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**Assessment and recovery of visually guided reaching deficits
following cerebellar stroke.**

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Abstract:

The cerebellum is known to play an important role in the coordination and timing of limb movements. The present study focused on how reach kinematics are affected by cerebellar lesions to quantify both the presence of motor impairment, and recovery of motor function over time. In the current study, 12 patients with isolated cerebellar stroke completed clinical measures of cognitive and motor function, as well as a visually guided reaching (VGR) task using the Kinarm exoskeleton at baseline (~2 weeks), as well as 6, 12, and 24-weeks post-stroke. During the VGR task, patients made unassisted reaches with visual feedback from a central ‘start’ position to one of eight targets arranged in a circle. At baseline, 6/12 patients were impaired across several parameters of the VGR task compared to a Kinarm normative sample (n=307), revealing deficits in both feed-forward and feedback control. The only clinical measures that consistently demonstrated impairment were the Purdue Pegboard Task (PPT; 9/12 patients) and the Montreal Cognitive Assessment (6/11 patients). Overall, patients who were impaired at baseline showed significant recovery by the 24-week follow-up for both VGR and the PPT. A lesion overlap analysis indicated that the regions most commonly damaged in 5/12 patients (42% overlap) were lobule IX and Crus II of the right cerebellum. A lesion subtraction analysis comparing patients who were impaired (n=6) vs. unimpaired (n=6) on the VGR task at baseline showed that the region most commonly damaged in impaired patients was lobule VIII of the right cerebellum (40% overlap). Our results lend further support to the notion that the cerebellum is involved in both feedforward and feedback control during reaching, and that cerebellar patients tend to recover relatively quickly overall. In addition, we argue that future research should study the effects of cerebellar damage on visuomotor control from a perception-action theoretical framework to better understand how the cerebellum works with the dorsal stream to control visually guided action.

Keywords: visuomotor control, cerebellum, limb ataxia, optic ataxia, reaching, dorsal stream, recovery

Introduction:

Over 30 years ago, Goodale, Milner, and their colleagues published the first in series of influential papers in which they outlined what is now commonly referred to as the “perception-action model” (Goodale & Milner, 1992; Goodale, Milner, Jakobson, & Carey, 1991).

Specifically, they suggested that the ventral visual stream – which extends from V1 to the inferotemporal cortex – was specialized for conscious visual perception. In contrast, they argued that the dorsal visual stream – which extends from V1 to the dorsal posterior parietal cortex (PPC) – was involved in the real-time control of visually guided actions (Goodale & Milner, 1992; Milner & Goodale, 2006). This model has had a tremendous impact on the study of visuomotor control, and vision science more generally, receiving thousands of citations, and appearing in many introductory textbooks on topics ranging from perception and cognition to brain and behavior and neuropsychology.

Although a great deal of research supports the importance of the dorsal visual stream in both the programming (i.e., feed-forward) and online (i.e., feedback) control of visually guided reaching and grasping (e.g., Astafiev et al., 2003; Connolly, Andersen, & Goodale, 2003; Culham, Cavina-Pratesi, & Singhal, 2006; Culham et al., 2003; Desmurget et al., 1999; Goodale, Jakobson, & Keillor, 1994; Goodale & Milner, 2004; Grea et al., 2002; Jakobson, Archibald, Carey, & Goodale, 1991; James, Culham, Humphrey, Milner, & Goodale, 2003; Jeannerod, Decety, & Michel, 1994; Milner, Dijkerman, McIntosh, Rossetti, & Pisella, 2003; Milner et al., 2001; Milner & Goodale, 2008; Perenin & Vighetto, 1988; Pisella et al., 2000; Striemer, Chouinard, & Goodale, 2011), the role of subcortical motor structures that both send outputs to, and receive inputs from the dorsal visual stream, have not been extensively studied from a similar perception-action theoretical framework. One prime example of this is the cerebellum.

The potential importance of the interaction between the dorsal stream and the cerebellum was acknowledged by Milner and Goodale in their now classic book, *The Visual Brain in Action* (first published in 1995; Milner & Goodale, 2006).

“These pontine nuclei are closely linked with the cerebellum and pontocerebellar circuits such as these have been implicated in the subcortical organization of visuomotor skills (Glickstein and May 1982), a view which would reinforce the suggestion that the dorsal stream may provide a modulatory influence (perhaps by virtue of more refined visual coding) on the activity of subcortical visuomotor networks. The extent of these corticopontine projections from dorsal-stream areas contrasts strikingly with the apparently complete absence of such projections from the inferotemporal cortex (Glickstein et al. 1980, 1985; Baizer et al. 1993; Schmahmann and Pandya 1993) (page 57).”

The cerebellum is known to play a critical role in controlling gait and balance, motor learning, as well as the timing and coordination of limb movements (e.g., Bodranghien et al., 2016; Glickstein, Strata, & Voogd, 2009; Glickstein, Sultan, & Voogd, 2011; Holmes, 1917). In addition, a growing body of research over the past 30 years has suggested that the cerebellum also plays a role in a variety of non-motor functions including language, working memory, executive control, emotion, motion perception, attention, and spatial processing (e.g., Adamaszek et al., 2017; Baier, Dieterich, Stoeter, Birklein, & Muller, 2010; Baumann et al., 2015; Baumann & Mattingley, 2022; Bellebaum & Daum, 2007; Craig, Morrill, Anderson, Danckert, & Striemer, 2021; Marvel & Desmond, 2010; Nankoo, Madan, Medina, Makepeace, & Striemer, 2021; Peterburs & Desmond, 2016; Schmahmann, Guell, Stoodley, & Halko, 2019; Schmahmann & Sherman, 1998; Stoodley, MacMore, Makris, Sherman, & Schmahmann, 2016; Stoodley & Schmahmann, 2009; Stoodley & Stein, 2011; Striemer, Cantelmi, Cusimano, Danckert, & Schweizer, 2015; Striemer, Chouinard, Goodale, & de Ribaupierre, 2015; Townsend et al., 1999; Verbitsky, Anderson, Danckert, Dukelow, & Striemer, 2023).

The cerebellum’s role in such a vast array of motor and non-motor functions is accomplished through its multitude of connections with a variety of cortical and subcortical

networks (for reviews see Buckner, 2013; Strick, Dum, & Fiez, 2009; Voogd, Schraa-Tam, van der Geest, & De Zeeuw, 2012). Of particular importance to the current study is the rich set of anatomical connections between the cerebellum and regions that are in the dorsal visual stream (i.e., the dorsal PPC), or structures that are known to project to the dorsal visual stream, such as the superior colliculus (SC) and the motion sensitive area MT (e.g., Kaas & Lyon, 2007; Lyon, Nassi, & Callaway, 2010). Specifically, in mammals, the SC, area MT, the superior temporal sulcus (STS), as well as the intraparietal sulcus (IPS) and the parieto-occipital sulcus, send visual inputs to contralateral posterior-inferior cerebellar regions (i.e., dorsal paraflocculus, uvula, paramedian lobe, Crus II) via projections through the pons (Glickstein, 2000; Glickstein et al., 1980; Glickstein & Doron, 2008; Glickstein et al., 1994; Mower, Gibson, & Glickstein, 1979; Mower, Gibson, Robinson, Stein, & Glickstein, 1980; Stein & Glickstein, 1992; Voogd et al., 2012). In contrast, there are very few visual projections from V1, or other ventral stream structures, to the cerebellum (Glickstein, 2000; Glickstein et al., 1980; Glickstein et al., 1994; Stein & Glickstein, 1992).

In humans, functional connectivity MRI studies have demonstrated that extrastriate visual areas like MT, and dorsal stream regions in the PPC, are functionally connected to regions in the posterior-inferior cerebellum, whereas early visual areas such as V1 are not (Buckner, 2013; Buckner, Krienen, Castellanos, Diaz, & Yeo, 2011). In addition, recent fMRI work has demonstrated that regions within each cerebellar hemisphere contain a visual map of ipsilateral space (van Es, van der Zwaag, & Knapen, 2019). One of these maps, located in lobule VIIIb (i.e., the paramedian lobe), is the same region that receives visual inputs from area MT and dorsal stream regions (Glickstein, 2000; Glickstein et al., 1994). Thus, the cerebellum is well positioned to assist the dorsal stream in controlling visually guided actions in the relative absence

of visual input from ventral stream structures (for reviews see Glickstein, 2000; Stein & Glickstein, 1992). One illustrative example of this is a study by Striemer and colleagues (Striemer, Enns, & Whitwell, 2019) in which a patient who was cortically blind to static visual stimuli following bilateral lesions to V1 and ventral temporal cortex, was nevertheless still able to adapt to optically displacing prisms – a task which is known to rely heavily on the cerebellum (e.g., Martin, Keating, Goodkin, Bastian, & Thach, 1996; Weiner, Hallett, & Funkenstein, 1983; Werner, Bock, Gizewski, Schoch, & Timmann, 2010).

Degeneration or damage to the cerebellum results in cerebellar ataxia which is characterized by deficits in posture, balance, and gait, as well as problems coordinating movements of the limbs (for a review see Bodranghien et al., 2016). In the case of limb control, three of the most common symptoms that are apparent during neurological examination are slowness of movement, intention tremor (tremoring during movement), and dysmetria – or the overshooting or undershooting of a target location (Bodranghien et al., 2016).

Although deficits in limb control are apparent at the bedside in patients with cerebellar damage, only a handful of studies have examined the kinematics of visually guided actions in these patients. In particular, a number of previous studies have demonstrated that cerebellar damage can disrupt predictive grip force when lifting objects (Brandauer et al., 2008; Kuper, Brandauer, et al., 2011; Kuper, Hermsdorfer, et al., 2011 ; Nowak, Hermsdorfer, Marquardt, & Fuchs, 2002; Nowak, Timmann, & Hermsdorfer, 2007; Rost, Nowak, Timmann, & Hermsdorfer, 2005). In contrast, only a few studies have examined the kinematics of visually guided reaching and grasping following cerebellar damage. With the limited data available, it appears that cerebellar damage can disrupt both the transport (i.e., lower peak velocity) and grasp (i.e., larger grip apertures) components during visually guided grasping (Brandauer et al., 2008; Kuper,

Brandauer, et al., 2011; Kuper, Hermsdorfer, et al., 2011; M. K. Rand, Shimansky, Stelmach, Bracha, & Bloedel, 2000; Zackowski, Thach, & Bastian, 2002). In addition, during visually guided reaching, cerebellar damage can result in longer movement times, increased curvature in reach trajectories, and overshooting or undershooting the target (i.e., dysmetria), particularly during faster movements (Day, Thompson, Harding, & Marsden, 1998; Konczak et al., 2010; Topka, Konczak, & Dichgans, 1998).

Of particular relevance to the current investigation, even fewer studies have examined the recovery of reach kinematics over time in patients with cerebellar injury. In one study of patients with cerebellar stroke, Konczak and colleagues (2010) found that lesions of the cerebellar cortex in the paravermal regions of lobules IV and V, and lesions affecting the deep cerebellar nuclei (dentate and interposed), were associated with longer movement times, lower peak velocities, and increased curvature in reach trajectories. In addition, their data revealed that motor recovery was relatively quick, with the majority of gains in upper limb function occurring early, within 3-4 weeks (on average) post-stroke (Konczak et al., 2010).

Given the small number of studies that have examined the assessment and recovery of reach kinematics following cerebellar injury, additional research is needed. In the current study, we carried out a retrospective analysis of visually guided reaching performance in a group of patients with isolated cerebellar stroke ($N = 12$). Patients completed a visually guided reaching task using the Kinarm Exoskeleton (Kinarm, Kingston, Ontario), and several clinical measures of cognitive and motor function at baseline (~2 weeks post-stroke), as well as at 6-, 12-, and 24-weeks post-stroke. If the cerebellum is involved in both the programming (i.e., feed-forward) and online (i.e., feedback) control of visually guided reaching (Konczak et al., 2010), then we should see evidence of this in terms of slower movement speeds and increased path curvature (i.e., feed-

forward), as well as longer overall movement times and an increased reliance on visual feedback in the later stages of the movement (i.e., feedback). In addition, based on previous research (Konczak et al., 2010), these deficits should recover relatively quickly, within the first 1-2 months post-stroke.

