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Visuo-motor gain adaptation and generalization following left hemisphere stroke

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Abstract

During gain adaptation, participants must learn to adapt to novel visuo-motor mappings in which the movement amplitudes they produce do not match the visual feedback they receive. The aim of the present study was to investigate the neural substrates of gain adaptation by examining its possible disruption following left hemisphere stroke. Thirteen chronic left hemisphere stroke patients and five healthy right-handed control subjects completed three experimental phases involving reaching with the left hand, which was the less-affected hand in patients. First, participants reached without visual feedback to six different target locations (baseline phase). Next, in the adaptation phase, participants executed movements to one target under conditions in which the perceived movement distance was 70% of the produced movement distance. Last, in order to test the generalization of this new visuomotor mapping, participants made movements without visual feedback to untrained target locations (generalization phase). Significant between-patient differences were observed during adaptation. Lesion analyses indicated that these between-patient differences were predicted by the amount of damage to the supramarginal gyrus (Brodmann area 40). In addition, patients performed more poorly than controls in the generalization phase, suggesting that different processes are involved in adaptation versus generalization periods.

Keywords

Kinematic adaptation; Gain perturbation; Motor Generalization; Neuropsychology; Stroke

Introduction

The motor system's rapid adaptability is critical for movement production. The processes underlying this flexible mapping between motor commands and expected perceptual results have been studied using motor adaptation paradigms which modify the relationship between the movement produced and the visual feedback received by the participant. This adaptation can involve a modification of movement direction by rotating the displayed path

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(visuomotor rotation adaptation [3]) or a modification of movement amplitude by changing the gain between movement amplitude and the displayed visual feedback (gain adaptation [2,14,26]). Although visuomotor rotation adaptation and its neural correlates have been studied extensively, less consideration has been given to the neural substrates of gain adaptation. Neuroimaging and patient studies of rotation adaptation report a large number of critical regions, including the cerebellum, the posterior parietal cortex [7,8,16,22,25], the premotor cortex [8,16] and the basal ganglia [18,19,22]. In contrast, the single study on the neuroanatomic basis of gain adaptation [16] reported that only subcortical structures (bilateral putamen) and left cerebellum were activated.

A complimentary approach to understanding the neuroanatomic bases of gain adaptation is to study patients with brain damage to examine if there is a consistent relationship between area of damage and behavioural deficits [20]. The aim of the present study was to investigate the neural substrates of gain adaptation by examining its possible disruption following left hemisphere stroke. Stroke patients and healthy controls made reaching movements under conditions in which the perceived movement distance was 70% of the produced movement distance. In response to the perturbation, participants had to learn to produce larger amplitude movements. Then, we tested the patients' capacity to generalize this gain adaptation to untrained movement directions and amplitudes. Given that gain adaptation in healthy controls generalizes across both direction and amplitude [17,26], testing for generalization in patients allowed us to confirm that the same sensorimotor processes were utilized in both participant groups. Based on the single previous neuroimaging study of gain adaptation [16], we predicted that left hemisphere stroke patients with putamen damage would exhibit poor gain adaptation. It is important to note that this study used relatively simple, small amplitude movements of a joystick, which may have reduced neural activation. Thus, we also considered the possibility that other regions involved in rotation adaptation (posterior parietal cortex, premotor cortex, and the basal ganglia) may also be critical for gain adaptation. Such an outcome would not be surprising since many of these regions are also involved in other sensorimotor functions. For example, the posterior parietal lobe seems to be involved in state estimation and for updating state estimates under conditions of mismatch [24], which is required for motor adaptation. Therefore, it is reasonable to predict that damage to these areas could disrupt gain adaptation.

Material and methods

Thirteen left hemisphere cerebral vascular accident (LCVA) patients (7 male, 6 female; mean age: 54 years and 3 months) and five healthy right-handed control subjects (5 female; mean age: 68 years and 7 months) participated in a single session, one-hour experiment. We limited our recruitment to LCVAs because this project was completed as part of a larger body of research in our laboratory focusing on this population. Recruiting from this population afforded us the access to research-quality brain scans required to complete the lesion analyses described below. Demographic and clinical information about the patients is shown in Table 1. Although the control group was on average older than the LCVA group, analyses included below show that age was not significantly correlated with the dependent measures described below (p 's $\geq .15$). Participants consented to the study in accordance with IRB guidelines of Albert Einstein Medical Center and were paid for their participation.

