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ORIGINAL ARTICLE

Preserved motor skill learning in acute stroke patients

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Abstract

Recovery is dynamic during acute stroke, but whether new motor skills can be acquired with the paretic upper limb (UL) during this recovery period is unknown. Clarifying this unknown is important, because neurorehabilitation largely relies on motor learning. The aim was to investigate whether, during acute stroke, patients achieved motor skill learning and retention with the paretic UL. Over 3 consecutive days (D1–D3), 14 patients practiced with their paretic UL the CIRCUIT, a motor skill learning task with a speed/accuracy trade-off (SAT). A Learning Index (LI) was used to quantify normalised SAT changes in comparison with baseline. Spontaneous motor recovery was quantified by another task without SAT constraint (EASY), by grip force (GF), and the Box and Blocks test (BBT). In patients, CIRCUIT LI improved 98% ± 66.2 (mean ± SD). This improvement was similar to that of young healthy individuals ($n = 30$) who trained with a slightly different protocol for 3 consecutive days (83.8% ± 58.8%). Generalisation of SAT gains to an untrained circuit was observed in both groups. From D1 to D3, stroke patients improved their performance on EASY, while changes in GF and BBT were heterogeneous. During acute stroke, patients retained SAT gains for a motor skill learned with the paretic UL in a manner similar to that of healthy individuals. These results demonstrate acute stroke patients achieved motor skill learning and retention that exceeded paretic UL improvements explained by spontaneous recovery.

Keywords Acute stroke · Hemiparesis · Motor skill learning · Upper limb function · Neurorehabilitation · Motor recovery

Introduction

Ongoing therapeutic progress has equated to more patients who survive the acute phase of stroke. Stroke thus became the primary cause of chronic disability in the Western World [1, 2]. Beyond the acute phase, a large proportion of stroke survivors (30–66%) suffer from chronic impairments [3, 4], with an approximate 40% dependence for daily life activities [5]. Motor impairments, mostly hemiplegia and hemiparesis, are especially frequent and have a major impact on independence and the ability to resume work [5, 6].

During the acute (post-stroke days 1–7) and early subacute (post-stroke day 7–3 months) phases of stroke [7], spontaneous recovery follows a stereotypical evolution which depends upon a cascade of biological events, which are little influenced by most neurorehabilitation therapies [8]. The amount of damage to the corticospinal tract is one of the major factors determining whether patients can expect to recover ~ 70% of their normal upper limb (UL) function based on the "proportional recovery" model [8] or whether recovery will be limited [9]. The proportional

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recovery model has been recently criticized and it is still unclear whether it should be refined or re-designed [10, 11]. Anyway, whereas the recovery of motor function has been extensively monitored from stroke onset to the chronic stage, much less is known about the capacity to achieve motor skills learning during (sub)acute stroke [8, 9, 12–14].

Motor learning is one of the driving forces of neurorehabilitation, particularly during the first post-stroke months during which rehabilitation interacts with the endogenous mechanisms of neural repair [13, 15–17]. Motor learning is a general concept which includes several "modules" that likely interact, such as sensorimotor adaptation (learning to recalibrate internal models and motor outputs to counteract a perturbation) and reinforcement learning (selection and improvement of actions through reward and punishment) that leads to the acquisition and retention of new sensorimotor skills such as lacing shoes [18]. When learning a motor skill, performance improves over time in terms of speed, accuracy, and speed/accuracy trade-off (SAT), with the capacity to generalise the skill to different contexts. The capacity to learn and retain motor skills is particularly important to neurorehabilitation, because stroke patients somehow face the challenge to (re-)learn how to perform everyday actions with an impaired UL which is controlled by limited neural resources.

It is currently unknown whether the capacity to achieve motor skill learning is enhanced, left unmodified, or impaired during (sub)acute stroke [13, 14]. This is a key issue, because neural repair and behavioural recovery interact with each other which should theoretically "boost" learning capacities [16]. If motor learning is enhanced during (sub)acute stroke, it is crucial to capitalise upon it. Conversely, (1) some forms of plasticity and motor learning might actually be maladaptive [17, 19, 20] and (2) because the physiology of the central nervous system is intensely disturbed after a stroke [21], neurobiological substrates of motor learning may be perturbed as well. The capacity to achieve motor learning may thus be transiently depressed early after a stroke. If so, it would be crucial to delineate the time-course of this phenomenon and to tailor neurorehabilitation accordingly.

The primary goal of this investigation was to determine whether, during the acute stroke phase, patients using their paretic UL achieved within- and between-session improvements in a complex motor skill learning task (CIRCUIT) involving a speed/accuracy trade-off (SAT). The secondary goal was to compare the changes in CIRCUIT with motor recovery.