Methods

Participants

The current study involved a retrospective analysis of previously collected data from individuals who experienced isolated cerebellar stroke and were recruited from the Foothills Hospital in Calgary, Alberta, Canada. Inclusion criteria were: Patients had to be age 18+ with an isolated cerebellar stroke (as confirmed by neuroimaging) and had to be able to follow task instructions. Exclusion criteria were: A previous history of stroke (prior to their current stroke) or other neurological disease, orthopedic injuries to the upper extremities at the time of testing, apraxia, or significant cognitive impairment, as indicated by a score of ≤ 19 on the Montreal Cognitive Assessment (MoCA). A total of 12 patients met our inclusion criteria which consisted of 92% males ($n=11$), with an average age of 51.83 years ($SD = 13.07$) that ranged from 31 to 68 years. Ten of the patients were right-handed, while the rest were left-handed. Out of the 12 patients, two had a left cerebellar stroke, one had bilateral lesions, and the rest ($n = 9$) had a right cerebellar stroke.

The initial clinical assessment, and the baseline visually guided reaching task (see below for details) for each patient, were carried out an average of 8 days post-stroke ($SD = 7.85$ days, ranging from 2 to 32 days). Additionally, patients completed the same clinical assessments and the visually guided reaching task at 6-, 12-, and 24-weeks post-stroke to examine recovery. A

description of the patient characteristics, and scores for the baseline clinical assessments, are provided in Table 1. All participants provided written informed consent, and the study was approved by the MacEwan University Research Ethics Board and Conjoint Health Research Ethics Board of the University of Calgary.

-- Insert Table 1 here --

Table 1: Demographic data and clinical data for the baseline assessment. **Bolded and underlined values indicate impaired performance.** The letter preceding the patient ID indicates whether the patient has a left (L-), right (R-), or bilateral (B) cerebellar lesion. N/A suggests that there is no patient data for the specific measure. M = Male; F = Female; L = Left arm; R = Right arm; CC = Cubic centimeter; MoCA = Montreal Cognitive Assessment; FIM = Functional Independent Measure; CMSA = Chedoke McMaster Stroke Assessment; PPT = Purdue Pegboard Test; TLT = Thumb Localization Test; *SD* = Standard deviation. Damage in the left deep cerebellar nuclei. **Damage in the right deep cerebellar nuclei.

Patient ID	Age	Sex	Handedness	Days post stroke	Lesion side	Lesion volume (CC)	MoCA	FIM	CMSA	PPT (L; R)	TLT (L; R)
R-905	37	M	R	7	R	3.68	<u>25</u>	89	7	13; <u>3.5</u>	0; 0
R-1037	63	M	R	13	R	22.63	<u>23</u>	83	7	10; <u>7.5</u>	<u>1</u> ; 0
R-1062	68	M	R	10	R	2.59	<u>20</u>	122	7	<u>9.5</u> ; <u>9.5</u>	0; 0
R-1071	58	M	R	7	R	0.23	<u>23</u>	101	7	10.5; 11.5	0; 0
B-1095	64	M	R	32	B*	34.33	<u>22</u>	113	7	10.5; <u>9</u>	0; 0
R-1299	31	M	L	2	R	3.11	<u>24</u>	126	7	13.5; 14	<u>1</u> ; 0
R-1558	59	M	L	5	R	10.36	30	126	7	14; <u>9.5</u>	0; <u>1</u>
L-1832	58	M	R	5	L	2.27	26	113	6	<u>4.5</u> ; <u>7</u>	<u>1</u> ; 0
R-1945	54	M	R	7	R	3.09	26	95	7	11.5; <u>4.5</u>	0; 0
L-3163	58	M	R	7	L	0.44	27	109	6	<u>8</u> ; 12	0; 0
R-3185	39	F	R	2	R**	10.03	28	112	7	15; <u>9</u>	0; 0
R-3334	33	M	R	4	R	2.89	N/A	124	7	13.5; 14.5	0; 0
<i>Mean</i>	51.83			8.42		7.97	24.91			-1.58; -2.76	
<i>SD</i>	13.07			8.05		10.40	2.88			1.56; 2.09	

Patient data from the Kinarm visually guided reaching task was compared to a normative sample included with the Kinarm Standard Tests (Kinarm, Kingston, Ontario; see description of the Kinarm task below). For the clinical measures, unless otherwise noted, we compared the patient data to established norms for each test.

Clinical measures

The Montreal Cognitive Assessment

The Montreal Cognitive Assessment (MoCA) is a widely used cognitive screening tool that measures an individual's basic cognitive functions, including visuospatial/executive function, episodic memory, attention, language, naming, and orientation (Nasreddine et al., 2005). The MoCA is highly sensitive to mild cognitive impairment as well as dementia (Carson, Leach, & Murphy, 2018; Nasreddine et al., 2005). The MoCA is scored out of 30 points, and a score of less than 26 indicates a mild cognitive impairment. Higher scores reflected better performance.

The Chedoke-McMaster Stroke Assessment

The Chedoke-McMaster Stroke Assessment (CMSA) is a two-part measure used by rehabilitation personnel to assess the impairment and activity of an individual following a stroke (Gowland et al., 1993). For this study, we only used the arm impairment portion of the CMSA. This determined the presence and severity of physical impairments to classify patients when planning and selecting interventions and evaluating their effectiveness. It consisted of six dimensions, each measured on a 7-point scale. These dimensions included shoulder pain,

postural control, the arm, the hand, the leg, and the foot. Scores for each dimension were determined by the quality of one's movement, wherein 1 indicated severe motor impairment and 7 indicated normal movement. Our data only measured the recovery stage of the patients' arms, and hence, the maximum score for CMSA was 7.

The Functional Independence Measure

The Functional Independent Measure (FIM) is used for evaluating the functional status of patients throughout their rehabilitation (Chumney et al., 2010). It is an 18-item screening tool that assesses the patient's degree of disability and burden of care. Thirteen items evaluated the disability in motor functions, while five items assessed any disability in cognitive functions. Each item was rated on a 7-point scale, with 1 indicating a total assist while 7 is complete independence (Chumney et al., 2010). Ratings were accumulated across items and used to determine the degree of dependency that the patients needed to accomplish basic, routine daily tasks. The total score for FIM ranged from 18 to 126, with a maximum score of 35 in FIM's cognitive functions and maximum of 91 in the motor functions (Chumney et al., 2010).

Thumb Localization Test

The Thumb Localization Test (TLT) measured the patient's proprioception, which is critical for movement control (Otaka et al., 2020; D. Rand, Weiss, & Gottlieb, 1999). In this test, the examiner moved the participant's affected arm to 4 different locations in space, and each time, the participant located and grasped their affected thumb with their less-affected hand while their eyes were closed. The scoring for each of the four attempts was based on the estimated distance

that they missed their affected thumb in space and discriminated between no (precisely grasps the thumb), mild (just misses to grasp the thumb and immediately corrects), moderate (able to find the arm and uses the arm to find the thumb), or severe proprioception deficits (cannot locate thumb).

The Purdue Pegboard Test

The Purdue Pegboard Test (PPT; LaFayette Instrument Company, LaFayette, IN) is a test that measures the fine motor dexterity of an individual's hands, fingers, and arms (Desrosiers, Hébert, Bravo, & Dutil, 1995; Tiffin & Asher, 1948). During the test the participant had to place as many pegs as possible into the holes on the board in a 30 second period. Each hand was tested separately. A short practice period was completed prior to the main test to familiarize patients with the task. Patient performance was compared to the normative data from Yeudall and colleagues (Yeudall, Fromm, Reddon, & Stefanyk, 1986) and Desrosiers and colleagues (Desrosiers et al., 1995).

Kinarm: Visually guided reaching task

Robotic assessments of the visually guided reaching (VGR) task were conducted using the Kinarm Exoskeleton (Kinarm, Kingston, ON, Canada; <https://kinarm.com/>) which allows the tracking of arm kinematics with high spatial and temporal precision. Patients were assessed at baseline (within ~2 weeks of their stroke), as well as 6-, 12-, and 24-weeks post-stroke to examine recovery over time.

Comparison to normative data.

When a patient completed the VGR task, a report was automatically generated at the end of the session by the Dexterit-E program (Version 3.7; Kinarm, Kingston, Ontario), which provided the performance scores for each of the movement parameters noted below. Each parameter score was converted to a Z-score, based on normative data included as part of the Kinarm Standard Tests. The normative dataset consisted of 307 healthy adults between the ages of 18 and 84 (152 males, 155 females; 36 left-handed) who were tested using the same 8-target adult version of the VGR task with their left and right arms. The Z-scores for each parameter controlled for the effects of age, sex, and handedness (Dexterit-E, Version 3.7, Kinarm, Kingston, ON, Canada; for details see <https://kinarm.com/support/user-guides-documentation>). For all parameters described below, a Z-score value of greater than or equal to ± 1.96 was used to identify impairments on a participant's performance on that specific parameter.

Visually guided reaching task procedure.

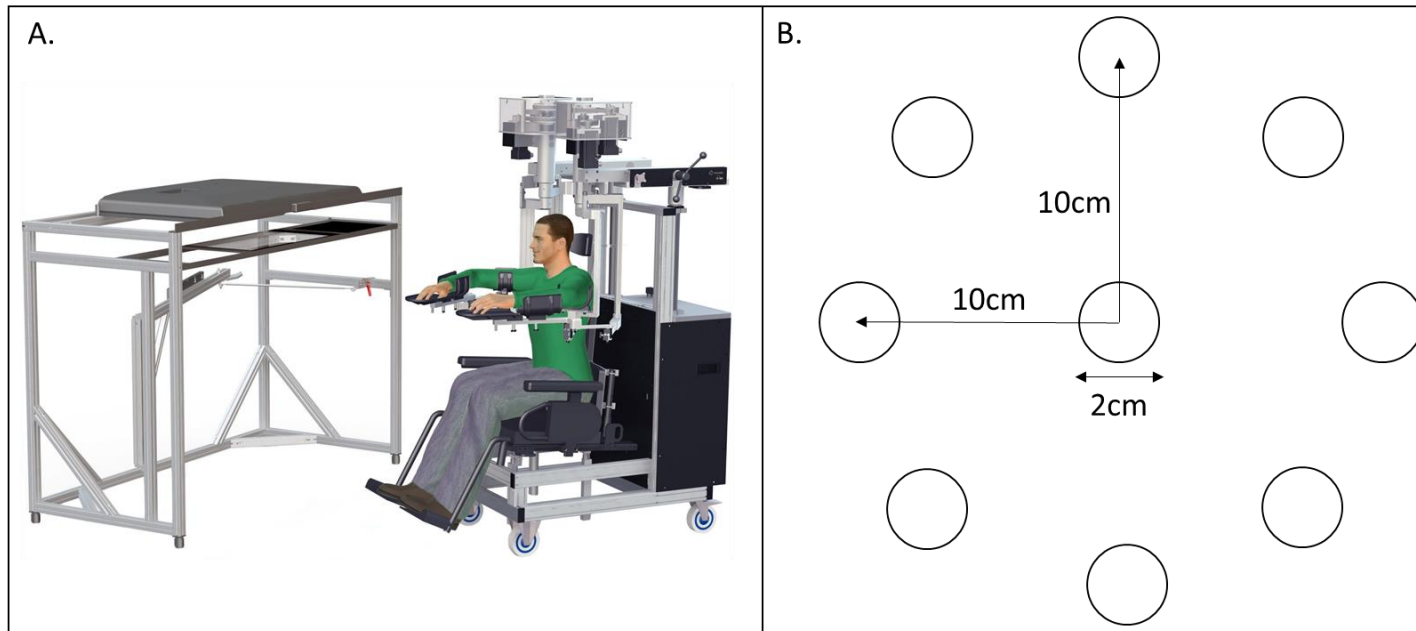
The VGR task was carried out using the procedures described in Coderre and colleagues (Coderre et al., 2010). To summarize, participants were seated in the Kinarm chair with their arms abducted into the horizontal plane, and the angle of abduction ($\sim 80^\circ$) was chosen to ensure that the arm, forearm, and hands were in the same plane as the shoulder. During the task, the Kinarm provided gravitational support of the arms, forearms, and hands, and only allowed horizontal movements, involving both flexion and extension of the elbow and shoulders (Figure 1). It is important to note that despite the gravitational support provided by the robotic device, it did not assist the participants in making movements and completing the task. A virtual reality

system displayed visual targets appearing in the same plane as the arm, and direct vision of the participant's arm was occluded. Hand position feedback was provided by a computerized representation of the tip of the participant's index finger (i.e., small white circle, 0.4 cm radius). Both the left and right arms were tested during separate blocks of trials within each testing session.

As described in Coderre and colleagues (2010), during the VGR task the participant's goal was to make unassisted reaches from a central circular "start" position (2cm diameter) to one of eight peripheral targets (2cm diameter; Figure 1). The targets were located at a distance of 10cm and were distributed uniformly around the circumference of a circle (Coderre et al., 2010). The center target was selected closer to the center of the arm's workspace (30° of shoulder flexion and 90° of elbow flexion). During a single trial, participants held the tip of their left or right index finger within the central target for 1250 to 1750 milliseconds. Then, once one of the peripheral targets was illuminated, participants were instructed to move as quickly and accurately as possible to the target. Participants were given 3000 milliseconds to complete the reach. Participants completed eight reaches to each of the eight target positions (presented in a random order) for a total of 64 reaches per arm, per testing session.

