Error correction strategies for motor behavior after unilateral brain damage: short-term motor learning processes

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Abstract

In order to identify the mechanisms underlying motor impairments and motor learning following stroke-related brain damage, we analyzed correction strategies used by hemiparetic individuals to produce precise elbow flexion movements of the paretic arm and compared them to those of healthy individuals. Participants made rapid elbow flexion movements to a 6° wide target and were instructed to correct movement errors as quickly as possible when a spring-like load was unexpectedly introduced. Angular positions and torques before correction were used to identify error patterns. Results showed that participants with mild hemiparesis minimized movement errors within three trials, as did healthy participants. In contrast, severely affected individuals needed more trials to diminish errors and their movements were inconsistent. Participants with a moderate motor disability used both typical and atypical correction strategies. The differences in correction behaviors likely reflect deficits in arm motor function (r = 0.79) and executive function (r = 0.58) rather than levels of intellectual function (IQ ratings). Results indicate that the deficits that individuals with stroke experience when adapting their movements to changed load conditions may be due to difficulty in rapidly integrating visual and proprioceptive information. Deficits in executive function could also contribute to problems in producing accurate and consistent movements from trial to trial. Taken together, these results imply that all hemiparetic individuals would not benefit equally from the same motor re-training approaches. © 2002 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Motor skill acquisition in healthy individuals is well documented. For example, repeated practice of even very simple movements, such as rapid single-joint elbow displacements to a target results in a decrease of movement time and an increase of peak velocity, both of which have been associated with motor learning [13]. In assuming that movement repetition leads to functional improvement in individuals with stroke-related brain damage as it does in healthy individuals, therapists encourage patients to practice different patterns of movement or different specific movements. However, well-controlled and systematic studies of how new motor skills are acquired in patients with central nervous system (CNS) damage due to stroke are few and have mainly focused on the analysis of the effects of different treatment approaches on specific motor outcome measures [2,12,19,30,37]. There is little empirical evidence supporting the notion that the same type of motor learning as that seen in healthy individuals occurs in the hemiparetic arm of stroke patients. Indeed, some or all of the components necessary for the acquisition of new movements may be impaired after a stroke [31]. In addition, the presence of cognitive impairments may have considerable influence on adapting movements to new conditions since modification of the motor plan requires the integration, storage and use of sensory information from the previous movement for the initiation of subsequent movements. This plasticity of thought, which requires working memory within the domain of executive functions has been
described as specific to the frontal lobes. Working memory is used to hold information on-line in order to achieve basic cognitive abilities such as comprehending, reasoning and problem solving [29]. It has been postulated that the working memory system consists of three components, a central executive or limited capacity workspace and two slave subsystems that support it [1]. Briefly, one such system consists of a phonological loop that allows rehearsal or recycling of small amounts of information and the second is likened to a visual “sketchpad”, thought to be involved in the short-term retention of visual and spatial information with an underlying role of the visual association cortex, inferior parietal lobe and inferior prefrontal cortex. The frontal cortex as well as the striatum and cerebellum have in turn been implicated in visuospatial motor learning [4,26,27]. It is, therefore, reasonable to assume that stroke-related damage in sensorimotor cortical areas affects working memory within the domain of executive functions when motor learning takes place.

To examine the relationship between cognitive and sensorimotor learning in hemiparetic patients, we used a simple experimental paradigm by which short-term motor learning in healthy participants was studied using repetitive 50° single-joint elbow flexion movements [39]. After training elbow flexion without an external load, a spring-like load that resisted the movement was unexpectedly introduced, leading to an undershoot movement error. Healthy participants eliminated this error after one or two trials in the new opposing load conditions suggesting that during successful task performance, the CNS stores in memory the values of specific parameters (control variables, CV's), that determine the relationship between muscle force and joint position. These CV's, which are reproduced in subsequent trials, are context-specific such that a change in context (i.e. the introduction of a load) results in a movement error, requiring subsequent adjustment of the CV's. The system is then able to reproduce a precise movement in the next loaded trial by using the adjusted CV's, a modification that could provide an explanation for the phenomenon of “one-trial learning” [39]. The hypothesis that movement reproduction depends on information about CV's stored and recalled from memory also implies that the pattern of movement errors may be history-dependent when load conditions change. According to this hypothesis, individuals may produce precise movements in a set of trials with or without a load but the first movement after an unpredicted change in external condition will always result in a movement error.