Methods

Patients

After approval by the local Ethics Committee (CHU UCL Namur Mont-Godinne), patients were prospectively recruited from the Stroke Unit. This study complied with the Helsinki Declaration. The inclusion criteria were an acute stroke on brain imaging (MRI/CT) and a motor deficit of the contralesional UL. The exclusion criteria were (1) severe motor impairment of the UL (MRC scale < 3/5), (2) inability to follow instructions due to aphasia or cognitive impairment, or (3) medical instability.

Study design

After providing informed consent, the patients were enrolled between post-stroke days 1 and 7 (4.3 ± 1.7 days, mean ± SD). On the first experimental day (D1), a baseline evaluation of motor function was performed. Then, the patients trained on CIRCUIT on D1 and D2. On D3, after an evaluation identical to baseline, CIRCUIT retention and generalisation were evaluated.

Motor skill learning

The motor skill learning task (CIRCUIT) involves an SAT and has been used previously in healthy individuals and chronic stroke patients [22–24]. The patient sat in front of a computer's monitor on which a circuit was displayed (Fig. 1), and they controlled a cursor through a computer's mouse placed in the paretic hand. At the beginning of each session, the patients started with a 1 min habituation run (a square). Next, they were instructed to move the cursor through a complex circuit to complete as many laps as possible within 30 s (velocity constraint) while keeping the cursor within the track (accuracy constraint). On both D1 and D2, they practiced 30 training blocks with interleaved rest blocks (30 s). On D3, only five CIRCUIT blocks were performed to assess retention. Afterwards, they practiced five blocks with a new circuit of identical length and difficulty to assess generalisation.

Motor control tasks

To quantify motor recovery within the same environment, patients performed on D1 and D3 a control task without SAT (EASY): displacing the cursor between two large targets with continuous left–right movement (60 s, neither velocity nor accuracy constraints) and, after 30 s of rest, anteroposterior movement (Fig. 1).

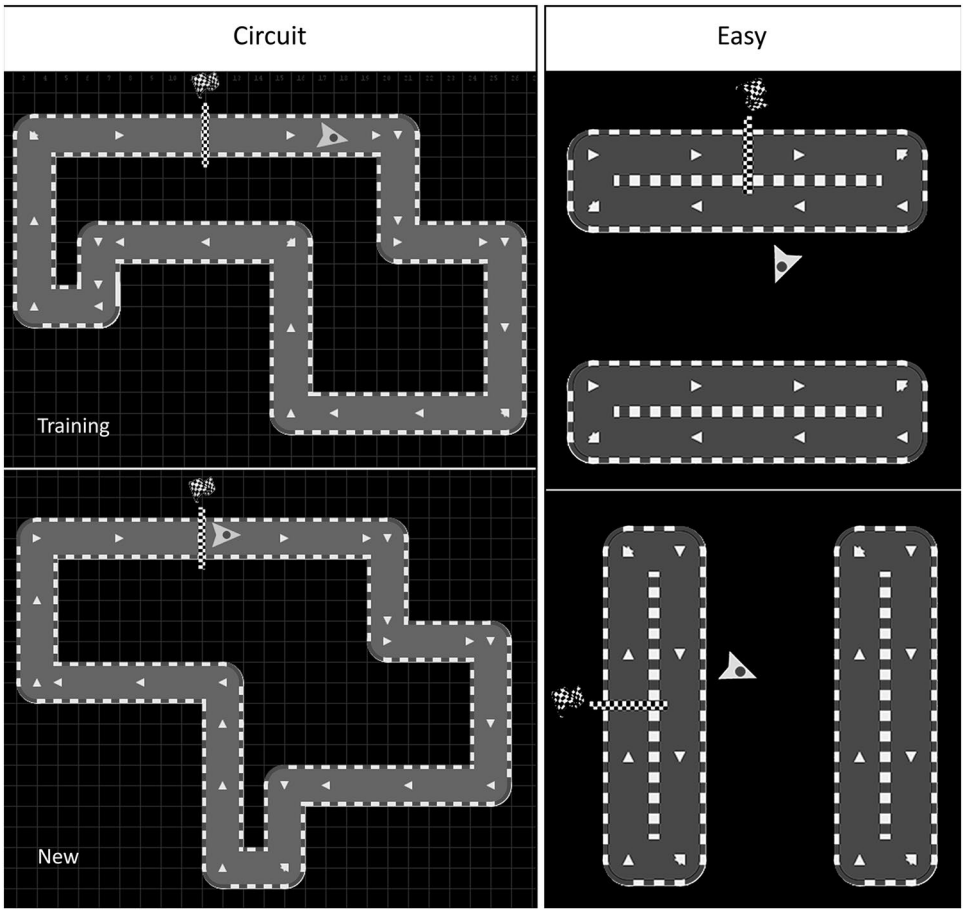

Fig. 1 CIRCUIT and EASY. Left column: the CIRCUIT task (upper row: the circuit used for training and retention; lower row: the circuit used for generalisation "new"). Right column: the EASY task

Moreover, the baseline (D1) and final (D3) assessments included the following measures: Box and Blocks (BBT) [25], grip force (GF) measured with a whole-hand Jamar dynamometer [26], and the NIH Stroke Scale (NIHSS) [27].