-- insert Figure 1 here—

Figure 1. A) Depiction of the Kinarm Exoskeleton Robot adapted from Wood et al. (2021). B) Layout of the Kinarm Visually Guided Reaching (VGR) Task. Participants begin by moving to a central start position. Participants were then asked to reach, as quickly and accurately as possible, to one of eight circular targets (each 2cm in diameter) arranged in a circle at a distance of 10cm from the central start position.



Movement parameters.

The Kinarm's Dexterit-E Software Version 3.7 (Kinarm, Kingston, ON, Canada) was used to automatically calculate a number of different movement parameters in order to characterize the participants' performance in the task compared to a large normative sample included with the software package. As described in Coderre and colleagues (2010), these parameters are broadly categorized into five aspects of sensorimotor control: 1) upper-limb postural control, 2) reaction time, 3) feed-forward control, 4) feedback control, and 5) total movement metrics. However, for the present study, we only focused on three categories of sensorimotor control: feed-forward control, feedback control, and total movement metrics, as these were the categories that demonstrated consistent impairment across a number of patients in our sample. The descriptions of the following task parameters were taken from the Kinarm Standard Tests Summary for Dexterit-E 3.7 (Kinarm, Kingston, Canada), as well as Coderre and colleagues (2010).

Feed-forward control.

The parameters in this category characterized the initial phase of the movement which was the time period from the onset of the movement to the first minimum hand speed, and the first minimum hand speed was the first local minimum after the first maximum hand speed. As described in Coderre and colleagues (2010), movement onset was determined algorithmically for each participant by: 1) calculating a lower speed threshold by taking the median hand speed across all trials for the 500ms prior to reach target onset, while the hand was still resting in the central target 'start' position as the hand tends to move very slightly at rest; and 2) calculating the upper speed threshold which was the 95th percentile for hand speed during this same time

period. The algorithm first determined when the hand exited the central ‘start’ position after the illumination of the reach target, then using this time point to work backwards, the algorithm determined reach onset as the first instance of either: 1) a local minimum hand speed that was below the upper speed threshold or 2) when hand speed fell below the lower speed threshold.

Initial direction angle (IDA) measured the angular deviation between (a) a straight line from the hand position at movement onset to the peripheral target and (b) a vector from the hand position at movement onset to the hand position after the initial phase of the movement.

Initial distance ratio (IDR) measured the ratio of (a) the distance the hand traveled during the participant’s initial movement to (b) the distance the hand traveled between movement onset and offset.

Feedback control.

This attribute characterizes how participants adjusted their reach in the later phase of the movement.

Speed maxima count (SMC) measured the smoothness of the movement, and the number of corrective movements made, by counting the number of hand speed peaks between the onset and offset of movement.

Total movement metrics.

In addition to analyzing measures of feed-forward and feedback control, we also examined *movement time (MT)* which was defined as the total time that elapsed from movement onset to termination.

Finally, we also examined *Task score* which measured the participant's overall performance for the VGR task. As described in the Kinarm Standard Tests manual (Kinarm.com), Task score provides a global measure of how far the participant's performance was from normal performance by converting all VGR task parameters scores into Z-scores (based on the Kinarm normative data). All parameters are given equal weighting except in circumstances where an unequal number of "successful trials" were completed for different parameters. The parameter Z-scores are initially transformed into a one-sided score, where a score of 0 indicates average performance, and higher scores reflect poorer performance. The root sum-of-squares (RSS) values are calculated using all the transformed parameter Z-scores, and then converted to a normal distribution by Box-Cox transformations. Thus, Task scores can be interpreted similarly to a Z-score where a Task score ≥ 1.96 represents impaired overall performance on the VGR task compared to the normative sample.

Lesion data

To explore the association between lesion location and behaviour, we acquired existing clinical MRI or CT scan data for each patient. Each patient's lesion was traced by Dr. Britt Anderson, an experienced behavioural neurologist, using MRIcron software

(<http://people.cas.sc.edu/rorden/mricron/index.html>). It is important to note that Dr. Anderson

was blind to the patients' performance on each measure prior to completing the lesion tracing. Each patients' anatomical scan and lesion masks were normalized into Montreal Neurological Institute (MNI) space onto a high-resolution computed tomography (CT) template using the Clinical Toolbox for SPM 12 (Rorden, Bonilha, Fridriksson, Bender, & Karnath, 2012). The normalized individual lesion masks were then combined to create a group lesion mask in MRIcron, which was overlaid onto the same high-resolution CT template. In addition, to examine which cerebellar regions were associated with impaired performance on the Kinarm VGR task, we used a subtraction analysis contrasting patients who were impaired versus unimpaired on the task. For both analyses, we extracted the MNI coordinates for the regions where most patients had overlapping lesions and converted them into Talairach coordinates. In turn, these Talairach coordinates were used to localize the lesioned regions using the Talairach Daemon Atlas (<http://www.talairach.org/>).

Results

The patients' demographic information (i.e., age, sex, handedness) and scores on the clinical measures at the baseline assessment are presented in Table 1.

Clinical Measures

Baseline assessments of CMSA and FIM

Our results indicated that our patient group did not show any impairments in the CMSA or the FIM.

Baseline assessment of MoCA

For the MoCA, a score of less than 26 indicates a mild cognitive impairment (Nasreddine et al., 2005). Data from the baseline assessments suggest that neither patient with a left sided lesion (i.e., patients L-1832 and L-3163) showed any significant impairments on the MoCA.

However, five patients with right cerebellar lesions showed significant impairments in this measure at the baseline assessment (Table 1). We were not able to obtain MoCA data for patient R-3334.

Our data also indicated that the patient with bilateral lesions (i.e., B-1095) was also impaired on the MoCA at baseline (Table 1).

Baseline assessment of TLT.

In TLT, a score of 0 indicates normal performance, and 1 or greater suggests impaired performance. For those with left sided lesions, only one patient (i.e., L-1832) showed impairments in finding their left thumb, with normal performance in finding their right thumb. Patient L-3163 did not have any impairments in TLT.

Among patients with right-sided lesions, two patients (i.e., patients R-1037 and R-1299) showed impairments while trying to find their left thumb, and patient R-1558 had impairments trying to find their right thumb in the TLT. The rest of the participants with right cerebellar lesions did not have any impairment.

Similarly, bilateral patient B-1095 showed normal performance in the TLT (refer to Table 1).

Purdue Pegboard Test (PPT).

The patients' scores in the PPT were compared to respective norms depending on their specific ages and sex, wherein higher scores indicate better performance in this task (Table 1; Figure 2). The norms used in the analyses were from the data used by Yeudall and colleagues (1986) and DesRosiers and colleagues (1995), and each patient's limb performance was compared to these norms.

Baseline impairment. For those with left sided lesions, patient L-3163 showed impairments in the PPT while using left limb ($z = -3.13$), whereas patient L-1832 was impaired in both limbs (left $z = -5.47$; right $z = -3.8$).

Five patients with right sided lesions had impairments in their right limbs (i.e., patients R-905, $z = -6.89$; R-1037, $z = -3.46$; R-1558, $z = -2.13$; R-1945, $z = -5.47$; and R-3185, $z = -4.31$), whereas patient R-1062 was impaired in both limbs (left $z = -2.13$; right $z = -2.13$).

Bilateral patient B-1095 also had impairments in the PPT when using their right limb ($z = -2.47$).

Recovery of function. As mentioned, PPT was the only clinical measure included when we examined the recovery of patients' motor functions. However, it is important to note that not all individuals completed all four assessments (i.e., from baseline to 24-weeks post-stroke assessment). To examine recovery, we focused only on patients who were impaired at baseline and returned for at least one of the subsequent follow-ups (i.e., patients R-905, R-1037, R-1558, R-1945, R-1832, L-3163, L-3185). It is also important to note that three patients who were impaired at PPT (i.e., patients B-1095, R-1558, and R-3185) did not show any impairment in the

Kinarm VGR task. The red outline in each patient's symbol denotes an impairment at the PPT but not in the Kinarm VGR task.

Baseline to 6-weeks post-stroke assessments. For those who completed two time points, three individuals showed significant impairments at the initial assessment (i.e., patients L-3163, R-905, and R-3185; Figure 2). The examination of their recovery revealed that, among the patients with left cerebellar lesions, patient L-3163 was no longer impaired by the second assessment ($z = -1.13$; Figure 2).

On the other hand, for those with right cerebellar lesions, the results indicated that patient R-905 was still impaired in his right arm six weeks following the stroke; however, as depicted in Figure 2, his score decreased from the initial (i.e., $z = -6.89$) to the second assessment (i.e., $z = -5.73$). Similarly, patient R-3185's impairment in her right limb persisted until 6-weeks post-stroke. However, her score in PPT also improved significantly from the baseline ($z = -4.31$) to 6-weeks post-stroke assessment ($z = -2.14$; Figure 2).

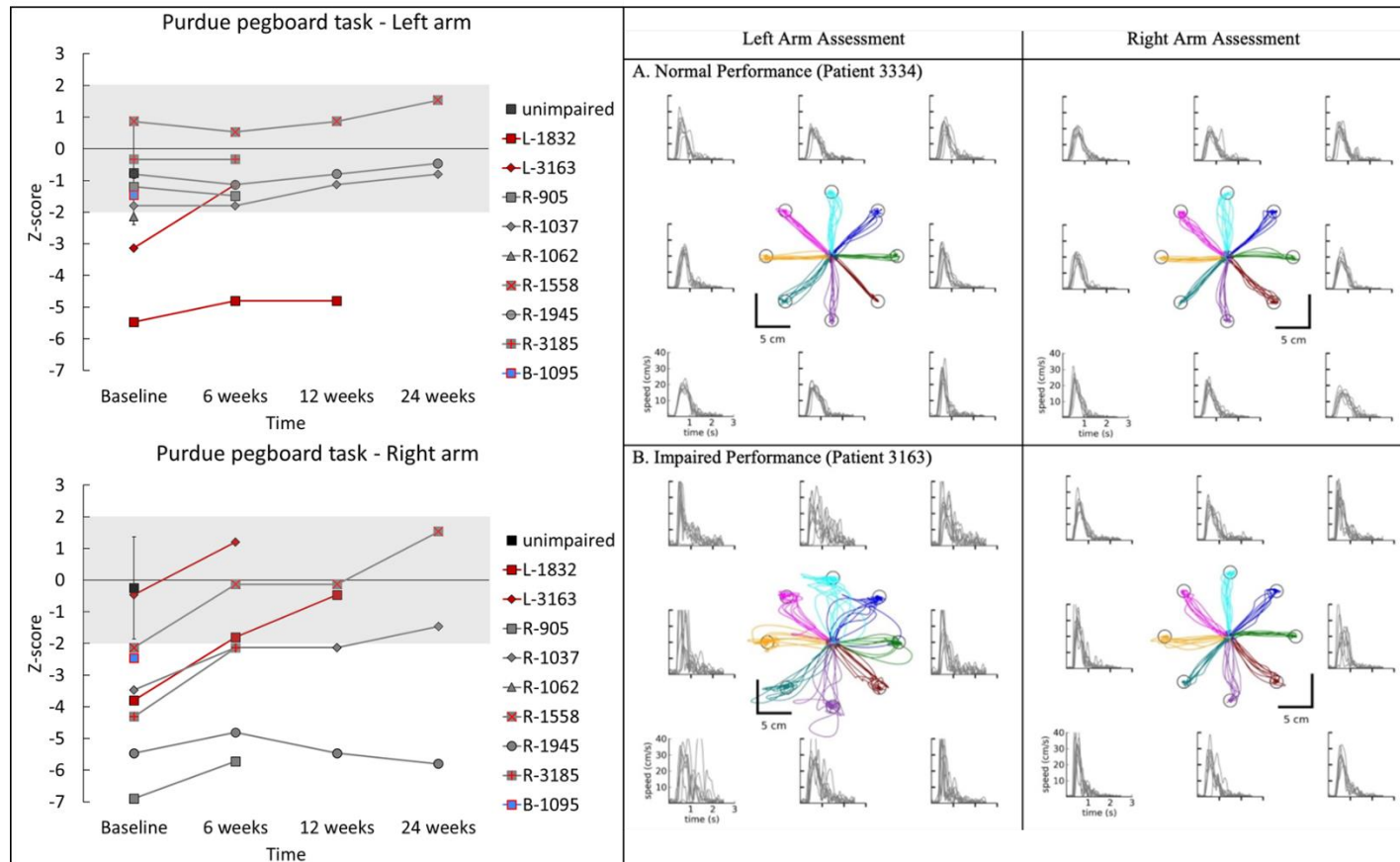
Baseline to 12-weeks post-stroke assessments. Patient L-1832, who had a left sided lesion, only completed the assessments until the third time point (Figure 2). Our findings revealed that he remained impaired in his left limb until 12 weeks after the stroke ($z = -4.8$); however, he recovered his functions in his right arm six weeks post-stroke ($z = -0.47$; Figure 2).