All participants performed the task while seated on a height adjustable chair in front of a digitizing tablet (CalComp, Drawing Board III, GTCO CalComp Inc, USA) placed horizontally 71 cm above the floor and positioned above the navel. The participants' heads and torsos were free to move. An image from a liquid crystal display (LCD) projector was presented on a back projection screen and viewed in a semi-silvered mirror 23 cm above the

digitizing table. The position of the mirror was adjusted so that when looking down at the mirror, the participant saw the virtual image of the target in the plane of the pointing surface. At the same time the mirror prevented participants from seeing their hand. In their left hands participants held a digitizing pen whose position was registered by the digitizing tablet (60 Hz, 0.1 mm resolution) and was displayed to the subject in real time as a 0.2 cm radius cursor.

Participants were tested in three blocks of trials (i.e., baseline, adaptation, and generalization) run in a single session. In the baseline block, participants were required to reach from a common start circle (S circle in Fig. 1, 0.8 cm in diameter) to six peripheral targets (0.8 cm in diameter) separated by 45° and placed at either 10 or 15 cm from the start circle. Each block began with the participant moving the digitizing pen to position the cursor (which indicated their hand position) inside the start circle. Once inside the start circle for 1500 ms, a target appeared prompting participants to execute their movement. The cursor disappeared after the participants moved 1.6 cm from the centre of the starting circle. Therefore participants executed movements without online visual feedback of their hand location. Once the participants thought their hand was in the target, they were asked to stop there briefly and then return to the start target. After 1500 ms in the start target the peripheral target disappeared and the next trial began. Participants executed 12 movements to each of the 6 targets, and the target presentation order was randomly varied. To match the feedback used in the adaptation block (see below), endpoint knowledge-of-results were provided for movements to a single target (C10, see Fig. 1). At the end of the movement to only this target, a circle indicating the participant's hand position (i.e., the terminal knowledge-of-results) was presented for 1500ms.

Next, in the adaptation block participants adapted to a modified gain relationship between the movements they produced and the visual feedback they received. A single target ("training target", C10, see Fig. 1) was used during these 50 trials. Trial presentation was identical to the knowledge-of-results trials in the baseline condition for the C10 target except that the relationship between the knowledge-of-results feedback and the actual position of the hand was modified with a gain of 0.7. Thus, the amplitude of the cursor's movement displayed during knowledge-of-results was 70% of the amplitude of the produced movement. Participants had to learn to produce larger amplitude movements (a movement 42% larger than the original). We used knowledge-of-results feedback because a stronger kinematic adaptation is observed under this condition [12].

Finally, the generalization block examined two issues. First, it tested how well participants were able to maintain the adaptation that occurred in the previous block. These 12 "adaptation maintenance" trials were made only to the training target (C10) and were identical to the adaptation phase and included endpoint knowledge-of-results (with a gain of 0.7). Second, 60 "generalization" trials to the other five, unadapted, targets examined how well the gain adaptation generalized to untested targets. As for the baseline block, participants executed these 60 "generalization" movements without online visual feedback of their hand. The order of the 72 target presentations was randomly varied.

Our analyses focused on measures derived from movement amplitude. Amplitude Accuracy (AA) of the i th movement during the baseline and the adaptation phase was calculated as a percentage of the target amplitude : $AA_i = 100 \cdot (A_i - T) / T$, where A_i is the i th movement amplitude and T the target amplitude. Perfect AA values were 0 in the baseline block and 42 during the adaptation block. Generalization was assessed separately for each participant by calculating the change in the amplitude of each trial from the average movement amplitude obtained for that target in the baseline period. The generalization of amplitude (GenA) of the i th movement was then calculated as a percentage: $GenA_i = 100 \cdot (A_{gen_i} - MeanA) / MeanA$,

where A_{gen_i} is the i th movement amplitude recorded during the Generalization period and $MeanA$ is the average movement amplitude performed by the subject for the corresponding target in the baseline phase.

Lesion locations were identified from T1-weighted MRI scans. Lesions were then segmented by an experienced research team member (who was blind to the behavioral data) using the MRIcro image analysis program. Lesion segmentations were confirmed by an experienced neurologist who was also blind to the behavioral data. After segmentation, proportion of damage to Brodmann's areas was calculated by using Brodmann maps included in the MRIcro program, which were based on the templates developed by Damasio & Damasio [6].

Results

Both groups produced relatively accurate movement amplitudes during the baseline phase. AA indexes showed no significant difference ($F(1,16) = 0.75, p = .41$) between patients ($M = -3.13, SD = 5.09$) and control participants ($M = 0.26, SD = 6.33$).

Marked between-patient variability was observed during both adaptation and generalization. To illustrate this point, Figure 2 shows the relationship for both controls and patients between an individual's adaptation performance (last 10 trials of adaptation) and generalization. As expected, these two variables were correlated for control participants ($r = 0.95, p = 0.013$) and for patients ($r = 0.77, p = 0.002$).