The experimental paradigm and the theoretical framework of Weeks et al. [39] were used in the present study to analyze the short-term motor learning strategies in hemiparetic individuals. Their performances were compared to those of healthy participants in order to determine whether hemiparetic individuals use similar adaptive strategies when learning a simple movement. This is particularly important for clinicians interested in refining clinical approaches to maximize treatment effects. Some of these results have previously appeared in abstract form [3].

2. Methods

2.1. Participants and clinical testing

Ten hemiparetic (mean age: 47.1 ± (S.D.) 13.4 years; Table 1) and six healthy individuals (mean age: 23.6 ± 2.3 years) participated after signing a consent form approved by the Ethics Committee of the Rehabilitation Institute of Montreal (RIM). The hemiparetic patients had sustained a single unilateral stroke between 6 months and 2.3 years previously and had a contralateral hemiparesis ranging from mild to severe (between 2 and 6 out of a maximum score of 7 on the Chedoke-McMaster stroke assessment scale [14]). They were screened for cognitive and perceptual deficits and excluded if these interfered with their ability to understand instructions or to carry out the task. Seven of the 10 participants had sustained a left hemispheric stroke and three had a right hemispheric stroke. Lesion location studies (MRI, PET imaging) revealed that four participants had cortical lesions only, two had cortical/sub-cortical lesions and four had restricted sub-cortical lesions (Table 1).

All hemiparetic participants underwent a battery of clinical, physical and neuropsychological tests prior to the
limb as well as from a cursor on a computer screen. A 6° final target window was chosen since it has proven to be positioned at 70° axis of rotation of the manipulandum and the shoulder was flexion/extension axis of the elbow was aligned with the wrist in the neutral position between supination and pronation by a polypropylene splint. The splint held the forearm and platform (manipulandum, Fig. 1) attached to a torque motor of an external load applied to the manipulandum by a torque motor function to 66 (maximum function) (Table 1).

Sensorimotor impairment of the arm was assessed using the Fugl-Meyer scale (FM) [9]. Scores ranged from 19 (low motor function) to 66 (maximum function) (Table 1).

Neuropsychological testing [3] was done to determine the relationship between motor behavior of the arm and standardized cognitive measures. First, the level of intellectual function was determined with a standardized abbreviated version of the WAIS-R and used as a reference for the interpretation of results in four specialized areas: (1) semantic (verbal) memory (Wechsler memory scale (revised), digit span, short prose passages); (2) figural or visuospatial (non-verbal) memory (Rey-Osterrieth complex figure); (3) attention (cancellation test for focused attention; trail making tests A and B for sustained attention and visuomotor tracking speed); (4) executive function (Wisconsin card sorting test, Tower of London).

All participants, except one, underwent neuropsychological testing either at the RIM or the Montreal Neurological Institute (MNIH). The results in each specialized area were ranked in four categories based on a comparison of test scores with established norms derived from a population of 122 neurologically intact adults tested at the MNIH: above average (four); average (three); below average (two); or well below average (one).

2.2. Experimental procedure

Participants were seated in a chair that fully supported the trunk. The forearm, wrist and hand rested on a horizontal platform (manipulandum, Fig. 1) attached to a torque motor by a polypropylene splint. The splint held the forearm and wrist in the neutral position between supination and pronation and between flexion and extension, respectively. The flexion/extension axis of the elbow was aligned with the axis of rotation of the manipulandum and the shoulder was positioned at 70° flexion and 70° abduction. The arm position ensured that participants could easily perform isolated single degree-of-freedom (d.f.) elbow movements. Participants made 50° flexion movements from an initial (3°) to a final (6°) target with the dominant right arm for healthy individuals and the hemiparetic arm for the experimental group. Since previous studies have shown a variable range in forearm motion control in hemiparetic individuals [21], the initial position was predetermined for each participant according to their ability to control elbow flexion through a 50° arc. Thus, the initial position differed for each participant and ranged from 110 to 170° with full extension at 180°. Participants had visual feedback from the moving limb as well as from a cursor on a computer screen. A 6° final target window was chosen since it has proven to be wide enough for healthy individuals to make both rapid and accurate movements [8,39].

