Movement Disorders
Vol. 13, No. 6, 1998, pp. 958–964

Gait in Patients With Cerebellar Ataxia

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Summary: The gait pattern in 10 patients with cerebellar degenerations was studied and the results were compared with 10 matched normal subjects, seeking the principal patterns in this disorder. Gait at natural speed was studied in a biomechanics laboratory using a video-based kinematic data acquisition system for measuring body movements. Patients showed a reduced step and stride length with a trend to reduced cadence. Heel off time, toe off time, and time of peak flexion of the knee in swing were all delayed. Range of motion of ankle, knee, and hip were all reduced, but only ankle range of motion reached significance. Multijoint coordination was impaired, as indicated by a relatively greater delay of plantar flexion of the ankle compared with flexion of the knee and a relatively late knee flexion compared with hip flexion at the onset of swing. The patients also showed increased variability of almost all measures. Although some of the deviations from normal were simply the result of slowness of walking, the gait pattern of patients with cerebellar degeneration shows incoordination similar to that previously described for their multijoint limb motion. **Key Words:** Cerebellum—Degeneration—Gait—Ataxia—Multijoint coordination.

One of the hallmarks of a cerebellar disorder is ataxic gait. The principal features are irregularity of stepping, both in direction and distance. This may lead to veering to one side or lurching in different directions. Stability of upright stance is poor and patients may fall. Just as with standing balance, the base, or distance between the feet, is said to be broad. Despite numerous clinical descriptions, there has been no quantitative assessment. Such assessment might be useful to diagnose or quantify the ataxia. Here we present a detailed analysis using standard gait laboratory methodology; several preliminary descriptions have already been published.[1,2]

PATIENTS AND METHODS

Patients

We studied six men with hereditary cerebellar cortical atrophy and three men and one woman with olivopontocerebellar atrophy (Table 1). Their ages ranged from 21–64 years with a mean of 46 years. Height ranged from 156–185 cm with a mean of 175 cm; weight ranged from 121–213 pounds with a mean of 177 pounds. Patients showed various degrees of disability, as assessed by a formal scale used in our group and also previously reported.[3] All patients exhibited clinically unequivocal ataxia of arm movements, speech, and gait, which was most prominent in those severely affected. They had no weakness, tremor, signs of cognitive disturbances, or marked abnormalities of the "pyramidal" or "extrapyramidal" systems or of the cranial or peripheral nerves. Brisk reflexes, but not extensor plantar responses, and mildly increased tone elicited only through the use of reinforcement maneuvers were not grounds for exclusion. Cerebellar cortical atrophy was diagnosed if patients showed one or both of the following: only upper or lower limb ataxia, or midline ataxia, dysarthria, and cerebellar oculomotor dysfunction without radiologic evidence of additional brain stem atrophy. If additional mild extracerebellar signs (pyramidal signs, rigidity) were present or if magnetic resonance imaging scans showed brain stem involvement, the diagnosis of olivopontocerebellar atrophy was made. None of the patients were taking anticonvulsants, muscle relaxants, antiparkinsonism medication, or psychotropic agents.

Ten neurologically normal volunteers served as age- and sex-matched control subjects. Ages of the control

Received November 18, 1997; revision received March 27, 1998. Accepted June 8, 1998.
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TABLE 1. *Patient characteristics*

Patient no.	Sex/age	Weight (lbs)	Height (cm)	Diagnosis	Balance score*	Gait score*	Gait base†
1	M/21	121	178	OPCA	3	2	Wide
2	M/43	190	175	CCA	3	4	Wide
3	M/33	160	178	OPCA	0.5	0.5	Normal
4	M/35	156	183	OPCA	1	2	Wide
5	M/64	172	175	CCA	2	4	Wide
6	M/52	213	185	CCA	1	4	Wide
7	M/64	173	178	CCA	2.5	2	Wide
8	M/55	227	168	CCA	3	3	Wide
9	M/49	200	175	CCA	1	1	Normal
10	F/39	156	158	OPCA	0.5	1.5	Normal

OPCA, olivopontocerebellar atrophy; CCA, cerebellar cortical atrophy.

