Recovery of Upper Limb Function After Cerebellar Stroke
Lesion Symptom Mapping and Arm Kinematics

Jürgen Konczak, PhD; Daniela Pierscianek, MD; Sarah Hirsiger, MS; Uta Bultmann, MD; Beate Schoch, MD; Elke R. Gizewski, MD; Dagmar Timmann, MD; Matthias Maschke, MD; Markus Frings, MD

Background and Purpose—Loss of movement coordination is the main postacute symptom after cerebellar infarction. Although the course of motor recovery has been described previously, detailed kinematic descriptions of acute stage ataxia are rare and no attempt has been made to link improvements in motor function to measures of neural recovery and lesion location. This study provides a comprehensive assessment of how lesion site and arm dysfunction are associated in the acute stage and outlines the course of upper limb motor recovery for the first 4 months after the infarction.

Methods—Sixteen adult patients with cerebellar stroke and 11 age-matched healthy controls participated. Kinematics of goal-directed and unconstrained finger-pointing movements were measured at the acute stage and in 2-week and 3-month follow-ups. MRI data were obtained for the acute and 3-month follow-up sessions. A voxel-based lesion map subtraction analysis was performed to examine the effect of ischemic lesion sites on kinematic performance.

Results—In the acute stage, nearly 70% of patients exhibited motor slowing with hand velocity and acceleration maxima below the range of the control group. MRI analysis revealed that in patients with impaired motor performance, lesions were more common in paravermal lobules IV/V and affected the deep cerebellar nuclei. Stroke affecting the superior cerebellar artery led to lower motor performance than infractions of the posterior cerebellar artery. By the 2-week-follow-up, hand kinematics had improved dramatically (gains in acceleration up to 86%). Improvements between the 2-week and the 3-month-follow-ups were less pronounced.

Conclusion—in the acute stage, arm movements were mainly characterized by abnormal slowness (bradykinesia) and not dyscoordination (ataxia). The motor signs were associated with lesions in paravermal regions of lobules IV/V and the deep cerebellar nuclei. Motor recovery was fast, with the majority of gains in upper limb function occurring in the first 2 weeks after the acute phase. (Stroke. 2010;41:2191-2200.)

Key Words: arm movement • ataxia • brain imaging • cerebellum • human

Infarction of the cerebellum represents ≈15% of all cerebral strokes.1,2 The “classical” cerebellar symptom is a loss of coordination that may affect speech, postural, locomotor, oculomotor, and upper limb control.3 The resulting ataxic signs in gait, posture, arm, and oculomotor function may not be fully detected during neurological examination, particularly when symptoms might not suggest a central nervous system cause. Based on the pooled data from 6 separate studies, a recent meta-review4 computed the mortality rate of 7% after cerebellar stroke. In the largest of these studies, 69% of patients were classified as independent by the investigators at 3 months.5

Few studies examined the motor deficits in the acute stage after a cerebellar stroke. In addition to the classical descriptions of acute symptoms after cerebellar injury,3,5,6 several recent reports have produced a more detailed account about the effects of acute lesions on oculomotor and speech-motor control,7,8 language function,9 and wrist motion.10

However, to our knowledge, there are no objective biomechanical data available regarding how a cerebellar infarction affects multi-joint upper limb function in the acute stage. The ataxic symptoms and control problems of cerebellar patients in their postacute or chronic stage are well-documented. With respect to arm movements, several studies have provided...
detailed kinematic and kinetic analyses showing that the patients’ inability to compensate for passive motion-dependent torques are at the heart of their problems to control a multi-limb system.

In addition, recent advances in MRI-based lesion-symptom mapping of the human cerebellum provided insights of how lesion site and motor dysfunction are linked in cerebellar injury. For example, investigating chronic lesions in young patients after tumor resection showed that the sparing of the deep cerebellar nuclei is most critical for the recovery of upper limb and postural function even at a young age. Another lesion-symptom mapping study using the International Cooperative Ataxia Rating Scale as a marker of motor dysfunction revealed that upper limb ataxia was significantly correlated with lesions of the interposed nuclei (NI) and part of the dentate nuclei (ND), ataxia of posture, and gait with lesions of the fastigial nuclei, including NI. Lesions of vermial, paravermial, and hemispheral lobules IV, V and VI showed the highest correlations with markers of upper limb ataxia.