Baseline to 24-weeks post-stroke assessments. Three patients who were impaired at baseline returned for the 24-week follow-up (i.e., patients R-1037, R-1558, and R-1945). As can be seen in Figure 2, patient R-1037's impairment in the right limb continued to persist until 12-weeks post-stroke ($z = -2.13$), but displayed recovery of functions by 24-weeks post-stroke ($z = -1.47$). By 6-weeks post stroke, patient R-1558 no longer showed impairment in his right limb as

his Z-scores improved significantly from baseline ($z = -2.13$) to 24-weeks post-stroke assessment ($z = 1.53$). Lastly, patient R-1945 consistently showed impairment in his right limb until 24-weeks post-stroke ($z = -5.80$; Figure 2). Based on his performance, patient R-1945 was the only patient still impaired by the end of the assessments.

-- insert Figure 2 here --

Figure 2. The **left panel** depicts Z-scores for the Purdue Pegboard Task (PPT) for impaired vs. unimpaired patients for the left (top) and right (bottom) arms at baseline, as well as 6, 12, and 24-weeks post-stroke. Left cerebellar patients are depicted in red symbols whereas right cerebellar patients are depicted in grey symbols. The bilateral patient is depicted in blue. The mean and standard deviation of the unimpaired patients is depicted in black for the baseline session only. The grey shaded region denotes the range of normal performance of ± 2 Z-scores. The **right panel** depicts performance on the Kinarm Visually Guided Reaching (VGR) task for left and right arms for two representative patients. The patient at the top (R-3334) depicts normal performance on the task as indicated by the relatively straight reach trajectories, as well as the normal velocity profiles. The patient at the bottom (L-3163) is a left cerebellar patient who is impaired at the VGR task when using their left arm, but not when using their right arm. Impairment is evident in the increased curvature in their reach trajectories as well as their abnormal velocity profiles.



Kinarm: Visually guided reaching task

As mentioned previously, we only focused on the Kinarm movement parameters where several patients showed significant impairments, specifically, IDA, IDR, SMC, and MT, and the overall Task score. Impairments were defined as participant's performance falling below 95% of controls (i.e., IDR) or above 95% of controls (i.e., IDA, SMC, and MT). Specifically, a Z -score $\geq \pm 1.96$ were indicative of impairment in a patient's performance in a specific parameter.

See Figure 2 (right panel) for representative examples of impaired and unimpaired patient performance in the VGR task at the initial assessment. Here, by comparing the performance of patient R-3334 (unimpaired; Figure 2, top right panel) and patient L-3163 (impaired; Figure 2, lower right panel) using their left limbs, we can see significant differences in their reach trajectories and velocity profiles when completing the VGR task. Specifically, patient L-3163's reach trajectories showed increased curvature, longer movement times, and an increased number of speed peaks compared to normative data. In contrast, patient R-3334's performance was unimpaired relative to normative data.

During the initial assessment, a total of six patients were unimpaired in any of the parameters (i.e., patients R-1071, B-1095, R-1299, R-1558, R-3185, and R-3334). In contrast, both patients with left cerebellar stroke (i.e., patients L-1832 and L-3163) and four patients with right cerebellar stroke (i.e., patients R-905, R-1037, R-1062, and R-1945) showed significant impairments in one or more parameters, in either one or both limbs (Table 2).

To examine recovery, we only focussed on the patients who were impaired at baseline and completed at least one or more of the follow-up sessions (i.e., R-905, R-1037, R-1062, L-1832, R-1945, L-3163). Refer to Figures 3-5 for patient performance in each of the VGR task

parameters at each time point. Z-score data for each Kinarm parameter at baseline are presented in Table 2. Z-score data for each Kinarm parameter for each time point are available online as Supplementary Data.

--insert Table 2 here--

Table 2. Z-scores for each of the Kinarm movement parameters at the baseline assessment. **Bolded and underlined values indicate impaired performance at initial assessment.** The letter preceding the patient ID indicates whether the patient has a left (L-) or a right (R-) cerebellar lesion. *SD* = standard deviation; IDA = Initial direction angle; IDR = Initial distance ratio; SMC = Speed maxima count; MT = Movement time. The letter following each of the parameter labels indicates values for the left (-L) or right (-R) arm.

Patient ID	IDA-L	IDA-R	IDR-L	IDR-R	SMC-L	SMC-R	MT-L	MT-R	Task score-L	Task score-R
R-905	-1.09	<u>4.16</u>	0.20	<u>-4.26</u>	-0.41	<u>2.48</u>	1.26	<u>4.09</u>	-0.16	<u>3.79</u>
R-1037	1.00	<u>2.97</u>	<u>-2.16</u>	<u>-3.08</u>	1.15	<u>3.61</u>	<u>2.04</u>	<u>3.39</u>	1.59	<u>3.99</u>
R-1062	<u>2.21</u>	0.41	<u>-2.53</u>	-0.50	<u>2.08</u>	-0.01	<u>1.97</u>	0.60	<u>2.02</u>	-0.05
R-1071	0.01	0.31	0.55	0.23	-0.49	-0.04	0.42	0.11	-1.25	-1.08
B-1095	1.67	0.35	0.70	-0.63	-0.51	-0.62	-0.27	0.51	-0.22	-0.36
R-1299	0.56	-0.06	0.26	1.26	-1.03	-1.22	-0.34	-0.25	-0.16	-0.68
R-1558	-1.35	0.74	1.37	-0.71	-1.06	-1.04	0.24	0.67	-1.71	0.50
L-1832	<u>2.27</u>	1.67	<u>-3.01</u>	-1.66	1.65	<u>2.08</u>	<u>2.92</u>	<u>2.72</u>	<u>2.71</u>	<u>2.01</u>
R-1945	0.93	<u>2.53</u>	-0.52	<u>-2.60</u>	0.96	0.47	1.48	1.13	0.53	<u>2.50</u>
L-3163	<u>3.46</u>	1.82	<u>-2.60</u>	-1.28	<u>2.48</u>	1.69	0.43	-0.13	<u>3.38</u>	0.92
R-3185	-0.20	0.82	0.47	-0.83	-0.34	-1.32	-0.11	-1.21	-0.45	1.07
R-3334	-1.05	-0.58	-0.14	-0.38	0.18	1.34	0.27	0.90	-0.73	-0.01
<i>Mean</i>	0.70	1.26	-0.62	-1.20	0.39	0.62	0.86	1.04	0.46	1.05
<i>SD</i>	1.52	1.40	1.53	1.51	1.23	1.61	1.05	1.57	1.61	1.69

Feed-forward control (i.e., Movement programming).

To identify impairments in the patient's feed-forward control, we examined both initial direction angle (IDA) and initial distance ratio (IDR; Figure 3).

Baseline impairment for IDA. Our findings revealed that both patients with left sided lesions (i.e., patient L-1832 and L-3163) were impaired in IDA using their left limbs ($z = 2.27$ and $z = 3.46$, respectively). For those with right cerebellar lesions, patient R-1062 was impaired in his left limb ($z = 2.21$), whereas patients R-905, R-1037, and R-1945 were impaired in their right limbs ($z = 4.16$, $z = 2.97$, and $z = 2.53$, respectively; Figure 3).

Recovery of function for IDA. Of those who completed two time points, only left cerebellar patient L-3163 and right cerebellar patient R-905 were impaired in the IDA at baseline (Figure 3). For patient L-3163, his impairments in his left limb persisted until six weeks following the stroke ($z = 2.84$). Similarly, patient R-905 still showed impairments in his right limb six weeks after the stroke ($z = 2.36$), although his performance had improved from the initial assessment ($z = 4.16$).

Among the patients who completed all assessments in the study, only three patients showed impairments in IDA at baseline (L-1832, R-1037, and R-1945 in Figure 3). For the left cerebellar patient (i.e., patient L-1832), he maintained his impairment in the left limb until six weeks following the injury ($z = 2.87$) and recovered his functions by 12-weeks post-stroke ($z = 1.64$), which was maintained until the end of his assessments ($z = 1.42$; Figure 3).

For the right cerebellar patients, R-1037 showed significant recovery in his right arm with a substantial decrease in his scores. As depicted in Figure 3, he fully recovered his right limb function by 6-weeks ($z = 0.65$) and maintained his recovery by 24-weeks following the stroke (z

= -0.45). This case was similar to that of patient R-1945. He fully recovered function in his right arm by 6-weeks post-stroke ($z = 1.91$) and maintained his gains over time (Figure 3). Although his scores went up from the third ($z = 1.22$) to fourth assessment ($z = 1.72$), it was still not enough to be considered impaired (Figure 3).

-- insert Figure 3 here --

Baseline impairment for IDR. Similar to IDA, we found that both left cerebellar patients (i.e., patient L-1832 and L-3163) showed significant impairments using their left limbs ($z = -3.01$ and $z = -2.59$, respectively; Figure 3). Among the right cerebellar patients, patient R-1062 showed impairments in his left limb ($z = -2.53$), patients R-905 ($z = -4.26$) and R-1945 ($z = -2.60$) showed impairments in their right limbs, and patient R-1037 was impaired in both limbs ($z = -2.16$ for left and $z = -3.08$ right limbs, respectively; Figure 3).

Recovery of function for IDR. Similar to the results for IDA, among those who completed two assessments, left cerebellar patient L-3163 and right cerebellar patient R-905 were the only patients who showed impairments in IDR at baseline (Figure 3). Patient L-3163 still seemed to show borderline impairment in his left arm by 6-weeks post-stroke ($z = -1.93$). Additionally, patient R-905's right limb was still impaired when he came in for his second assessment, although his scores dropped by 50% from baseline (i.e., from $z = -4.26$ to $z = -2.83$; Figure 3).

For those who completed all four assessments, only patients L-1832, R-1037, and R-1945 showed impairments in IDR at baseline (Figure 3). Our findings revealed that left cerebellar patient L-1832 was consistently impaired in IDR using his left limb at baseline ($z = -3.01$) to 24 weeks after stroke ($z = -3.79$; Figure 3). On the other hand, among the right cerebellar patients, patient R-1037 was impaired in both limbs initially, but fully recovered six weeks after the stroke ($z = -1.39$ in left and $z = -0.21$ in right limb). Additionally, patient R-1945 was still impaired with his right arm when he completed the second assessment ($z = -2.03$), but our findings revealed that he regained his function 12-weeks post-stroke and was no longer impaired in his third and fourth assessments ($z = -1.53$ and $z = -1.71$, respectively; Figure 3).

Feedback control (i.e., Online control).

Baseline impairment. To examine feedback control during the VGR task, we examined SMC. The results indicated that among those with left sided lesions, patient L-3163 showed impairments in left limb ($z = 2.48$), while patient L-1832 revealed impairments in SMC using his right limb ($z = 2.08$). For those with right cerebellar lesions, patient R-1062 had impairment in his left limb ($z = 2.08$), whereas both patients R-905 and R-1037 had impairments in SMC using their right limbs ($z = 2.48$ and $z = 3.61$, respectively; Figure 4).

Recovery of function for SMC. For those patients who completed only two time points, left cerebellar patient L-3163 and right cerebellar patient R-905 were the only ones who showed impairments in feedback control at the baseline assessment. We found that patient L-3163 initially displayed impairment in the left arm and maintained this impairment in the second assessment ($z = 2.32$). In addition, he was within the range of normal performance for the right arm at the initial assessment ($z = 1.69$); however, his right hand was considered impaired at the 6-week follow-up ($z = 2.46$; Figure 4). On the other hand, patient R-905 had recovered function in his right arm six weeks following the stroke ($z = 1.49$; Figure 4).