* Balance score and gait score came from the Human Motor Control Scale and were rated from 0 (normal) to 4 (severely affected).

† The base of the gait was assessed clinically as either normal or wide before formal measurement.

subjects ranged from 25–68 years with a mean of 46, height ranged from 162–180 cm with a mean of 172 cm, and weight ranged from 130–225 pounds with a mean of 167 pounds. The protocol was approved by the Institutional Review Board, and all subjects gave their written informed consent for the study.

METHODS

Gait was analyzed by a bilateral sagittal plane technique, as described previously.[4] Kinematic data were sampled within a stationary orthogonal laboratory coordinate system defined by a vertically oriented Z-axis and a Y-axis parallel to the path of progression. A calibration volume extending 0.606, 2.24, and 2.04 meters along the X, Y, and Z axes of the laboratory coordinate system, respectively, was defined using 20 spherical retroreflective targets (3M Scotchlite high-grain 7610 sheeting, Industrial Optics/3M, St. Paul, MN, U.S.A.), 25.4 mm in diameter, equally distributed among four plumb bob wire sets. Two strain gauge force plates (AMTI type OR6-3A, Advanced Mechanical Technology, Newton, MA, U.S.A.) were positioned along the Y-axis such that consecutive contralateral stance phase kinetics could be measured. Gait events, such as heel strike and toe off, were determined visually from the kinetic and kinematic data.

A five-camera video-based kinematic data acquisition system (VICON, Oxford Metrics, Tampa, FL, U.S.A.) synchronously collected the unprocessed kinematic and force plate data at 50 Hz. AMASS software (AMASS, Adtech, Adelphi, MD, U.S.A.) was used for camera calibration and the generation of three-dimensional data. The National Institutes of Health "Get Temporal and Distance Values" software was used to generate sagittal plane (Y- and Z-axes) gait variables.

Spherical retroreflective targets, 25.4 mm in diameter, were used to define the segment ends of a 12-segment body model. Targets were bilaterally attached to the skin surface at the following sites: anterior to the tragus of the ear, greater tubercle of the humerus, lateral epicondyle of the humerus, posterior surface of the forearm between the styloid processes of the radius and ulna, greater trochanter of the femur, lateral femoral condyle, lateral malleolus, and fifth metatarsophalangeal joint.

The subjects, dressed in shorts, walked with bare feet at a self-determined pace. Data acquisition began approximately 1 second before the subject entered the calibrated volume and ended approximately 1 second after the subject left the volume. Six to 10 gait trials were analyzed for each subject. Camera nonlinearities, calibration errors, and resultant target residual values were all within acceptable limits.

The following gait descriptors were determined:

- Cadence: The number of steps per minute
- Step length: The Y-axis displacement between the lateral malleolus target positions of the two feet between heel strike and the subsequent contralateral heel strike
- Stride length: The sum of two consecutive step lengths, measured as the Y displacement of the lateral malleolus of one foot from heel strike to subsequent heel strike of the same foot
- Step width: The X-axis displacement between the lateral malleolus target positions of the two feet between heel strike and the subsequent contralateral heel strike
- Step length symmetry: The step length on the right divided by the step length on the left, figured as a percentage

