commands appropriate for the desired motion. These processes are a key part of the movement planning process and performance errors due to inadequate transformations have been shown to increase with the dynamic complexity of the movement. This hypothesis predicts that apraxic patients should show planning deficits when reaching to visual targets, especially when the coordination and/or dynamic requirements of the task increase. Three groups (18 healthy controls, 9 non-apraxic and 9 apraxic left hemisphere damaged patients) performed reaching movements to visual targets that varied in the degree of interjoint coordination required. Relative to the other two groups, apraxic patients made larger initial direction errors and showed higher variability during their movements, especially when reaching to the target with the highest intersegmental coordination requirement. These problems were associated with poor coordination of shoulder and elbow torques early in the movement, consistent with poor movement planning. These findings suggest that the requirement to transform extrinsic visual information into intrinsic motor commands impedes the ability to accurately plan a visually targeted movement in ideomotor limb apraxia.

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1. Introduction

Ideomotor limb apraxia, a disorder that commonly occurs following left hemisphere damage, has been described as an inability to perform skilled, purposeful movements (Haaland, Harrington, & Knight, 1999, 2000; Ochipa & Gonzalez Rothi, 2000; Pazzaglia, Smania, Corato, & Aglioti, 2008; Rothi, Ochipa, & Heilman, 1991). The cardinal deficits associated with the disorder are spatiotemporal errors (e.g. jerky vertical movements rather than smooth horizontal motion when performing a bread slicing gesture) observed across a variety of movement contexts (Clark et al., 1994; Poizner et al., 1995; Poizner, Mack, Verfaellie, Rothi, & Heilman, 1990). These deficits are commonly identified by errors made during imitation or pantomime of meaningful movements (object-related and intransitive gestures) and/or imitation of meaningless movements. In order to diagnose apraxia, most studies rely upon a composite score that reflects errors across these different types of movements, and relatively few studies have directly examined differences among them. However, because several studies have shown that apraxic patients make more errors in the context of object-related actions (Haaland & Flaherty, 1984; Haaland et al., 2000; Mozaz, Rothi, Anderson, Crucian, & Heilman, 2002) and the view that planning of object-related movements is partially distinct from meaningless movements due to their dependence on previously stored knowledge about objects and their functions (see Frey, 2007 for a review), recent research has focused largely on understanding deficits in object-related motion (see Daprati & Sirigu, 2006 for a review).

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However, many studies, including some from our laboratory have demonstrated that spatiotemporal deficits in apraxia are not limited to object-use movements. For example, we have previously shown that apraxic patients make errors that are consistent with impaired integration of spatial and temporal features of movements during reaching (Haaland et al., 1999) and sequencing (Harrington & Haaland, 1992). Importantly, apraxic patients also make more errors than non-apraxic patients and healthy subjects when asked to imitate meaningless movements, such as touching the nose, and also intransitive gestures, such as saluting (Goldenberg, 1999; Haaland et al., 2000; Toraldo, Reverberi, & Rumiati, 2001). In fact, numerous studies have used deficient imitation of intransitive and/or meaningless movements as a basis for classifying patients as apraxic, without considering object-use movements at all (Buxbaum, Johnson-Frey, & Bartlett-Williams, 2005; De Renzi, Motti, & Nichelli, 1980; Pazzaglia et al., 2008). These results have thus demonstrated that deficits in apraxia are evident during movements made in a variety of contexts, and are not simply limited to object-use actions.

More recent studies have focused on elucidating the mechanisms underlying the different types of deficits observed in apraxic patients. Several authors have proposed that these deficits arise from damage to, or impaired retrieval of previously stored representations of learned actions. Accordingly, lesion and imaging studies have shown the involvement of left parietal regions for recognition as well as imitation of object-related actions and meaningful/intransitive gestures, which has led to the conclusion that these parietal regions form the neural substrate for storage of representations for learned actions (Buxbaum et al., 2005; Buxbaum, Kyle, Grossman, & Coslett, 2007; Buxbaum, Sirigu, Schwartz, & Klatzky, 2003; Chao & Martin, 2000; Creem-Regehr, 2009; Swinnen et al., 2010). Damage to these regions would also explain the impairment observed in apraxic patients during the pantomime and command-evoked gesturing of such learned actions. However, Goldenberg (2009) has reviewed extensive evidence supporting the view that damage to left parietal regions adversely impacts imitation of not just highly learned actions, but also meaningless movements and motor performance on other tasks.