Definitions

There is no unequivocal means to disentangle motor learning from motor recovery, especially during (sub)acute stroke [13, 28, 29]. "Motor skill learning" is defined as the retention of SAT gains on CIRCUIT or the generalisation of SAT gains to an untrained circuit. "Motor recovery" is defined as SAT improvement on EASY or positive changes in GF or BBT.

Data processing and analysis

For each patient, the software divided each training block into 3 s bins ("repetition Time", rTime). Within rTime, it computed the mean error (E) and the total distance (D) travelled by the cursor. Error (the opposite of accuracy) was quantified as the surface between the cursor trajectory and the midline of the track, defined as the ideal trajectory. Velocity was defined as the first derivative of position, quantified by the formula $V = D/\text{rTime}$. Velocity and error were combined to compute a Performance Index (PI) using the formula $\text{PI} = V/E$ (where V = velocity and E = mean error). An increase in PI, driven by an improvement in velocity and/or a reduction in error [22, 23], reflects enhanced SAT, i.e., more skilled performance. Individual data were group-averaged into bins of 150 s (5 blocks of 30 s) for CIRCUIT and into two bins of 60 s for EASY.

For CIRCUIT, the Learning Index (LI) was computed by normalising PIs to the Baseline PI, with the formula $\text{LI} = [(\text{PI}_{\text{RETENTION}} - \text{PI}_{\text{BASELINE}})/\text{PI}_{\text{BASELINE}})] \times 100$. The primary measure was LI as retention on D3. The Generalisation Index (GI) was computed as $\text{GI} = [(\text{PI}_{\text{NEW}} - \text{PI}_{\text{BASELINE}})/\text{PI}_{\text{BASELINE}})] \times 100$, which expressed D3 performance on the new circuit as a percentage of the D1 Baseline on the trained circuit. The SAT difference between retention and generalisation on D3 was computed as [GI − LI]. For EASY, the SAT change from D1 to D3 was computed as $\text{EASY} = [(\text{EASY}_{\text{D3}} - \text{EASY}_{\text{BASELINE}})/\text{EASY}_{\text{BASELINE}})] \times 100$. The BBT and GF changes were computed with a similar formula; for the NIHSS only, the absolute difference was calculated.

Association between the D1–D3 changes on SAT (LI), BBT, GF, and EASY was measured by Pearson's correlation coefficient (statistical analyses performed with R 3.3.2 and the *ggplot2* package).

To provide an example of typical motor skill learning, the data from a group of young, right-handed healthy individuals (HI, $n = 30$, age 23.2 ± 5.3 years) who learned the CIRCUIT with the non-dominant left hand over 3 consecutive days were processed identically. Because our aim was not to compare the amount of motor skill learning between groups and because this protocol was slightly different, no formal statistical comparison was computed. The HI were recruited through the Faculty of Medicine of the UCLouvain with the same ethical regulations as patients. The inclusion criteria were being (1) > 18 years of age and (2) right-handed. The exclusion criteria were (1) a neurological condition and (2) use of medications modulating brain function.

Results

Of 364 consecutively screened patients, 31 matched the criteria. Four decided not to participate, 13 did not complete the experiment, because they worsened (2), dropped out (3), or were discharged (8) before the protocol's completion. Fourteen patients completed the study, seven men and seven women (Table 1). One female patient had a haemorrhagic stroke; the others had an ischemic stroke. The dominant hemisphere was involved in ten patients. One patient (#11; see Fig. 2) had a cardio-embolic stroke with multiple small cortical and sub-cortical lesions—the symptomatic one (left UL paresis) being in the right corona radiata (Fig. 2).

From D1 to D3, LI on CIRCUIT improved $98\% \pm 66.2\%$ (mean ± SD; median 71.9%, min 5.5%, max 267.5%) compared with baseline (Table 2). Although there was large inter-patient variability, the magnitude of LI evolution was comparable with that of HI ($83.8\% \pm 58.8\%$; median 79.1%, min 8.7%, max 266.6%; Fig. 3). One patient (#9; see Table 2) showed no clear SAT improvement on D3 (+5.5%), many of her PIs worsened when compared with baseline and her LI never reached 10%.