- Stride length symmetry: The stride length measured from the right foot divided by the stride length measured from the left foot, figured as a percentage
- Step width symmetry: The step width on the right divided by the step width on the left, figured as a percentage
- Stance time: The time that the foot is on the floor, measured as the time between heel strike and toe or heel off, whichever is last
- Swing time: The time that the foot is in the air, measured as the time between toe off and heel strike
- Step time: The time between heel strike of one foot to the subsequent heel strike of the contralateral foot
- Gait cycle or stride time: The time between two consecutive heel strikes of the same foot
- Average gait velocity: The stride length divided by the stride time
- Knee joint angle: The posterior measurement of the angle formed by the projection of the greater trochanter, the lateral femoral condyle, and the lateral malleolus targets into the Y-, Z-plane
- Ankle joint angle: The anterior measurement of the angle formed by the projection of the lateral femoral condyle, the lateral malleolus, and the fifth metatarsophalangeal joint targets into the Y-, Z-plane
- Hip joint angle: The anterior measurement of the

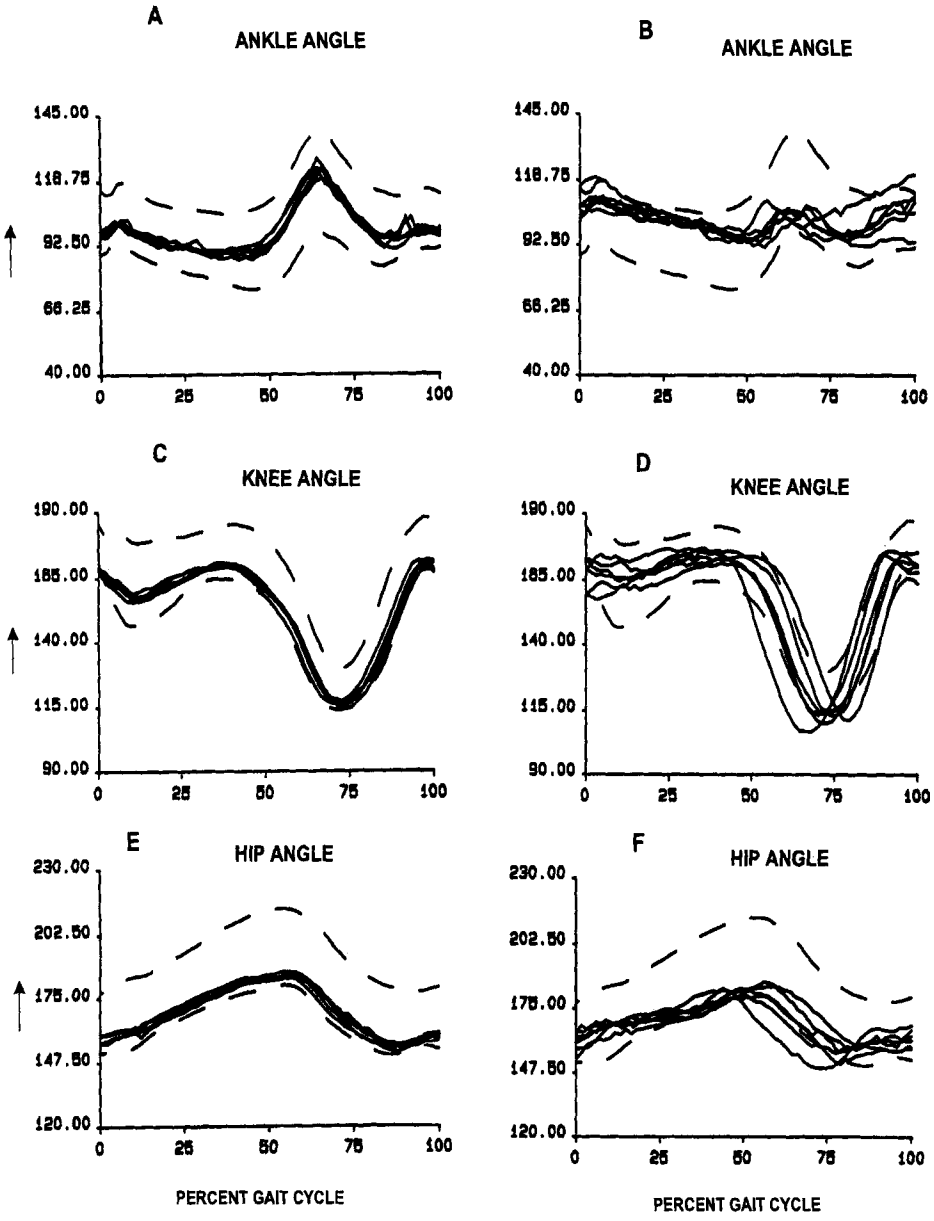

FIG. 1. Comparison of right-sided angular motions of patient 1 (**B**) with his matched control (**A**). Single trials (solid lines) are plotted together with the normal confidence limits (dashed lines) of ± 2 standard deviations.

angle formed by the projection of the greater tubercle of the humerus, the greater trochanter, and the lateral femoral condyle targets into the Y-, Z-plane