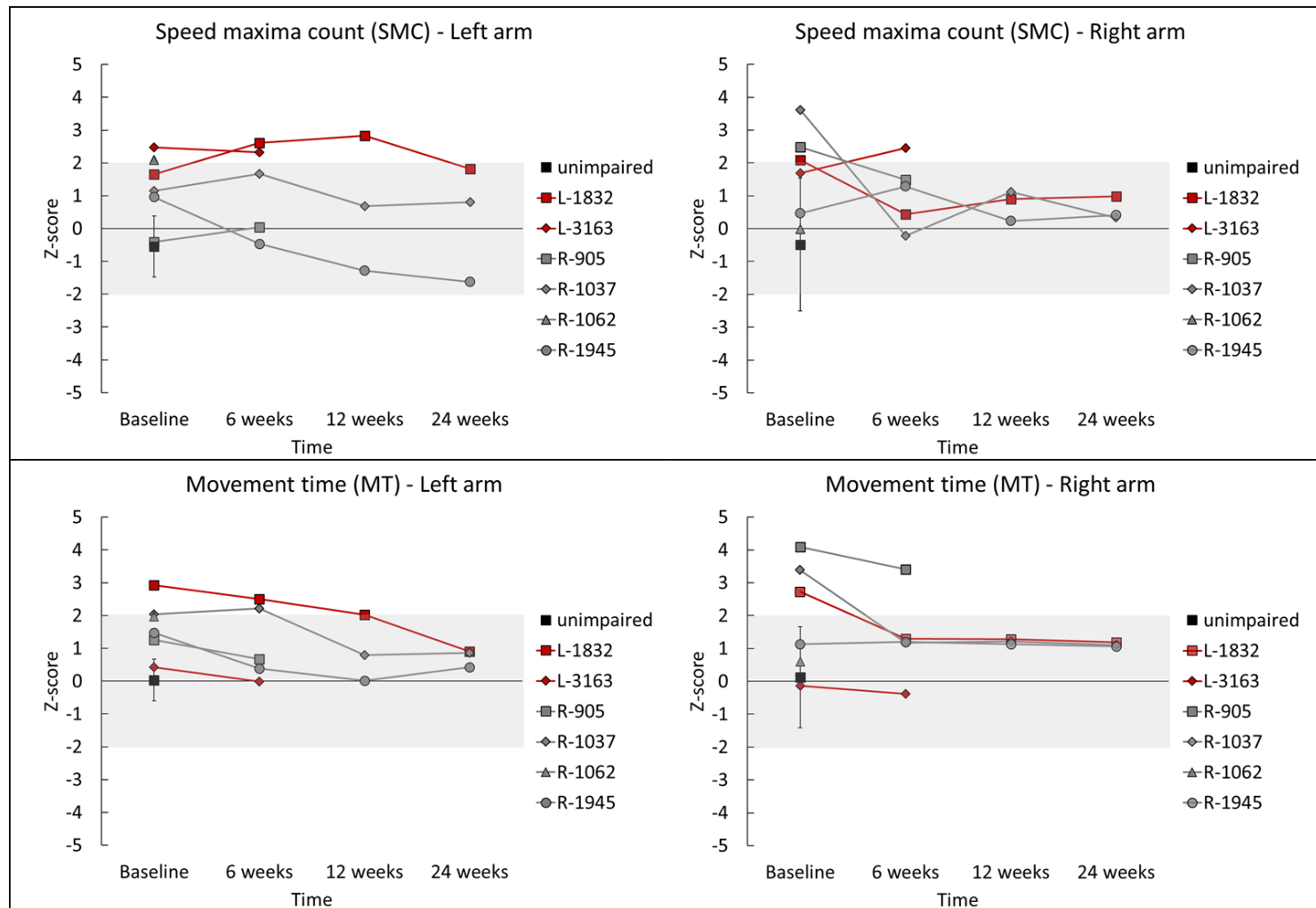
Unlike in the first two parameters, right cerebellar patient R-1945 showed normal performance for SMC (Figure 4). Left cerebellar patient L-1832 and right cerebellar patient R-1037 were the only ones who showed impairments in feedback control among those who completed all four assessments. Specifically, left cerebellar patient L-1832 regained the function in his right arm 6-weeks post-stroke ($z = 0.44$). Although his left arm was within the normal limits at baseline ($z = 1.65$), he developed an impairment in the same arm at both six weeks ($z = 2.61$) and 12 weeks ($z = 2.82$) follow-up. By 24 weeks after stroke, patient L-1832 showed

recovery in SMC for both limbs ($z = 1.82$ and $z = 0.99$ in left and right arms, respectively; Figure 4).

For right cerebellar patient R-1037, his impairment at baseline was no longer present by the second assessment (Figure 4). Specifically, his performance using his right limb significantly improved from baseline ($z = 3.61$) to last assessment ($z = 0.33$).

-- insert Figure 4 here --

Figure 4. Depicts Z-scores for speed maxima count (SMC; **top panel**) and movement time (MT; **bottom panel**) for impaired vs. unimpaired patients for the left and right arms at baseline, as well as 6, 12, and 24-weeks post-stroke. Left cerebellar patients are depicted in red symbols whereas right cerebellar patients are depicted in grey symbols. The mean and standard deviation of the unimpaired patients is depicted in black for the baseline session only. The grey shaded region denotes the range of normal performance of ± 2 Z-scores from the Kinarm normative sample. See Methods for descriptions of these measures.



Movement time (MT).

Baseline impairment for MT. Two of the six patients who displayed impairments in other parameters showed normal performance for MT (i.e., patients R-1945 and L-3163; Figure 4). However, left cerebellar patient L-1832 showed impairments in both limbs ($z = 2.92$ in left limb and $z = 2.72$ in right limb). Among those with right-sided lesions, patient R-1062 had impairment in his left limb ($z = 1.97$), patient R-905 in his right ($z = 4.09$), and patient R-1037 in both limbs ($z = 2.04$ in left and $z = 3.39$ in right limb).

Recovery of function for MT. Among the patients who only completed two time points, only right cerebellar patient R-905 showed impairment in his right arm at baseline ($z = 4.09$; Figure 4). This impairment in his right arm was maintained until six weeks after the stroke, but his scores decreased somewhat from baseline ($z = 3.41$; Figure 4).

For those who completed all assessments, left cerebellar patient L-1832 and right cerebellar patient R-1037 were the only individuals with initial impairments. Specifically, patient L-1832 was initially impaired in both arms, but his right arm recovered by six weeks post-stroke ($z = 1.30$). However, his left arm did not fully recover until he returned for his 24-week assessment ($z = 0.91$; Figure 4). Similarly, patient R-1037 showed impairments in both arms at baseline. Although his right arm recovered by six weeks ($z = 1.17$), our findings revealed that he still maintained an impairment in his left arm ($z = 2.22$). However, by the 12-week follow-up, patient R-1037 recovered his functions in both limbs. This recovery was maintained until the 24-week follow-up ($z = 0.87$ in left and $z = 1.10$ in right limb; Figure 4).

Task score.

Baseline impairments. All six patients who displayed impairments in one or more previously discussed parameters also revealed impairments in the overall task score (Figure 5). For those with left-sided lesions, patient L-3163 had impairment in his left arm ($z = 3.38$), whereas patient L-1832 had impairments in both limbs ($z = 2.71$ in left and $z = 2.01$ in right limb). All four right cerebellar patients showed impairments in task score. More specifically, our findings revealed that patient R-1062 had impairment in their left arm ($z = 2.02$), whereas the rest (i.e., patients R-905, R-1037, and R-1945) showed significant impairments in their right arm ($z = 3.79$, $z = 4.00$, and $z = 2.50$, respectively; Figure 5).

Recovery of function for task score. Left cerebellar patient L-3163 and right cerebellar patient R-905 were the only participants who were impaired in their overall task score among those who completed two time points. Patient L-3163 showed impairment in his left arm, while patient R-905 showed impairment in his right and these impairments were still present at the 6-week follow up. Although they were still impaired at the 6-week follow-up, their scores had decreased somewhat from the initial assessment ($z = 2.21$ and $z = 3.27$ for patients L-3163 and R-905, Figure 5).

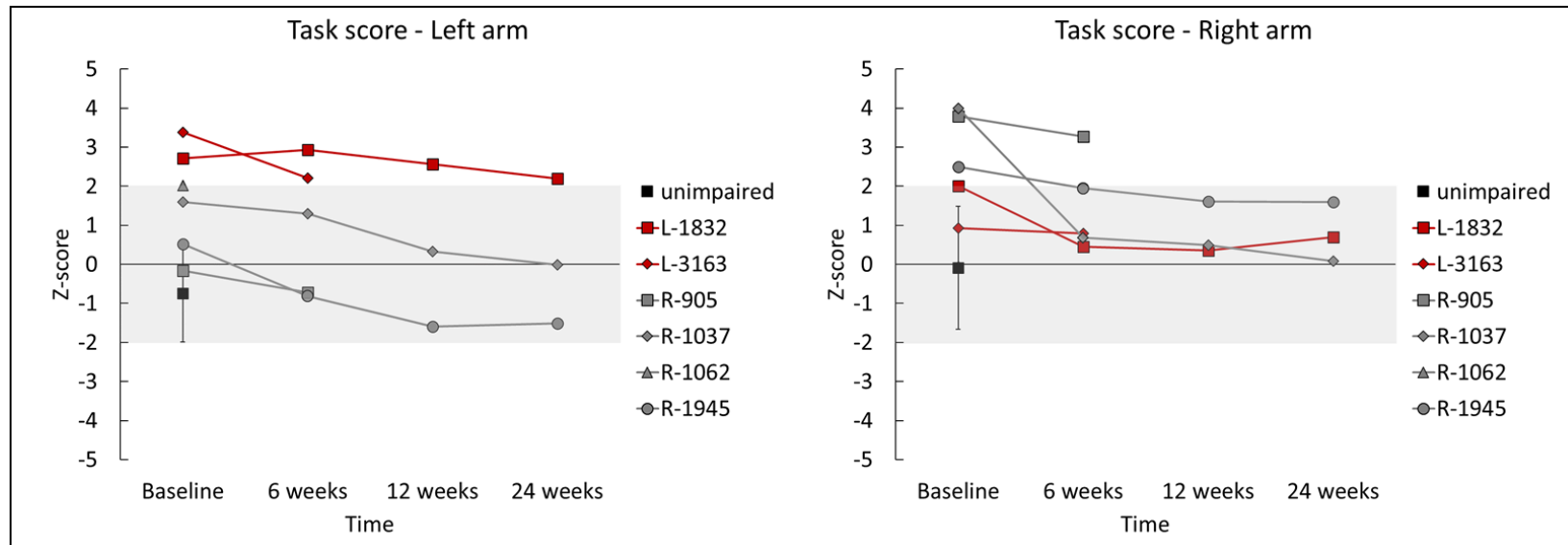
For the patients who were impaired at baseline and completed all of the follow-up sessions, left cerebellar patient L-1832 consistently showed impairment in his left arm from baseline ($z = 2.71$) to 24-weeks post stroke ($z = 2.19$). However, the results showed that the impairment in his right arm recovered by the 6-week follow-up assessment ($z = 0.45$).

On the contrary, for right cerebellar patients (Figure 5), patient R-1037's impairment in his right arm at baseline was no longer present by six weeks post stroke ($z = 0.68$) and was

maintained until 24 weeks post stroke ($z = 0.08$). Additionally, patient R-1945's impairment in the right arm regained its function six weeks after stroke ($z = 1.95$) and was maintained throughout the end of the assessments ($z = 1.59$ at 24-weeks follow up; Figure 5). Overall, our findings suggested that, among the impaired patients who completed all of the assessments, patients 1037 and 1945 fully recovered their functions by 24 weeks, while patient 1832 still showed some impairment in his left limb (Figure 5).

-- insert Figure 5 here --

Figure 5. Depicts Z-scores for Task score for impaired vs. unimpaired patients for the left and right arms at baseline, as well as 6, 12, and 24-weeks post-stroke. Left cerebellar patients are depicted in red symbols whereas right cerebellar patients are depicted in grey symbols. The mean and standard deviation of the unimpaired patients is depicted in black for the baseline session only. The grey shaded region denotes the range of normal performance of ± 2 Z-scores from the Kinarm normative sample. See Methods for descriptions of this measure.



Correlation analysis.

Correlation analyses were also conducted separately for the left and right arms to examine the relationship between the clinical and demographic variables and VGR task parameters. These data are presented in Supplementary Tables 1 and 2. The findings indicated that, for both limbs, scores on the PPT and Kinarm variables were not significantly correlated with the patients' age, days post-stroke, or scores on the MoCA (i.e., all p 's $\geq .058$). In addition, patients' scores on the MoCA were also not correlated with age, days post-stroke, lesion volume, scores on the PPT, or performance in Kinarm movement parameters (i.e., all p 's $\geq .11$). There was, however, a significant positive correlation between days post-stroke and the patient's lesion volume ($r = .83$, $p < .001$).