Feedback about movement accuracy was given at the end of each trial by visualization of the final position of the cursor. Feedback about movement speed was also available at the bottom of the screen from a horizontal bar of different lengths (Fig. 1). Since, they were instructed to move at a self-paced speed on all trials, the feedback ensured that participants reproduced a similar range of velocities from trial to trial in the training session. For experimental trials, they were asked to correct movement errors resulting from the sudden and unexpected introduction or removal of an external load applied to the manipulandum by a torque motor attached to its axis of rotation. For loaded trials, the load was zero when the arm was in the initial position and it was increased as a linear function of arm displacement to the target. The presence of the load could not be detected before the arm left the initial window. In order to compare results between healthy and hemiparetic participants, the value of the load at the target position was chosen as a percentage (30%) of the participant’s maximal voluntary isometric strength at that position, determined prior to the start of the experiment (mean of three trials). The stiffness constant of the load ranged from 0.03 to 0.08 N m/° (corresponding to 1.5–4.0 N m at the target position) for hemiparetic individuals and from 0.06 to 0.10 N m/° (corresponding to 3.0–5.0 N m at the target position) for healthy participants.

Prior to the start of the experiment, all participants practiced making fast and accurate movements without the load.
Training stopped when performance reached 70% accuracy in a series of 10 non-loaded trials. For both groups, 20–40 training trials were required to meet criteria before experimental testing began. The experiment consisted of a series of trials divided into blocks. A block of trials in which the opposing load was applied alternated with a block of trials without an opposing load. All trials in a block had the same load condition such that we were able to establish the number of trials necessary to minimize error. The number of trials in each block was varied randomly between five and 10 to prevent anticipation of the change in load and preparatory adjustments. Blocks were alternated 12–15 times for a maximum of 150 trials. Trials in each block were classified according to type: (1) test trial (T), the first trial in a changed load condition; (2) trials P1 and P2, the second and third post-test trials, respectively; (3) C trials, all other trials in the same load condition. For healthy participants, C trials were control trials in which movements were fully adapted to new load conditions, i.e. no errors made.

2.3. Data analysis

Movement behavior was analyzed under two conditions: blocks of trials with a load and blocks of trials with no load. For each trial, torque, velocity and position data were recorded with separate transducers at 1000 Hz from 0.2 s before movement onset for a total duration of 2.2 s. Thus, participants had 2 s to complete the initial movement and the correction. Trials were analyzed individually on an interactive video display to determine the initial position, the peak velocity and the final position before correction.

Criteria for the identification of the final position before correction were established after careful qualitative inspection of angle–time, velocity–time and velocity–angle (phase) curves from T, P1 and P2 trials compared to C trials. In non-loaded C trials, the arm made a smooth transition from one position to another after rapid (Fig. 2A and B) or comparatively slow (Fig. 2C) decaying terminal oscillations. Similar differences were also observed in velocity–time (Fig. 2D–F) and phase curves (Fig. 2G–I). The phase diagrams in C trials were spiral-like and eventually terminated at the final position. The term “phase” refers to the time along these diagrams.