The current study substituted clinical markers of upper limb ataxia with objective biomechanical markers of motor performance. Although clinical scores are an important outcome of a neurological examination, clinical rating scales are necessarily coarse and may fail to detect subtle differences in behavior. In addition, we investigated arm motor function in patients with acute lesions and followed-up their recovery during the first 4 months after stroke. This is important because previous work had focused on patients with chronic stage and no detailed biomechanical data were available documenting the early process of recovery in patients with cerebellar stroke. Finally, we used lesion-symptom mapping analyses to relate a patient’s motor performance to the MRI-based lesion sites to gain an understanding of how lesion location relates to arm motor deficit.

### Subjects and Methods

Sixteen patients with cerebellar stroke (5 females and 11 males; mean age, 60.1 ± 14.4 years) and 11 age-matched healthy controls (4 females and 7 males; mean age, 59.0 ± 10.3 years) participated in the study. All patients and 10 control subjects were right-hand-dominant based on the results of the Edinburgh Handedness Inventory. Another lesion-symptom mapping study using the International Cooperative Ataxia Rating Scale as a marker of motor dysfunction revealed that upper limb ataxia was significantly correlated with lesions of the interposed nuclei (NI) and part of the dentate nuclei (ND), ataxia of posture, and gait with lesions of the fastigial nuclei, including NI. Lesions of vermial, paravermial, and hemispheral lobules IV, V and VI showed the highest correlations with markers of upper limb ataxia.

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from the ceiling in front of them at shoulder height. The distance to the target was individually adjusted to ~90% of the subject's total arm length to avoid trunk motion by leaning forward. At the beginning of each trial, the arm assumed a relaxed position at the subject's side. Subjects were instructed to point to the target as fast and as accurately as possible.

In the no target condition, subjects pointed as fast as possible to the same location in absence of the target. This condition revealed if patients were able to perform fast movements at all and whether slowing in the target condition was the manifestation of a compensation strategy. Both arms were tested. Participants performed a total of 10 trials per arm in both conditions.

To examine the motor recovery over time, measurements took place at 3 poststroke intervals, first in the acute stage (session 1), then 2 weeks after session 1, and then 3 months after session 1. The dates of initial testing differed between the patients because of their individual conditions after stroke (mean time, 14.5 days after stroke; range, 1–33 days).

Measurements
The kinematic data analysis was performed using customized software routines based on MATLAB Technical Programming Language. The recorded time–position data of each marker were filtered offline using a fourth-order low-pass Butterworth filter with a cut-off frequency of 8 Hz. Onset of movement and movement end were determined for each pointing movement. Movement onset was defined as the point in time when the resultant velocity exceeded 5% of the peak resultant velocity starting from the resting position. Movement end at the target condition was defined as the point in time when the resultant velocity of the target exceeded the 25 mm/s threshold for the first time (ie, it was moved). We set this threshold to indicate that the target was fully hit. In the no target condition, movement end was defined as the point in time when the resultant velocity decreased to 5% of the peak resultant velocity after peak velocity was reached.

Initial analysis of the spatial hand trajectories revealed little evidence of intention tremor. We thus focused on the analysis of the higher-order kinematics velocity and acceleration. Five kinematic variables were analyzed for each trial: maximum hand velocity (Vel Max) and maximum hand acceleration (Acc Max) during the large transport phase of the hand. Subsequently, acceleration time, defined as the time between movement onset and peak resultant velocity, and deceleration time, defined as the time between peak resultant velocity and movement end, were computed. Total movement time (MT) was calculated as the sum of the acceleration and the deceleration times.

To obtain a variable describing the overall kinematic performance, we derived a motor score taking into account 3 different kinematic variables: MT, Vel Max, and Acc Max. We computed this motor control score (MCS) as follows:

\[
MCS = (a \times (1/MT)) + b \times \text{Vel Max} + c \times \text{Acc Max} \times 10^6
\]

with \(a = 10^3\), \(b = 1\), and \(c = 0.15\) as constants. These constants were chosen because MT, peak hand velocity, and acceleration have largely different magnitudes during pointing. Thus, the weightings \(a\), \(b\), and \(c\) served to adjust the impact of each kinematic variable on the motor score in a way that all 3 kinematic variables contributed approximately equal to the overall score. We used the MCS to assign patients to an “impaired” and “not impaired” subgroup for later lesion symptom analysis (see Results).