On D3, after retention assessment, the patients practiced a new circuit to assess generalisation. Their GI was $98.9\% \pm 59.2\%$ (median 92.5%, min 2.4%, max 198.2%) compared with baseline. The SAT difference between retention and generalisation [GI − LI] was $0.8\% \pm 38.6\%$ (median 4.7%, min −76.8%, max 51.1%). From retention to generalisation, eight patients improved and six worsened. This "worsening" was *relative to* the final retention on D3, and improvement on the new circuit was maintained compared to Baseline on D1. In HI, the GI was $67.2\% \pm 58.6\%$ (median 40.4%, min −8.5%, max 233.6%), and the [GI − LI] was $-16.6\% \pm 29.0\%$ (median −16.4%, min −93.8%, max 42.9%).

For EASY, the patients improved $101.2\% \pm 70.5\%$ (median 99.2%, min 1.6%, max 229.3%) (Table 2). From D1 to D3, the mean BBT improved $16.5\% \pm 15.8\%$, but three patients did not improve. The mean GF improved $4.4\% \pm 10.4\%$ on D3; however, GF worsened in five patients and did not change in one. The NIHSS improved (-0.5 ± 0.5 point) on D3 in seven patients and remained unchanged in the others.

There was a slight association between D1–D3 changes in LI, BBT, and EASY (Pearson's *r* [95%CI]: 0.26 [−0.32 to 0.69] and 0.23 [−0.34 to 0.68], respectively). There was no association between LI and GF (Pearson's *r*: 0.00 [−0.53 to 0.53]) (Table 3). The correlations between change in EASY and BBT or GF were slight (Pearson's *r*: 0.27 [−0.30 to 0.70] and 0.23 [−0.31 to 0.70], respectively), while the correlation between BBT and GF was moderate (Pearson's *r*: 0.54 [0.01 to 0.83]).

Discussion

During the acute phase of stroke, motor performances with the paretic UL improved over 3 days. Whether these improvements on CIRCUIT, EASY, BBT, GF, and NIHSS were driven by spontaneous recovery or whether CIRCUIT improvements reflect genuine motor skill learning is the heart of the following discussion.

Changes in paretic UL performance

From D1 to D3, improvements were large for CIRCUIT and EASY, whereas GF, BBT, and NIHSS improved less or even deteriorated. Smaller/absent improvements may reflect a ceiling effect for some measures (e.g., NIHSS) compared with the continuous CIRCUIT and EASY data. Several mechanisms may underlie these changes in paretic UL performance. Spontaneous recovery undoubtedly sustained improvements in the different tasks. Spontaneous recovery is driven by the biological phenomena triggered by stroke and the plastic reconfiguration of the spared neural resources [12, 16, 30]. Theoretically, motor recovery could have been the sole mechanism sustaining improvement in tasks requiring no motor skill learning (or to a minor extent) such as EASY, GF, or BBT. Habituation to the experimental procedures may have contributed to overall improvement, but it is unlikely to explain the full results. Patients may have developed compensatory strategies [15, 17, 31], e.g., increased trunk movements and UL synergies [32, 33]. The current data do not permit differentiating compensatory strategies from true recovery, i.e., the restitution of normal motor patterns [15, 17, 31]. Fluctuations in residual motor aptitudes

Table 1 Demographic and clinical characteristics of the stroke patients

Patient	Age (years)	Gender	Time @ incl (day)	Handn	Damaged hem	Stroke type	Localisation	Initial UL impairment	NIHSS Adm	NIHSS D1	NIHSS D3	IVT	TBY
1	69	M	5	R	Non-dom (R)	I	sub-cx	Useless hand	3	2	2	–	–
2	52	M	1	R	Dom (L)	I	sub-cx	Useless hand	6	0	0	YES	–
3	43	F	3	R	Dom (L)	I	cx	Useful hand	1	1	1	–	–
4	44	F	3	R	Dom (L)	H	sub-cx	Minimal mvts	4	1	1	–	–
5	50	M	6	R	Dom (L)	I	sub-cx	Useful hand	0	0	0	–	–
6	76	M	7	L	Dom (R)	I	sub-cx	Useless hand	5	4	3	–	–
7	64	M	5	L	Dom (R)	I	sub-cx	(Near) Normal	1	1	0	–	–
8	47	F	3	R	Dom (L)	I	sub-cx	Useless hand	1	0	0	–	–
9	42	F	4	L	Non-dom (L)	I	cx	Minimal mvts	15	1	0	YES	YES
10	82	F	4	R	Dom (L)	I	sub-cx	(Near) Normal	20	1	0	YES	–
11	75	M	5	R	Non-dom (R)	I	sub-cx/multiple cx	(Near) Normal	9	1	0	–	–
12	84	F	4	R	Non-dom (R)	I	cx	(Near) Normal	6	2	1	YES	–
13	78	M	3	R	Dom (L)	I	sub-cx	Useful hand	1	1	1	–	–
14	59	F	7	R	Dom (L)	I	cx	Minimal mvts	15	2	1	–	–
Mean	61.8	7F/14	4.3	2L/14	10 Dom/14	13 I/14	4 cx/14	–	6.2	1.2	0.7	4/14	1/14
SD	15.5		1.7					–	6.3	1.1	0.9		