In T, P1 or P2 trials, angle–time curves showed that the forearm approached an intermediate position and then moved to a secondary position, indicating a movement correction. The exact times marking the end of the primary movement and the onset of the correction could not be clearly identified from angle–time and velocity–time curves. They could, however, be easily identified as inflection points on phase diagrams combining position, velocity and time information. In Fig. 3, the primary movement is represented...
by the smooth right to left U-shaped trajectory of the phase diagram that is similar in shape to the uncorrected C trial (Fig. 3A, compare right and left panels). In the same figure, the final position of the primary movement is represented by an inflection point (Fig. 3A, right, short vertical arrow) in the phase diagram. The diagram also shows the secondary or corrective movements made to reach the target. Generally, an undershoot error in the primary movement of the T trial occurred when the load condition was changed from no load to load (Fig. 3A and B) and an overshoot error occurred when the load condition changed from load to no load (Fig. 3C). For the hemiparetic group, undershoot errors occurred in 87.1% of the T trials when conditions changed from no load to load. The mean undershoot error for T trials was 14.9° compared to 12.4° for the healthy group (Table 2). The mean overshoot error (66.6% of trials) when conditions changed from load to no load was 5.5° compared to 9.5° for the healthy group. The change in final positions in T trials was significantly different from that in P1 trials for both conditions (load, $F = 23.72, d.f. = 3, P < 0.01$; no load, $F = 12.07, d.f. = 3, P < 0.01$).

For hemiparetic participants, the mean time of correction in T trials was 704 ± 270 ms when conditions changed from no load to load and 950 ± 378 ms when they switched from load to no load ($P < 0.05$). Overall, the shortest correction latency for all hemiparetic participants was 393 ms. In contrast, the mean error correction time in T trials for the healthy
Second, final positions in P1, P2 and C trials for the load condition were significantly less than those in the no load condition. The average peak velocity in no load C trials for the hemiparetic participants was 262 ± 397 ms.

Table 2

<table>
<thead>
<tr>
<th>Participant Type of trial</th>
<th>From no load to with load (load)</th>
<th>From with load to no load (no load)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T</td>
<td>P1</td>
</tr>
<tr>
<td>Hemiparetic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>23.5 ± 9.9</td>
<td>36.0 ± 6.1</td>
</tr>
<tr>
<td>2</td>
<td>36.0 ± 4.2</td>
<td>38.7 ± 4.0</td>
</tr>
<tr>
<td>3</td>
<td>29.6 ± 1.9</td>
<td>34.3 ± 2.6</td>
</tr>
<tr>
<td>4</td>
<td>37.5 ± 4.6</td>
<td>39.3 ± 5.6</td>
</tr>
<tr>
<td>5</td>
<td>34.2 ± 10.2</td>
<td>45.6 ± 3.2</td>
</tr>
<tr>
<td>6</td>
<td>43.0 ± 5.5</td>
<td>46.9 ± 8.7</td>
</tr>
<tr>
<td>7</td>
<td>42.7 ± 7.1</td>
<td>49.6 ± 2.3</td>
</tr>
<tr>
<td>8</td>
<td>33.4 ± 3.3</td>
<td>40.8 ± 2.2</td>
</tr>
<tr>
<td>9</td>
<td>33.7 ± 5.4</td>
<td>46.1 ± 3.0</td>
</tr>
<tr>
<td>10</td>
<td>36.3 ± 3.6</td>
<td>46.8 ± 7.0</td>
</tr>
<tr>
<td>Mean ± S.D.</td>
<td>35.1 ± 5.9</td>
<td>42.3 ± 5.4</td>
</tr>
<tr>
<td>Healthy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± S.D.</td>
<td>37.6 ± 4.5</td>
<td>47.4 ± 2.7</td>
</tr>
</tbody>
</table>

*Perfect final position is 50°.

Group was over 240 ms faster than for the hemiparetic group (455 ± 64 ms) when load conditions changed from no load to load and 603 ± 133 ms when the switch was from load to no load. For this group, the shortest correction time was 397 ms.