To assess the degree of motor recovery across time, we calculated the relative change in MT, Vel Max, and Acc Max observed at 2 weeks and 3 months in respect to the acute stage using the following formula:

\[
\text{Relative change}_{i} = \frac{\text{mean, session}/\text{mean, acute stage}}{100}
\]

where \(i = \text{MT, Vel Max, or Acc Max}\), and \(j = 2\) weeks or 3 months.

MRI
MR images of all cerebellar participants were acquired with a 1.5-T Siemens Sonata scanner. In the acute stage, a 3D sagittal volume of the entire brain was made using a T2-weighted sequence (repetition time, 19.4 ms; echo time, 5.71 ms; field of view, 240 mm; slice thickness, 1.5 mm; voxel size, 1.3x0.9x1.5 mm³). After 3 months, a 3D sagittal volume of the entire brain was obtained using a T1-weighted, magnetization prepared, rapid-acquisition, gradient-echo sequence (repetition time, 2400 ms; echo time, 4.38 ms; field of view, 256 mm; 160 slices; voxel size, 1.0x1.0x1.0 mm³). Images were examined by an experienced neuroradiologist and extracerebel lar pathology was excluded. Ischemic lesions were traced manually in non-normalized 3D MRI datasets and saved as regions of interest using MIRcro software (http://www.cabiatl.com/micro/micro). The individual regions of interest and complete 3D MRI datasets were simultaneously spatially normalized into a standard proportional stereotaxic space using SPM2 (http://www.fil.ion.ucl.ac.uk/spm/). Based on the Montreal Neurological Institute spatial coordinates of cerebellar lesions, the affected cerebellar lobules and nuclei were defined with the help of 3D MRI atlases of the cerebellum.23,24 Note that none of the MRI scans used for this study had been acquired within the first hours after stroke onset. Effects of penumbra tissue were negligible, but edema may still have been present. Acute lesions were determined by increased signal intensity on T2-weighted sequences, which do not always allow for a precise distinction between infarction size and edema. However, in the chronic stage at the 3-month follow-up, T1-weighted sequences had been applied. Thus, the lesion volumes for the acute and chronic stages could not be compared meaningfully. Despite these limitations, the volumes of the chronic lesion site remain informative, indicating the extent of the infarcted region. We have added the volumes of cerebellar lesions in the chronic stage in Table 2.

For statistical analysis of the effect of the ischemic lesions on kinematic performance, the effects of lesions were evaluated using lesion map subtraction analysis based on MIRcro software. Based on their MCS, cerebellar patients were divided into 2 subgroups of “impaired” and “not impaired” patients (see Results for details). A patient’s lesion was labeled “consistent” for a particular voxel either if the patient was classified as motorically “impaired” and had a lesion in that voxel or if the patient was “not impaired” and had no lesion in that voxel. MIRcro calculated the percentage of patients who were “consistent” for each voxel by taking the percentage of impaired patients with a lesion in that voxel and subtracting the percentage of unimpaired patients with a lesion at the same voxel. This yielded a value between 100% (all patients consistent) and −100% (all patients inconsistent). MRI subtraction analysis was only performed for the acute stage and not for the 3-month follow-up, because only 1 patient was left after 3 months with a MCS out of the range of controls.