During adaptation (x-axis of Figure 2) both patients and controls generally modified their movement amplitudes according to the knowledge-of-results. Although there was a tendency toward lower levels of final adaptation in all patients (33.08 vs. 43.95 - AA index- for patients and controls, respectively), this difference did not reach significance ($F(1,16) = 2.43, p = 0.13$). The group analyses, however, fail to illustrate the notable patient variability, with the one patient (L3) who was completely unable to adapt and two other patients (L7 and L10) with notably lower levels of adaptation than the control group (their performance were 2 standard deviations below the control mean).

At the group level, generalization -GenA- (y-axis of Figure 2) was significantly less complete ($F(1, 16) = 4.91, p < 0.05$) for the patients ($M = 17.57, SD = 14.84$) than for control participants ($M = 31.53, SD = 7.82$). Similarly large between-patient differences were observed, with the three patients with poor adaptation also exhibiting poor generalization (L3, L7, and L10) as well as two additional patients (L9 and L13) who had relatively normal adaptation but poor generalization, as indicated by their performance being 2 standard deviations below the control generalization mean.

To try to explain this patient variability in both phases we examined the relationship between the behavioral measures shown in Figure 2 and the patients' lesion locations. We first identified potential regions of interest (ROIs) based on previous reports in other motor adaption studies (see introduction). These ROIs included BA 5, 6, 7, 39, 40, 44, 45 and 46 as well as four subcortical structures (Pallidum, Caudate Nucleus, Putamen and Thalamus). To avoid undue influence from a single patient, we limited our ROI analysis to those ROIs in which at least 33% of patients ($n = 4$) had damage to at least 10% of the voxels in that ROI. Five ROIs met this criterion: BA 6, 39, 40, 44, and 45 and the Putamen.

Table 1 displays the correlation between number of voxels damaged and adaptation performance in the final 10 trials (left column) and generalization trials (right column). We observed a significant negative correlation between the number of damaged voxels in BA 40 and adaptation performance ($r = -0.67, p < 0.02$). Thus, increasingly greater damage to BA

40 was associated with increasingly poor adaptation. No other significant correlations between other ROIs and adaptation or generalization performance were observed.

Discussion

The aim of the present study was to investigate the neural substrates of gain adaptation by examining its possible disruption following stroke. The primary result is that significant between-patient variability was observed during both adaptation and generalization (see Figure 2). This level of patient variability is commonly observed in stroke patients, with many analysis techniques, such as voxel-based lesion symptom mapping [1], relying on significant between-patient variability (see [15] for an example). Although some patients were unimpaired during learning and generalization, others had poor performance during the adaptation and/or the generalization period. Lesion analyses showed a significant negative correlation between the amount of damage to the supramarginal gyrus (SMG; BA 40) and performance during the adaptation period. This result is consistent with other adaptation studies implicating the SMG in motor adaptation [8,16]. However, all of these studies investigated rotation adaptation. Thus, a novel finding of the present study is identification of the role of the SMG in gain adaptation. This finding extends the results of an earlier imaging study showing only subcortical (bilateral putamen and left cerebellum) activation in a gain adaptation task [16], and suggests that the SMG may be involved in both rotation and gain adaptation.

Previous researchers have described a variety of roles to the SMG. The region is a part of the inferior parietal lobule which has been shown to be involved in the selection and preparation of complex skilled actions [4,9]. In our study, since participants did not receive visual feedback during the entire movement, they could adapt to the visuo-proprioceptive mismatch only by changing motor planning for the following movements. Interestingly, some authors have proposed that this specific region is implicated in motor attention. For example, Hesse et al. [11] showed that SMG is involved in the selection of the correct movement and the preparation of the respective action, and not just in the disengagement of motor attention from one action to another. This result is consistent with the hypothesis that patients with lesions to the SMG may have problems in preparation and/or selection of the correct movement in a situation where the relation between the visual feedback and the motor commands is altered, as it is in our paradigm.

The second main result of the present experiment was that patients have poorer generalization than controls when tested with untrained movement directions and distances. Our results show that it is possible to adapt to a gain modification but fail to generalize this new visuo-motor mapping. Krakauer and colleagues [16] found that different structures may be involved in different portions of motor adaptation. For example they showed that the early phase of rotation adaptation activated the preSMA, and that later phases of adaptation activated right ventral premotor cortex, right posterior parietal cortex, and the left lateral cerebellum. For gain adaptation, these investigators observed a specific pattern of activation during the early course of adaption (bilateral putamen and left cerebellum), but failed to observe any significant activations during later stages of adaptation. The present study may indicate that during gain adaptation different processes, and potentially different neural structures, are involved in different aspects of adaptation (e.g., training and generalization).

