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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 and generalization periods.

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 (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 [18,19,22,23]. 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 adaption is to study patients with brain damage to examine if there is a consistent relationship between area of damage and behavioral 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 adaption 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

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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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.

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, 1-h 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 ≥ 0.15). 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 1500 ms.

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].

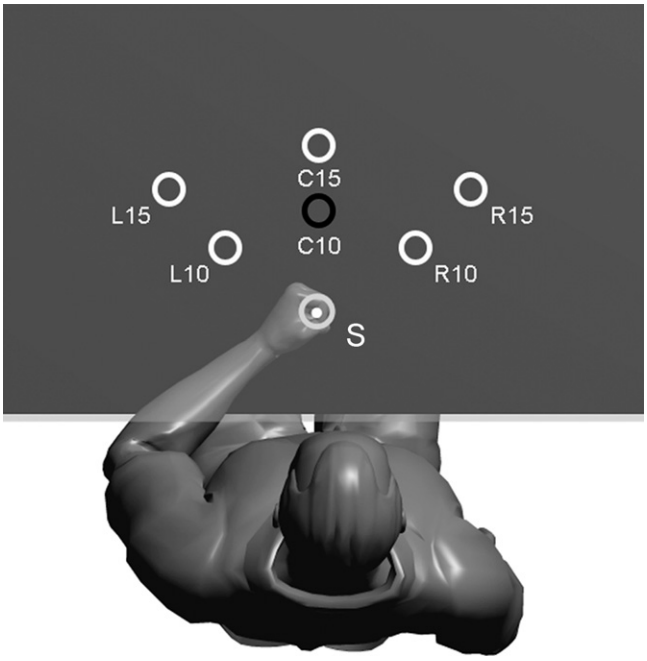

Fig. 1. Top view with starting and target positions used in the baseline and in the generalization blocks. Targets were disposed either at the left (L), the centre (C) or at the right (R) respective to the subject's body axis, and were separated by 45°. Target distance was 10 cm or 15 cm. In the adaptation block only movements toward the C1O target were executed (see details in the text).

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 in the baseline block, participants executed these 60 "generalization" movements without online visual feedback of their hand. The order of the 72 targets presentations was randomly varied.

Our analyses focused on measures derived from movement amplitude. Amplitude Accuracy (AA) of the ith movement during the baseline and the adaptation phase was calculated as a percentage of the target amplitude: $AA_i = 100 \times (A_i - T)/T$, where A_i is the ith 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 ith movement was then calculated as a percentage: $GenA_i = 100 \times (Agen_i - MeanA)/MeanA$, where $Agen_i$ is the ith 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