In general, movements made by the hemiparetic participants were slower than those made by the healthy group, although this difference did not reach statistical significance. The average peak velocity in no load C trials for the hemiparetic participants was 262.4 ± 74.7 °/s compared to 334.1 ± 20.5 °/s for healthy individuals. The latter completely adapted their movements to new load conditions as evidenced by a similar peak velocity in load (341.5 ± 9.6 °/s) compared to no load C trials and by an accurate final position (Table 2). The stroke differed from the healthy group in two ways. First, movement speed for load trials (247.7 ± 83.9 °/s) was still significantly slower than in no load C trials (paired t-test, t = 2.98, d.f. = 9, P < 0.02). Second, final positions in P1, P2 and C trials for the load condition were significantly less than those in the no load condition as shown by a mean undershoot of the target position of 6.5, 7.3 and 5.4 °, respectively (P < 0.01; Table 2).

In healthy individuals, the error in T trials was, on average, corrected in the subsequent P1 trial in 63% of cases. This pattern is consistent with a one trial learning process. By the third trial (P2), the percentage of accurate final positions rose to an average of 87% and was maintained for the remaining C trials. This is illustrated by a series of final positions of 60 trials before correction in a healthy participant (Fig. 4C). For this individual, errors resulting from the change in load condition were eliminated in fewer than three trials in 100% of cases and in one trial, by P1, in 77% of cases.

Three hemiparetic participants (S7, S9 and S10) with well-recovered motor function (FM scores from 62 to 66) behavied similarly to healthy individuals in more than 60% of blocks in which load conditions changed. Fig. 4B shows final positions before correction in 60 of the 120 trials for S9. For this participant, positional errors resulting from the change in load condition were eliminated in fewer than three trials in 69% of cases and in one trial in 46.5% of cases. The other seven participants made inadequate attempts to correct the error with less than 40% of cases showing similar correction patterns as those of healthy participants. Fig. 4C shows final positions before correction in 60 trials in the participant with the lowest functional score (S1). Movement errors were eliminated in fewer than three trials only 33% of the time, in one trial in only 6.3% of the cases and no stable correction pattern could be identified. Her final elbow positions before corrections for all trials demonstrate that: (a) movement errors were eliminated mainly after more than three trials (Fig. 4C, arrow 1); (b) even when the target was reached in fewer than three trials the behavior was inconsistent (Fig. 4C, arrow 2); (c) in some blocks, accurate movements did not occur in any of the C trials (Fig. 4C, arrow 3).

Correction strategies used by most participants with mild sensorimotor deficits (high FM scores) resembled those of healthy individuals while those of participants with more severe sensorimotor deficits (low FM scores) were atypical. For example, errors in C trials were more often seen in hemiparetic participants. For the healthy group, only about one error occurred in C trials within a single experiment in the no load condition, while 2.3 ± 1.5 errors occurred in the load condition. For hemiparetic participants, the number of errors was greater and averaged more than two errors in C trials in the no load condition and exceeded six errors in the load condition.
Four different strategies of movement corrections when load conditions changed were identified (Fig. 5): (1) in pattern 1 (Fig. 5A), the error in the T trial was corrected in the subsequent trial (P1) and movements were made without corrections in the C trials; (2) pattern 2 (Fig. 5B) was characterized by a T and P1 error, followed by a correct position in P2 maintained in the C trials; (3) pattern 3 (Fig. 5C) was described by a T trial error, a correction to the target within the next two trials but more than one error in the subsequent C trials (inconsistent behavior); (4) pattern 4 (Fig. 5D) was characterized by a T trial error that remained uncorrected for the remaining trials in a block.

Table 3 shows the percentage of cases for both load conditions in which each of the four patterns occurred for each participant. Percentages of patterns 1 and 2 were then pooled together because they included all the strategies seen in healthy individuals and were judged as appropriate adaptive behaviors. Patterns 3 and 4, not seen in healthy individuals [39], were pooled and considered to be non-adaptive since they represented unsuccessful attempts to correct the error evoked by the load change.

Error correction strategies were categorized according to the frequency of use of each pattern. Category 1 included participants using patterns 1 and 2 more than 60% of the time. Category 2 grouped participants using typical strategies in approximately half the trials (between 40 and 60%) and category 3 included those using typical patterns in less than 40% of trials. Frequencies of patterns for each participant and the categorization of individuals are shown in Fig. 6. There was a significant relationship between correction behaviors and the level of residual arm motor function (FM scores and category, \( r = 0.84, P < 0.05; \) Spearman).