M male, *F* female, *handn* handedness, *R* right, *L* left, *Hem* hemisphere, *Dom* dominant, *Non-dom* non-dominant, *I* ischemic, *H* haemorrhagic, *cx* cortical, *sub stroke-cx* sub-cortical stroke, *UL* upper limb, *mvts* movements, *adm* at admission, *IVT* intravenous thrombolysis, *TBY* thrombectomy

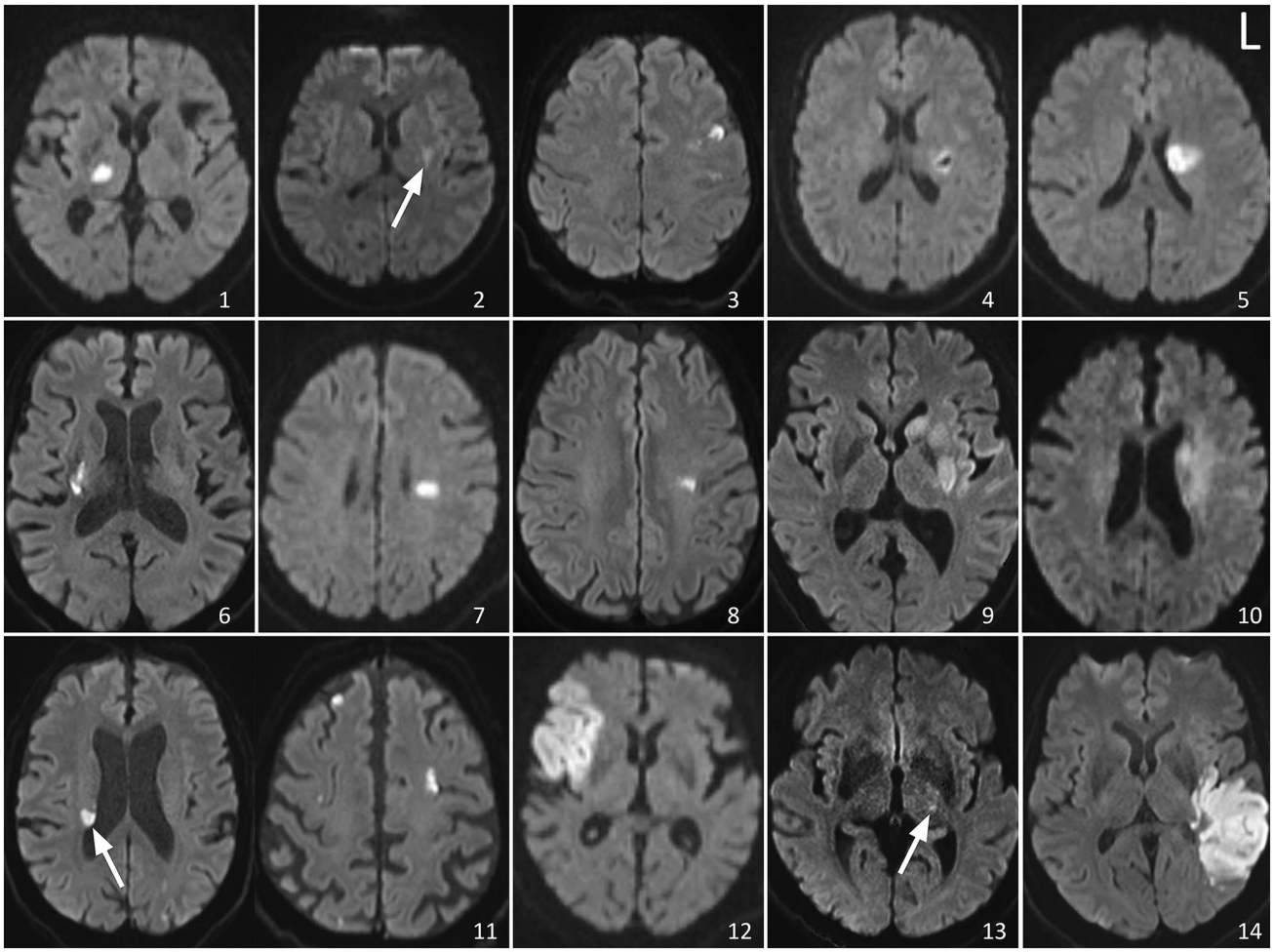

Fig. 2 Magnetic resonance imaging (MRI) scans. Note that patient #11 suffered from multiple cardio-embolic lesions in both hemispheres (small cortical and sub-cortical lesions); the sub-cortical stroke in the right corona radiata was responsible for the slight left-sided hemiparesis. *L* left

may explain some of the changes in BBT, GF, or NIHSS. Fluctuations and transient deteriorations may be driven by fatigue, hemorrhagic transformation or secondary bleeding, extension of the infarcted zone, etc. However, such fluctuations could not explain the (normalised) improvements in CIRCUIT similar to that of HI. Finally, motor skill learning may underlie a large part of CIRCUIT improvements.

Motor skill learning

It is unlikely that spontaneous recovery underlay the entire improvement in CIRCUIT for several reasons. Evolution's curves on CIRCUIT were typical of motor skills learning in stroke patients and HI [18, 23, 34, 35], with a relatively sharp early improvement followed by shallower asymptotic-like progress. During subacute-to-chronic stroke, recovery may spontaneously follow a similar evolution [4, 12, 17, 28–30]. However, improvements in CIRCUIT reflected both within-session progress and between-session retention, which suggests that it entailed some form of motor memory.

By design, the improvements in CIRCUIT were quantified as (1) within-session SAT gains and (2) between-session retention of SAT gains, a hallmark of motor skill learning [18, 35]. Despite a considerable overnight retention, there was a slight performance drop for the first block of the next day. Again, spontaneous recovery could theoretically underlie these changes, but the same evolution was observed in young HI achieving typical motor skill learning.