Data Reduction and Correction
In the acute stage, we obtained brain imaging data and examined the kinematic performance of 16 patients. Because of attrition we lost 5 patients during follow-up. Thus, the description of the motor recovery over time was based on data of 11 patients. A post hoc analysis comparing the acute stage motor performance of the 5 patients with the remaining 11 patients revealed that the mean MCS of these 2 subgroups were not significantly different from each other. Thus, the attrition of these patients during the follow-up session did not introduce a bias to the follow-up analysis. A total of 2840 pointing movements were recorded during this experiment. A first analysis of the kinematics of patients’ less affected arms (all patients had unilateral lesions) revealed that they were essentially normal (all kinematics within the range of the control group). Thus, the further analysis focused on the patients’ movements with their affected arms. Because each patient had a matched control subject, only data of the same side as the patient’s affected side are presented here (ie, if the left arm was affected, only the left arm of the control subject entered the analysis). After this first data reduction, a combined total of 1420 pointing movements across all subjects were then used for further analysis (760 patient
trials and 660 control group trials). Target and no target trials were evenly split within each group (380 trials in the patient group, 330 trials in the control group). In 254 out of the 760 patient trials (33%) and in 209 out of 660 control group trials (32%), the automatic analysis program was not able to set the movement end point, either because subjects did not hit the target at all or because subjects only had made contact with target at the return movement, or the target was moved, but target velocity did not exceed the 25 mm/s threshold criterion (ie, subject barely touched the target). We were able to reanalyze a subset of trials for each group (patients, 56 trials; controls, 38 trials) by determining movement end using the time–series data of the finger trajectory instead. Thus, a total of 1051 trials (patients, 562 trials; controls, 489 trials) were used for further analysis, with each subject having at minimum 4 trials for each condition. To illustrate the scope of the patients’ motor performance, Figure 1 shows exemplar hand trajectories and the associated higher kinematics for 1 control subject and 2 patients.

### Results

#### Lesion Location and Volume

A summary of each patient’s lesion location and volume is given in Table 2. All lesions were unilateral. In the acute stage, all patients had lesions in the paravermal region. The vermis was affected in 14 out of 16 patients (88%). Two patients (13%) with vermal lesions showed lesions in all 3 deep cerebellar nuclei. Four patients (26%) revealed nuclear...
lesions restricted to ND, whereas 2 patients had lesions in ND and NI. In the chronic stage, 3 months after the first examination, only 1 out of 11 re-examined patients (patient 9) still presented with a nuclear lesion affecting ND.

To understand the link between lesion volume and motor performance, we correlated lesion volume at the 3-month chronic stage with the corresponding MCS for the target and no target conditions. Neither correlation reached the level of significance ($P > 0.05$), indicating no strong association between lesion volume and kinematic motor performance.

Relating Lesion and Motor Performance in the Acute Stage
Fifteen out of the 16 patients (94%) showed mild clinical signs of upper limb ataxia during the acute phase with their upper limb International Cooperative Ataxia Rating Scale scores (range, 2–14). During pointing to a target, 6 patients (38%) exhibited MT exceeding the range of the control group. The pointing gestures of 12 patients (75%) had lower peak hand velocities in comparison to controls, whereas 11 patients (69%) exhibited reduced peak hand accelerations outside the range of the control group (Figure 2). Four of the 6 patients with prolonged MT had lesions affecting the deep cerebellar nuclei.

In the no target condition, 7 out of 16 patients (44%) showed prolonged MT, 50% revealed lower peak velocities, and 56% of the patients showed reduced peak accelerations with respect to controls. The kinematic performance of 6 patients (patients 1, 2, 3, 9, 12, 14) was outside the range of the control group for any kinematic variable in both conditions.

To obtain a measure of overall motor performance, we computed a MCS. We used the MCS for the target condition to assign each patient to 1 of 2 subgroups: a “not impaired” group, whose MCS was within the MCS range of the control group and an “impaired” group, whose MCS value was outside the range of the control group. One patient (patient 6) was excluded from this analysis because he had a higher MCS than controls. MRI subtraction analysis between “impaired” and “not impaired” patients was used to identify cerebellar areas unique to patients with impaired kinematic performance. In patients with MCS values outside the range