- Foot height: The maximum distance of the fifth metatarsophalangeal joint from the floor during swing
- Heel off time: The moment in stance when the heel leaves the floor, measured in percent of gait cycle when the ankle angle begins to plantar flex
- Toe off time: End of stance phase when the toe leaves the floor, measured in percent of gait cycle when the ankle angle shows a maximum of plantar flexion and begins to dorsiflex.

Stride length, step length, and step width were normalized by the subject's height.

Variability of the measurements was determined using the coefficient of variation, defined as the standard deviation of a measurement for each subject divided by the corresponding mean value and expressed as a percentage.

Statistical Analysis

Overall differences in measurements between the patients and the control subjects were assessed with an unpaired t test (two-tailed). Statistical significance was defined as $p < 0.05$. Because this study was exploratory, there was no correction for multiple comparisons, and the results should be considered as descriptive.

RESULTS

Although all the trials from the control subjects could be analyzed, many of the trials from the patients could not. If the patients veered out of view of the cameras or stumbled, the data could not be properly assessed. Hence, the patient trials analyzed are their best performances. There were almost always six trials per subject (rarely five or seven).

Examples of angular motion at the ankle, knee, and hip from two patients compared with their matched control subjects are shown in Figures 1 and 2. In these figures, ankle, knee, and hip angles are displayed together