The SAT gains largely transferred to a new circuit compared to baseline, a typical generalisation, i.e., another hallmark of motor skill learning [18, 36]. Theoretically, the transfer of SAT gain may "simply" reflect spontaneous motor recovery. However, the SAT gain transfer on the new circuit was comparable to the generalisation found in HI, and in chronic stroke patients learning CIRCUIT under sham/real non-invasive brain stimulation [22, 23, 34].

Table 2 Changes in motor performance

Patient	LI retention (%)	GI new circuit (%)	GI – LI (%)	EASY (%)	BBT (%)	GF (%)	Delta NIHSS (point)
1	69.9	2.4	−67.5	45.5	36.1	19.4	0
2	122.5	90.3	−32.2	23.9	10.4	−5.8	0
3	66.2	106.9	40.7	50.3	0.0	15.0	0
4	48.6	65.6	17.1	100.5	15.9	6.3	0
5	79.2	116.3	37.1	1.6	12.3	−10.5	0
6	267.5	190.8	−76.8	192.9	50.0	17.6	−1
7	70.2	61.9	−8.3	46.1	8.9	10.3	−1
8	147.1	198.2	51.1	165.9	27.5	17.4	0
9	5.5	10.5	4.9	176.9	7.5	4.2	−1
10	73.6	78.1	4.5	229.3	0.0	0.0	−1
11	190.9	180.1	−10.8	107.7	9.1	−7.5	−1
12	64.6	101.5	36.9	139.3	40.0	5.6	−1
13	98.6	94.6	−4	39.6	0.0	−3.6	0
14	68.1	87.1	19.1	97.8	12.7	−7.4	−1
Mean	98.0	98.9	0.8	101.2	16.5	4.4	−0.5
SD	66.2	59.2	38.6	70.5	15.8	10.4	0.5

LI (Learning Index) at retention (% from baseline), *GI* (Generalisation Index), *EASY* motor task, *BBT* Box and Blocks Test, *GF* whole-hand grip force, *NIHSS* delta D1–D3 (point)

The correlations between the D1–D3 changes in LI, BBT, and GF were slight at best. Furthermore, there was either no change or even worsening in some patients for BBT, GF, and/or NIHSS. Beyond the technical issues already discussed (e.g., ceiling effects), it would be surprising that the same spontaneous biological processes would drive large and consistent recovery in some aspects of UL motor function while letting others deteriorate. The simplest explanation is that at least another process was involved in CIRCUIT improvements in addition to spontaneous recovery, namely motor skill learning.