Figure 1. Exemplar 3-dimensional hand trajectories and velocity and acceleration profiles of the hand during goal-directed finger pointing. Shown are trajectories from a healthy control participant, a patient with mild limb ataxia with no signs of motor slowing, and an impaired patient showing reduced peak velocities and accelerations. The graphs all include the return movement back to the starting position after the target was hit. Because the target was suspended, it moved slightly after contact. To illustrate the anthropometric dimensions of the arm and to appreciate the movement amplitude, the left graph includes a stick figure of the arm depicting the actual recorded joint marker positions for a single frame.
of the control group, lesions were up to 45% more common in lobules IV and V and the dentate or interposed nuclei in both conditions (Montreal Neurological Institute coordinates for the target condition: \(x = -8 - 22, y = -40 - 60, z = -14 - 32\); Montreal Neurological Institute coordinates for the no target condition: \(x = -8 - 22, y = -40 - 56, z = -14 - 32\); Figures 3, 4A). The affected cerebellar lobules were at first defined using with the help of the atlas by Schmahmann,\(^24\) which refers to the system introduced by Larsell.\(^25\) In a second step, the affected lobules were considered and attributed either to the territory of the PICA or to the territory of the SCA according to the description by Tatu.\(^26\) The territory of the SCA includes anterior lobe, simple lobule, and superior semilunar lobule, which correspond to the hemispheric lobules I to VI and Crus I of lobule VII A,\(^25\) as well as the vermal lobules I to VII A and most of the cerebellar nuclei. The territory of the PICA includes inferior semilunar, gracile, and biventer lobules and the tonsil of the cerebellum, which are Crus II of hemispheric lobule VII A, lobule VII B, and lobules VIII to X, as well as the vermal lobules VII B to X and parts of the dentate nucleus (Figure 4A).

**Kinematic Indicators of Motor Recovery**

When compared to the control group, the patients’ pointing movements during the acute stage were characterized by increased MT and reductions in peak hand velocity and acceleration. Kinematic signs of motor recovery were most pronounced between the acute stage and the 2-week follow-up session.

**Link Between Motor Recovery and Lesion Site**

Figure 4B reveals how individual patients recovered kinematically from the acute stage to the 3-month follow-up using the MCS as a global marker of performance. Based on the MRI subtraction analysis, the majority of patients with abnormal MCS values had lesions in the paravermal region of lobules IV and V or the deep cerebellar nuclei (ND, NI). Both lesion sites led to significant initial motor impairments, but most patients had recovered by the 3-month follow-up (Figure 4B).

**Comparing Motor Recovery for SCA and PICA Infarctions**

In the acute stage, the mean MCS of patients with an infarction of the SCA was significantly higher than those of...
PICA patients ($P = 0.002$). This statistical difference vanished at both follow-up sessions ($P > 0.05$).

**Pointing to a Target**

During target pointing, 8 of 11 patients were able to reduce MT after 2 weeks, and 4 of those patients achieved further improvements in session 3. The relative improvement with respect to the acute stage ranged between 9% and 55% for the 2-week follow-up and between 14% and 64% for the 3-month follow-up. Mean MT was 19% lower after 2 weeks and 28% lower after 3 months (Figure 5, top graph).

Motor improvements were also found with respect to peak hand velocity and acceleration. In comparison to their acute stage performance, 9 of 11 patients (82%) increased Vel Max during target pointing in the 2-week follow-up and 6 patients showed further improvements in session 3 (Figure 5, middle graph). Mean Vel Max for the patient group increased by 15% after 2 weeks and by 23% after 3 months.

The most pronounced relative change in kinematics was seen in the ability of patients to accelerate their hand during the arm transport phase. Ten out of 11 patients increased peak hand acceleration at 2 weeks (by up 86%), and 9 patients continued to improve slightly in the 3-month follow-up. In relation to the acute stage, mean change in Acc Max of all patients was 35% after 2 weeks and 49% after 3 months (Figure 5, bottom graph).

**Pointing as Fast as Possible**

Motor impairments extended to the no target condition (Figure 2), but recovery followed the same trend as seen for the target condition. With respect to the acute stage, mean MT decreased by 18% at 2 weeks and by 15% at 3 months. Motor recovery was also observable in the patients’ peak hand velocity, with 45% of the patients (5/11) achieving higher mean peak velocities between 10% and 54% in session 2. The increase in peak hand velocity was accompanied with concurrent increase in hand acceleration during the hand transport phase. With respect to the acute state, 55% of the patients (6/11) revealed increases in peak hand acceleration ranging between 12% and 120% at the 2-week follow-up.

**Discussion**

This study investigated the recovery of upper limb function in patients after cerebellar infarction. It pursued the following 3 aims: (1) to give a detailed biomechanical movement analysis of the upper limb symptoms in the acute stage after cerebellar stroke that has been lacking in the literature; (2) to perform lesion-symptom mapping by linking observed movement deficits of arm motor control to lesion location; (3) to provide a comprehensive assessment of the course of upper limb motor recovery for the first 4 months after a cerebellar stroke.