In contrast, we did observe significant correlations between performance scores on the PPT and the Kinarm VGR task parameters. Specifically, for the left arm (Supplementary Table 1), we found that poorer scores on PPT were correlated with poorer performance on all Kinarm movement parameters (range: $r = -.64$ to $r = .82$; all p 's $< .027$). For the right arm (Supplementary Table 2), poorer scores on the PPT were also correlated with poorer scores on all Kinarm movement parameters (range: $r = -.58$ to $r = .82$; all p 's $< .048$), except SMC ($r = -.29$, $p = .36$). These differences in correlation patterns between the PPT and VGR parameters for the left and right arms could be linked to the fact that only two of our patients had left cerebellar lesions that would mostly affect their left limbs, whereas nine patients had right cerebellar lesions that mostly affected their right limbs.

We also conducted a simple linear regression analysis to examine the extent to which scores on the PPT could predict the overall Kinarm VGR task scores for both arms. Here, we used the overall task score because it is a single value that integrates all the impairments across

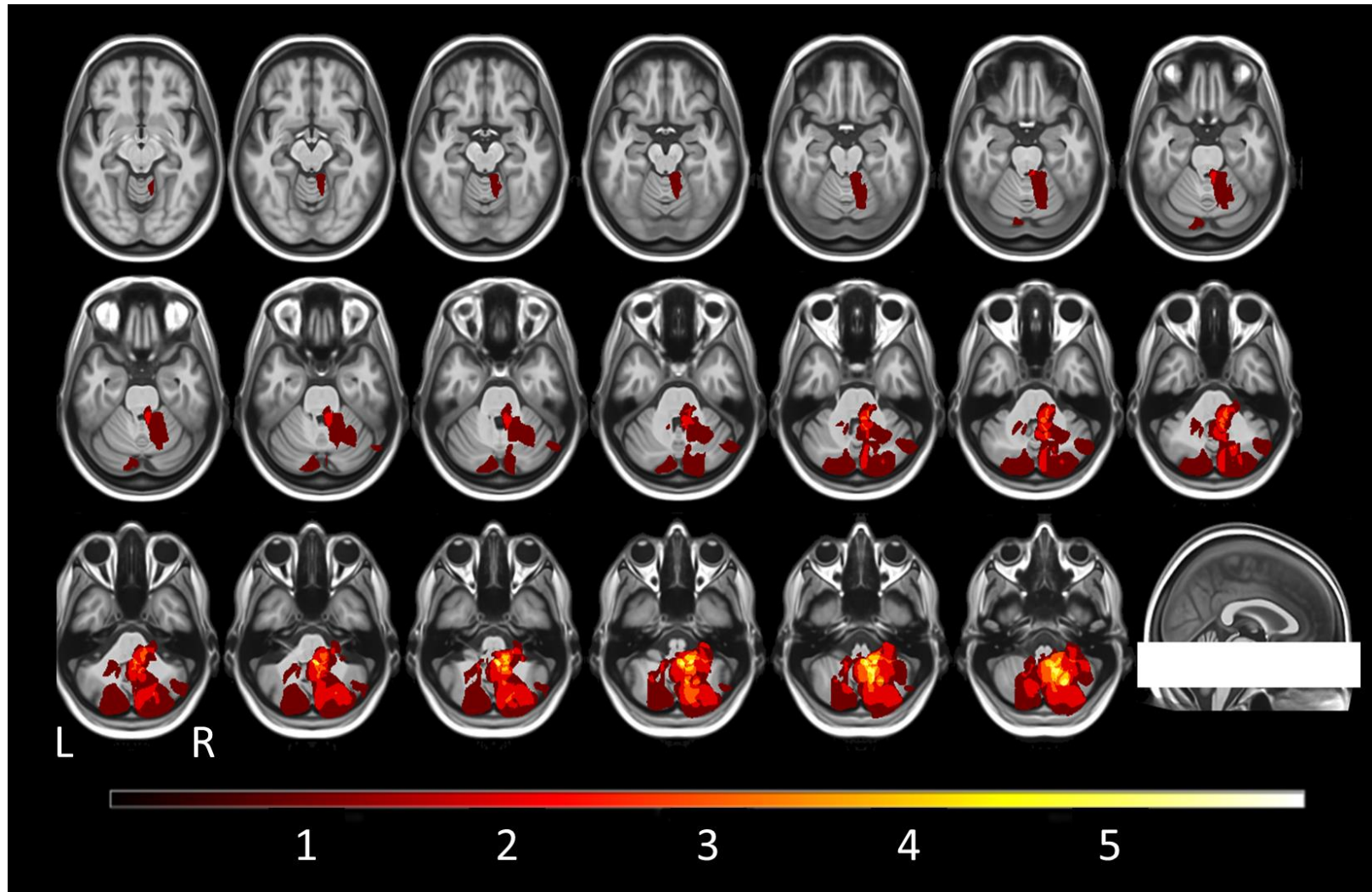
all the different Kinarm movement parameters. Our findings indicated that scores on the PPT for the left arm accounted for 66% of the variance in Kinarm VGR task scores for the left arm ($R^2 = .66$, $F(1,10) = 19.11$, $p = .001$). Similarly, for the right arm we found that scores in the PPT accounted for 61% of the variance in Kinarm VGR task scores for right arm ($R^2 = .61$, $F(1,10) = 15.66$, $p = .003$).

Lesion analysis.

Lesion maps of each individual patient can be found in Supplementary Figure 1. The mean lesion volume for the overall group was 7.97 cm^3 ($SD=10.40 \text{ cm}^3$). Based on the MRI atlas of the interposed/dentate nuclei from Dimitrova and colleagues (Dimitrova et al., 2006), two of our patients appeared to have damage in the deep cerebellar nuclei. Specifically, bilateral patient B-1095 had possible damage to the interposed/dentate nuclei in the left cerebellum, and right cerebellar patient R-3185 had damage to the interposed/dentate in the right cerebellar hemisphere (Table 1). Our results revealed that the regions of maximum lesion overlap in 5/12 patients (42%) were the cerebellar tonsil (i.e., HIX; MNI: $x = 10$, $y = -53$, $z = -47$), and the inferior semilunar lobule (i.e., Crus II; MNI: $x = 20$, $y = -64$, $z = -49$) in the right posterior-inferior cerebellum (Figure 6).

-- insert Figure 6 here --

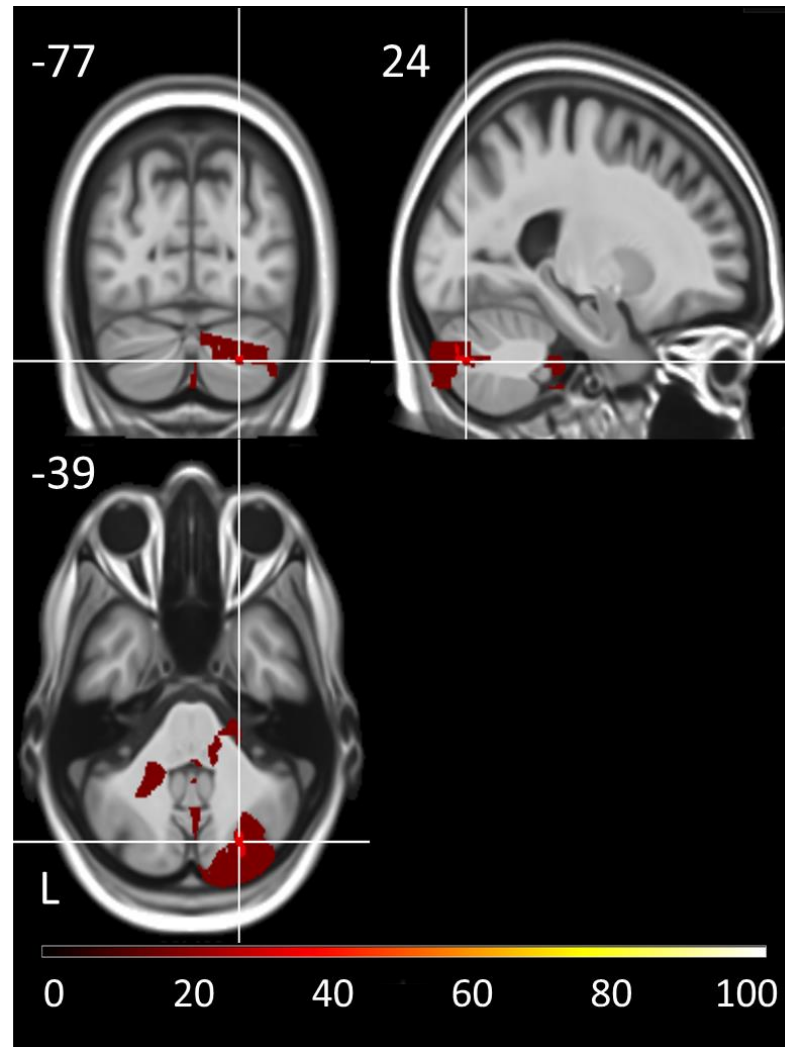
Figure 6. Group lesion overly for the cerebellar patients (n=12). The scale indicates the number of patients (out of 12) with a lesion in a specific region. Based on this analysis the regions of maximum lesion overlap in 5/12 patients (42%) were the cerebellar tonsil (i.e., HIX; MNI: $x = 10, y = -53, z = -47$), and the inferior semilunar lobule (i.e., Crus II; MNI: $x = 20, y = -64, z = -49$) in the right posterior-inferior cerebellum.



In addition, we carried out a lesion subtraction analysis by contrasting lesions in patients who were impaired (based on overall task score) at the VGR task at baseline (n=6), to those who were not impaired (n=6). This subtraction analysis revealed that the region that was associated with impaired performance on the VGR task in 40% of patients was lobule VIII of the right cerebellum (MNI: x=24, y= -77, z= -39; Figure 7). Note, that given the relatively small sample size, this analysis should be interpreted with caution.

-- insert Figure 7 here --

Figure 7. Results of the lesion subtraction analysis for patients impaired (n=6) vs. unimpaired (n=6) on the Kinarm VGR task. The region that was associated with impaired performance on the VGR task in 40% of patients was lobule VIII of the right posterior-inferior cerebellum (MNI: $x=24$, $y=-77$, $z=-39$).



Discussion

In the current study we examined the assessment and recovery of visually guided reaching (VGR) deficits in patients with cerebellar stroke (n=12) using the Kinarm exoskeleton, as well as a variety of clinical measures. Overall, the only clinical measure that reliably detected motor impairment in our patient group was the Purdue Pegboard Task (PPT), with 9/12 patients showing an impairment in one or both arms at baseline (Table 1). This is consistent with previous research showing that patients with cerebellar damage show impairments in fine motor control as measured by the PPT (e.g., Beuriat et al., 2020; Klinke et al., 2010). Six patients who were impaired at baseline completed the 6-week follow-up, and 3 patients completed the 24-week follow-up. Of the patients who completed the subsequent follow-ups, only one patient (R-1945) failed to show any significant recovery on the PPT (Figure 2).

In addition, 6/12 patients were impaired on the Montreal Cognitive Assessment (MoCA) at baseline. This is consistent with the notion that, in addition to its role in motor control, the cerebellum is also involved in a variety of different cognitive functions (for a review see Schmahmann et al., 2019). However, MoCA scores were not significantly correlated with any of our other clinical measures, or VGR task performance.

Kinarm VGR task performance.

For the Kinarm VGR task, a sub-group of patients showed significant impairment at baseline in one or both limbs relative to normative data in measures of both feed-forward (i.e., movement programming), and feedback (i.e., online) control (Table 2). For feed-forward control, consistent with previous work (Brown, Hefter, Mertens, & Freund, 1990; Day et al., 1998; Konczak et al.,

2010; Topka et al., 1998; Zackowski et al., 2002), 6/12 of our patients showed increased curvature in their reach trajectories during the initial phase of the reach (as measured by IDA), and covered less distance in the initial phase of the reach (as measured by IDR), relative to normative data (Figure 3).

Again, similar to previous studies (Bastian, Martin, Keating, & Thach, 1996; Day et al., 1998; Konczak et al., 2010; Zackowski et al., 2002), at baseline 5/12 patients showed an increase in the number of corrective movements (as indexed by SMC) compared to normative data (Figure 4), possibly reflecting an increased reliance on visual and/or proprioceptive feedback in the final phase of the reach (Bastian et al., 1996; Day et al., 1998). Finally, consistent with previous work (Bastian et al., 1996; Konczak et al., 2010; Topka et al., 1998; Zackowski et al., 2002), at baseline, 4/12 patients showed a significant increase in overall MT compared to normative data (Figure 4).