The SAT improvements for the paretic UL were similar to those in young HI learning the CIRCUIT task, both for raw (PI) or individually normalised (LI) data. The mean baseline PI was lower and the inter-individual variability larger in the patients compared with the HI, as expected. The purpose of Fig. 3 was not to compare the amount of learning between stroke patients and HI but to provide evidence that a similar process was observed in both groups. If the process driving consecutive within-session SAT gain, between-session retention, and generalisation in HI is motor skill learning, then it should be—at least in part—the same process in patients. Alternatively, the most conservative interpretation would be that spontaneous motor recovery for an SAT task during (sub)acute stroke would be virtually identical to motor skills learning for the same task in HI. Such a training-induced recovery may be a fundamentally different form of motor learning, i.e., learning to activate the residual motor substrates to control the recovering motor repertoire of the paretic UL.

To sum up, from a behavioural point of view, the characteristics of SAT within-session gain, between-session retention, and generalisation for acute stroke patients, practicing the CIRCUIT with their paretic UL, are virtually indistinguishable from motor skill learning, retention, and generalisation in young HI, practicing the same task with their non-dominant UL.

Inter-individual differences

Motor skill learning was consistent across HI. Why did some patients achieve very little improvement with CIRCUIT? First, given patient heterogeneity, large inter-individual variability was expected, e.g., whether the dominant or non-dominant hemisphere was damaged. Second, some level of spontaneous recovery may be mandatory to achieve motor skill learning, which may not have been reached in some patients. However, there was a lack of BBT improvement or even GF deterioration in several patients who improved on CIRCUIT, which is inconsistent with this hypothesis. Third, it is possible that some patients were enrolled too early and that on-going negative processes (oedema, disrupted synaptic transmission, etc.) prevented the enhanced plasticity typical of the (sub)acute stroke period. Fourth, because damage to the cerebellum