Our main findings are the following. First, in the acute stage, arm movements were mainly characterized by abnormal slowness (bradykinesia) and not dyscoordination (ataxia). Second, the kinematic signs of arm motor dysfunction were associated with paraverminal cortical lesions in lobules IV/V or the deep cerebellar nuclei, especially ND and NI. Third, motor recovery was fast, with majority of gains in upper limb function occurring in the first 2 weeks after the acute phase.
Motor Performance in the Acute Stage

Ataxic symptoms have been well-described in several seminal articles. The characteristic deficits include dysarthria, nystagmus, loss of balance, and loss of movement coordination. Ataxia becomes especially prominent during fast goal-directed movements and is most pronounced during multi-joint arm motion, with patients showing a decomposition of multi-joint motion and intention tremor in the terminal phase of the movement. In the early stages of cerebellar injury, signs of abnormal slowness, especially in the arms, have been observed in patients with gunshot wounds and have been related to loss of muscle tone. Movement slowness has also been measured in patients with chronic cerebellar lesions, especially during fast movements. In patients with chronic conditions, movement slowness or bradykinesia usually has not been considered to be a genuine cerebellar deficit but has been interpreted as a patient’s deliberate compensation strategy of attempting to avoid high accelerations and the associated high passive interaction torques that the patient may not be able to control when attempting to move the hand to a target in space. That is, to avoid an intention tremor at the end of a goal-directed movement, a cognitive strategy is to move slowly toward it. Here, we report that patients in the acute stage showed signs of motor slowing and not of dyscoordination. Patients performed pointing movements at lower peak velocities and accelerations than did healthy controls. It seems unlikely that this slowing resulted from adopting a compensation strategy, because this deficit was also observed when patients simply had to point in the air (Figures 2, 4), and no spatial accuracy requirements challenged cerebellar patients. Further, none of the patients reported that they slowed deliberately to better-perform the task.

In our opinion, the slowed motor performance in the no target condition indicates that motor slowing is a genuine deficit in the acute stage after cerebellar infarction, which is an interpretation consistent with classical descriptions of symptoms after acute cerebellar injury. Moreover, it underlines the notion that acute lesions of neural structures involved in motor control lead initially to slowness and signs of bradykinesia, which is a finding well-described for lesions affecting the motor areas of the neocortex.

However, some caution is warranted when generalizing these findings. Clinically, our patient sample presented with relatively mild signs of cerebellar ataxia, even in the acute stage. Because it is known that upper limb ataxia might be more severe after cerebellar injury, it might well be that in...
more severely affected patients any signs of bradykinesia are masked by the more severe ataxic symptoms and they are not clinically recognized.

**Motor Performance and Associated Lesions**

Our MRI subtraction analysis revealed that lesions of the cerebellar cortex in the paravermal regions of lobulus IV and V and lesions affecting the deep cerebellar nuclei, especially the dentate and interposed nuclei, were associated with abnormal kinematic performance (Figure 3; Table 2). This deficit is consistent with the notion that lobules IV and V as well as ND and NI contain fractured somatotopic maps of the arm and, thus, are involved in arm motor control. Lobules IV and V and most parts of the deep cerebellar nuclei are supplied by the SCA, which explains that SCA patients exhibited a greater degree of upper limb impairment than patients with PICA-related infarcts (Figure 4A).

With respect to nuclear lesions, the motor impairments are also consistent with recent findings demonstrating that ND and NI lesions led to persistent problems in arm control that are not compensated well at any developmental age. In monkeys, the temporary inactivation of the NI and the adjacent portion of ND are known to affect arm motor control, leading to impairments in reaching and grasping gestures.32–34

With respect to cortical lesions, functional MRI data had shown earlier that the lateral areas of the anterior cerebellum, including lobulus V, are involved in hand and arm function.55 Further, converging evidence from brain imaging, neurophysiological, and clinical observations indicates that lobuli V–VI and VIII contain fractured somatotopic maps of the ipsilateral arm. In addition, neuroanatomical and electrophysiological data from the cat revealed significant reciprocal connections between the paravermal lobule Vbc and the interposed nuclei. Based on the latter finding, it becomes plausible that an interruption of these projections either at the cortical level (lobulus V) or at the level of the nuclei may lead to impairments in arm motor control. This conclusion is fully consistent with our human findings showing that patients with lesions affecting cortical lobules IV/V or NI presented with the most persistent kinematic problems.