A mechanism that could account for these deficits following parietal damage is a failure to convert visual information, such as experimenter actions during imitation, into motor commands necessary to produce the desired motion. This hypothesis is particularly compelling as an explanation for the deficits seen in apraxic patients during the imitation of novel and meaningless movements, for which neural representations may not be developed (Rothi et al., 1991; Tessari, Canessa, Ukmar, & Rumiati, 2007). This view is also consistent with the extensive evidence supporting the role of posterior parietal circuits in the transformation of extrinsic visual information into intrinsic motor commands required to produce the desired motion during visually targeted movements (see Buneo & Andersen, 2006; Fogassi & Luppino, 2005; Jackson & Husain, 2006 for reviews). For visually targeted movements, such visuomotor transformations have been shown to be a key component of the movement planning process (see Sarlegna & Sainburg, 2009 for a review). Moreover, inaccuracies during this transformation process have been shown to adversely impact motor planning (Sarlegna, Przybyla, & Sainburg, 2009; Schlicht & Schrater, 2007; Sober & Sabes, 2005), with more recent results suggesting that movement coordination errors that occur as a consequence of inadequate transformations are larger for movements involving more complex movement dynamics (Sarlegna et al., 2009). It is important to emphasize that this hypothesis does not imply that deficits in apraxia cannot occur due to a loss in stored knowledge of learned actions, or that such actions do not involve visuomotor transformation processes. Rather, we suggest that deficits should be evident due to impaired sensorimotor transformation mechanisms that operate in parallel or downstream to mechanisms involved in the representation of learned actions. If this is true, we predict: (1) planning deficits in patients with ideomotor apraxia should be evident during simple reaching movements to visual targets and (2) these deficits should be magnified as the dynamic requirements of the movement are increased. We test this hypothesis in a task in which healthy control, and non-apraxic and apraxic left hemisphere damaged subjects reach to three visually presented targets that vary in the degree of interjoint (shoulder and elbow) coordination required. We expect that apraxic patients will show planning deficits associated with impaired specification of movement dynamics early in the movement, prior to the time that feedback can influence movement patterns. In addition, we predict that these deficits will be greatest for the movement requiring maximal interjoint coordination.

2. Methods

2.1. Participants

Because ideomotor limb apraxia is more prevalent following left rather than right hemisphere damage (De Renzi et al., 1980; Haaland & Flaherty, 1984; Kertesz & Ferro, 1984), we restricted our patient group to 18 left hemisphere damaged (LHD) stroke patients. We compared the performance of these stroke patients with that of 18 age- and education-matched healthy control subjects. Prior to participation, all subjects gave informed consent according to the Declaration of Helsinki. The study was approved by the Human Research Review Committee of the University of New Mexico and the New Mexico Veterans Affairs Healthcare System. All subjects were pre-morbidly right-handed; handedness was determined using a 10-item version of the Edinburgh inventory (Oldfield, 1971). None of the participants had a history of (i) substance abuse within the past 10 years and/or significant psychiatric diagnosis; (ii) any neurological diagnoses for control subjects and non-stroke related neurological problems for stroke patients, or (iii) movement restrictions for peripheral reasons, such as neuropathy or orthopedic disorders. Language comprehension (Kertesz, 1982) and grip strength were assessed in all subjects, and stroke patients were also given the Fugl-Meyer test of motor function in the contralesional arm (Fugl-Meyer, Jaasko, Leyman, Olsson, & Steglind, 1975).

The LHD stroke patients were divided into two groups, apraxic or non-apraxic, determined using a standardized test for limb apraxia (Haaland & Flaherty, 1984). Patients were asked to imitate five meaningless (e.g. index finger on earlobe), five intransitive (e.g. salute) and five transitive (e.g. brush teeth) movements with their ipsilesional (left) arm. When errors in internal hand configuration (e.g. fist versus flat palm), orientation (e.g. vertical versus horizontal) or target (e.g. brushing nose instead of teeth) were made, or when a body part was used as an object (e.g. extend finger to brush teeth), the item was scored as incorrect. Thus, more than one type of error could be made on a single movement, but only one error per movement was scored. Patients were considered apraxic if they made spatio-temporal errors on four or more of the fifteen movements (2 SD greater than normal controls) (Haaland & Flaherty, 1984; Haaland et al., 2000). Movements were videotaped for scoring by two raters. Using this test, 9 of the 18 LHD patients were classified as apraxic, while the remaining 9 were classified as non-apraxic. The average number of errors made by the apraxic group was 4.88 ± 0.35 (mean ± S.E.), while the non-apraxic stroke subjects made 1.22 ± 0.32 (mean ± S.E.) errors during apraxia testing. The same battery was given to control subjects, who made 1.7 ± 0.29 (mean ± S.E.) errors.

MRIs were obtained in 16 stroke patients, while CT scans were done for 2 patients (1 apraxic, 1 non-apraxic) due to medical contraindications for MRI (e.g. cardiac pacemakers). The area of damage for each patient was traced on 11 standardized horizontal sections derived from the DeArmond atlas (DeArmond, Fusco, & Dewey, 1989) using T1-weighted MRI images for anatomical detail and T2-weighted images to specify borders of the damaged tissues. This was done by a board certified neurologist, who was blinded to the behavioral characteristics of the patients. These outlines were retraced on a digitizing tablet for input into a computer program to allow the overlapping of lesions within each stroke group and to calculate lesion volume (Frey, Woods, Knight, Scabini, & Clayworth, 1987).

2.2. Experimental setup and task

The experimental setup (Fig. 1) and task were the same as in our recent study (Schaefer, Haaland, & Sainburg, 2009b). Briefly, subjects sat facing a table with their palms facing downwards and hands supported over the table using an air sled system to eliminate the effects of gravity and minimize friction. A cursor representing the position of the index finger tip, a start circle and targets were projected using a ceiling mounted projector onto a back projection screen, and a mirror was placed beneath this screen. The mirror blocked direct vision of the subjects arm, but reflected the visual display to give the illusion that the display was in the same horizontal plane as the fingertip. Position and orientation of the forearm and upper-arm segments were sampled using a Flock of Birds (Ascension Technology) system. The positions