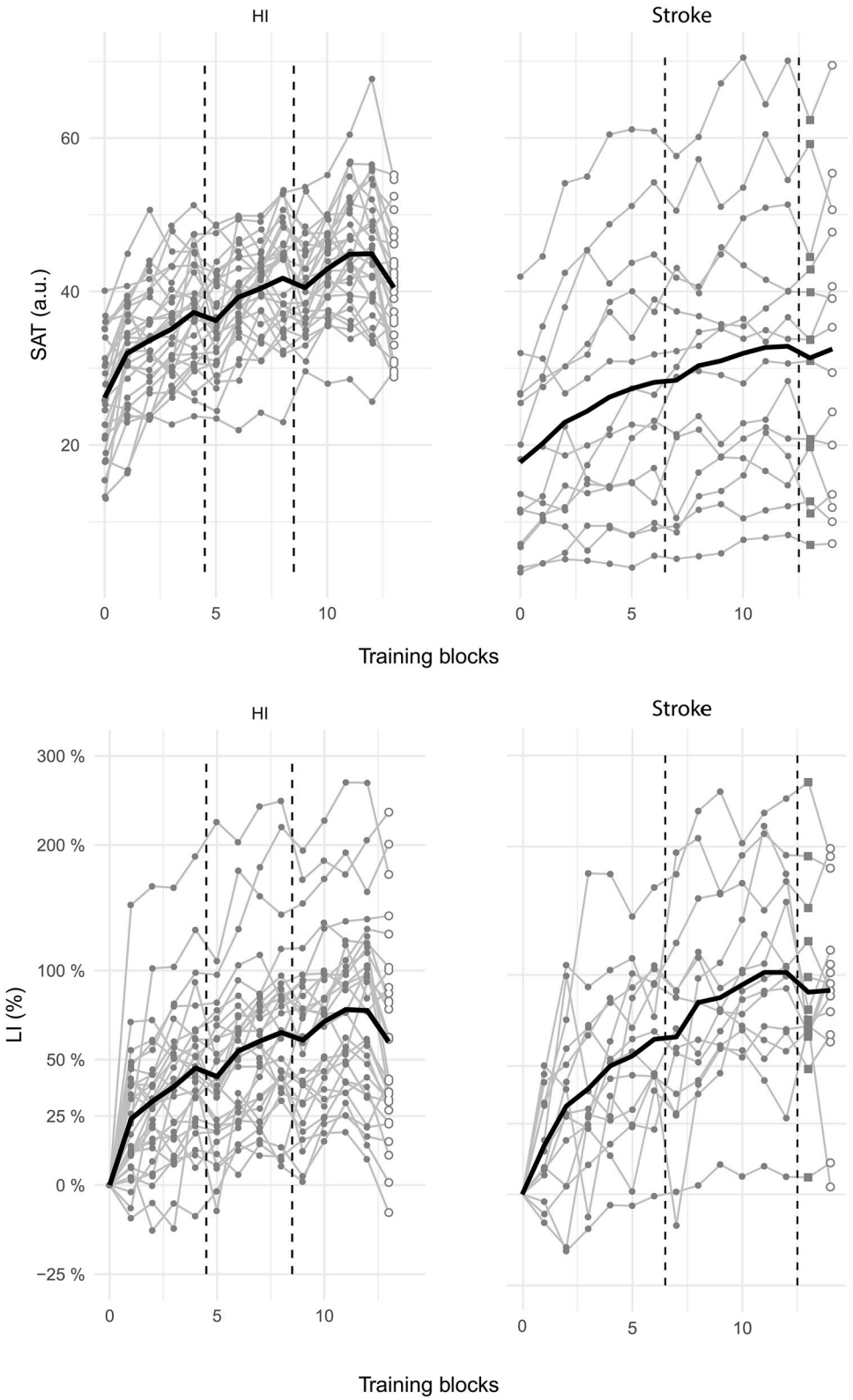

Fig. 3 SAT CIRCUIT changes. *x* axis: number of CIRCUIT blocks (baseline: 120 s, training: 3600 s, retention: 300 s, generalisation: 300 s), the vertical dotted lines separate D1, D2, and D3. Upper panel: the speed/accuracy trade-off (SAT) changes are expressed as Performance Index (PI) in arbitrary units (a.u.) for the 30 young healthy individuals (HI, left) using their non-dominant left UL and 14 acute stroke patients (right) using their paretic UL. Light grey: individual data; black: group means; white dots: generalisation block. Lower panel: SAT expressed in LI as percentage change from Baseline. Note that the experimental protocol (e.g., blocks/day) was different between the HI and patient groups. Despite this difference, both the SAT evolution in stroke patients and in HI shows continuous improvement, with a slight drop on the next day, and a large generalisation of SAT performance on an untrained circuit

or left parietal cortex impairs sensorimotor adaptation [37, 38], a stroke involving some crucial part of the brain may theoretically disturb motor skill learning. This has not been formally demonstrated [13], but some aspects of motor skill learning might be transiently impaired during the first post-stroke days. This would explain why some

Table 3 Associations between SAT, BBT, GF, and EASY

Pair	*r*	CI95	*p* Value
LI–BBT	0.26	[−0.32 to 0.69]	0.38
LI–GF	0.00	[−0.53 to 0.53]	1.00
LI–EASY	0.23	[−0.34 to 0.68]	0.43
BBT–GF	0.54	[0.01 to 0.83]	0.05
BBT–EASY	0.27	[−0.3 to 0.7]	0.35
GF–EASY	0.27	[−0.31 to 0.7]	0.36

Pearson's correlation coefficient for each pair of variables

CI95 limits of the 95% confidence interval, *LI* Learning Index, *BBT* Box and Blocks Test, *GF* whole-hand grip force, *EASY* motor task

patients appear to be "poor learners". Our small sample size precludes further analysis.

Therapeutic implications

Training with CIRCUIT resulted in large improvements, suggesting that (sub)acute stroke patients achieved motor skill learning with their paretic UL. A more restrictive interpretation would be that training with a complex SAT task led to generalised within- and between-session improvements in motor recovery. Nevertheless, whether involving motor skill learning or not, practicing complex SAT tasks such as CIRCUIT may be important to neurorehabilitation.

Limitations

First, the sample size was small and relatively heterogeneous (e.g., one haemorrhagic stroke, four patients with paresis of the non-dominant UL, etc.). As such, conclusions may not be applicable to all stroke patients. Replication in studies with a larger sample size would be appropriate. Second, stroke patients suffered mild-to-moderate motor impairment. Results might be different in patients with severe hemiparesis. Third, it would be interesting to screen for a correlation between motor skill learning and the timing of inclusion after stroke onset.

Conclusions

Practicing the complex CIRCUIT task with the paretic UL for 3 consecutive days led to consistent within-session, between-session, and generalisation of SAT improvements in acute stroke patients which were very similar to motor skill learning, retention, and generalisation in young HI using their non-dominant UL. These improvements contrasted with more heterogeneous changes in other motor outcome measures in stroke patients. In summary, on top of the well-described spontaneous recovery, acute stroke patients achieved motor skill learning and retention for a novel task.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical standards All procedures performed in this study were approved by the local Ethics Committee (CHU UCL Namur Mont-Godinne) and were in accordance with the 1964 Helsinki declaration and its later amendments.

Informed consent All patients (or the next of kin, on the behalf of the patient) provided a written informed consent prior to participating in the study.

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