**Time Course of Motor Recovery**

To our knowledge, this is the first study examining the kinematics of upper limb control in human patients in the acute stage after cerebellar stroke and also after their recovery for the first months after the infarction. Our data revealed that motor recovery is fast after cerebellar infarction, with most improvements seen in the arm kinematics occurring early after the infarct. Two weeks after the acute stage assessment, 9 of 11 patients showed higher peak hand velocities and accelerations. Performance gains were substantial, ranging between 10% and 54% for peak velocity and between 12% and 120% for peak acceleration in the no target condition, and slightly lower when pointing toward a target. Some patients continued to improve until the 3-month follow-up, but the rate of improvement was generally lower (Figure 5).

**Conclusion**

In summary, the present study provided, for the first time to our knowledge, a detailed account of the acute deficits in arm motor control in patients after cerebellar stroke. Arm movements in the acute stage were mainly characterized by bradykinesia and not ataxia. These motor impairments were associated with lesions in the paravermal region of lobulus IV and V, or the deep cerebellar nuclei, especially the NI and ND. Motor recovery was fast, with the majority of gains in upper limb function occurring in the first 2 weeks after the acute phase. The degree of improvements with respect to hand velocity and acceleration were also largest in the early phase of recovery.

**Sources of Funding**

The study was supported by a grant from the German Federal Ministry for Science and Research (BMBF 01 GA 0508).

**Disclosure**

None.

**References**

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*Stroke*. 2010;41:2191-2200; originally published online September 2, 2010; doi: 10.1161/STROKEAHA.110.583641

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://stroke.ahajournals.org/content/41/10/2191

Data Supplement (unedited) at:

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小脳の脳卒中後にみられる上肢機能回復
—病変・症状マッピングと上肢の運動力学

Recovery of Upper Limb Function After Cerebellar Stroke
—Lesion Symptom Mapping and Arm Kinematics

Jürgen Konczak, PhD1; Daniela Pierscianek, MD2; Sarah Hirsiger, MS1,3; Uta Bultmann, MD2; Beate Schoch, MD4; Elke R. Gizewski, MD5,6; Dagmar Timmann, MD2; Matthias Maschke, MD7; Markus Frings, MD2

1 Human Sensorimotor Control Laboratory, University of Minnesota, Minneapolis, Mn; 2 Department of Neurology, University of Duisburg-Essen, Essen, Germany; 3 Institute of Human Movement Sciences and Sport, ETH Zürich, Zürich, Switzerland; 4 Department of Neurosurgery, Liebig University, Giessen, Germany; 5 Department of Neuroradiology, University of Duisburg-Essen, Essen, Germany; 6 Department of Neuroradiology, UKGM, Justus-Liebig University, Giessen, Germany; 7 Department of Neurology, Brüderkrankenhaus, Trier, Germany.

背景および目的：脳梗塞後の上肢機能回復は小脳病変後で特に重要である。運動機能回復の経過については既に記述されているが、急性期の運動失調や運動力学的特性についての討議は少ない。本研究では、急性期の上肢運動障害の病変部位の関連について包括的評価を行い、梗塞発症後最初の4ヶ月間における上肢の運動機能回復の経過について検討する。

方法：被験者は、小脳の脳卒中を経験した成人患者16例で、急性期の運動を問う症候を有する症例を対象とした。急性期の運動の測定から2週間および3ヶ月後の追跡調査時に、運動の測定を実施した。運動機能の評価方法として、急性期および3ヶ月後の追跡調査時の測定の際にMRIデータを収集した。ボクセル単位の病変マッピング解析を実施し、運動力学的能力に対する病変部位の影響を検討した。

結果：急性期には70%近くの患者が、対照群と比較して手の最大速度および最大加速度の低下を示した。MRI解析の結果、運動機能の障害のある患者は小脳血管障害群において、運動機能の障害は静的であった。手の運動機能の回復は、最終的に6ヶ月で改善した。

結論：急性期の上肢運動の主要な特徴は、異常な運動反応（bradykinesia）であり、運動障害（ataxia）で見られなかった。運動機能の回復は、運動機能の改善が見られなかった。

Stroke 2010; 41: 2191-2200