Participants with correction behaviors classified as category 1 had FM scores between 62 and 66. Those in category 2 had scores ranging from 22 to 65 and those in category 3 scored between 19 and 26. In general, three out of four participants with mild sensorimotor impairments fell into category 1 and three out of four participants with more severe sensorimotor deficits were classified in category 3.

### 3.1. Correlations with clinical measures

To determine how multiple variables interact, we used logistic regression analysis. Since logistic analysis requires a dichotomous dependent variable, we grouped participants into two types according to response behaviors: those with adaptive behavior (category 1) were assigned a score of 0 and those with non-adaptive behaviors (categories 2 and 3) were scored 1. Using response type as the dependent variable, the combination of scores on verbal memory, non-verbal memory and intelligence (IQ) tests explained only 10.3% of the variance (\( r = 0.32 \)). The addition of the score for executive function significantly improved the correlation (variance explained = 52.3%, \( r = 0.72 \)) while the addition of a fifth variable (visual-spatial attention) did not further strengthen the model (variance explained = 52.4%, \( r = 0.72 \)).
The best correlations were obtained with the covariance of FM scores and executive functions on error correction behaviors. The relationship between the types of motor behavior and the FM score explained 62.8% of the variance ($r = 0.79$), while that between motor behavior and executive function explained only 33.1% of the variance ($r = 0.58$). However, when both were combined and compared to the motor behavior, the percent of variance explained by the model climbed to practically 100% with a regression coefficient of 1.

4. Discussion

Hemiparetic participants required more time within a trial to begin the corrective movement. When adapting to the addition of a load, they did not fully adjust movement speed or achieve the non-loaded final position within a block of loaded trials. These movement deficits have been previously described [34] but the present results provide new information about error correction strategies in which CVs specifying individual movements are learned, stored in memory and recalled appropriately for accurate movement production. We were able to measure a subject’s ability to choose an appropriate CV or motor command by analyzing movement errors. Using this paradigm based on a model of motor control, we have shown that participants with mildly impaired cognitive and sensorimotor functions were able to use correction strategies similar to those of healthy individuals and retained the capacity for one-trial learning. Those with more severe impairments used atypical correction strategies reflecting maladaptive behavior.

4.1. Motor learning

Our data suggest that the persistent C trial errors committed by the moderately to severe hemiparetic participants are accounted for by impaired interpretation of proprioceptive information about limb position and load as well as by deficient mnemonic and problem-solving ability. Lesions in the prefrontal cortex, primary motor cortex, posterior parietal cortex or basal ganglia may explain errors related to movement direction and extent [10,11,16,17], while deficits in force production in the hemiparetic limb may also contribute to movement error [34]. The correlation between patterns of motor behavior, functional motor deficits and executive problem solving ability in individuals with moderate to severe clinical motor impairment supports the view that the aptitude to use somatosensory experience in each load condition cannot be considered independent of cognitive processes involved in motor memory and problem-solving ability.

Evidence for the co-dependence of these subsystems stems from findings of numerous reciprocal connections...
between regions of the medial temporal lobe critical for mnemonic processing, inputs to specific regions of the prefrontal cortex originating from the visual prestriate cortex, the auditory and the visual temporal association cortex and the posterior parietal cortex involved in motor planning [28].