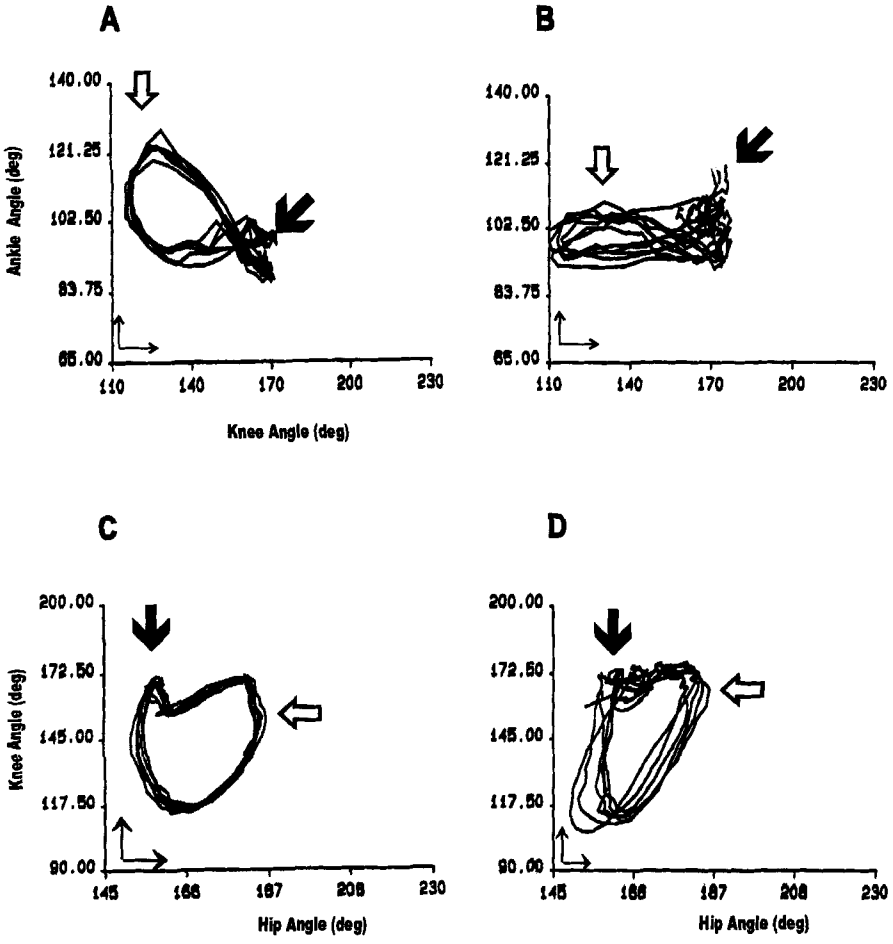

FIG. 2. Comparison of left-sided angular motions of patient 8 (**B**) with his matched control (**A**). Single trials (solid lines) are plotted together with the normal confidence limits (dashed lines) of ± 2 standard deviations.

TABLE 2. *Kinematic descriptors of gait*

Descriptor	Control (mean ± SD)	Patient (mean ± SD)	p from *t* test
Cadence (steps/min)	111.0 ± 7.6	102.2 ± 15.9	0.14
Step length (% height)	0.48 ± 0.20	0.29 ± 0.07	0.02*
Stride length (% height)	0.96 ± 0.40	0.59 ± 0.14	0.02*
Step width (% height)	0.16 ± 0.08	0.14 ± 0.03	0.68
Step length symmetry	1.00 ± 0.06	0.97 ± 0.17	0.64
Stride length symmetry	1.02 ± 0.02	1.00 ± 0.04	0.35
Step width symmetry	0.98 ± 0.06	1.02 ± 0.12	0.31
Stance time (sec)	0.66 ± 0.12	0.79 ± 0.17	0.07
Swing time (sec)	0.39 ± 0.05	0.43 ± 0.06	0.18
Step time (sec)	0.54 ± 0.04	0.61 ± 0.11	0.13
Stride time or gait cycle (sec)	1.08 ± 0.08	1.21 ± 0.22	0.11
Gait velocity (mm/sec)	0.90 ± 0.39	0.47 ± 0.17	0.01*
Ankle angle range of motion	31.5 ± 6.2	23.2 ± 5.1	0.004*
Ankle angle at heel strike	102.5 ± 5.9	103.0 ± 8.5	0.90
Heel off time (% gait cycle)	44 ± 4	50 ± 8	0.04*
Toe off time (% gait cycle)	66 ± 1	68 ± 3	0.04*
Knee angle range of motion	58.5 ± 2.1	53.9 ± 7.7	0.10
Knee angle range of motion during stance	11.5 ± 4.9	7.5 ± 3.8	0.07
Time of peak flexion of knee during swing (% gait cycle)	2.7 ± 1.9	75.5 ± 3.2	0.02*
Hip angle range of motion	34.3 ± 5.9	31.3 ± 4.7	0.23
Foot height (cm)	11.9 ± 1.2	11.5 ± 1.9	0.62

with angle/angle diagrams for ankle and knee and knee and hip. In the angle versus time plots, the dashed line indicates the normal tolerance band of mean ± 2 standard deviations. In the plots of the control subjects, the normal events are clear. The ankle shows a brief plantar flexion at heel strike, followed by a gentle dorsiflexion in stance as the body moves over the foot. The ankle then shows a brisk plantar flexion, producing push-off lasting from heel off to toe off. During swing there is a dorsiflexion of the ankle to avoid hitting the toe on the ground and to prepare for heel strike. The knee shows a slight sag during stance and a more pronounced flexion during swing. The hip extends in stance and flexes during swing.