The PPT was the only clinical variable that was associated with performance on the Kinarm VGR task. Specifically, PPT scores for the left arm predicted 66% of the variance in VGR performance for the left arm, and PPT scores for the right arm predicted 61% of the variance in VGR performance for the right arm. These results suggest that, while clinical measures like the PPT are useful in characterizing fine motor impairments in cerebellar patients, kinematic measures may provide additional objective data that are useful for diagnosis, as well as monitoring recovery over time (Coderre et al., 2010).

Recovery of VGR performance.

In terms of recovery, 5/6 patients who showed kinematic impairments in the VGR task at baseline returned for the 6-week follow-up, and 3 returned for the 12 and 24-week follow-ups. Overall, most patients showed good recovery over the 24-week period. Of the two patients who only completed the 6-week follow-up, left cerebellar patient L-3163 improved somewhat, with a task score that was close to the normal range for their left hand, and was within the normal range for their right hand (Figure 5). Although right cerebellar patient R-905's overall task score was still outside the normal range for their right hand at the 6-week follow-up (Figure 5), they did show some improvements in IDA, IDR (Figure 3), and SMC (Figure 4), compared to their baseline assessment. For the remaining three patients (L-1832, R-1037, and R-1945), when examining overall task score (Figure 5), all showed recovery at the 24-week follow-up compared to the baseline assessment. Only left cerebellar patient L-1832 had an overall task score that was just outside the normal range for their left arm. However, L-1832 did show improvements in IDA (Figure 3), SMC (Figure 4), and MT (Figure 4) over the 24-week period.

Our recovery data are generally consistent with Konczak and colleagues (2010) which is, to our knowledge, is the only previous study to look at the recovery of reach kinematics over time in patients with cerebellar damage. Specifically, Konczak and colleagues (2010) observed that many of the cerebellar patients in their study (n=16) showed significant improvements within the first few weeks post-stroke, and continued to show improvement until the 3-month follow-up. The relatively quick recovery we observed is consistent with the notion of “cerebellar reserve” – or the capacity of the cerebellum to compensate for damage (for a review see Mitoma et al., 2020).

Lesion data.

In terms of the lesion data, the region of greatest overlap (42% or 5/12 patients) in the overall patient group was the cerebellar tonsil (lobule HIX), and the inferior semilunar lobule (Crus II) of the right posterior-inferior cerebellum (Figure 6). A lesion subtraction analysis comparing patients who were impaired at the VGR task at baseline (n=6), to those who were not impaired (n=6) found that the region of greatest overlap that differentiated the two groups was lobule VIII in the right posterior-inferior cerebellum (Figure 7). Previous research in cerebellar patients has demonstrated that deficits in reaching and grasping are typically associated with damage to lobules IV and V of the anterior lobe (Konczak et al., 2010; Kuper, Brandauer, et al., 2011), as well as lesions to the interposed and dentate nuclei in the medial cerebellum (Kuper, Hermsdorfer, et al., 2011). This is consistent with the notion that the anterior lobe, as well as the medial cerebellum, send outputs to motor cortex to control limb movements (for a review see Glickstein et al., 2011). At first glance, this seems at odds with results of our lesion analysis. However, it is well-known that the cerebellum contains two motor representations, the first being housed in the anterior lobe (lobules I-V; as noted above), and the second being housed in lobules VIIB and VIII in the posterior-inferior cerebellum (Glickstein et al., 2011; Manni & Petrosini, 2004). The results of our lesion subtraction analysis (lobule VIII) overlap with the location of this second motor representation in the posterior-inferior cerebellum. This is consistent with a previous study by Kuper and colleagues (Kuper, Brandauer, et al., 2011) in which they suggested that lesions of the “inferior” motor representation (i.e., lobule VIII in the inferior cerebellum) were associated increased curvature in reach trajectories during a visually guided grasping task. Thus, our data lend additional support to the notion that damage to the inferior motor representation in the cerebellum can lead to impairments in visually guided action.

Limitations.

Although our data have clearly demonstrated that damage to the cerebellum results in deficits in both feed-forward and feedback control during visually guided reaching, our study is not without its limitations. First, we have a relatively modest sample size of $n=12$ patients. However, it is worth noting that this sample size is similar to previous studies examining the effects of cerebellar damage on reaching and grasping (Bastian et al., 1996; Nowak et al., 2002; Rost et al., 2005; Topka et al., 1998; Zackowski et al., 2002). It is also important to point out that we only included patients with isolated cerebellar stroke. Such patients comprise less than 3% of all stroke cases, making it challenging to recruit larger groups of these patients (Kelly et al., 2001; Macdonell, Kalnins, & Donnan, 1987; Tohgi, Takahashi, Chiba, & Hirata, 1993).

A related criticism is that fact that only 5/6 of the patients who were impaired at the VGR task at baseline were able to return for the 6-week follow-up, and only 3/6 patients impaired at baseline were able to return for the 24-week follow-up. Although our results for reaching recovery are consistent with previous work (Konczak et al., 2010), given the small sample size, these results should be interpreted with caution.

Another limitation of the current study is that, because this was a retrospective analysis of data that was collected as part of a larger project, we were not able to obtain scores from standardized ataxia screening questionnaires that are typically used to assess cerebellar ataxia symptoms, such as the Scale for the Assessment and Rating of Ataxia (SARA) (Schmitz-Hübsch et al., 2006), or the International Cooperative Ataxia Rating Scale (ICARS) (Trouillas et al., 1997). However, we were able to obtain data from the PPT which is often used to assess deficits in fine motor control. Previous work in patients with cerebellar damage has demonstrated that patients score poorly on both ataxia screening questionnaires, as well as the PPT (Beuriat et al.,

2020; Klinke et al., 2010). In fact, one of these previous studies observed a high correlation ($r = .60$) between scores on the PPT and the SARA in patients with cerebellar damage (Klinke et al., 2010). In the current study, PPT scores for the left and right arms predicted 66% and 62% of the variance in Kinarm task score for the left and right arms (respectively). Given this, we are confident that performance on the Kinarm VGR task would have correlated significantly with ataxia screening measures had we been able to obtain them.

Implications for the perception-action model.

In the introduction, we noted that the inferior cerebellum receives ipsilateral visual inputs from the SC, area MT as well as dorsal stream regions such as the IPS and the parieto-occipital sulcus, via the pons (for reviews see Glickstein, 2000; Stein & Glickstein, 1992). In contrast, the cerebellum receives few (if any) visual inputs from V1, or the ventral visual stream (Glickstein, 2000; Stein & Glickstein, 1992). Thus, the cerebellum has the ability to work closely with the dorsal stream to control visually guided actions. Although these two structures must work closely with one another, very few studies have attempted to examine this.

In the current study, a lesion subtraction analysis showed that damage to lobule VIII, which receives visual input from the dorsal stream (Glickstein, 2000; Stein & Glickstein, 1992), and contains the inferior motor representation in the cerebellum (Glickstein et al., 2011; Manni & Petrosini, 2004), was associated with impaired performance on the VGR task. Specifically, cerebellar patients had increased curvature in their reach trajectories (IDA), covered less distance in the initial phase of the reach (IDR), and showed an increased number of corrective movements in the late phase of the reach (SMC). In addition, previous research has also shown that

cerebellar damage can disrupt both the transport and grasp components during visually guided grasping tasks (Brandauer et al., 2008; Kuper, Brandauer, et al., 2011; Kuper, Hermsdorfer, et al., 2011; M. K. Rand et al., 2000; Zackowski et al., 2002). Collectively, these symptoms are reminiscent of many of the reaching and grasping deficits commonly observed in patients with optic ataxia following dorsal stream lesions (e.g., Jakobson et al., 1991; Jeannerod et al., 1994; Karnath & Perenin, 2005; Milner et al., 2003; Milner et al., 2001; Milner & Goodale, 2008; Perenin & Vighetto, 1988; Pisella et al., 2009; Pisella et al., 2007).

Given the similarities in reaching and grasping deficits that can arise following damage to either dorsal stream, or the cerebellum, we believe it would be of great value for future studies to examine the effects of cerebellar damage on visually guided action using a perception-action theoretical framework. For example, one could compare grip apertures when cerebellar patients grasp objects of different sizes, to when they perform manual size estimations of the very same objects in isolation (e.g., Goodale et al., 1991), or in the context of visual illusions (e.g., Aglioti, DeSouza, & Goodale, 1995). One previous study by Zackowski and colleagues (Zackowski et al., 2002) provides some interesting clues for future investigation. Specifically, they observed that grip apertures were larger in cerebellar patients compared to controls for both manual estimates and grasping, but there was no correlation between the two. This suggests that the increase in grip aperture during grasping was not the result of a perceptual deficit. However, this study used only one object size, and thus it was not possible to compare grip scaling across different object sizes between the two groups.

There are many other interesting questions one could examine which are rooted in predictions from the perception-action model (Goodale & Milner, 1992; Milner & Goodale, 2006, 2008). For example, one could examine how reach and grasp kinematics in cerebellar

patients might be altered when performing actions in “real-time,” versus after a delay period where vision of the object is occluded (Milner et al., 2003; Milner et al., 2001; Rossetti et al., 2005). In addition, one could examine kinematics of reaching to targets in central vs. peripheral vision (Rossetti, Pisella, & Vighetto, 2003), or reaching to targets that either remain in the same position, or suddenly jump to a new target location during reach (the so-called “automatic pilot”) (Goodale, Pelisson, & Prablanc, 1986; Pisella et al., 2000). In such cases, if the cerebellum is working closely with the dorsal stream, then one might expect grip aperture to be more affected for grasping compared to manual estimates, for actions to be more impaired in real-time compared to delayed conditions, and for reaching to be more impaired for peripheral compared to central targets, and for trials where targets unexpectedly jump to a new location. Of course, none of these outcomes is guaranteed, but examining these questions will help us better understand how cerebellar damage can influence perception vs. action, and, ultimately, will enable a better understanding of how the cerebellum works with the dorsal stream to control visually guided actions.

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Supplementary Table 1. Correlation analysis for the left arm. Montreal Cognitive Assessment (MoCA), Purdue Pegboard Task (PPT), Initial direction angle (IDA), Initial distance ratio (IDR), Speed maxima count (SMC), Movement time (MT) and Task score. * $p < .05$, ** $p < .01$, *** $p < .001$.

Pearson's Correlations

		n	Pearson's r	p
Age	- Days post-stroke	12	0.534	0.074
Age	- MoCA	11	-0.317	0.342
Age	- Lesion volume	12	0.340	0.279
Age	- PPT_L_Z-score	12	-0.304	0.336
Age	- IDA_L_Z-score	12	0.560	0.058
Age	- IDR_L_Z-score	12	-0.414	0.181
Age	- SMC_L_Z-score	12	0.485	0.110
Age	- MT_L_Z-score	12	0.424	0.169
Age	- Task score_L_Z-score	12	0.396	0.202
Days post-stroke	- MoCA	11	-0.510	0.109
Days post-stroke	- Lesion volume	12	0.833 ***	< .001
Days post-stroke	- PPT_L_Z-score	12	-0.058	0.857
Days post-stroke	- IDA_L_Z-score	12	0.312	0.323
Days post-stroke	- IDR_L_Z-score	12	0.055	0.865
Days post-stroke	- SMC_L_Z-score	12	-0.011	0.974
Days post-stroke	- MT_L_Z-score	12	-0.088	0.785
Days post-stroke	- Task score_L_Z-score	12	0.038	0.906
MoCA	- Lesion volume	11	-0.220	0.516
MoCA	- PPT_L_Z-score	11	0.253	0.453
MoCA	- IDA_L_Z-score	11	-0.352	0.288
MoCA	- IDR_L_Z-score	11	0.271	0.421
MoCA	- SMC_L_Z-score	11	-0.204	0.548
MoCA	- MT_L_Z-score	11	-0.210	0.536
MoCA	- Task score_L_Z-score	11	-0.197	0.562
Lesion volume	- PPT_L_Z-score	12	0.184	0.568
Lesion volume	- IDA_L_Z-score	12	0.039	0.905
Lesion volume	- IDR_L_Z-score	12	0.219	0.495
Lesion volume	- SMC_L_Z-score	12	-0.244	0.445
Lesion volume	- MT_L_Z-score	12	-0.191	0.552
Lesion volume	- Task score_L_Z-score	12	-0.147	0.649
PPT_L_Z-score	- IDA_L_Z-score	12	-0.735 **	0.006
PPT_L_Z-score	- IDR_L_Z-score	12	0.817 **	0.001
PPT_L_Z-score	- SMC_L_Z-score	12	-0.691 *	0.013
PPT_L_Z-score	- MT_L_Z-score	12	-0.635 *	0.027
PPT_L_Z-score	- Task score_L_Z-score	12	-0.810 **	0.001

Pearson's Correlations

		n	Pearson's r	p
IDA_L_Z-score	- IDR_L_Z-score	12	-0.770 **	0.003
IDA_L_Z-score	- SMC_L_Z-score	12	0.782 **	0.003
IDA_L_Z-score	- MT_L_Z-score	12	0.346	0.270
IDA_L_Z-score	- Task score_L_Z-score	12	0.873 ***	< .001
IDR_L_Z-score	- SMC_L_Z-score	12	-0.939 ***	< .001
IDR_L_Z-score	- MT_L_Z-score	12	-0.752 **	0.005
IDR_L_Z-score	- Task score_L_Z-score	12	-0.953 ***	< .001
SMC_L_Z-score	- MT_L_Z-score	12	0.657 *	0.020
SMC_L_Z-score	- Task score_L_Z-score	12	0.920 ***	< .001
MT_L_Z-score	- Task score_L_Z-score	12	0.615 *	0.033

* p < .05, ** p < .01, *** p < .001

Supplementary Table 2. Correlation analysis for the right arm. Montreal Cognitive Assessment (MoCA), Purdue Pegboard Task (PPT), Initial direction angle (IDA), Initial distance ratio (IDR), Speed maxima count (SMC), Movement time (MT) and Task score (Task score). * p < .05, ** p < .01, *** p < .001.