4.2. Explanations based on models of motor control

Current models of motor control can explain how these deficits may be involved in producing the atypical correction behaviors observed in our hemiparetic participants. Two classes of models provide frameworks for the explanation of motor learning. One assumes that the nervous system plans the desired movement trajectories and then, based on some internal model of the motor apparatus, computes appropriate muscle torques and forces to perform the movement using inverse dynamics [15,22,23,32,40]. The other class is based on the experimental finding that intentional movements or isometric torque exertions emerge from the resetting of the activation thresholds ($\lambda$'s) of muscles, a finding resulting in the formulation of the equilibrium-point ($\lambda$ model) hypothesis [6,39]. In this model, proprioceptive information about the current state of the system as well as of the moving limb is integrated into the motor command. Both models are consistent with current theories of motor learning that suggest that information stored in short-term memory is accumulated and transferred to long-term memory as a “motor schema” which represents the system’s “knowledge” of how to perform a specific task [33,36]. According to Schmidt [33], the CNS integrates in the schema rules that forecast the effects of a motor command in a given environment. Learning consists of a refinement or refreshment of the schema with repeated practice. The ‘schema’ would be represented by the “forward model” in the inverse dynamics framework and by CVs according to the $\lambda$ model. In particular, according to the $\lambda$ model, participants initially issue CVs adapted to no load conditions. These CVs determine the appropriate relationship between muscle torque and joint angle resulting in an accurate movement to the target. When conditions are changed to load, the arm is prevented from reaching the zone (undershoot) and the system appropriately adjusts the CVs resulting in a movement correction in the T trial. In the P1 and subsequent trials with load, healthy participants generally reproduce the adjusted CVs to guarantee correct movements. Our results show that some hemiparetic participants (category 1), like healthy individuals, were able to use information from the previous movement (T, P1 and P2 trials) to adjust subsequent movements in the same load condition (C trials). This implies that they were able to perceive the movement error in the T trial, store the information in short-term memory and update the motor schema or CVs in subsequent trials.

For moderately and severely affected patients, a similar explanation may be used. Because appropriate correction behaviors were seen in a proportion of trial blocks, these patients to some extent retained the capacity to perceive the movement error and use previously stored sensorimotor information in subsequent movements. However, their ability to reproduce the appropriate forward model or CVs was incomplete resulting in more difficulty making simultaneously rapid and accurate movements against a novel load. In these individuals, under-compensation or partial correction strategies were more often observed (patterns 3 and 4) resulting in the occurrence of positional errors in the same direction (range effects [18]) or in the opposite direction (assimilation effects [35]). This latter behavior may be due to impaired central processing in which sensory information cannot be translated into internal coordinates or appropriate commands cannot be selected causing inadequate or incomplete updating of CVs from trial to trial. The unstable behavior in the C trials can be explained by a random reproduction of motor commands or by an inability to memorize and reproduce previously specified commands. Such inconsistent behaviors...
were observed in C trials of moderately severe hemiparetic participants (category 3 patients).

4.3. Clinical considerations

Our results suggest a relationship between the ability of hemiparetic individuals to adapt motor responses to new external conditions, the degree of motor impairment in the arm and to some extent, cognitive deficits. The latter may also be linked to deficits in decision-making such as problems with shifting mental sets or learning from external cues. However, further studies with larger numbers of patients with circumscribed lesions are necessary.

The ability of hemiparetic patients to use appropriate error correction strategies was associated with their level of sensorimotor impairment. Participants with high FM scores (>60; category 1) predominantly used correction patterns 1 and 2. The frequency of use of adaptive patterns can be used as an indicator of which patients may benefit from movement repetition therapy and which will require a greater amount of repetition to learn new tasks since it reflects the potential to make appropriate corrections after a change in load conditions. In addition to suggesting the presence of a motor control mechanism by which motor learning occurs, our results concur with those of previous studies showing different motor learning capacities and treatment outcomes in hemiparetic patients [2,7,38].

There was a good correlation between the grouped correction behaviors (patterns 1 and 2, and patterns 3 and 4) and residual arm function according to the FM scale. This suggests that the FM score may be a good physical indicator of the capacity of patients to benefit from movement repetition treatments. Since there was some overlap between categories based on FM scores (S3 and S8 in Fig. 6B), it is possible that other factors not measured by this scale might also influence the patient’s ability to make accurate corrections. Combining residual motor function and cognitive assessment may lead to the creation of a new global clinical score for motor learning capacities that would be more representative of the patient’s potential to benefit from movement repetition. Such information would be of interest for clinicians in choosing the best treatment approaches for individual patients.

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