The angle/angle diagrams can best be interpreted in terms of the normal gait cycle. In the ankle/knee diagram, heel strike is at the upper right. Stance phase is C-shaped, ending at the lower right, resulting from the dorsiflexion of the ankle and simultaneous sag of the knee. Swing phase is a counterclockwise oval, with the initial movement up and to the left coming from plantar flexion of the ankle with simultaneous knee flexion. In the knee/hip diagram, heel strike is at the upper left. Stance phase is located at the top of the oval, with the early dip resulting from knee sag and the general motion to the right resulting from hip extension. Swing phase shows a return of the graph to the left resulting from hip flexion, whereas the graph goes down and up as a result of flexion and extension of the knee.

The first impression of the patient graphs is the marked variability compared with the control graphs. The general pattern of angular motion shows the major normal characteristics, but some systematic abnormalities can be appreciated. Some events appear late, such as the push-off of the ankle and time of peak knee flexion. The knee sag in stance is deficient. The angle/angle diagrams look more deviant. The ankle/knee diagram is flattened as a result of reduced range of motion of the ankle. Also, the upward movement of the graph at the onset of swing is delayed because of relatively more delay of the ankle than of the knee. The knee/hip diagram is compressed in the hip angle direction, suggesting reduced hip angle range of motion. The lower right part of the oval is not as round, indicating relatively tardy knee flexion compared with hip flexion.

The mean quantitative measures of performance are given in Table 2. The first observation is that the patients are slower than the control subjects. Step and stride length are significantly reduced, and there is a trend toward reduced cadence. These factors together lead to a significant diminution of gait velocity. Events significantly delayed are heel off time, toe off time, and time of peak flexion of the knee in swing. All these late events could be the result of prolonged stance time in the patients, for which there was a trend that just missed significance. Ankle range of motion, knee range of motion, knee range of motion during stance, and hip range of motion all were reduced in the patients, but only ankle range of motion reached significance. Despite clinical impression, there was no significant difference in step width or height that the toe was lifted from the floor in swing phase.

Patients differed significantly from control subjects in terms of variability of performance for virtually all measures (Table 3), including step and stride length, stride length symmetry, cadence, step time, and stance time. However, there was no increased variability with either step width or step width symmetry. There was increased variability of ankle range of motion, the ankle angle at heel strike, and the distance of the foot from the floor in swing. The knee showed increased variability with range of motion during stance and for the time of peak flexion during swing.

DISCUSSION

A stepping generator in the spinal cord is probably responsible for the gross pattern of rhythmic movements in locomotion, and evidence for the existence of such a generator in humans has been strengthened by the recent

observations of locomotion patterns in patients with complete spinal cord injury.[5] Nevertheless, the spinal generator is clearly controlled and modified by supraspinal signals, and the cerebellum probably plays a part in this process.

Patients with cerebellar dysfunction clearly have abnormal gait, characterized by stumbling, scraping the toe, lurching to one side, and falls. Because such performance cannot be captured and quantified easily, the data we have analyzed represent the best performance of the patients, not their average performance. The patients walked more slowly than the matched control subjects, usually as a result of reduced step length, but there was also a trend toward a slower cadence. Slowness characterizes limb movement in patients with cerebellar dysfunction; certainly some is the result of prolongation of electromyographic burst patterns required for movement,[2,6] but the majority of the slowness is likely the result of compensation. Balance is poor in these patients,[3,7] and the movements while walking and the need to balance periodically on one foot stress the system. Slowing down should make the balance problem less severe. Compensatory slowness of walking is seen in most neurologic conditions affecting the motor system.