Pearson's Correlations

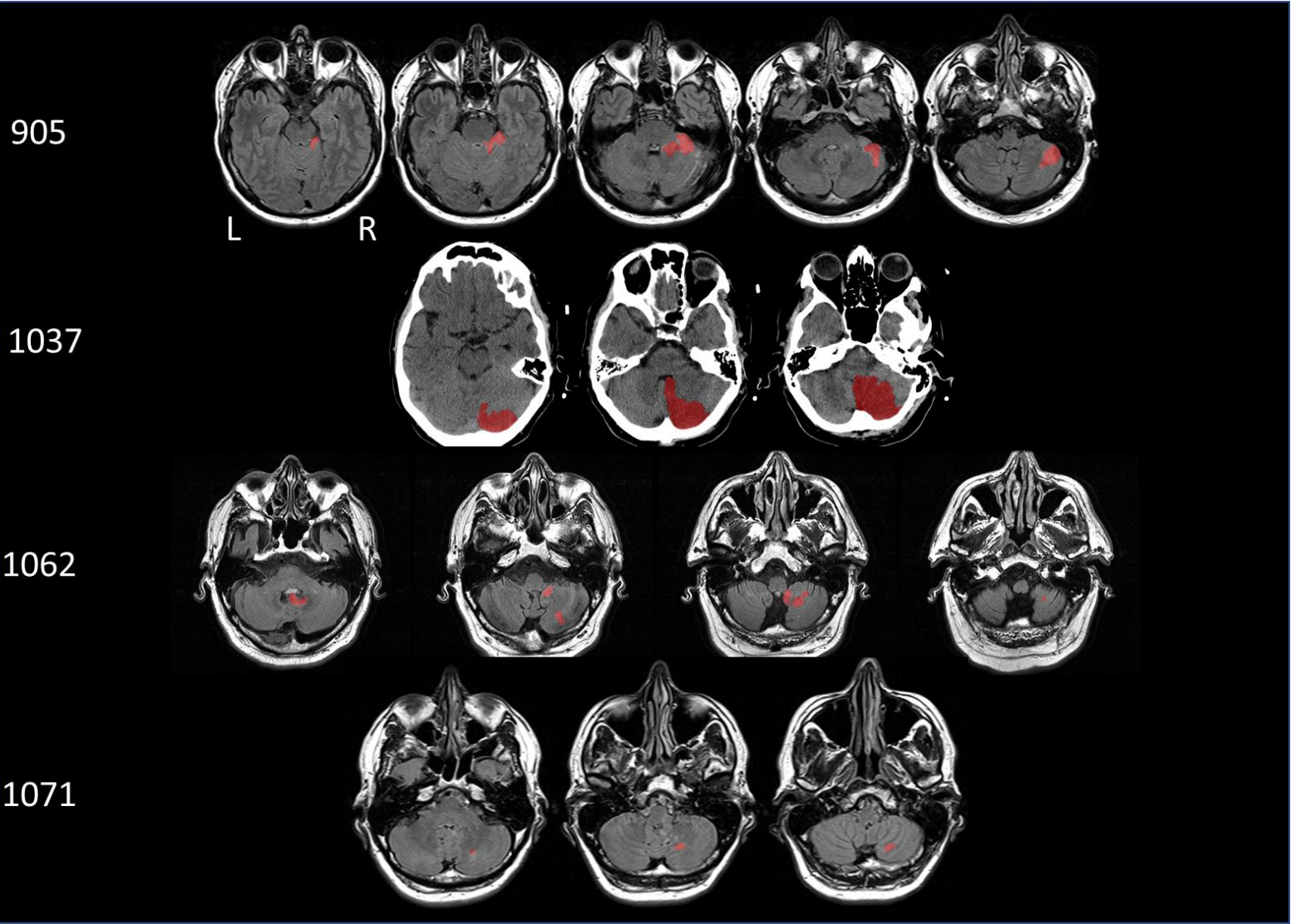
		n	Pearson's r	p
Age	- Days post-stroke	12	0.534	0.074
Age	- MoCA	11	-0.317	0.342
Age	- Lesion volume	12	0.340	0.279
Age	- PPT_R_Z-score	12	0.061	0.850
Age	- IDA_R_Z-score	12	0.090	0.780
Age	- IDR_R_Z-score	12	-0.106	0.743
Age	- SMC_R_Z-score	12	0.128	0.693
Age	- MT_R_Z-score	12	0.092	0.776
Age	- Task score_R_Z-score	12	0.017	0.958
Days post-stroke	- MoCA	11	-0.510	0.109
Days post-stroke	- Lesion volume	12	0.833 ***	< .001
Days post-stroke	- PPT_R_Z-score	12	-0.021	0.947
Days post-stroke	- IDA_R_Z-score	12	-0.005	0.987
Days post-stroke	- IDR_R_Z-score	12	-0.087	0.789
Days post-stroke	- SMC_R_Z-score	12	0.016	0.961
Days post-stroke	- MT_R_Z-score	12	0.107	0.742
Days post-stroke	- Task score_R_Z-score	12	-0.068	0.835
MoCA	- Lesion volume	11	-0.220	0.516
MoCA	- PPT_R_Z-score	11	-0.161	0.637
MoCA	- IDA_R_Z-score	11	0.151	0.658
MoCA	- IDR_R_Z-score	11	-0.118	0.730

Pearson's Correlations

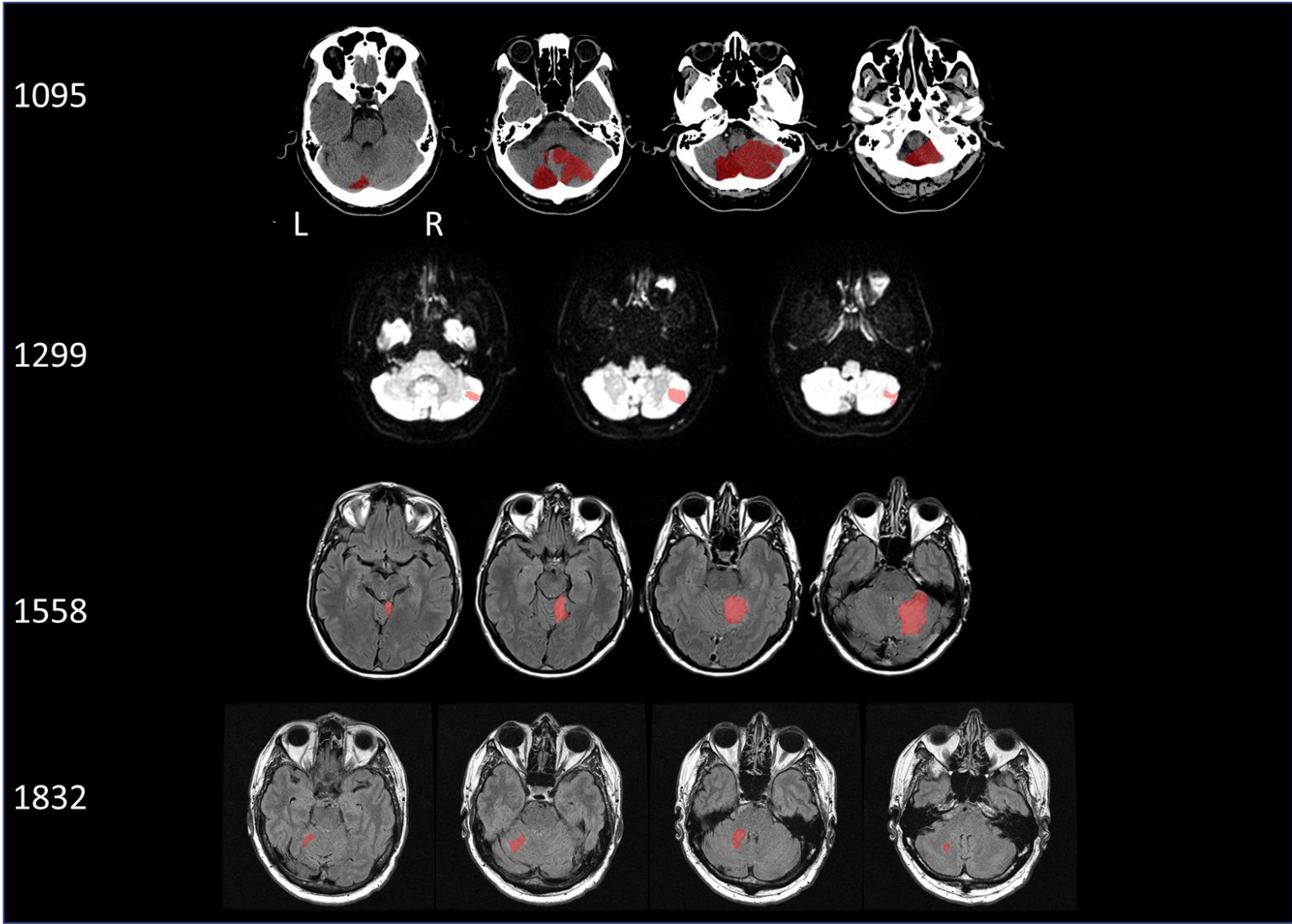
		n	Pearson's r	p
MoCA	- SMC_R_Z-score	11	-0.139	0.683
MoCA	- MT_R_Z-score	11	-0.145	0.670
MoCA	- Task score_R_Z-score	11	0.188	0.580
Lesion volume	- PPT_R_Z-score	12	-0.105	0.745
Lesion volume	- IDA_R_Z-score	12	0.008	0.980
Lesion volume	- IDR_R_Z-score	12	-0.106	0.742
Lesion volume	- SMC_R_Z-score	12	-0.036	0.912
Lesion volume	- MT_R_Z-score	12	0.095	0.770
Lesion volume	- Task score_R_Z-score	12	0.090	0.780
PPT_R_Z-score	- IDA_R_Z-score	12	-0.780**	0.003
PPT_R_Z-score	- IDR_R_Z-score	12	0.816**	0.001
PPT_R_Z-score	- SMC_R_Z-score	12	-0.292	0.357
PPT_R_Z-score	- MT_R_Z-score	12	-0.580*	0.048
PPT_R_Z-score	- Task score_R_Z-score	12	-0.781**	0.003
IDA_R_Z-score	- IDR_R_Z-score	12	-0.937***	< .001
IDA_R_Z-score	- SMC_R_Z-score	12	0.671*	0.017
IDA_R_Z-score	- MT_R_Z-score	12	0.739**	0.006
IDA_R_Z-score	- Task score_R_Z-score	12	0.920***	< .001
IDR_R_Z-score	- SMC_R_Z-score	12	-0.727**	0.007
IDR_R_Z-score	- MT_R_Z-score	12	-0.797**	0.002
IDR_R_Z-score	- Task score_R_Z-score	12	-0.941***	< .001
SMC_R_Z-score	- MT_R_Z-score	12	0.815**	0.001
SMC_R_Z-score	- Task score_R_Z-score	12	0.739**	0.006
MT_R_Z-score	- Task score_R_Z-score	12	0.784**	0.003

* p < .05, ** p < .01, *** p < .001

Supplementary Figure 1, part 1. Individual patient lesion maps.



Supplementary Figure 1, part 2. Individual patient lesion maps.



Supplementary Figure 1, part 3. Individual patient lesion maps.