Although we did observe a significant relationship between BA40 damage and adaptation performance, our small sample of LCVA patients limited our power to detect similar relationships in other regions or in the right hemisphere. A recent study by Schaefer and colleagues [21], showing that end position control is more disrupted in right hemisphere patients than left, suggests that gain adaptation may be even more disrupted in right

hemisphere stroke patients. In addition, we found only a weak relationship ($r = -.19$) between amount of putamen damage and adaptation performance, even though this was one of only a few regions activated in a previous neuroimaging study of gain adaptation [12]. Several methodological differences could be responsible for this difference, such as our use of knowledge-of-results feedback rather than continuous visual feedback [13], or the potential use of both implicit and explicit knowledge for adaptation [10]. Future studies would be needed to address these issues. The fact that left-hemisphere patients with parietal damage exhibited deficits in acquiring internal models is also in accordance with Buxbaum et al. [5] who showed that left hemisphere stroke patients with parietal lesions may have a specific deficit in generating and maintaining internal representations of goal directed movements. This hypothesis appears compatible with our observation that patients with left BA 40 lesions were impaired at developing a new solid internal model.

Abbreviations

LCVA	left hemisphere cerebral vascular accident
LCD	liquid crystal display
AA	Amplitude Accuracy
GenA	Generalization of Amplitude
ROI	regions of interest
SMG	supramarginal gyrus
SMA	supplementary motor area

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Research highlights

- Left hemisphere stroke patients and controls completed movements to one target.
- The perceived movement distance was 70% of the produced movement distance.
- The between-patient differences were predicted by the amount of damage to BA 40
- Different processes are involved in adaptation versus generalization motor periods.

Fig. 1.

Top view with starting and target positions used in the baseline and in the generalization blocks. Targets were disposed either at the Left (L), the centre (C) or at the Right (R) respective to the subject's body axis, and were separated by 45° . Target distance was 10 cm or 15 cm. In the adaptation block only movements toward the C10 target were executed (see details in the text).

Fig. 2.
Relation between the adaptation period (last block of adaptation) and the generalization period for both controls and patients.

Table 1
Demographics, comprehension, gesture performance and lesion information of participants.

Group	Participant	Age (year,month)	Education	Gender	Testing date relative to stroke (year,month)	WAB	Apraxia Index Score	Brodman's areas lesioned
LCVA	L1	71,8	13	M	10,8	63,7	66,25	2,3,6,20,21,22,38,41,42,44,45,47
	L2	51,11	12	M	4,6	na	na	25
	L3	54	13	M	7,6	92,3	68,75	1,2,3,4,5,6,7,8,40
	L4	38,9	12	F	4,6	91,5	85	9,44,45
	L5	63,8	16	M	16,6	46,1	56	2,3,4,6,9,44,45
	L6	53,8	16	F	7,11	87,1	70	21,22,37,40,41,42,43,44
	L7	63,1	11	M	5	62,2	75	1,2,3,19,21,22,37,39,40,41,42,43
	L8	51,1	16	F	2,9	84,7	77,5	1,3,4,6,8,24,32,43,44,45
	L9	63,1	16	M	2	89	86	17,18,19,22,39,40,41,42
	L10	60,2	12	F	0,8	78	75	34
	L11	54,6	12	F	1,9	81,8	82,5	21,22,41,42
	L12	34,3	14	F	3,10	na	na	45
	L13	46,6	12	M	9,1	na	na	1,2,3,4,6,22,43,44,45
Control	Mean	54,3	13,4		5,11	77,6	74,5	
	S.D.	10	1,8		4,2	14,6	10	
	C1	74,11	14	F				
	C2	70,11	12	F				
	C3	64,10	16	F				
	C4	65,4	12	F				
	C5	66,11	16	F				
	Mean	68,7	14					
	S.D.	3,10	1,8					

WAB: Western Aphasia Battery (see [5] for details); Apraxia Index Score: Apraxia score was determined through testing gesturing to the sight of objects and meaningless imitation (see [5] for details); Brodman's areas lesioned: ≥ 10% damage to listed regions; na: not available.

Table 2

Correlation matrix between the adaptation (AA) and generalization (GenA) periods and the number of voxels damaged in neuroanatomic ROIs.

Correlation coefficients (r)		
Brodmann area (BA)	Adaptation	Generalization
BA 6	−0.26 (p = 0.38)	−0.23 (p = 0.48)
BA 39	−0.17 (p = 0.57)	−0.22 (p = 0.48)
BA 40	− 0.67 (p = 0.01)	−0.35 (p = 0.25)
BA 44	0.25 (p = 0.42)	0.47 (p = 0.12)
BA 45	0.26 (p = 0.38)	0.27 (p = 0.40)
Putamen	−0.19 (p = 0.52)	−0.15 (p = 0.63)

Note: Significant *p* value appears in bold characters.