Some of the "abnormalities" observed here are a simple consequence of slowness of walking. Slowing down will increase the percent of the gait cycle devoted to stance.[8] This is certainly the main explanation of the delayed motions of the ankle and knee. Other abnormalities, including reduced sag of the knee in stance and decreased range of motion at the ankle, cannot be ascribed to slowness of walking. Such abnormalities might reflect a regression to earlier stages of ontogeny of locomotion, as described by Forssberg et al.[9]

We were unable to demonstrate a wide base while walking, despite clinical impression of this and the fact that these patients prefer to stand with a widened base of support. This may be in part the result of the requirement that the patients had to step on the force platform for us to measure the trial.

An analysis of interjoint coordination, best displayed with the angle/angle diagrams, reveals problems that are not immediately apparent by looking at one joint at a time. For example, the diagrams showed that there was relative delay of plantar flexion of the ankle compared with knee flexion at the end of stance and relative delay of knee flexion to hip flexion at the onset of swing. Patients with cerebellar dysfunction have particular difficulty coordinating multijoint movement,[10,11] and this finding is another example.

The variability of even the best performances of the patients is a striking abnormality. Although increased variability may characterize all movement disorders, it is particularly marked in cerebellar dysfunction.[2] The explanation for the variability is unknown. However, the variability is clearly the source of problems in gait. Thus, for example, although the foot on average clears the floor with a good distance, the variability is such that the foot will hit the floor on some occasions.

TABLE 3. *Coefficient of variability of kinematic descriptors of gait*

Descriptor	Control (mean ± SD)	Patient (mean ± SD)	p from *t* test
Cadence	3.2 ± 1.8	5.4 ± 1.9	0.01*
Step length	3.2 ± 2.1	7.2 ± 4.3	0.02*
Stride length	2.0 ± 1.5	6.1 ± 3.4	0.005*
Step width	9.5 ± 4.1	12.5 ± 5.1	0.16
Step length symmetry	5.5 ± 4.5	9.1 ± 4.8	0.10
Stride length symmetry	1.9 ± 1.4	6.5 ± 4.8	0.02*
Step width symmetry	11.4 ± 6.2	18.8 ± 10.4	0.07
Stance time	2.9 ± 1.4	6.5 ± 3.4	0.009*
Swing time	8.6 ± 10.7	9.6 ± 3.7	0.78
Step time	3.1 ± 1.5	5.4 ± 2.0	0.008*
Stride time or gait cycle	3.0 ± 2.5	4.3 ± 2.7	0.30
Gait velocity	3.8 ± 4.5	5.7 ± 2.2	0.24
Ankle angle range of motion	5.5 ± 2.3	9.9 ± 4.8	0.02*
Ankle angle at heel strike	1.1 ± 0.3	3.0 ± 1.8	0.008*
Heel off time	4.4 ± 2.7	7.0 ± 4.4	0.14
Toe off time	1.9 ± 0.9	3.0 ± 1.6	0.08
Knee angle range of motion	2.6 ± 0.7	5.7 ± 5.5	0.12
Knee angle range of motion during stance	15.3 ± 12.2	57.1 ± 59.4	0.05*
Time of peak flexion of knee during swing	1.7 ± 0.8	3.0 ± 1.5	0.02*
Hip angle range of motion	5.1 ± 2.8	7.9 ± 4.7	0.13
Foot height	2.8 ± 0.8	5.7 ± 2.2	0.002*

We previously described the quantitative aspects of gait in a group of autistic patients.[4] At that time, we speculated that their abnormalities (decreased range of motion of the ankle and decreased knee flexion in early stance) were consistent with cerebellar dysfunction, although data on gait in patients with cerebellar deficits were limited. The present results confirm that speculation.

There are limited studies of quantification of locomotion in animals with cerebellar lesions.[12,13] These confirm the clumsiness of this type of movement but cannot be easily compared with our findings.

Acknowledgments: The authors thank the personnel of the Biomechanics Laboratory of the Department of Rehabilitation Medicine, particularly Dr. Steve Stanhope, without whose help this work would have been impossible. The authors also thank D. Schoenberg, MSc, for skillful editing.

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